



Atlas of Gastrointestinal Endoscopy

(Fascinating Images for Clinical Education; FICE)

5th Edition




Edited by
Sombat Treeprasertsuk, M.D.
Linda Pantongrag-Brown, M.D.
Rungsun Rerknimitr, M.D.



The 5th Edition of the Atlas for GI Endoscopy (Fascinating Images for Clinical Education; FICE)

All endoscopic pictures were taken by staffs of Excellent Center for GI Endoscopy (ECGE),
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Atlas of Gastrointestinal Endoscopy (Fascinating Images for Clinical Education; FICE) 5th Edition

Editors

Sombat Treeprasertsuk, M.D.

Linda Pantongrag-Brown, M.D.

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Fifth edition

Thai Association for Gastrointestinal Endoscopy (TAGE)

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Preface from TAGE President



Dear Fascinating Readers,

The Fascinating Images for Clinical Education (FICE) Atlas is the latest book series of “Atlas in GI Endoscopy” by TAGE. To date, “Enhanced Image Endoscopy” has become our routine practice and we can see what we did not clearly before. All images from this atlas have been captured from the latest 4450HD series with 1080i HDTV output from Fujifilm Corporation. Many of these clinical images are well displayed by the beautiful flexible spectral imaging color enhancement (FICE). The atlas is nicely separated in parts according to the organs of interest. All clinical contexts are well relevant to the current practice and can be utilized easily.

Needless to say, I would like to express my deeply thank to the editors, Professor Rungsun Rerknimitr, Dr.Linda Pantongrag-Brown, Associated Professor Sombat Treeprasertsuk, and all contributors for their great efforts to create this fascinating book.

Finally, I hope we altogether can learn from this collection and apply it to the best care in GI endoscopy.

Best,

Dr.Thawee Ratanachu-ek, M.D.

The president of the Thai Association for
Gastrointestinal Endoscopy (TAGE) (2012-present)



From Editors

The 5th edition of the atlas for gastrointestinal (GI) Endoscopy (**Fascinating Images for Clinical Education; FICE**) is our latest edition to share knowledge and experience in GI endoscopy. All endoscopic images have been produced by the cutting edge technology with high definition (HD) format that solely supported by Fujifilm Corporation, Tokyo, Japan. With valuable feedback from our readers and colleagues, we aim to improve our atlas to share and update knowledge for many GI endoscopists worldwide and hope this education material will indirectly lead to a better care of patients.

With various contributions from many authors, the book is clearly representing the series of interesting gastrointestinal diseases as we split them into 5 categories; Esophagus, Stomach, Small bowel, Colon, and Endosonography (EUS). We are grateful to all contributors for their excellent support to make this atlas happens. As our tradition, we have incorporated a comprehensive review of essential or advanced developments in GI endoscopy and radiology in this atlas. For all readers, your comments and feedbacks are very valuable to us as they motivate us to raise the educational level in GI endoscopy.

Last but not least, please do not forget to visit us and download all previous issues from our website (<http://www.thaitage.com/Thai/home.html>)

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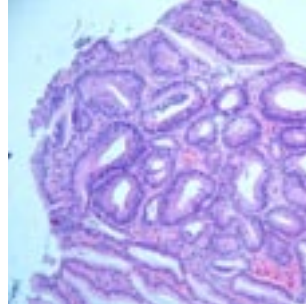
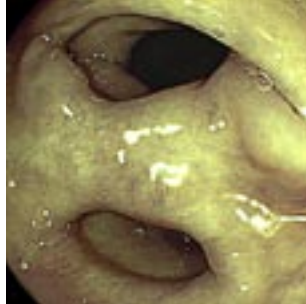
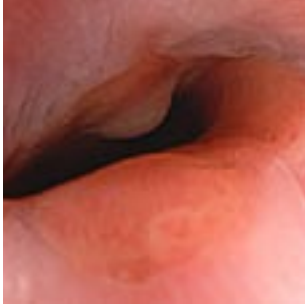
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Part 1

Esophagus

Case 1

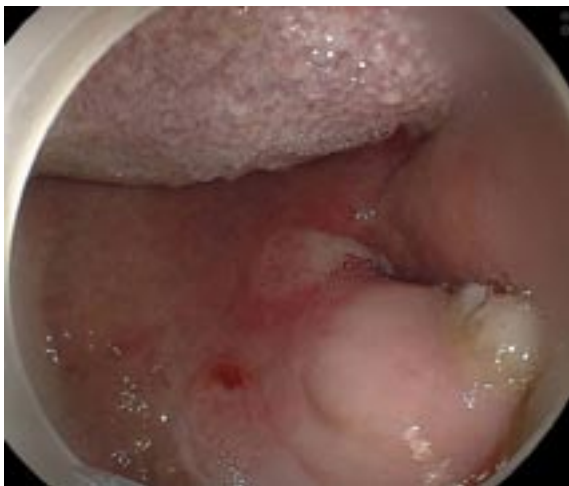
Suparat Khemnark, M.D.

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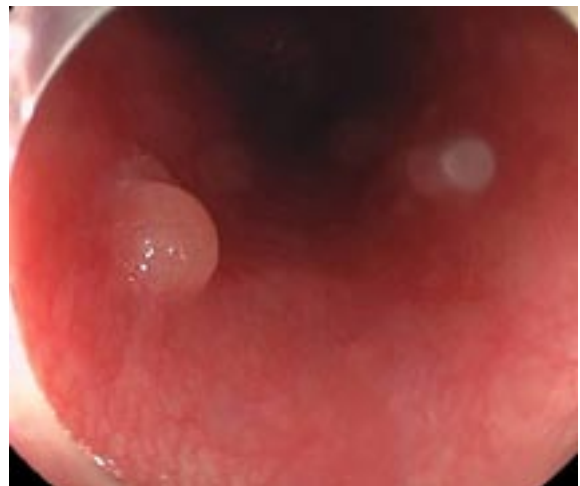
Rungsun Rerknimitr, M.D.

A 21-year-old man presented with chronic nonspecific conjunctivitis for 5 months. There were multiple oral ulcers and vesicular lesions at his face. Biopsy was done at the vesicular lesion and the pathological report was compatible with cicatricial

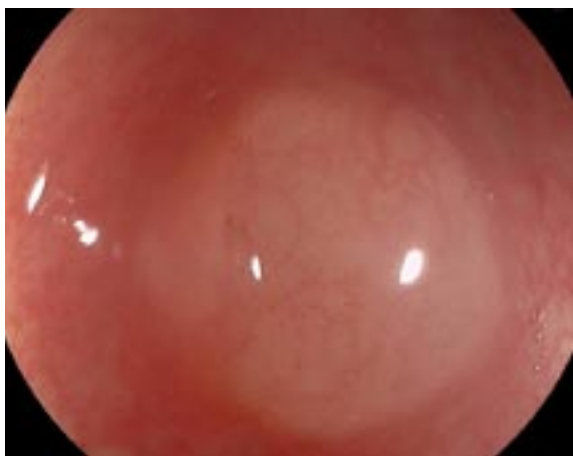
pemphigoid. Although the patient did not have any esophageal symptoms, EGD was performed due to patient's preference. Endoscopic findings showed multiple clean base ulcers at hard palate and one small bullous lesion at mid esophagus (Figure 1-4).



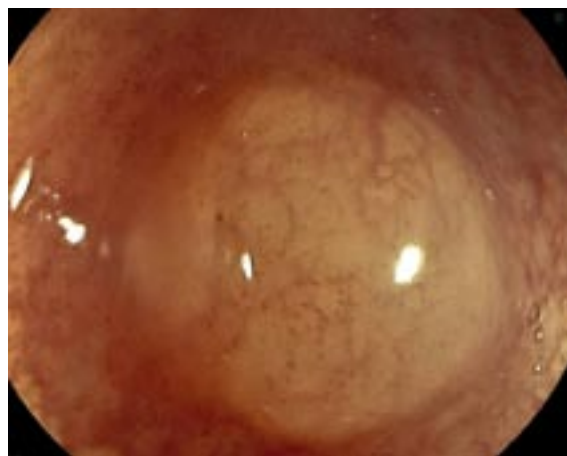
■ Figure 1: Hard palate ulcers



■ Figure 2: Small bullous lesions at mid esophagus



■ Figure 3: Bullous lesion under white light endoscopy



■ Figure 4: Bullous lesion under FICE station 6

Diagnosis:

Cicatricial pemphigoid

Discussion:

Cicatricial pemphigoid (CP) is defined as a heterogeneous group of rare chronic autoimmune blistering diseases that predominately affects the mucous membranes, including oral cavity, ocular mucosa, or mucous membranes of the nose, larynx, esophagus, rectum, penis, vagina¹, and occasionally the skin. The oral mucosa is involved in 89-100% of cases and the rate of conjunctival involvement is 61-71% with 25% of them having cicatrization that leads to blindness. Esophageal involvement varies from about 2.3 to 13%². Bullous lesions are rarely seen because endoscopy is usually performed at a late stage when scarring has already occurred. Esophageal changes can be classified to two major groups: 1) webs in the esophagus in early disease 2) stenosis of variable length and smooth contour, commonly in the upper esophagus, and representing the advanced stage of the disease³. The differential diagnoses are bullous pemphigoid (BP) and epidermolysis bullosa acquisita (EBA). The clinical presentations depend on the sites of involvement. Oral

cavity lesion presents as recurrent, painful erosions. The gingivae are most commonly involved, followed by the palate and the buccal mucosa; however, any mucosal site in the mouth may become a blister. Involvement of the oropharynx may present with hoarseness or dysphagia. Progressive scarring disease may lead to esophageal stenosis requiring dilatation procedures⁴.

References

1. Chan LS. Ocular and oral mucous membrane pemphigoid (cicatricial pemphigoid). *Clin Dermatol* 2012;30:34-7.
2. Al-Kutoubi MA Eliot C. Oesophageal involvement in benign mucous membrane pemphigoid. *Clin Radiol* 1984;35:131-5.
3. Nalbant S, Saracoglu E, Karaeren N, et al. A case of cicatricial pemphigoid with esophageal involvement. *Turk J Gastroenterol* 1999;10:74-77.
4. Chan LS, Ahmed AR, Anhalt GJ, et al. The first international consensus on mucous membrane pemphigoid: definition, diagnostic criteria, pathogenic factors, medical treatment, and prognostic indicators. *Arch Dermatol* 2002;138: 370-9.

Case 2

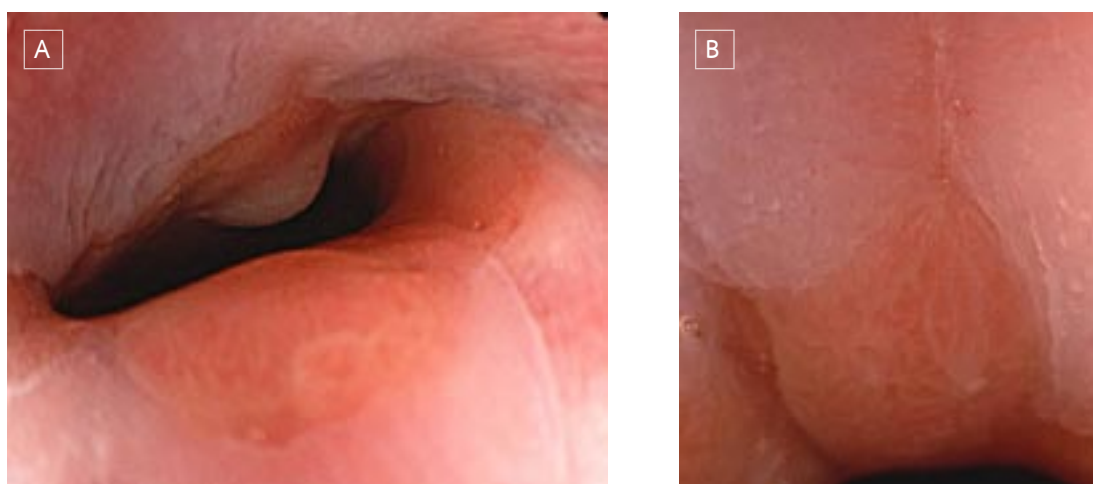
Surasak Aumkaew, M.D.

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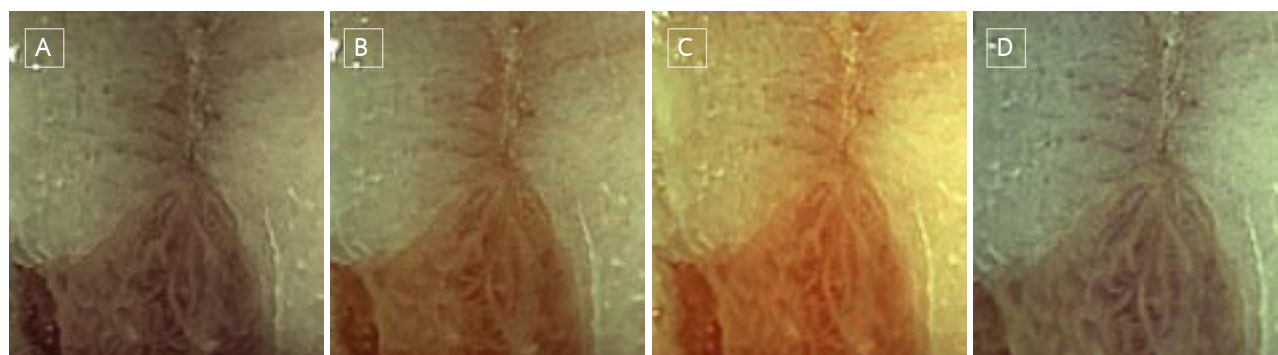
Rungsun Rerknimitr, M.D.

A 35-year-old woman presented with heart burn for 3 months. Her symptoms were partially improved with proton pump inhibitors. EGD was performed. It revealed minimal change of distal esophageal mucosa. FICE with magnification was applied. It showed triangular

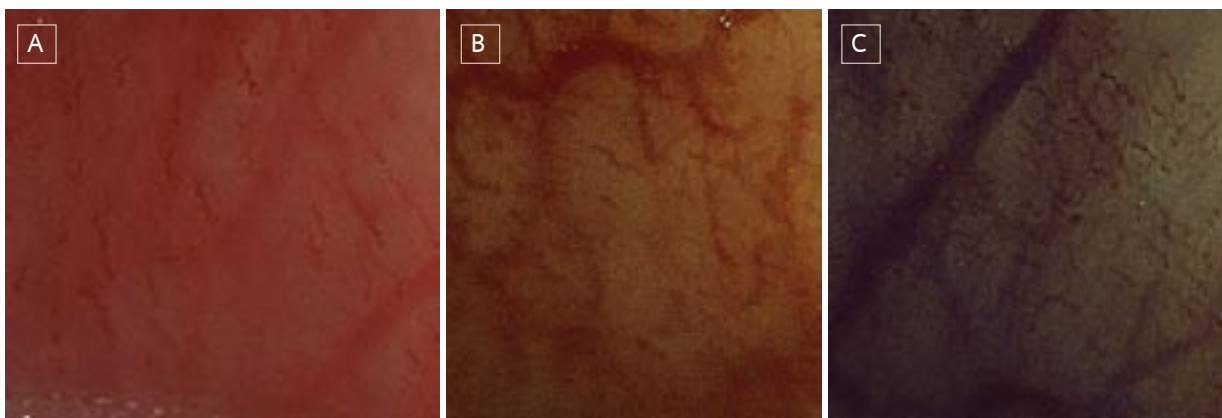
indentation at esophagogastric junction (EGJ) with increased number esophageal capillary loops (Figure 1-3). A 24-hr esophageal pH monitoring was compatible with esophageal reflux disease.



■ Figure 1: A) Normal white light endoscopy and B) 50x magnification showed minimal distal esophageal mucosal change with triangular indentation



■ Figure 2: FICE with 50x magnification showed triangular indentation A) FICE station 0, B) FICE station 1, C) FICE station 5 and D) FICE station 8



■ Figure 3: A) Normal esophageal capillary loop under white light endoscopy, B) FICE with 100x magnification station 5 showed increased, tortuous esophageal capillary loops and C) FICE with 100x magnification station 8 showed increased, tortuous esophageal capillary loops

Diagnosis:

Minimal change esophageal reflux disease (MERD)

Discussion:

Minimal change esophageal reflux disease (MERD) is in a spectrum of gastroesophageal reflux disease that the novel technology such as magnifying or chromoendoscopy demonstrates minimal change of previously called “non erosive esophageal reflux disease” (NERD)¹. FICE in cooperation with a high-resolution image processor can improve visualization of subtle NERD lesions including a triangular indentation at the Z-line and increasing numbers of tortuous and dilated intra papillary capillary loops². The previous data showed that FICE provided higher sensitivity, negative predictive value and accuracy than the standard white light endoscopy in the diagnosis of MERD^{3,4}.

References

1. Falk GW. Is conventional endoscopic identification of non-erosive reflux disease adequate? *Digestion* 2008;78 Suppl 1:17-23.
2. Chaiteerakij R, Rerknimitr R, Kullavanijaya P. Role of digital chromoendoscopy in detecting minimal change esophageal reflux disease. *World J Gastrointest Endosc* 2010;2:121-9.
3. Chaiteerakij R, Tangmankongworakoon N, Gonlachanvit S, et al. Efficacy of Intelligent Chromo Endoscopy for Detection of Minimal Mucosal Breaks in Patients with Typical Symptoms of Gastroesophageal Reflux Disease. *Gastrointest Endosc* 2008;67:AB86.
4. Aumkaew S, Pittayanon R, Rerknimitr R, et al. Feasibility of the New Fuji Intelligent Color Enhancement (FICE) and Probe-Based Confocal Laser Endoscopy (pCLE) in Diagnosing Minimal Change Esophageal Reflux Disease (MerD). *Gastrointest Endosc* 2012;75:AB449-AB450.

Case 3

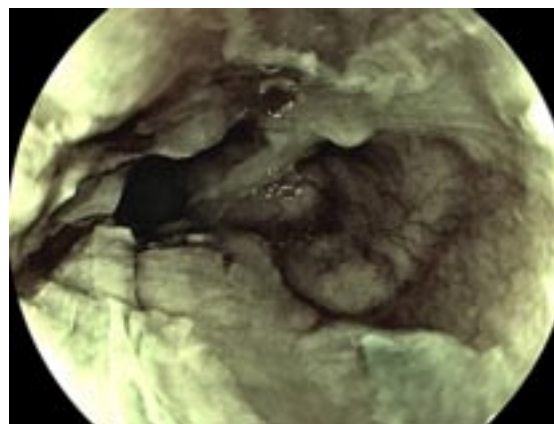
Kittiyod Poovorawan, M.D.

Rapat Pittayanon, M.D.

Rungsun Rerknimitr, M.D.

A 64-year-old woman was scheduled for a colonoscopy as a part of her colon cancer screening program. A day before procedure, by mistake, she suffered from heart burn sensation after taking higher than standard concentration (4x) of laxative solution for bowel preparation (Niflec, Composition of Macroglol,

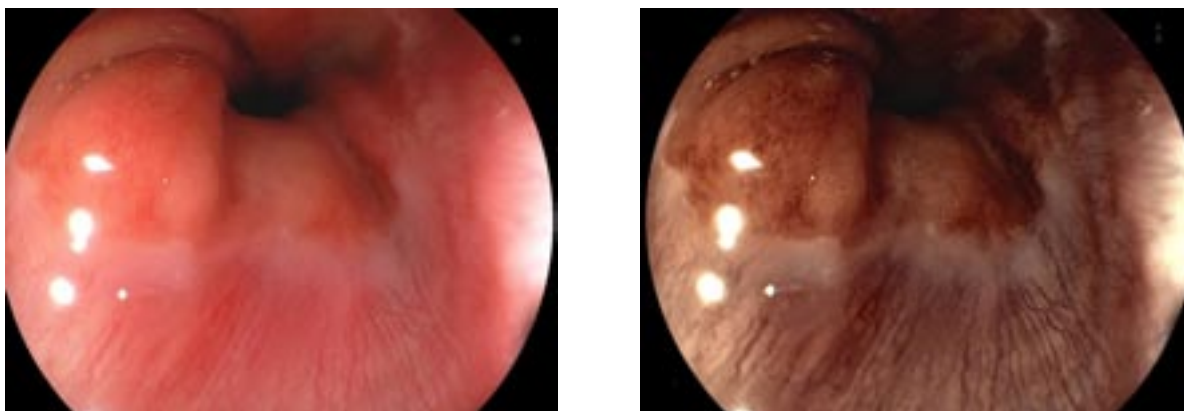
Sodium sulfate, Sodium hydrogen carbonate, Sodium chloride and Potassium chloride). Physical examination was unremarkable. EGD showed desquamation of superficial esophageal epithelium of the entire esophagus (Figure 1-4). The Z-line appeared normal (Figure 5-6).



■ Figure 1-2: Esophagitis with multiple longitudinal patchy superficial desquamation at distal esophagus by using standard white light endoscopy (WLE) and FICE system station 0



■ Figure 3-4: Esophagitis with multiple longitudinal patchy white superficial desquamation at distal esophagus by using FICE system station 2 and 6



■ Figure 5-6: Normal squamocolumnar junction (Z-line) by using WLE and FICE system station 2

Diagnosis:

Esophageal injury secondary due to high concentrated large bowel preparation solution

Discussion:

Caustic esophageal injury typically is usually resulted from either acid or alkali ingestion¹. Many drugs and mixtures such as tetracycline, NSAIDs and potassium chloride can also induce esophageal injury². Mechanisms of esophageal injury were direct irritant effect, prolonged transit time of hyperosmotic solutions and disruption of the normal cytoprotective barrier in the mucosa of the esophagus. Most cases of drug induce esophageal injury are self-limited and resolve without complications³.

In this patient, the cause of injury was probably due to a high concentration of electrolyte mixture in bowel preparation solution.

References

1. Poley JW, Steyerberg EW, Kuipers EJ, et al. Ingestion of acid and alkaline agents: outcome and prognostic value of early upper endoscopy. *Gastrointest Endosc* 2004;60:372-7.
2. Eng J, Sabanathan S. Drug-induced esophagitis. *Am J Gastroenterol* 1991;86:1127-33.
3. Zografos GN, Georgiadou D, Thomas D, et al. Drug-induced esophagitis. *Dis Esophagus* 2009;22:633-7.



Case 4

Pornphan Thienchanachaiya, M.D.

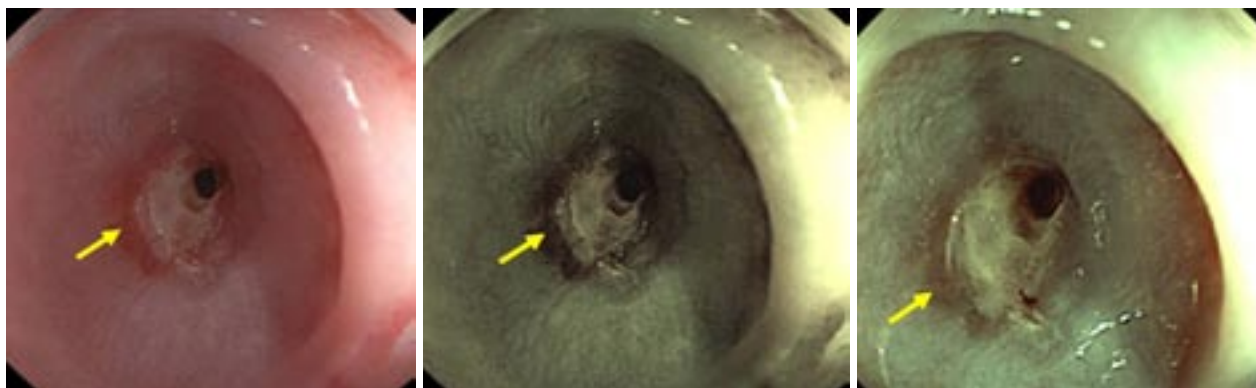
Satimai Aniwon, M.D.

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A 28-year-old man presented with progressive dysphagia for 3 months. He had been diagnosed with nasopharyngeal cancer for 2 years. He underwent surgery, radiation and chemotherapy 6 months ago. Esophagoscopy showed narrowing esophageal lumen with smooth surface at 33 cm from the incisor (Figure 1-

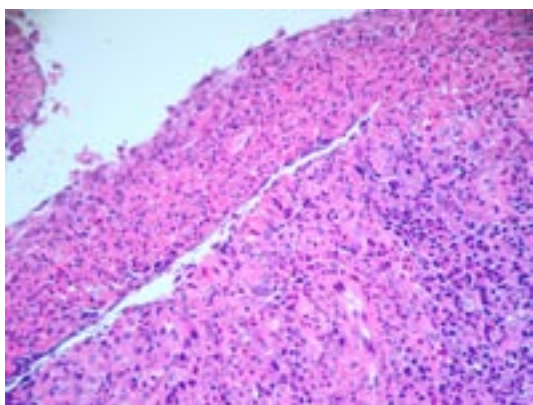
3). The stricture could not be passed by a 9.8 mm esophagoscope. Barium swallow revealed short and smooth narrowing segment of mid esophagus with proximal esophageal dilation (Figure 4). Microscopic examination showed organizing inflammation with granulation tissue (Figure 5).



■ Figure 1-3: Smooth taper narrowing esophageal lumen at 33 cm from the incisor (yellow arrow) under white light mode, FICE Station 0 (R525, B495, G455) and FICE Station 1 (R550, B500, G420)



■ Figure 4: Barium swallow found smooth narrowing short segment esophageal stricture at mid esophagus (red arrow) with proximal esophagus dilatation (yellow arrow)



■ Figure 5: Microscopic examination showed organizing inflammation with granulation tissue formation.

Diagnosis:

Post radiation esophageal stricture in patient with head and neck cancer

Discussion:

One adverse effect observed after external beam radiation therapy for squamous cell carcinoma of the head and neck is the stricture of esophagus. The incidence of radiation-induced esophageal stricture in patients treated with radiotherapy for head and neck cancer was 3.3%¹. The median duration between the end of radiation therapy and the time for diagnosis of esophageal stricture was 8 months (1-132 months). Total obliteration of the esophageal lumen was found in 14% of patients. After dilatation, the majority of stricture could be passed by 7x 10-mm scope¹.

The pattern of esophageal stricture after radiation is mainly characterized by fibrosis caused by progressive obliterative endarteritis, leading to ischemia of the esophageal wall. Clinical manifestations, ranged from a membranous ring to total obliteration of esophageal lumen². The significant risk factors of stricture formation included dosage of radiation of more than 45 Gy and the use of an NG tube or PEG during or immediately after radiation therapy^{1,3}.

Treatment of the stricture with either Savary-Gilliard bougienage or through the scope balloon dilatation is safe and effective. However, repeat dilations are often needed in order to achieve and maintain adequate dysphagia relief in the majority of patients. Time to onset of esophageal stricture is the most important factor for the treatment success. The median time to the onset of esophageal stricture after radiation therapy was significantly shorter in patients who did not respond to endoscopic bougie dilation.

References

1. Ahlberg A, al-Abany M, Alevronta E, et al. Esophageal stricture after radiotherapy in patients with head and neck cancer: experience of a single institution over 2 treatment periods. *Head Neck* 2010;32:452-61.
2. Laurell G, Kraepelien T, Mavroidis P, et al. Stricture of the proximal esophagus in head and neck carcinoma patients after radiotherapy. *Cancer* 2003;97:1693-700.
3. Alevronta E, Ahlberg A, Mavroidis P, et al. Dose-response relations for stricture in the proximal oesophagus from head and neck radiotherapy. *Radiother Oncol* 2010;97:54-9.

Case 5

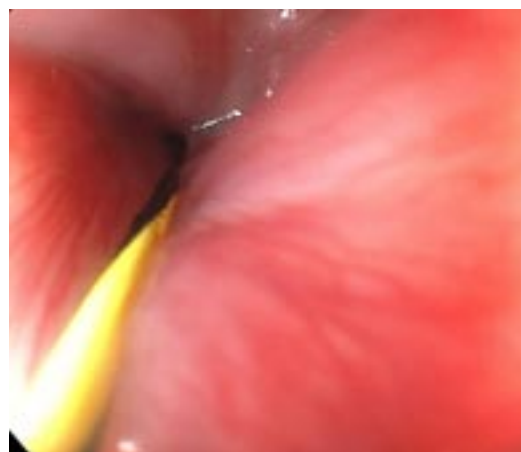
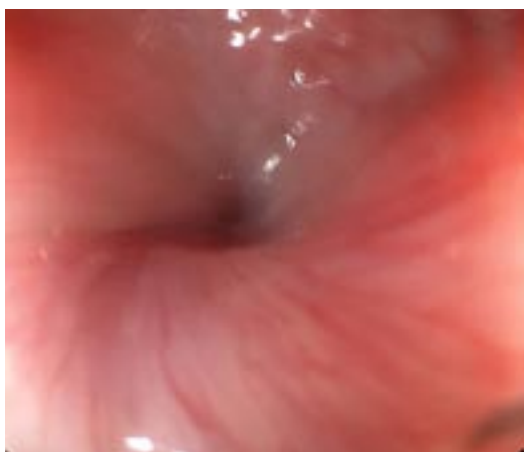
Suparat Khemnark, M.D.

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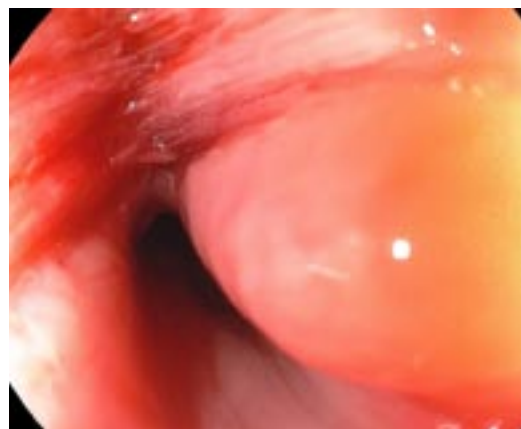
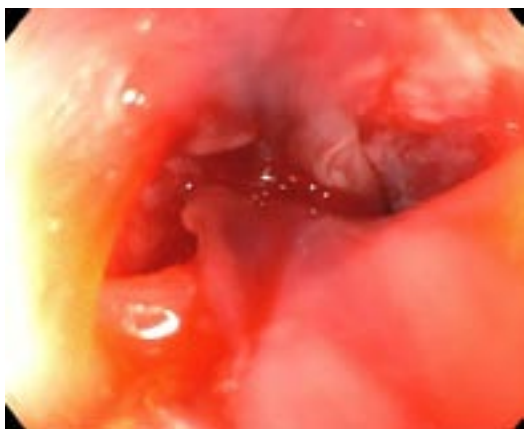
Rungsun Rerknimitr, M.D.

A 45-year-old man presented with dysphagia due to corrosive ingestion. Severe esophageal stricture following corrosive esophagitis occurred only 5 days after ingestion. Management with multisession of esophageal dilatation by Savary dilator had been done every 2 weeks. This time, he presented with dysphagia again.

EGD was done and found a pin-point esophageal lumen at 15 cm from incisor as shown in Figure 1-2. Management in this session, serial Savary dilators was used (9, 11, 14 mm). After finishing dilation, a diluted triamcinolone 2.5 mg per aliquot was injected around the dilation site to decrease the risk of relapse (Figure 3-4).



■ Figure 1-2: Pin-point esophageal lumen (at 15 cm from incisor)



■ Figure 3-4: After dilatation, esophageal tear

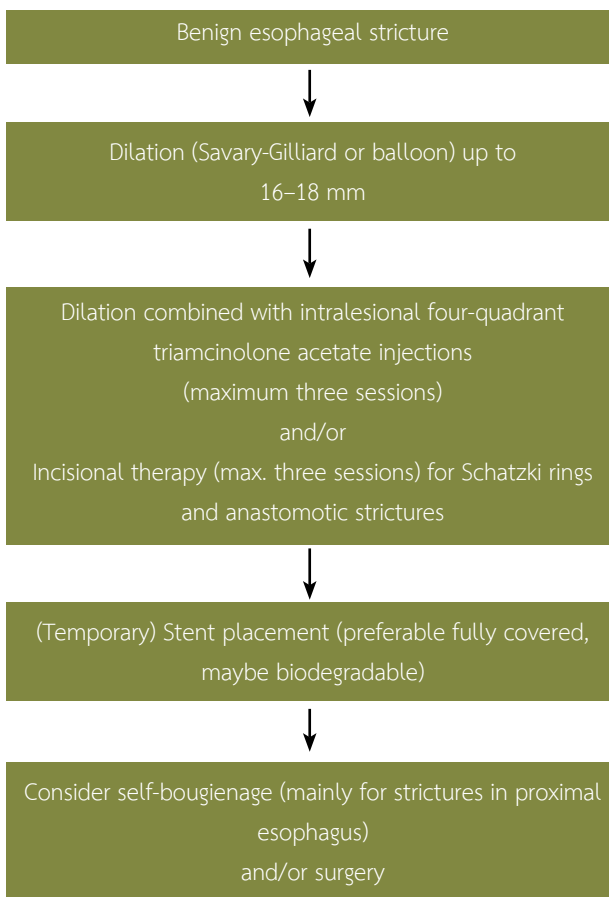
Diagnosis:

Corrosive esophageal stricture

Discussion:

Alkali ingestions typically damage the esophagus more than the stomach or duodenum whereas acids cause more severe gastric injury¹. Ingestion of alkali (such as ammonia or sodium hydroxide) acutely results in a penetrating injury called liquefactive necrosis. The injury extends rapidly (within seconds) through the mucosa and wall of the esophagus towards the mediastinum until tissue fluids buffer the alkali. The likelihood of stricture formation depends upon the depth of damage and degree of collagen deposition. Up to one-third of patients who suffer caustic esophageal injury develop esophageal strictures. The peak incidence of dysphagia due to esophageal stricture formation after corrosive esophageal injury is two months, although it can occur as early as two weeks or as late as years after ingestion².

According to the current guideline in 2011², the first-line option for treating benign esophageal strictures is dilation therapy. Dilation usually relieves symptoms of dysphagia; however, recurrent strictures occur in selective cases. A recurrent or refractory stricture is defined as an anatomic restriction caused by a cicatricial luminal compromise or fibrosis that result in symptoms of dysphagia in the absence of endoscopic evidence of inflammation. This may occur from either an inability to successfully remediate the anatomic problem to a diameter of 14 mm over 5 sessions at 2-week intervals (refractory) or as a result of inability to maintain a satisfactory luminal diameter for 4 weeks once the target diameter of 14 mm has been achieved (recurrent).



References

1. Poley JW, Steyerberg EW, Kuipers EJ, et al. Ingestion of acid and alkaline agents: outcome and prognostic value of early upper endoscopy. *Gastrointest Endosc* 2004;60:372-7.
2. De Wijkerslooth LR, Vleggaar FP, Siersema PD. Endoscopic Management of Difficult or Recurrent Esophageal Stricture. *Am J Gastroenterol* 2011; 106:2080-91.

Case 6

Nuttaporn Norrasetwanich, M.D.

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Rapat Pittayanon, M.D.

Rungsun Rerknimitr, M.D.

A 25-year-old male was admitted to the emergency department with a sore throat, dysphagia, and retrosternal pain. He had suicide attempt by ingesting 50 mL of a paraquat solution one day prior to admission. On physical examination, swelling and

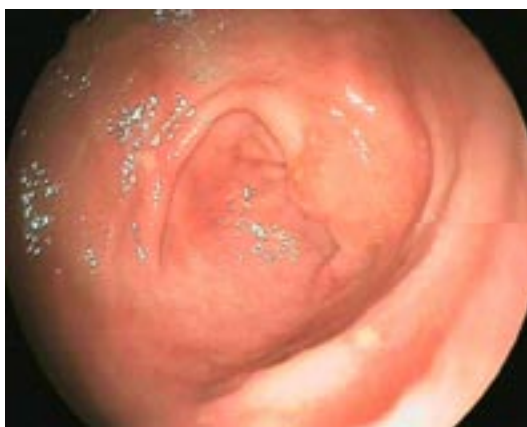
erythematous mucosa were observed in the mouth and on the tongue. The EGD showed multiple discrete necrotic areas at esophagogastric junction (EGJ) and erythematous mucosa at antrum (Figure 1-3).



■ Figure 1: Necrotic areas at EGJ



■ Figure 2: Normal retroflex view



■ Figure 3: Erythematous mucosa at antrum

Diagnosis:

Esophageal corrosive injury from paraquat ingestion

Discussion:

Paraquat is a widely used, nonselective contact herbicide of great toxicological importance. The very high case fatality of paraquat is due to inherent toxicity and lack of effective treatments. Common clinical findings are gastrointestinal symptoms, acute renal failure, pulmonary hemorrhage, and late pulmonary fibrosis. Generally, death, from respiratory failure, occurs within three weeks after the ingestion. In cases of massive intake (> 50 mg/kg of body weight), death occurs a few hours after ingestion, due to multiple organ failure¹.

Gastrointestinal toxicity is universal in those ingesting paraquat concentrate. Mucosal lesions of the mouth and the tongue ('paraquat tongue') begin to appear within the first few days and may become ulcerated with bleeding. These are of little prognostic significance as they occur even in those who spit paraquat out without swallowing. Mucosal lesions in the pharynx, esophagus and stomach are also very common

and much more sinister. These may result in perforation, mediastinitis and/or pneumomediastinum. The contribution of this direct caustic effect to mortality is probably underestimated².

The observational study from Yen TH, et al. included 16 of 1410 paraquat subjects who underwent endoscopies at Chang Gung Memorial Hospital between 1980 and 2007. They concluded paraquat, a mild caustic agent, produces only grades 1, 2a, and 2b esophageal injury. Their findings showed a potential relationship between the degree of hypoxia, mortality, and degree of esophageal injury³.

References

1. Neves FF, Sousa RB, Pazin-Filho A, et al. Severe paraquat poisoning: clinical and radiological findings in a survivor. *J Bras Pneumol* 2010;36: 513-6.
2. Gawarammana IB, Buckley NA. Medical management of paraquat ingestion. *Br J Clin Pharmacol* 2011;72:745-57.
3. Yen TH, Lin JL, Lin-Tan DT, et al. Spectrum of corrosive esophageal injury after intentional paraquat ingestion. *Am J Emerg Med* 2010;28: 728-33.



Case 7

Kittiyod Poovorawan, M.D.

Rapat Pittayanon, M.D.

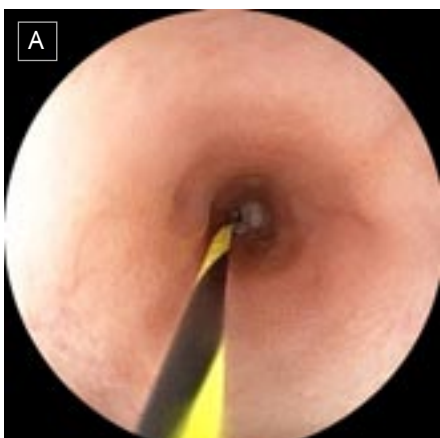
Rungsun Rerknimitr, M.D.

A 31-year-old man presented with progressive dysphagia. He had a history of corrosive ingestion with multiple episodes of recurrent dysphagia after esophageal dilatation. Due to refractory esophageal stricture, he was scheduled for EGD to insert a covered self expandable metallic stent (SEMS). Endoscopic finding (Nasogastroscope, EG 530N, Fujifilm, Japan) showed a stricture at middle esophagus (27 cm from

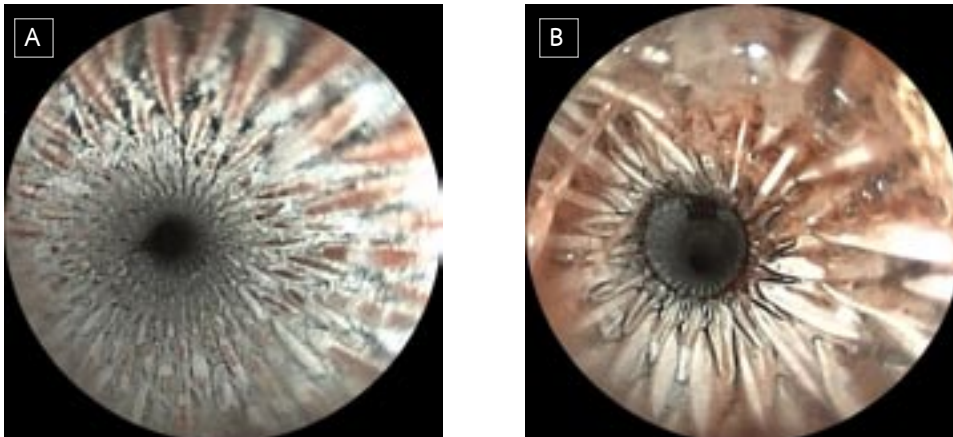
incisors), (Figure 1). Savary dilator over the Jag wire was performed by a 9 mm dilator (Figure 2). Subsequently, a fully covered SEMS was deployed under fluoroscopic guidance was successfully inserted (Figure 3). A follow-up EGD for stent removal showed a significant luminal opening and he reported less dysphagia during 6 months follow up.



■ Figure 1: Endoscopic finding: a stricture at middle esophagus 27 cm from incisor.



■ Figure 2: Endoscopic finding: Guide wire (Jag wire) passed through the stricture site. (A) Post dilatation with 9 mm Savary dilator. (B)



■ Figure 3: Endoscopic finding: Fully covered self-expanding removable stents was deployed under fluoroscopic guidance. (A) Proximal end of the stent. (B)

Diagnosis:

Corrosive esophageal stricture with esophageal covered stent placement

Discussion:

Caustic esophageal strictures are late complication of caustic injury. These are often difficult to treat, since relapsing is frequent after medical or endoscopic treatment¹. Treatment of this complication included endoscopic dilatation, endoscopic incisional therapy, intraliesional steroid injection, self-bougienage, surgery and placement of self-expanding metal stents, Polyflex stents, or biodegradable stents². Efficacy of Fully covered self-expanding removable stents (SERS) placement in benign refractory strictures is 46.2%, however, it is associated with migration rate of 26.4%³.

References

1. Manta R, Conigliaro R, Bertani H, et al. Self-expandable metal stenting of refractory upper gut corrosive strictures: a new role for endoscopy? *Case Rep Gastrointest Med* 2011;2011:1-5.
2. Kochhar R, Poornachandra KS. Intraliesional steroid injection therapy in the management of resistant gastrointestinal strictures. *World J Gastrointest Endosc* 2010;2:61-8.
3. Thomas T, Abrams KR, Subramanian V, et al. Esophageal stents for benign refractory strictures: a meta-analysis. *Endoscopy* 2011;43:386-93.

Case 8

Pornphan Thienchanachaiya, M.D.

Rapat Pittayanon, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

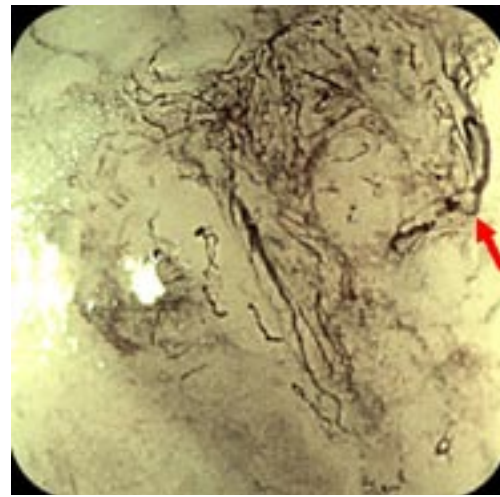
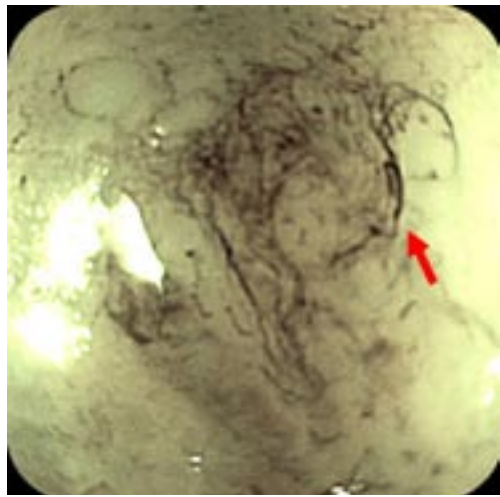
Rungsun Rerknimitr, M.D.

A 65-year-old woman underwent EGD because of the program of screening esophageal cancer. She had previously been diagnosed with stage 4 (T4N2M0) squamous cell carcinoma of tonsil 8 months ago. She responded to the treatment with radiation and the treatment was discontinued 7 months ago. EGD was performed with white light mode and discovered only mild irregular esophageal dimple. Then FICE station 0 was performed and it better depicted an ill-defined irregular surface elevated mucosa, 10 mm in diameter at mid-esophagus (Figure 1-2). Under FICE with 50 and 100 magnification demonstrated dilated, tortuous weaving,

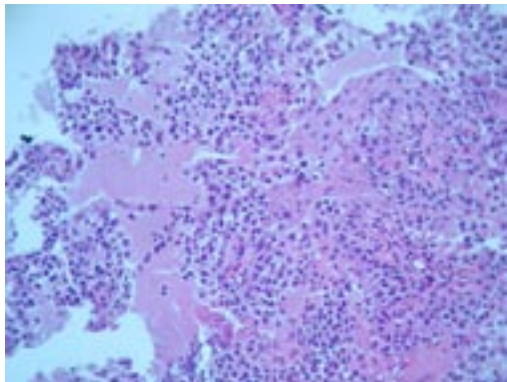
irregular caliber, and form variation in shape of intraepithelial papillary capillary loop (IPCL), compatible with type V according to Inoue's classification (Figure 3-4). Esophageal biopsy was obtained. The pathological report showed polygonal-shaped cells with pleomorphic basophilic nuclei and abundant eosinophilic cytoplasm, mitosis, focal necrosis, keratin pearl formation, mucosal ulceration with hemorrhage (Figure 5). The final diagnosis was moderately differentiated esophageal squamous cell carcinoma. No evidence of lymph node metastasis was detected by CT scan.



■ Figure 1-2: An ill define irregular surface elevated mucosa, 10 mm in diameter at mid-esophagus (yellow arrow) under white light mode and FICE Station 0 (R525, B495, G455).



■ Figure 3-4: Under FICE station 0 with magnification 50 and 100 power demonstrated dilatation, tortuous weaving, irregular caliber, and form variation in shape of intraepithelial papillary capillary loop (IPCL)



■ Figure 5: Microscopic examination showed tumor cells possess polygonal-shaped cells with pleomorphic basophilic nuclei with abundant eosinophilic cytoplasm

Diagnosis:

Metachronous esophageal squamous cell carcinoma in a previously diagnosed as tonsil cancer patient.

Discussion:

Esophageal squamous cell carcinoma (ESCC) remains the most common esophageal cancer in Asia. Risk factors for ESCC include tobacco use and alcohol consumption. Patients with a history of head and neck squamous cell cancers are the high risk populations of esophageal squamous cell carcinoma with odds ratio 2.6-3.8^{1,2}.

Diagnosis of early ESCC by white light endoscopy and lugol chromoendoscopy are limited. Currently, the

novel image system, digital chromoendoscopy (DC) provides a higher specificity for early esophageal squamous cell carcinoma detection when compared with lugol chromoendoscopy (86% vs. 72%)³. The suspicious neoplastic lesion was the brown-stained area depicted by magnifying DC and it represented an increase in vascularization. Magnification of endoscopy can provide the detail of early ESCC^{4,5}.

Flexible spectral Imaging Color Enhancement (FICE) is one of the novel image enhanced system that seems to be the useful equipment to detect many early GI neoplasms including esophageal neoplasia⁵. Magnification of FICE is helpful for further detailing of those lesions⁶.

References

1. Akhtar S, Sheikh AA, Qureshi HU. Chewing areca nut, betel quid, oral snuff, cigarette smoking and the risk of oesophageal squamous-cell carcinoma in South Asians: a multicentre case-control study. *Eur J Cancer* 2012;48:655-61.
2. Wang WL, Lee CT, Lee YC, et al. Risk factors for developing synchronous esophageal neoplasia in patients with head and neck cancer. *Head & neck* 2011;33:77-81.
3. Ide E, Maluf-Filho F, Chaves DM, et al. Narrow-band imaging without magnification for detecting early esophageal squamous cell carcinoma. *World J Gastroenterol* 2011;17:4408-13.
4. Chai NL, Ling-Hu EQ, Morita Y, et al. Magnifying endoscopy in upper gastroenterology for assessing lesions before completing endoscopic removal. *World J Gastroenterol* 2012;18:1295-307.
5. Inoue M, Miyake Y, Odaka T, et al. Objective evaluation of visibility in virtual chromoendoscopy for esophageal squamous carcinoma using a color difference formula. *J Biomed Opt* 2010;15:056019.



Case 9

Pornphan Thienchanachaiya, M.D.

Rapat Pittahyanon, M.D.

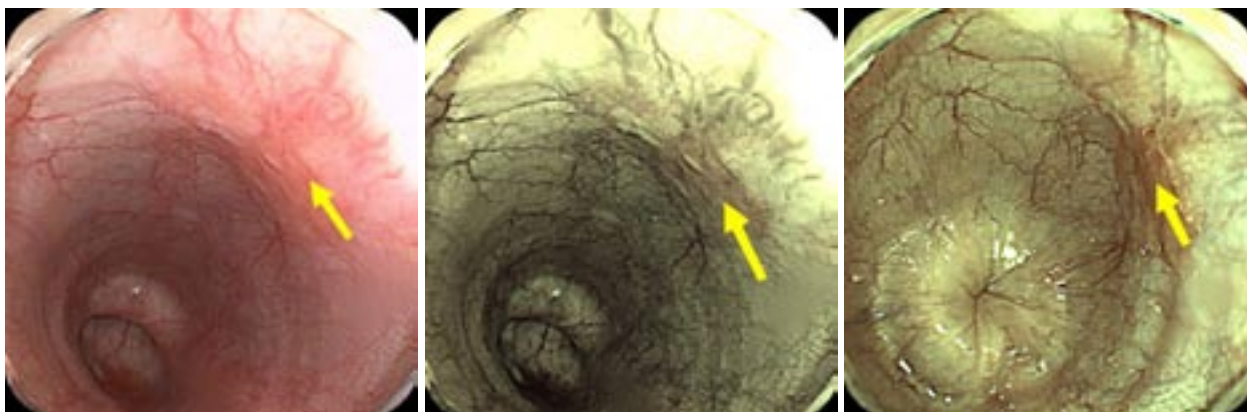
Naruemon Wisedopas-Klaikeaw, M.D.

Rungsun Rerknimitr, M.D.

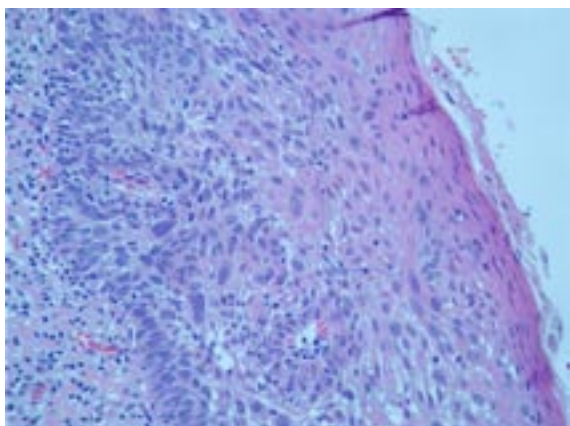
A 61-year-old man underwent esophageal cancer screening program. He had been diagnosed as squamous cell carcinoma of the base of tongue (T4N1M0) 18 months ago and had succeeded the treatment with 70 Gy radiation and carboplatin.

EGD was performed for metachronous cancer surveillance with white light and FICE modes. It showed

a well-defined erythematous flat mucosa, 1.0 cm in diameter at mid-esophagus (Figure 1-3). Esophageal biopsy showed disorganization and disorientation of the esophageal mucosa with scatter pleomorphism of nuclei, compatible with high grade dysplasia of esophagus (Figure 4).



■ Figure 1-3: A well define erythematous flat mucosa, 1.0 cm in diameter at mid-esophagus (yellow arrow) under white light mode, FICE Station 0 (R525, B495, G455) and FICE Station 1 (R550, B500, G420).



■ Figure 4: Microscopic examination showed disorganization and disorientation of esophageal mucosa with scatter pleomorphism of nuclei.

Diagnosis:

High grade esophageal dysplasia in patient with previously diagnosed tongue cancer

Discussion:

High-grade dysplasia is defined by revised Vienna consensus for diagnosis of early esophageal neoplasia. The relative risk of high grade esophageal dysplasia patient to develop esophageal squamous cell carcinoma is 28.3 (95%CI; 15.3-52.3)¹. Patients with a history of head and neck squamous cell cancers are also the high risk populations of esophageal squamous cell carcinoma with estimated risk at 4-9 times of normal populations¹.

Currently, the novel imaging system without magnification seems to provides a high sensitivity (100%) for detecting early esophageal squamous cell carcinoma and is comparable to 2.5% lugol chromoendoscopy². Flexible spectral Imaging Color Enhancement (FICE)

is one of the promising novel imaging system to detect many early GI neoplasms including esophageal neoplasia³.

References

1. Wang GQ, Abnet CC, Shen Q, et al. Histological precursors of oesophageal squamous cell carcinoma: results from a 13 year prospective follow up study in a high risk population. *Gut* 2005;54:187-92.
2. Ide E, Maluf-Filho F, Chaves DM, et al. Narrow-band imaging without magnification for detecting early esophageal squamous cell carcinoma. *World J Gastroenterol* 2011;17:4408-13.
3. Inoue M, Miyake Y, Odaka T, et al. Objective evaluation of visibility in virtual chromoendoscopy for esophageal squamous carcinoma using a color difference formula. *J Biomed Opt* 2010;15:056019.



Case 10

Pornphan Thienchanachaiya, M.D.

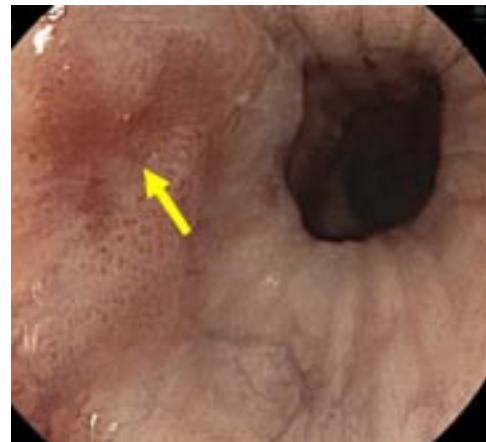
Rapat Pittahyanon, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

Rungsun Rerknimitr, M.D.

A 61-year-old man with a history of squamous cell carcinoma of tongue underwent surveillance esophagogastroscope (EGD). Standard white-light endoscopy and FICE detected a 10 mm in diameter of erythematous depressed mucosa, suspected of

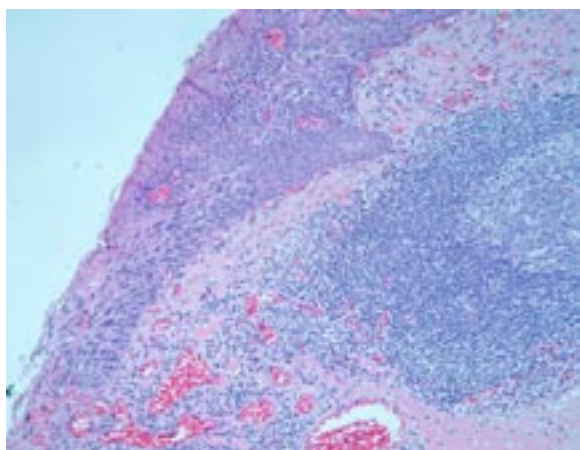
dysplasia, at 32 cm from the incisor (Figure 1-2). Cap-assisted esophageal mucosal resection (EMR) was performed en bloc without complication (Figure 3-4). Microscopic examination showed high grade esophageal dysplasia with free resection margin (Figure 5).



■ Figure 1-2: A well define erythematous depressed mucosa, 10 mm in diameter at 32 cm from incisor (yellow arrow) under white light mode and FICE Station 2 (R550, B500, G470).



■ Figure 3-4: Esophageal mucosal resection was performed with a cap-assisted EMR.



■ Figure 5: Microscopic examination showed a large dysplastic change of epithelium

Diagnosis:

Cap-assisted esophageal mucosal resection (EMR) for high grade esophageal dysplasia in patient with previously diagnosed with tongue cancer

Discussion:

High-grade dysplasia is a high risk lesion to develop esophageal squamous cell carcinoma¹. It has been shown to be associated with a low risk of lymph node metastasis, thus forming the basis for endoscopic mucosal resection (EMR)². Endoscopic resection should be performed for mucosal cancer of 3 cm or less without evidence of metastasis³. The limitation of EMR is the unpredictable depth of each resection⁴.

However, EMR is an effective treatment modality for high grade dysplasia of esophagus. Although the rate of stenosis development is significant after EMR, it is easily treated by endoscopic dilation⁵.

References

1. Wang GQ, Abnet CC, Shen Q, et al. Histological precursors of oesophageal squamous cell carcinoma: results from a 13 year prospective follow up study in a high risk population. *Gut* 2005;54:187-92.
2. Katada C, Muto M, Momma K, et al. Clinical outcome after endoscopic mucosal resection for esophageal squamous cell carcinoma invading the muscularis mucosae--a multicenter retrospective cohort study. *Endoscopy* 2007;39:779-83.
3. Choi JY, Park YS, Jung HY, et al. Feasibility of endoscopic resection in superficial esophageal squamous carcinoma. *Gastrointest Endosc* 2011; 73:881-9.
4. Teoh AY, Chiu PW, Yu Ngo DK, et al. Outcomes of endoscopic submucosal dissection versus endoscopic mucosal resection in management of superficial squamous esophageal neoplasms outside Japan. *J Clin Gastroenterol* 2010;44:e190-4.
5. Chennat J, Konda VJ, Ross AS, et al. Complete Barrett's eradication endoscopic mucosal resection: an effective treatment modality for high-grade dysplasia and intramucosal carcinoma--an American single-center experience. *Am J Gastroenterol* 2009;104:2684-92.



Case 11

Kriangsak Charoensuk, M.D.

Rapat Pittayanon, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

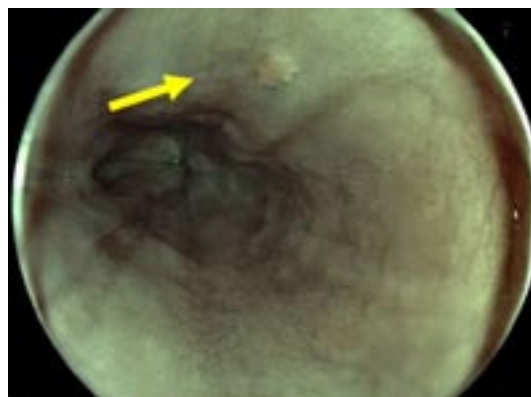
Rungsun Rerknimitr, M.D.

A 41-year-old man presented with dyspepsia and gastroesophageal reflux symptoms for 3 months. EGD was performed and revealed a small mucosal lesion at distal esophagus, 35 cm from incisor (Figure 1-2). FICE with 100 times magnification was applied and found a papule with verrucous surface with increased vasculatures as shown in Figure 3-4. Histology

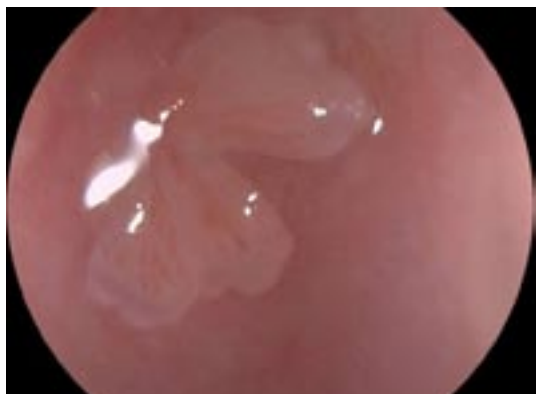
demonstrated papillary projection of the esophageal epithelium covered with fibrovascular core. The adjacent mucosa exhibited focal nodular lesion comprising enlarged epithelial cells containing clear cytoplasm (Figure 5), compatible with esophageal squamous papilloma.



■ Figure 1: Mucosal lesion at distal esophagus (White light)



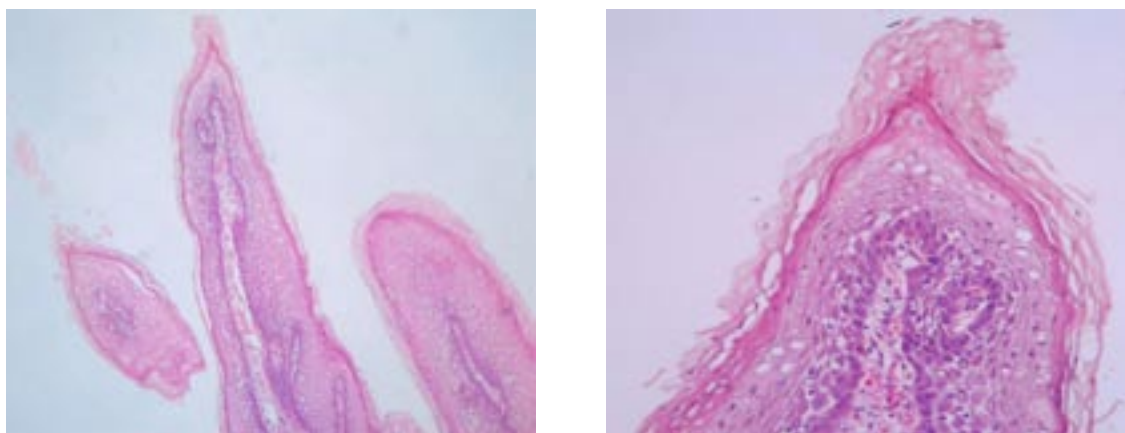
■ Figure 2: Mucosal lesion at distal esophagus (FICE Station1)



■ Figure 3: Lesion at distal esophagus (White light, 100 x magnifications)



■ Figure 4: Lesion at distal esophagus (FICE station 2, 100 x magnifications)



■ Figure 5: Showing finger-like projection and delicate vascularized tissue supporting broad layers of squamous epithelium.

Diagnosis:

Esophageal squamous papilloma

Discussion:

Esophageal papilloma is rare benign epithelial lesions characterized histologically by finger-like projections of tissue lined by an increased number of squamous cells and a core of connective tissue that contains small blood vessels¹. It is usually asymptomatic without characteristic symptoms although it may cause dysphagia. The etiology of esophageal squamous papilloma remains unclear. It has been suggested that chemical, viral or mechanical factors may contribute to the pathogenesis. Most etiology in humans is chronic irritation from reflux esophagitis; two-thirds of reported cases of esophageal papillomas are found in the distal esophagus^{1,2}. The enhanced image with magnification by

FICE can help for a better delineation of the lesion. Morphologically, it is a benign lesion, but there is much debate as to whether it is a premalignant lesion. At present, there is no evidence for this, and malignant changes have not been reported in humans^{2,3}.

References

1. Abeid M, Labib S, Neizamy E. Esophageal squamous papilloma. *Gastrointest Endosc* 2011;73:1035.
2. Mosca S, Manes G, Monaco R, et al. Squamous papilloma of the esophagus: long-term follow up. *J Gastroenterol Hepatol* 2001;16:857-61.
3. Orłowska J, Jarosz D, Gugulski A, et al. Squamous cell papillomas of the esophagus: report of 20 cases and literature review. *Am J Gastroenterol* 1994;89:434-7.



Case 12

Pornphan Thienchanachaiya, M.D.

Rapat Pittahyanon, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

Rungsun Rerknimitr, M.D.

A 45-year-old man presented with past history of squamous cell carcinoma of hypopharynx. He had been reported as cure after surgery. Esophagogastroduodenoscopy was performed for a surveillance of metachronous squamous cell carcinoma

under white light and FICE endoscopy. A well define annular salmon-colored velvety patch (1.5 cm in length) at upper-esophagus was found (Figure 1-2) and esophageal biopsy revealed regular antral gastric mucosa adjacent to normal esophageal mucosa.



■ Figure 1-2: A well define annular salmon-colored velvety patch (1.5 cm in length) at upper-esophagus (spanning 15-17 cm from incisor) with prominent margin (yellow arrow) under white light mode and FICE Station 1 (R550, B500, G420).

Diagnosis:

Esophageal Inlet patch

Discussion:

Inlet patch is a congenital anomaly of cervical esophagus consisting of gastric mucosa. It occurs most frequently in the postcricoid portion of the esophagus at

or just below the upper esophageal sphincter. The inlet patch found in 10% of the population with careful searching at endoscopy but it is often overlooked by endoscopists and radiologists and studies frequently report prevalence between 0.1 and 3%¹.

Most inlet patches are largely asymptomatic, but in problematic cases complications related to acid

secretion such as esophagitis, ulcer, web, and stricture may produce symptoms such as chest and throat pain, dysphagia, globus sensation, and shortness of breath. Amongst those with concurrent inlet patch and gastric *H. pylori* may infect inlet patch which exacerbate complications and related symptoms. Adenocarcinoma may arise in the ectopic gastric mucosa but this is rare and is considered sporadic. In contrast to Barrett's esophagus there is no increased risk for adenocarcinoma associated with inlet patches as they are not metaplastic¹⁻³.

At endoscopy, the lesion appears salmon-colored and velvety and is easily distinguished from the normal grey-white squamous epithelium of the esophagus⁴. Inlet patches range from 0.2 to 5 cm and can be round or oval with a flat, slightly raised, or depressed surface and may have heaped margins most often on the lateral or posterior surfaces⁵. Most inlet patches are solitary and extend longitudinally, affecting only part of the circumference, but some are annular and multiple lesions are not uncommon.

References

1. Behrens C, Yen PP. Esophageal inlet patch. *Radiol Res Pract* 2011;2011:460890.
2. Poyrazoglu OK, Bahcecioglu IH, Dagli AF, et al. Heterotopic gastric mucosa (inlet patch): endoscopic prevalence, histopathological, demographical and clinical characteristics. *Int J Clin Pract* 2009;63:287-91.
3. Weickert U, Wolf A, Schroder C, et al. Frequency, histopathological findings, and clinical significance of cervical heterotopic gastric mucosa (gastric inlet patch): a prospective study in 300 patients. *Dis Esophagus* 2011;24:63-8.
4. von Rahden BH, Stein HJ, Becker K, et al. Heterotopic gastric mucosa of the esophagus: literature-review and proposal of a clinicopathologic classification. *Am J Gastroenterol* 2004;99:543-51.
5. Jacobs E, Dehou MF. Heterotopic gastric mucosa in the upper esophagus: a prospective study of 33 cases and review of literature. *Endoscopy* 1997; 29:710-5.



Part 2

Stomach

Case 1

Kessarin Thanapirom, M.D.

Nopporn Anukularnkusol, M.D.

Rapat Pittayanon, M.D.

Rungsun Rerknimitr, M.D.

A 64-year-old woman presented with melena and chronic anemia for 4 months. She needs multiple sessions of blood transfusion. She has an underlying of non-alcoholic steatohepatitis. EGD was performed and showed in Figure 1-2.

Diagnosis:

Gastric antral vascular ectasia (GAVE)

Discussion:

Gastric antral vascular ectasia (GAVE), also named



■ Figure 1-2: Flat, erythematous punctuate lesions at antrum and pylorus.

watermelon stomach, is a rare entity, but is found in about 4% of all non-variceal upper gastrointestinal bleeding¹. The pathogenesis of GAVE remains poorly understood. Although GAVE is usually found in patients with severe co-morbidities like liver cirrhosis, it is also found in autoimmune connective tissue diseases, chronic renal failure and bone marrow transplantation. It has become clear, however, that portal hypertension does not play an important role in the development of GAVE. This is supported by findings that there is no significant correlation between the degree of vascular ectasia (mean mucosal capillary cross-sectional area) with the degree of portal hypertension and lack of response to measures reducing portal pressures (beta-blockade, TIPS)^{2,3}.

It is vitally important to distinguish between GAVE and portal hypertensive gastropathy. There are distinct entities that require different treatments. Whereas GAVE is most commonly limited to the antrum, portal hypertensive gastropathy (PHG) predominantly causes changes of the mucosa in the fundus and corpus⁴. GAVE patients have more severe liver disease, greater blood loss, lower serum gastrin levels and a higher incidence of previous sclerotherapy⁵. Biopsy was the best way of distinguishing between GAVE and PHG. Microvascular thrombi, vascular ectasia, spindle cell proliferation and fibrohyalinosis in antral biopsies have all been shown to be significantly more associated with

GAVE than PHG. Visible columns of red tortuous ectatic vessels along the longitudinal folds of the antrum are pathognomonic endoscopic findings for GAVE⁶. The typical lesion is limited to the antrum.

References

1. Dulai GS, Jensen DM, Kovacs TO, et al. Endoscopic treatment outcomes in watermelon stomach patients with and without portal hypertension. *Endoscopy* 2004;36:68-72.
2. Spahr L, Villeneuve JP, Dufresne MP, et al. Gastric antral vascular ectasia in cirrhotic patients: absence of relation with portal hypertension. *Gut* 1999; 44:739-42.
3. Perez-Ayuso RM, Pique JM, Bosch J, et al. Propranolol in prevention of recurrent bleeding from severe portal hypertensive gastropathy in cirrhosis. *Lancet* 1991;337:1431-4.
4. Burak KW, Lee SS, Beck PL. Portal hypertensive gastropathy and gastric antral vascular ectasia (GAVE) syndrome. *Gut* 2001;49:866-72.
5. Payen JL, Cales P, Voigt JJ, et al. Severe portal hypertensive gastropathy and antral vascular ectasia are distinct entities in patients with cirrhosis. *Gastroenterology* 1995;108:138-44.
6. Stotzer PO, Willen R, Kilander AF. Watermelon stomach: not only an antral disease. *Gastrointest Endosc* 2002;55:897-900.



Case 2

Kriangsak Charoensuk, M.D.

Linda Pantongrag-Brown, M.D.

Rungsun Rerknimitr, M.D.

A 62-year-old woman presented with progressive jaundice and acute cholangitis. She had no history of recurrent epistaxis and no episode of GI bleeding. Physical examination showed icteric sclera with multiple telangiectases at the lower lip (Figure 1). Computed tomography of the abdomen showed portal

AVM with mild intrahepatic duct dilatation secondary to vascular compression (Figure 2-3). She underwent ERCP that found choledocholithiasis with CBD dilatation and extraluminal compression. EGD revealed multiple angiodysplasias of gastric antrum, body, and duodenal bulb (Figure 3-8).



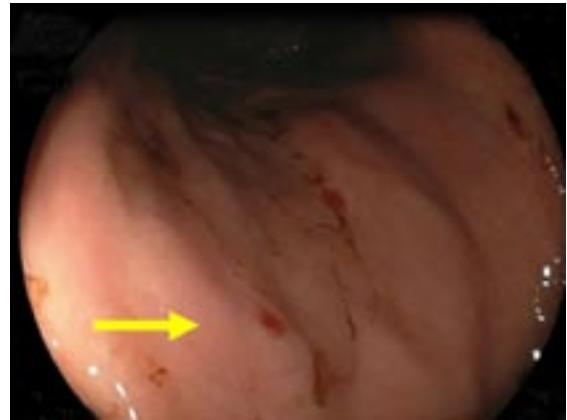
■ Figure 1: Telangiectasia at lower lip



■ Figure 2A-2B: Portal AVM and dilatation of left intrahepatic duct



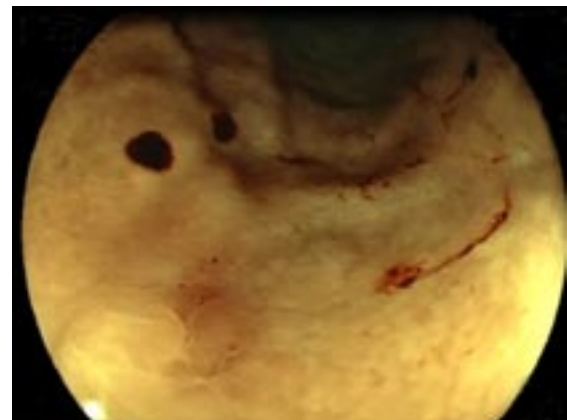
■ Figure 3: Multiple angiodysplasias in gastric antrum



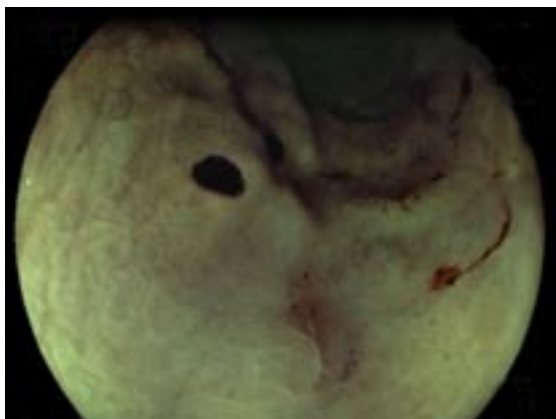
■ Figure 4: Angiodysplasias at lesser curvature of gastric body



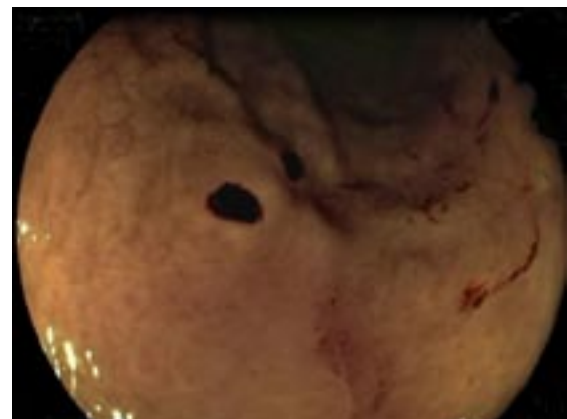
■ Figure 5: FICE image station 0



■ Figure 6: FICE image station 5



■ Figure 7: FICE image station 7



■ Figure 8: FICE image station 8

Diagnosis:

Osler-Weber-Rendu disease (Hereditary Hemorrhagic telangiectasia) with portal biliopathy from portal AVM

Discussion:

Hereditary hemorrhagic telangiectasia (HHT), inherited as an autosomal dominant trait, affects approximately 1 in 5,000 people¹⁻⁴. The spectrum of disease extends beyond the telangiectasia/AVM. This disease is diagnosed by the Curaçao criteria which is based on the presence of at least three of four main clinical features: nose bleeding history, mucocutaneous telangiectasia, visceral involvement (pulmonary, cerebral, hepatic and spinal arteriovenous malformation), and affected first degree relative¹⁻⁴. Liver involvement consists of extensive intrahepatic vascular malformation associated with blood shunting (arteriovenous, arterioportal and/or portovenous), which leads to significant systemic and hepatobiliary abnormalities. The prevalence of hepatic involvement in HHT was 8-

31% in many retrospective studies¹⁻³. The three most common initial clinical presentations are high-output heart failure, portal hypertension, and biliary disease. Biliary involvement characterized by right upper quadrant pain, cholestasis with or without cholangitis. Imaging studies demonstrates biliary stricture or obstruction from vascular impression, and/or bile cysts¹⁻⁴.

References

1. Larson AM. Liver disease in hereditary hemorrhagic telangiectasia. *J Clin Gastroenterol* 2003;36:149-58.
2. Khalid SK, Garcia-Tsao G. Hepatic vascular malformations in hereditary hemorrhagic telangiectasia. *Semin Liver Dis* 2008;28:247-58.
3. Garcia-Tsao G. Liver involvement in hereditary hemorrhagic telangiectasia (HHT). *J Hepatol* 2007; 46:499-507.
4. Shovlin CL. Hereditary haemorrhagic telangiectasia: pathophysiology, diagnosis and treatment. *Blood Rev* 24:203-19.



Case 3

Nuttaporn Norrasetwanich, M.D.

Rapat Pittayanon, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

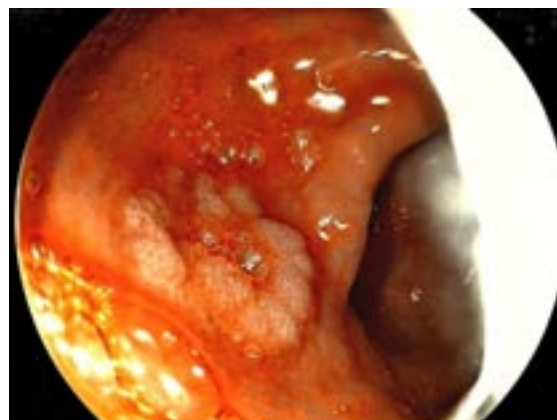
Rungsun Rerknimitr, M.D.

A 67-year-old woman with underlying of cryptogenic cirrhosis child B underwent an EGD for esophageal varices surveillance. Standard white light EGD and FICE with magnification showed 1 cm whitish

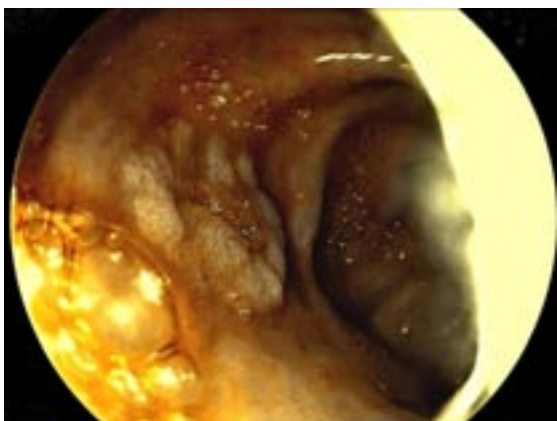
plaque, with irregular surface, at the lesser curvature of gastric antrum (Figure 1-6). Biopsy was performed. Histology demonstrated tubular adenomatous change of the gastric glands with focal high grade dysplasia (Figure 7).



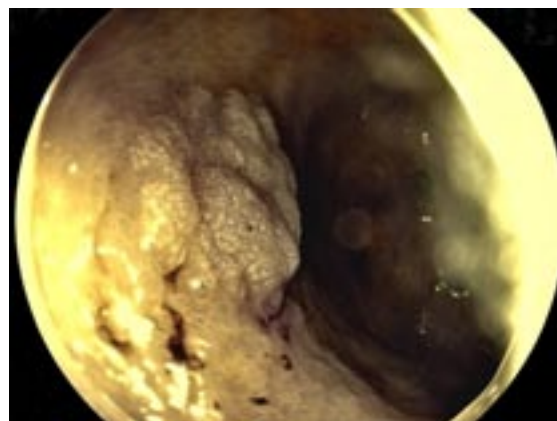
■ Figure 1



■ Figure 2: FICE no.2



■ Figure 3: FICE no.4



■ Figure 4: FICE no.4



■ Figure 5



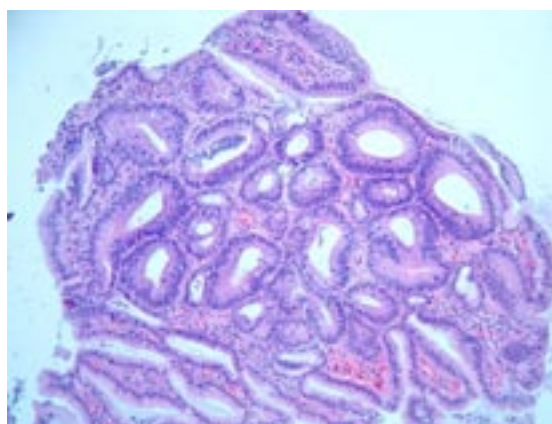
■ Figure 6: FICE no.2 with x50 magnification

Diagnosis:

Adenomatous polyp with focal high grade dysplasia, tubular proliferation

Discussion:

Gastric adenomas are precancerous neoplastic lesion. They are histologically classified as tubular, villous and tubulovillous types. Adenomatous polyps may occur sporadically or in association with FAP. Endoscopically, adenomatous polyps are typically velvety, lobulated solitary (82%), located in the antrum, typically with size less than 2 centimeters (cm) in diameter. These polyps can be circumscribed lesions, pedunculated or sessile. Histology reveals dysplastic epithelium without detectable invasion of the lamina propia. Their prevalence varies widely and is estimated to be 0.5–3.75% in western countries and 9–27% in prevalent areas of gastric carcinoma, such as China and Japan. Sporadic, gastric adenomatous polyps may be considered as one of the possible steps in the development of gastric adenocarcinoma. Both conditions are often found in patients with chronic, atrophic, metaplastic gastritis. In addition, they share a common epidemiological pattern. There is no proven association with *H. pylori* infection. The larger adenomatous polyp, the greater chance for polyp to contain foci of



■ Figure 7

adenocarcinoma. A synchronous adenocarcinoma in another area of the stomach has been found in up to 30% of patients with an adenomatous polyp, and up to 50% of adenomatous polyps larger than 2 cm in diameter harbor a focus of adenocarcinoma. Neoplastic progression is greater when polyps are larger than 2 cm in diameter and this occurs in 28.5–40% of villous adenomas and 5% of tubular adenomas.

The risk of association between adenomatous polyps and cancer increases with age^{1,2}. The guidelines of the American Society of Gastrointestinal endoscopy (ASGE) recommend that adenomatous gastric polyps are at increased risk for malignant transformation and should be resected completely. Surveillance endoscopy 1 year

after removing adenomatous gastric polyps is reasonable to assess recurrence at the prior excision site, new or previously missed polyps, and/or supervening early carcinoma. If the result of the examination is negative, repeat surveillance endoscopy should be performed not more frequently than at 3- to 5-year intervals. Follow-up after resection of polyps with high-grade dysplasia or early gastric cancer should be individualized³.

References

1. Goddard AF, Badreldin R, Pritchard DM, et al. The management of gastric polyps. *Gut* 2010;59:1270-6.
2. Carmack SW, Genta RM, Graham DY, Lauwers GY. Management of gastric polyps: a pathology-based guide for gastroenterologists. *Nat Rev Gastroenterol Hepatol* 2009;6:331-41.
3. Hirota WK, Zuckerman MJ, Adler DG, et al. ASGE guideline: the role of endoscopy in the surveillance of premalignant conditions of the upper GI tract. *Gastrointest Endosc* 2006;63:570-80.



Case 4

Suparat Khemnark, M.D.

Rapat Pittayanon, M.D.

Rungsun Rerknimitr, M.D.

A 76-year-old woman underwent an EGD in order to follow up a gastric ulcer. Endoscopy showed a

healed gastric ulcer with a large gastric diverticulum in the antrum (Figure 1-2).



■ Figure 1: Wight light endoscopy: gastric diverticulum at antrum



■ Figure 2: FICE station 2

Diagnosis:

Large gastric diverticulum

Discussion:

Gastric diverticulum is rare and commonly incidentally detected from screening EGD. Its prevalence ranges from 0.04% by contrast study radiographs to 0.01%-0.11% by EGD database. Mostly gastric diverticulum are asymptomatic however it may present with a vague sensation of fullness or discomfort in the upper abdomen¹. A gastric diverticulum should be

differentiated from a gastroduodenal fistula, or a double-channel pylorus, which is caused by a penetrating ulcer in the distal antrum that directly erodes into the base of the duodenal cap or into the bulb².

References

1. Gockel I, Thomschke D, Lorenz D. Gastrointestinal: Gastric diverticula. *J Gastroenterol Hepatol* 2004; 19:227.
2. Bhattacharya K. Gastric diverticulum - 'Double pylorus appearance'. *J Minim Access Surg* 2005;1:39.

Case 5

Tanassanee Soontornmanokul, M.D.

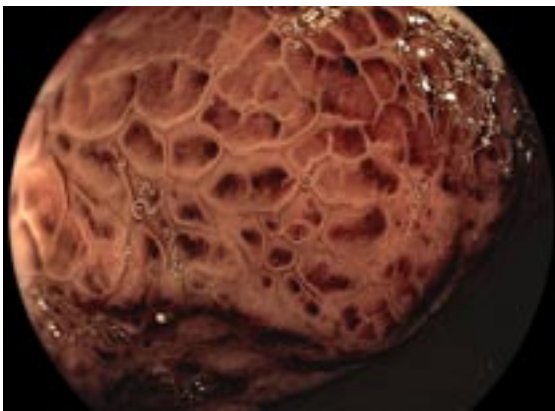
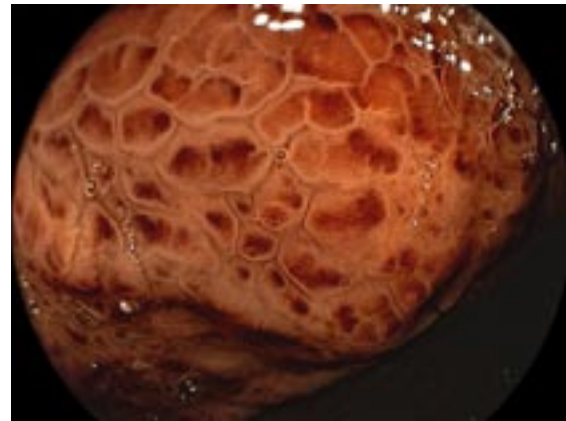
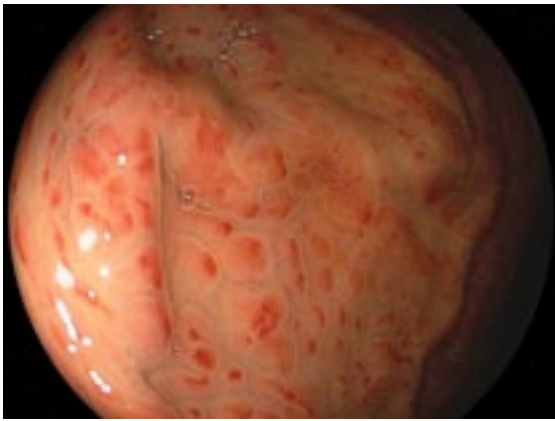
Rapat Pittayanon, M.D.

Sombat Treeprasertsuk, M.D.

Rungsun Rerknimitr, M.D.

A 52-year-old man with underlying disease of compensated alcoholic cirrhosis underwent upper endoscopic examination for esophageal varices surveillance. It revealed the snake-skin mosaic pattern with flat hemorrhagic spots in gastric fundus, which was

compatible with severe portal hypertensive gastropathy (PHG). White light endoscopy (WLE) and Flexible Spectral Imaging Color Enhancement (FICE) system (station 2 and 3) without magnification was applied to examine the lesions. Findings are shown in Figure 1-3.



■ Figure 1-3: White light endoscopic image showed hemorrhagic gastric mucosa arranging in mosaic pattern. The feature was better depicted by FICE at different stations

Diagnosis:

Severe portal hypertensive gastropathy

Discussion:

Portal hypertensive gastropathy (PHG) is characterized by typical gastric mucosal lesions associated with portal hypertension. Typical location is in gastric fundus and upper body of the stomach although it can present in entire gastric mucosa or even in other part of gastrointestinal tract, including the small bowel or the colon. PHG may mimic with diffuse form of gastric antral vascular ectasia (GAVE)¹. It is usually asymptomatic but, when symptomatic, it most frequently causes chronic gastrointestinal blood loss and iron deficiency anemia. PHG may present with hematemesis and/or melena, as an uncommon cause of acute gastrointestinal bleeding (GIB) in patients with cirrhosis². Diagnosis of acute GIB from PHG is established when active bleeding from gastropathy lesions or non-removable clots overlying these lesions is observed or

when there is PHG without other cause of GI bleeding can be demonstrated³. Non-selective beta-blockers have been shown to decrease bleeding from both acute and chronic forms of bleeding from PHG⁴.

References

1. Ripoll C, Garcia-Tsao G. The management of portal hypertensive gastropathy and gastric antral vascular ectasia. *Dig Liver Dis* 2011;43:345-51.
2. Gostout CJ, Viggiano TR, Balm RK. Acute gastrointestinal bleeding from portal hypertensive gastropathy: prevalence and clinical features. *Am J Gastroenterol* 1993;88:2030-3.
3. Ripoll C, Garcia-Tsao G. Management of gastropathy and gastric vascular ectasia in portal hypertension. *Clin Liver Dis* 2010;14:281-95.
4. Perez-Ayuso RM, Pique JM, Bosch J, et al. Propranolol in prevention of recurrent bleeding from severe portal hypertensive gastropathy in cirrhosis. *Lancet* 1991;337:1431-4.



Case 6

Suparat Khemnark, M.D.

Rapat Pittayanon, M.D.

Linda Pantongrag-Brown, M.D.

Sombat Treeprasertsuk, M.D.

Rungsun Rerknimitr, M.D.

A 61-year-old man, diagnosed as cirrhosis with hepatoma from hepatitis B virus, underwent an EGD for esophageal varices surveillance. EGD with Flexible Spectral Imaging Color Enhancement (FICE) system was performed. It revealed large gastric varices at the cardia

without recent bleeding stigmata (Figure 1-2). CT scan of the abdomen showed an enhancing tortuous, tubular structure on gastric fundus mucosa which was consistent with gastric varices (Figure 3).



■ Figure 1: WLE (Retroflex view)



■ Figure 2: FICE station 2



■ Figure 3: CT scan shows enhancing gastric varices at its fundus (arrow)

Diagnosis:

Gastric varices

Discussion:

Gastric varices are dilated submucosal veins within the wall of the stomach. Sarin et al. has classified gastric varices into four anatomical types; 2 types of gastroesophageal varices and 2 types of isolated gastric varices. Type 1 gastroesophageal varices (EGV) which

involve lesser curvature of the stomach are the most common type. Type 2 gastroesophageal varices located on greater curvature are associated with higher mortality rate and can bleed easily. Type 1; isolated gastric varices involve only gastric fundus; have a high incidence of bleeding. Type 2; isolated gastric varices; are mainly ectopic¹.

Splenomegaly, portal venous thrombosis, platelet count $<135,000/\text{mm}^3$, and albumin $<3.5 \text{ g/dl}$ are independent predictors of large EGV in hepatocellular carcinoma patients². Most of these predictors are related to the complications of portal hypertension².

On CT scan, varices appear round, tubular, or serpentine structures that are smooth with homogeneous

attenuation, and enhance with contrast material to the same degree as adjacent vessels³.

References

1. Sarin SK, Lahoti D, Saxena SP, et al. Prevalence, classification and natural history of gastric varices: a long-term follow-up study in 568 portal hypertension patients. *Hepatology* 1992;16:1343-9.
2. Yeh JL, Peng YC, Tung CF, et al. Clinical predictors of large esophagogastric varices in patients with hepatocellular carcinoma. *Dig Dis Sci* 2002;47:723-9.
3. Cho K C, Patel Y D, Wachsberg R H, et al. Varices in portal hypertension: evaluation with CT Radio Graphics 1995;15:609-22.



Case 7

Suparat Khemnark, M.D.

Rapat Pittayanon, M.D.

Sombat Treeprasertsuk, M.D.

Rungsun Rerknimitr, M.D.

A 65-year-old woman had been fed via balloon-type percutaneous gastrostomy tube for 2 years due to acute stroke and bed-ridden status. Over the last month, she suffered from peri-stoma leakage causing irritation on skin after gastrostomy tube was exchanged. Two weeks later, she came back to the hospital and the physician increased water volume to the balloon. Probably by

gravity the PEG tube accidentally migrated down and left with only a short distance of feeding end near the skin (Figure 1). EGD found migration of balloon gastrostomy tube to duodenum. Balloon was deflated and removed. Gastric ulcer under the area of compression was found (Figure 2-3). A new PEG (non-balloon type) placement was done.



■ Figure 1: Shortening of gastrostomy tube



■ Figure 2: PEG migration



■ Figure 3: Compression ulcer

Diagnosis:

Gastrostomy tube migration

Discussion:

PEG tube placement is associated with several complications. The migration of the balloon into the pylorus, duodenum or proximal jejunum can cause various symptoms including abdominal pain, recurrent vomiting and increased leakage around the stoma, jaundice, pancreatitis, and gastrointestinal obstruction¹⁻³.

Upper gastrointestinal study can confirm the diagnosis. In the case of balloon type PEG tube, deflating the balloon and pulling the tube back should be done in order to relieve the symptom¹.

References

1. Schrag SP, Sharma R, Jaik NP, et al. Complications related to percutaneous endoscopic gastrostomy (PEG) tubes. A comprehensive clinical review. *J Gastrointestin Liver Dis* 2007;16:407-18.
2. Date RS, Das N, Bateson PG. Unusual complications of ballooned feeding tubes. *Ir Med J* 2002;95:181-2.
3. Bhat M, Bridges E. Acute obstructive pancreatitis caused by a migrated balloon gastrostomy tube. *CMAJ* 2011;183:E759.



Case 8

Nuttaporn Norrasetwanich, M.D.

Rapat Pittayanon, M.D.

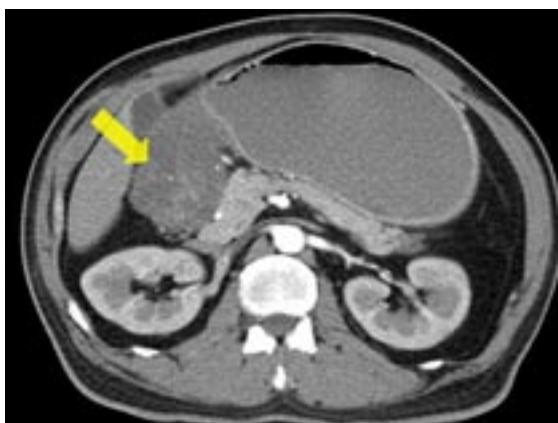
Linda Pantongrag-Brown, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

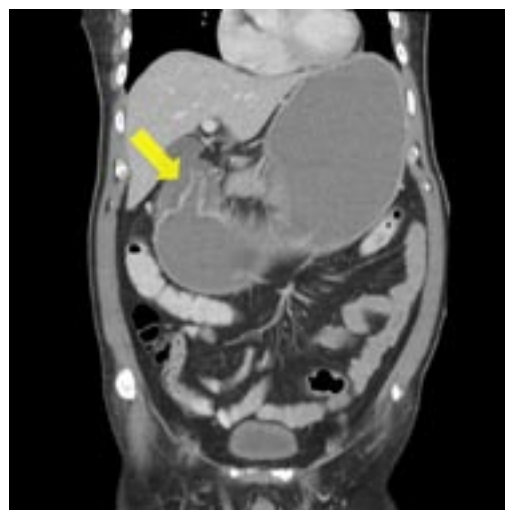
Rungsun Rerknimitr, M.D.

A 56-year-old man presented with bilious, post-prandial vomiting for 1 month. He had lost his weight, about 30 kgs in a few months. Physical examination revealed hyperactive bowel sound with succussion splash at the epigastrium. Epigastric mass was palpable. Thus, gastric outlet obstruction was diagnosed. CT of the whole abdomen demonstrated a circumferential mass with multiple small calcifications involving the antrum and pylorus, causing gastric outlet obstruction (Figure 1-2). EGD revealed a large circumferential ulcerative mass, containing necrotic tissue at the antrum of the

stomach, which was easily bled with contact. Scope could not pass through the pylorus due to complete obstruction by the antral mass (Figure 3-8). Biopsy specimen showed patchy infiltration of tumor cells possessing pleomorphic nuclei with vacuolated cytoplasm. Nuclei were placed at periphery. The tumor cells were surrounded with mild mucinous lake and inflammatory background. Focal necrosis and mitoses were also observed. The diagnosis was poorly-differentiated adenocarcinoma with signet-ring cell appearance (Figure 9-10).



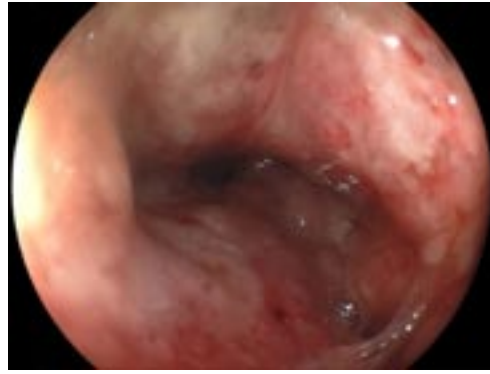
■ Figure 1



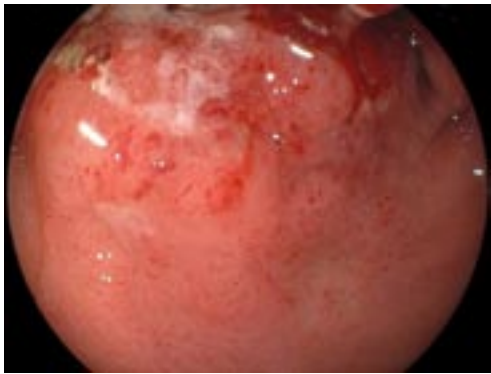
■ Figure 2



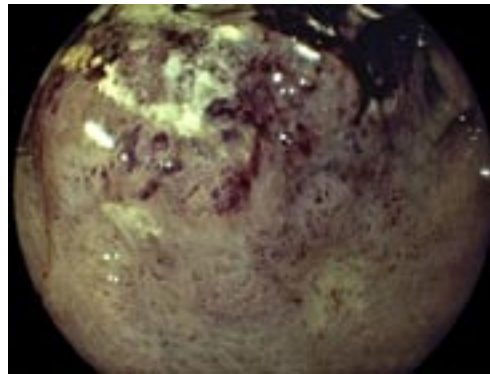
■ Figure 3



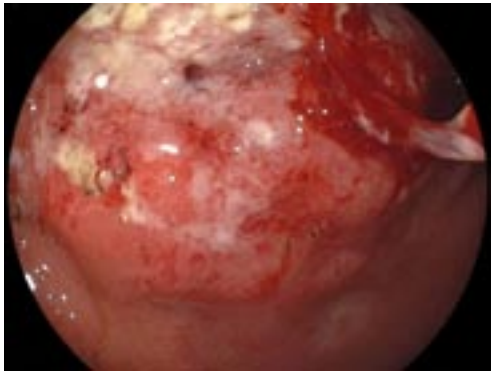
■ Figure 4



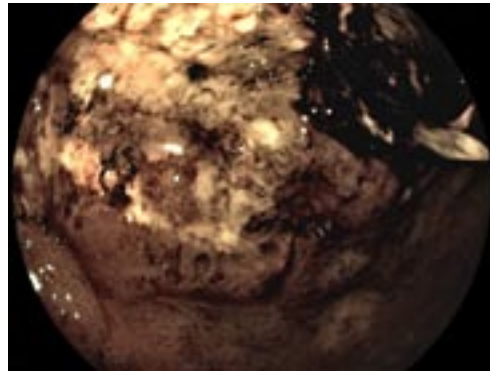
■ Figure 5



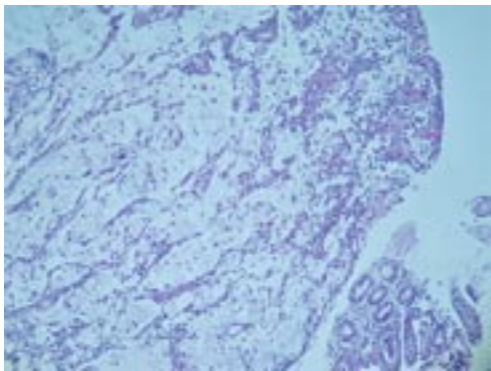
■ Figure 6: FICE station 8



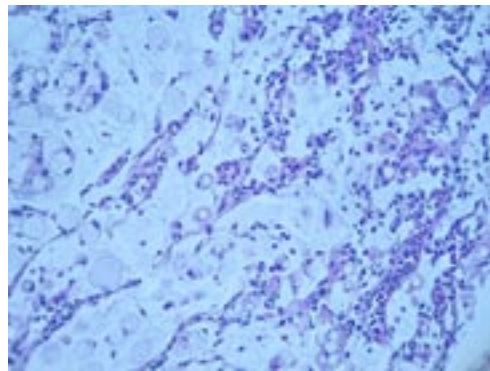
■ Figure 7



■ Figure 8: FICE station 3



■ Figure 9



■ Figure 10

Diagnosis:

Gastric adenocarcinoma (poorly-differentiated adenocarcinoma with signet-ring cell appearance)

Discussion:

Gastric signet ring cell carcinoma (SRC) is a histological diagnosis, based on the microscopic characteristics of the tumor as described by the World Health Organization (WHO). SRC is characterized by its poor prognosis and potential to infiltrate the stomach wall¹. Signet ring cell carcinoma of the stomach has wide range of incidence, from 3.4% in Japan to 29% in Western countries. It is reported to be more frequent in female patients and patients of younger age than those with non-signet ring cancers. Signet ring cancers typically diffusely infiltrate the gastric wall and cause marked desmoplastic reaction. They tend to be larger and to spread superficially in the mucosa and submucosa, making them amenable to early detection. The natural history of this disease is quite aggressive, although the majority of cases will not have transmural invasion².

SRC-resected patients exhibited higher rates of localized peritoneal carcinomatosis, lymph node involvement at diagnosis, lower R0 resection rate, and earlier tumor relapse. This study showed that SRC is a major and independent predictor of poor prognosis due to infiltrative nature of the tumor with high affinity for lymphatic tissue, and a higher rate of peritoneal carcinomatosis³.

References

1. Zhang M, Zhu G, Zhang H, et al. Clinicopathologic features of gastric carcinoma with signet ring cell histology. *J Gastrointest Surg* 2010;14:601-6.
2. Wu CH, Chen MJ, Chang WH, et al. Signet-ring type adeno-carcinoma arising from a tiny gastric polyp. *Gastrointest Endosc* 2008;67:724-5.
3. Piessen G, Messager M, Leteurtre E, et al. Signet ring cell histology is an independent predictor of poor prognosis in gastric adenocarcinoma regardless of tumoral clinical presentation. *Ann Surg* 2009; 250:878-87.



Case 9

Suparat Khemnark, M.D.

Rapat Pittayanon, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

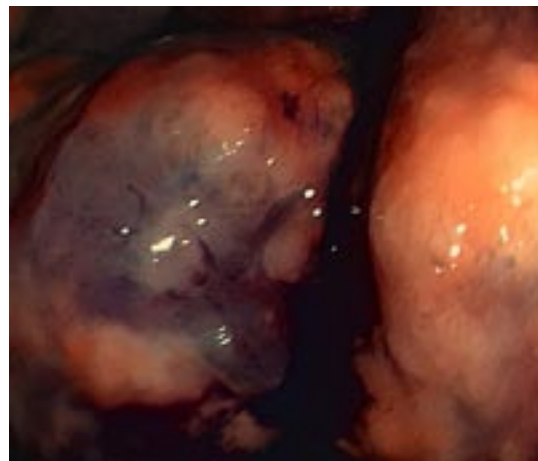
Rungsun Rerknimitr, M.D.

A 77-year-old female, presented with intermittent melena for 3 months. Her physical examination showed cervical lymphadenopathy. EGD with FICE system was done and found a huge

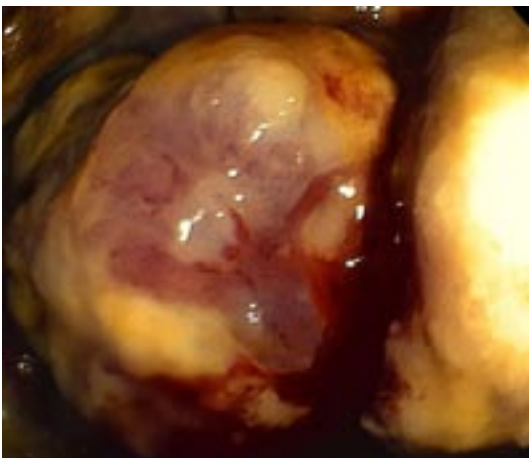
exophytic mass at gastric antrum as shown in Figure 1-4. Biopsy was done and the pathological report revealed moderately-differentiated adenocarcinoma (Figure 5).



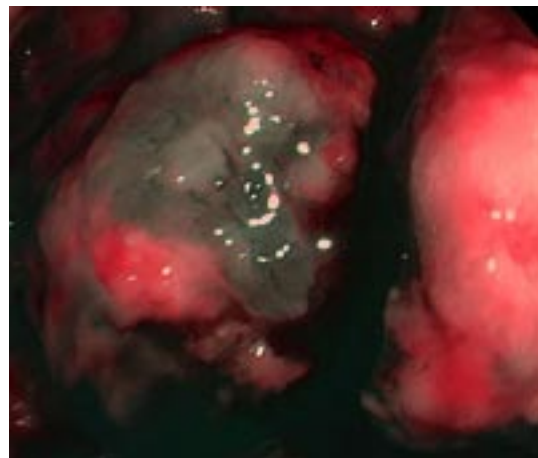
■ Figure 1: Huge exophytic mass at the antrum (White light endoscopy)



■ Figure 2: FICE station 2 station 8



■ Figure 3: FICE station 6



■ Figure 4: FICE



■ Figure 5: Histopathology showed markedly pleomorphic nuclei with visible nucleoli : Moderately-differentiated adenocarcinoma (No *H.pylori* seen)

Diagnosis:

Adenocarcinoma of stomach

Discussion:

Gastric cancer is often asymptomatic in early stage. If patients are symptomatic, they already have advanced incurable disease at the time of presentation. Weight loss and persistent abdominal pain are the most common symptoms at initial diagnosis (50-60%) and the second most common symptom is melena at 20%¹.

EGD is the currently the procedure of choice for the diagnosis of gastric cancer. Tissue diagnosis and anatomic localization of the primary tumor are best obtained by EGD. Distinct irregular mucosal surface and vascular patterns have been found to correlate with the

presence of dysplasia and carcinoma². About 90% to 95% of cancerous (malignant) tumors of the stomach are adenocarcinomas. Endoscopic findings of a gastric adenocarcinoma are in variety appearances such as mass (exophytic mass, circumferential mass) or depressed mucosal lesion (ulcerated mucosa).

References

1. Wanebo HJ, Kennedy BJ, Chmiel J, et al. Cancer of the stomach: A patient care study by the American college of surgeons. *Ann Surg* 1993;218:583-92.
2. Tanaka K, Toyoda H, Kadowaki S, et al. Surface pattern classification by enhanced-magnification endoscopy for identifying early gastric cancers. *Gastrointest Endosc* 2008;67:430-7.



Case 10

Rapat Pittayanon, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

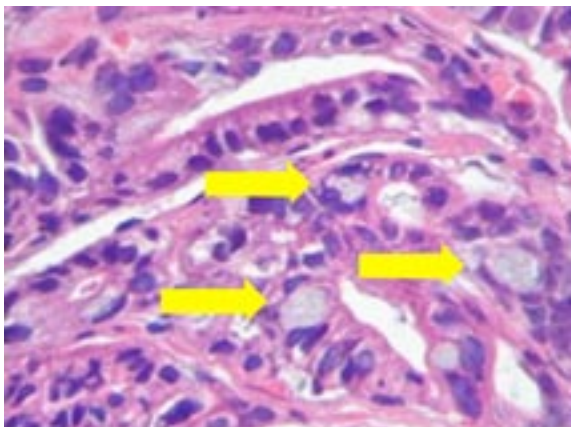
Rungsun Rerknimitr, M.D.

A 35-year-old woman presented with chronic intermittent dyspepsia for 2 years. Her symptom was not respond by PPI therapy. EGD with 50x to 100x magnified FICE was performed. It revealed light blue crest (LBC) which is a fine, blue-white line on the crest of epithelial surface (Figure 1A), large long crest (LLC) which is a

combination of linear dark and light areas (Figure 1B), and villous pattern (VP) which is a raised area of villi above the gastric mucosal surface (Figure 1C). Biopsies for gastric intestinal metaplasia (GIM) diagnosis from many abnormal areas were done (Figure 2).



■ Figure 1: FICE station 8 showed A) light blue crest (LBC) with 50x magnification, B) large long crest (LLC) with 50x magnification and C) villous pattern (VP) with 100x magnification



■ Figure 2: Histology revealed eosinophilic absorptive enterocytes, with well-defined brush borders, alternate with a well-formed goblet cells (arrows) (Complete-type GIM)

Diagnosis:

Gastric intestinal metaplasia (GIM)

Discussion:

GIM is a well known premalignant lesion for gastric cancer¹. From the current studies, the accuracy of digital chromoendoscopy (esp. NBI) for GIM diagnosis is varying from 78%-98% by using light blue crest (LBC) criteria²⁻⁴. Rerknimitr et al. proposed the other two findings for GIM diagnosis which are light long crest (LLC) and villous pattern (VP). However LLC and VP showed low sensitivity (17% and 29%, respectively) with good specificity (95% and 97%, respectively)⁴.

Reference

1. Correa P, Piazuelo MB, Wilson KT. Pathology of gastric intestinal metaplasia: clinical implications. *Am J Gastroenterol* 2010;105:493-8.
2. Uedo N, Ishihara R, Iishi H, et al. A new method of diagnosing gastric intestinal metaplasia: narrow-band imaging with magnifying endoscopy. *Endoscopy* 2006;38:819-24.
3. Capelle LG, Haringsma J, de Vries AC, et al. Narrow band imaging for the detection of gastric intestinal metaplasia and dysplasia during surveillance endoscopy. *Dig Dis Sci* 2010;55:3442-8.
4. Rerknimitr R, Imraporn B, Klaikeaw N, et al. Non-sequential narrow band imaging for targeted biopsy and monitoring of gastric intestinal metaplasia. *World J Gastroenterol* 2011;17:1336-42.



Part 3

Small bowel

Case 1

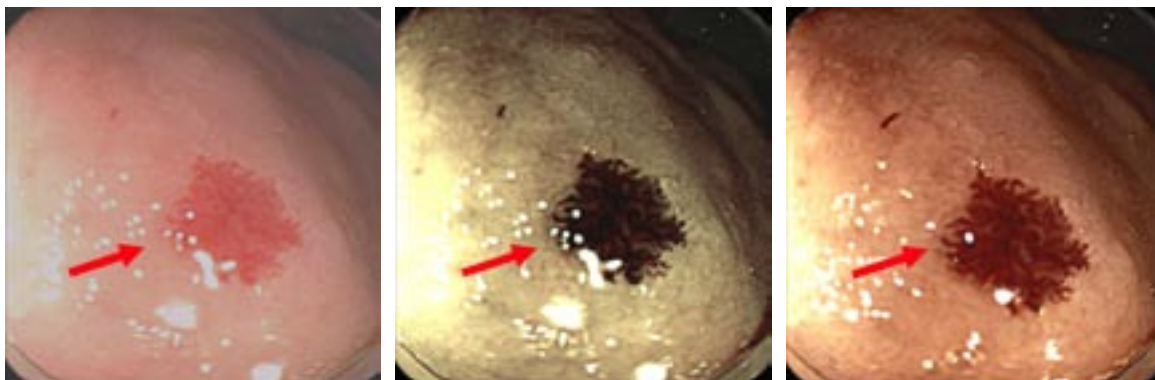
Pornphan Thienchanachaiya, M.D.

Satimai Aniwat, M.D.

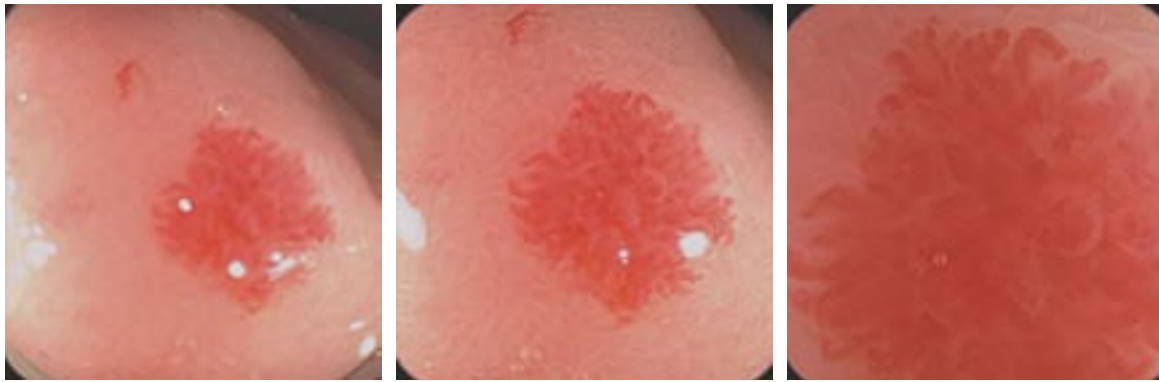
Rungsun Rerknimitr, M.D.

A 67-year-old man presented with iron deficiency anemia and congestive heart failure. He had an underlying disease of dilated cardiomyopathy, chronic atrial fibrillation, pulmonary hypertension, and alcoholic cirrhosis diagnosed for 8 years. Physical examination found mild pale conjunctiva

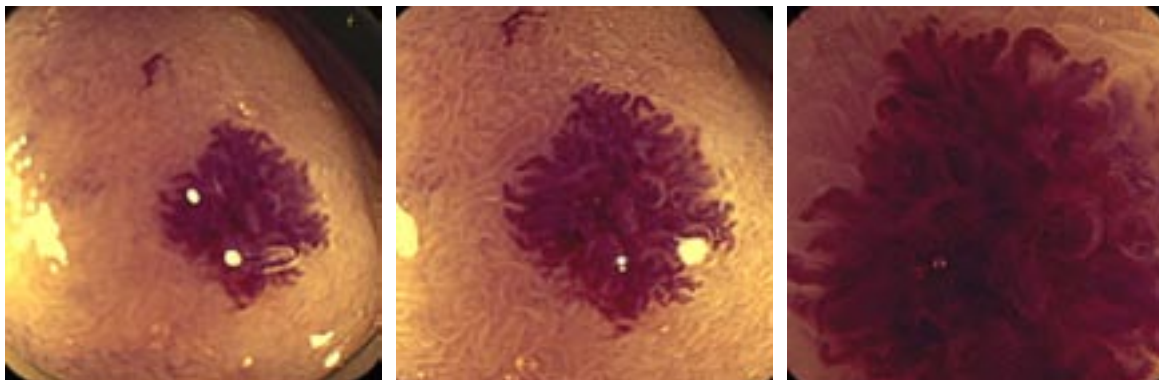
and signs of congestive heart failure. Blood tests were compatible with iron deficiency anemia. EGD was performed (Figure 1-9) and argon plasma coagulation was applied at the lesion.



■ Figure 1-3: The study showed an isolated angioectasia at duodenal bulb (red arrow), size 1 cm in diameter under white light, FICE station 0 (R525, B495, G455), and station 2 (R550, B500, G470). The lesion appeared more prominent under FICE.



■ Figure 4-6: An angioectasia under white light and at x50 and x100 magnification showed a coral reef-like pattern of small vessels.



■ Figure 7-9: Under FICE Station 9 (R550, B500, G400) and at x50 and x100 magnification, the detail of this vascular lesion was well demonstrated.

Diagnosis:

Duodenal angioectasia

Discussion:

Angioectasia, also named angiodysplasia, are vascular malformations that can be found throughout the gastrointestinal tract, with the most common site being in the right colon. These lesions may occasionally cause significant bleeding but they are usually found in symptom-free patients. In terms of patient presentation, angioectasias are most common in elderly patients undergoing an evaluation for gastrointestinal bleeding. Angioectasias are proposed to be the result of a degenerative process. The prevalence is estimated to 0.9-3.0% in non-bleeding patients and up to 6% in patients with evidence of blood loss¹.

The visibility of vascular ectasias depends on their size, hydration, hemoglobin level, blood flow, and use of narcotic drugs. This case showed the ability of

FICE to enhance the appearance of vascular malformation. The vessels appear much darker than the surrounding mucosa under FICE. The obscure lesion can be made more visualized under FICE².

Endoscopic treatments of angiodysplasia are thermal coagulation and argon plasma coagulation (APC). However, APC is more appropriate because the depth of coagulation is usually superficial and angiodysplasia always locates at the mucosal level³.

References

1. Foutch PG. Angiodysplasia of the gastrointestinal tract. *Am J Gastroenterol* 1993;88:807-18.
2. Ringold DA, Sikka S, Banerjee B. High-contrast imaging (FICE) improves visualization of gastrointestinal vascular ectasias. *Endoscopy* 2008;40 Suppl 2:E26.
3. Suzuki N, Arebi N, Saunders BP. A novel method of treating colonic angiodysplasia. *Gastrointest Endosc* 2006;64:424-7.

Case 2

Tanassanee Soontornmanokul, M.D.

Phonthep Angsuwatcharakon, M.D.

Vichai Viriyautsahakul, M.D.

Linda Pantongrag-Brown, M.D.

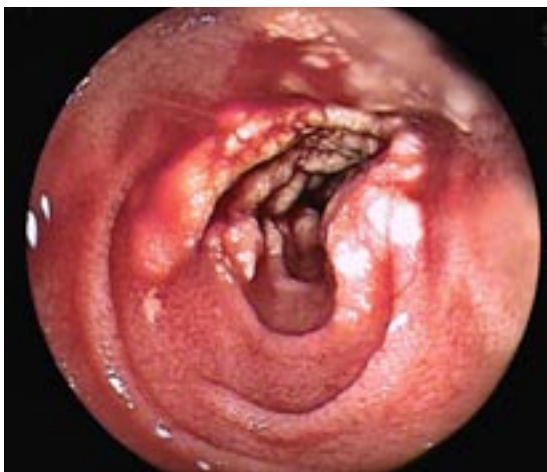
Naruemon Wisedopas-Klaikeaw, M.D.

Rungsun Rerknimitr, M.D.

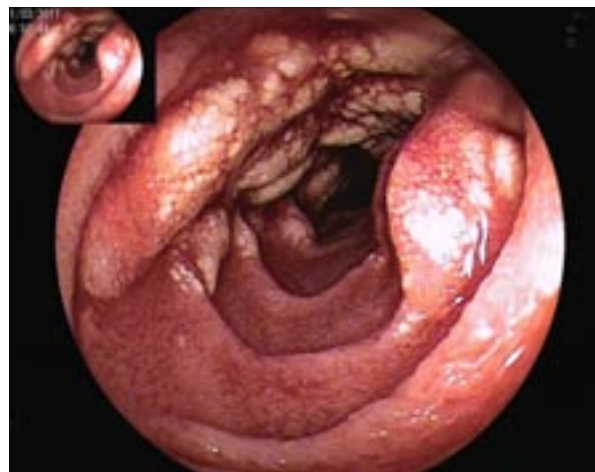
A 20-year-old Thai male presented with anemic symptoms for one week. He had the history of melena without hematemesis for a few days. There was no abdominal pain or other alarming symptoms. He came to the emergency room with another episode of acute bleeding. The esophagogastroduodenoscopy and colonoscopy revealed nothing. He was diagnosed and managed as overt obscure GI bleeding. Double Balloon enteroscopy was later performed in this patient via oral route. During double balloon enteroscopy (DBE), a segment of distal jejunum, approximately 15 cm

long, was visualized as a nearly circumferential lymphangiectatic change of its mucosa with oozing blood (Figure 1-5). The biopsy was performed at the lesion, and after water flushing, there was no further active bleeding.

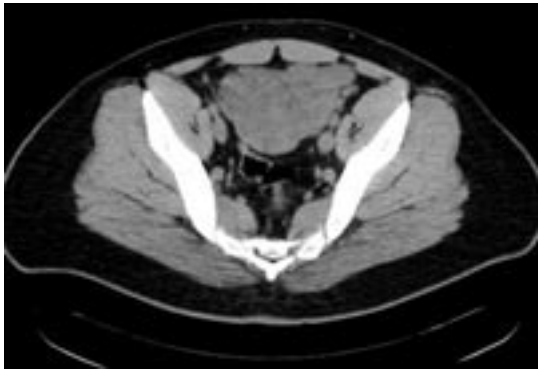
One month later, the patient underwent exploratory laparotomy of the abdomen and the mass at distal jejunum was totally removed and sent for histopathology (Figure 6-12). The operation was successful and the patient had no recurrence of GI bleeding.



■ Figure 1: Endoscopic findings included edematous mucosa, thickened fold, submucosal hemorrhages, and segmental lymphangiectasia.



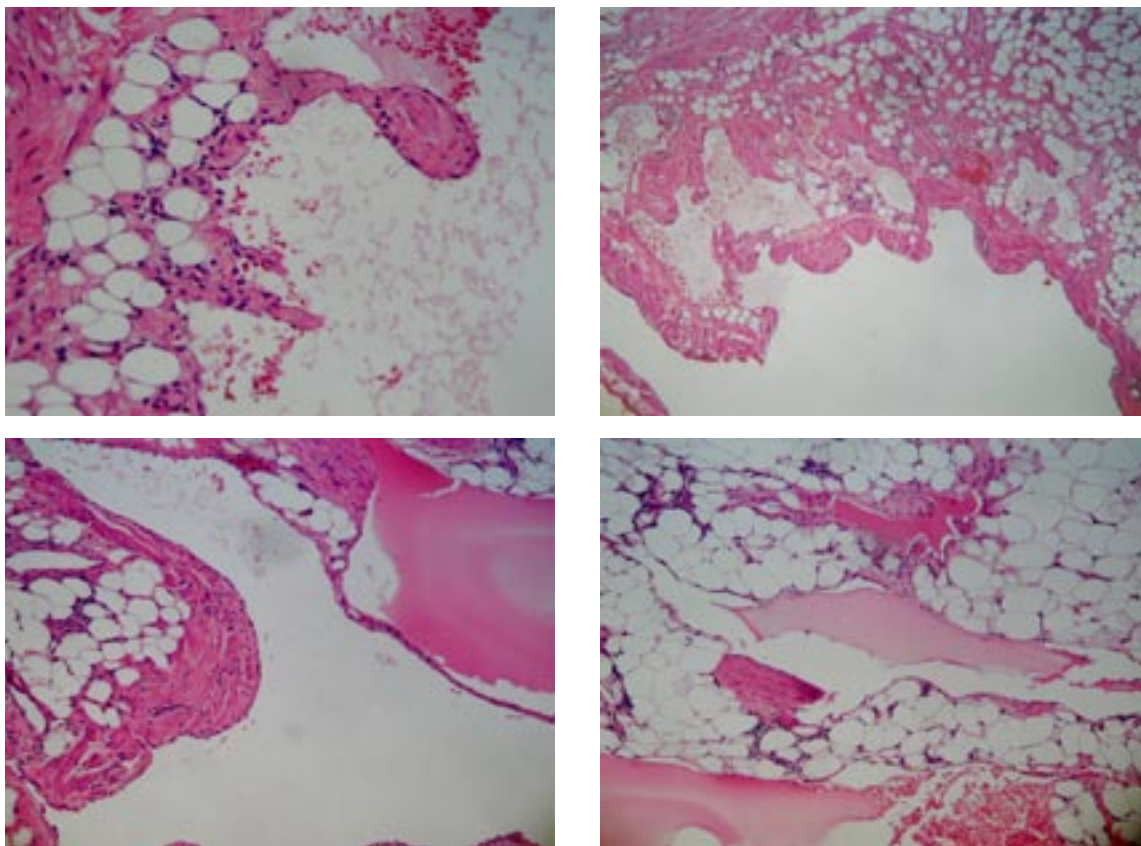
■ Figure 2: FICE demonstrated a clear area of lymphangiectasia



■ Figure 3-5: A 7.2x11.0x9.6 cm lobulated contour mass was noted attaching along the wall of the distal jejunum. This mass appeared as multiloculated cystic lesions without definite contrast enhancement. Findings were consistent with a lymphangioma.



■ Figure 6-8: Gross specimen of the jejunal mass showed lymphangiectatic change of the mucosa.



■ Figure 9-12: The microscopic examination showed dilated lymphatic channels located within the lamina propria, and extended through the muscularis mucosa into the submucosal layer. The channels contained homogenous fluid and few red cells.

Discussion:

Lymphangioma is a benign neoplastic lesion of the lymphatic system that usually present during infancy as subcutaneous lesion. Intraabdominal site accounts for less than 1% of the cases, and often occurs in the mesentery of the small bowel. Jejunum is a very rare location for this entity¹ since lymphangioma comprises as only 3% of benign small bowel tumor². They can lead to distinct symptoms including mid-gastrointestinal bleeding, abdominal pain and protein-losing enteropathy³. They are the malformation of sequestered lymphatic tissue that fails to communicate with the normal lymphatic system. Subsequent cellular proliferation and accumulation of fluid account for the cystic nature of this lesion³. Typically, endoscopic finding is an elevated

polypoid tumor, yellowish-white to tan. The surface is smooth, often with white specks, and can be impressed with a light touch by biopsy forceps⁴. Endoscopic examination may reveal satellite lesions undetected radiologically⁴. The histopathology showed thin-walled cystic masses with smooth gray, pink, tan, or yellow external surface. On a cut section, it may contain large macroscopic interconnecting cysts (often referred as cystic hygroma or cystic lymphangioma) or microscopic cysts (cavernous lymphangioma). The cyst may contain chylous, serous, hemorrhagic, or mixed fluid⁵. Surgery is the treatment of choice. The prognosis after complete excision is usually excellent⁶.

References

1. Griffa B, Basilio V, Feltri M, et al. [Submucosal jejunal lymphangioma: an unusual case with obscure gastrointestinal bleeding in an adult, detected by video-capsule endoscopy and treated by laparoscopy]. *Minerva Chir* 2006;61:529-32.
2. Hsu SJ, Chang YT, Chang MC, et al. Bleeding jejunal lymphangioma diagnosed by double-balloon enteroscopy. *Endoscopy* 2007;39 Suppl 1:E5-6.
3. Barquist ES, Apple SK, Jensen DM, et al. Jejunal lymphangioma. An unusual cause of chronic gastrointestinal bleeding. *Dig Dis Sci* 1997;42:1179-83.
4. Shigematsu A, Iida M, Hatanaka M, et al. Endoscopic diagnosis of lymphangioma of the small intestine. *Am J Gastroenterol* 1988;83:1289-93.
5. Levy AD, Cantisani V, Miettinen M. Abdominal lymphangiomas: imaging features with pathologic correlation. *AJR Am J Roentgenol* 2004;182:1485-91.
6. Wani I. Mesenteric lymphangioma in adult: a case series with a review of the literature. *Dig Dis Sci* 2009;54:2758-62.



Case 3

Suparat Khemnark, M.D.

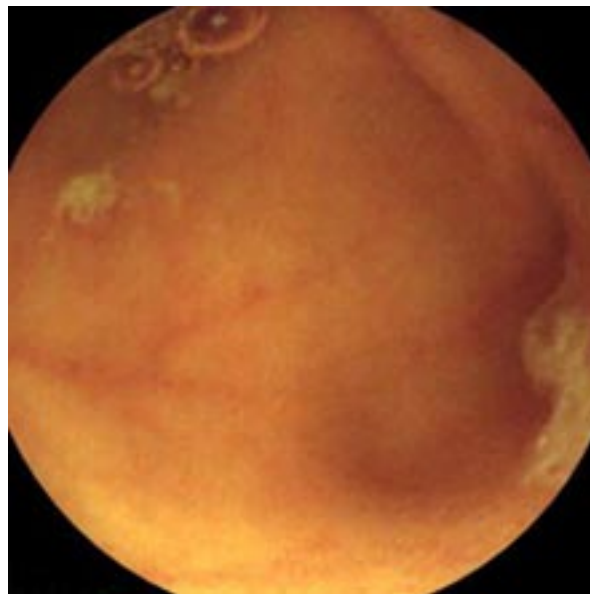
Satimai Aniwon, M.D.

Vichai Viriyautsahakul, M.D.

Rungsun Rerknimitr, M.D.

An 85-year-old man presented with hematochezia. He had been diagnosed as carcinoma of sigmoid colon and had undergone left hemicolectomy for 6 years. He presented with maroon stool and underwent EGD and colonoscopy. After non-diagnostic endoscopy, his bleeding persisted and required 4 units of transfusion. Capsule endoscopy (CE) was carried out for the evaluation small bowel. CE revealed multiple jejunal diverticuli (Figure 1), and patient reported no

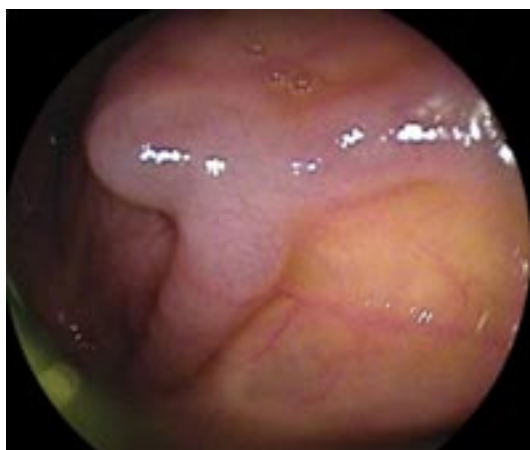
passage of the capsule. It was speculated that the capsule retained in one of diverticuli until the battery ran out. Four days later, double balloon endoscopy (DBE) was performed and revealed multiple huge small bowel diverticuli in the proximal jejunum (Figure 2-3), many of diverticuli contained dilated and congested vessel (Figure 4). Eventually, DBE discovered the retained capsule (Figure 5). Later a capsule was successfully retrieved by a basket.



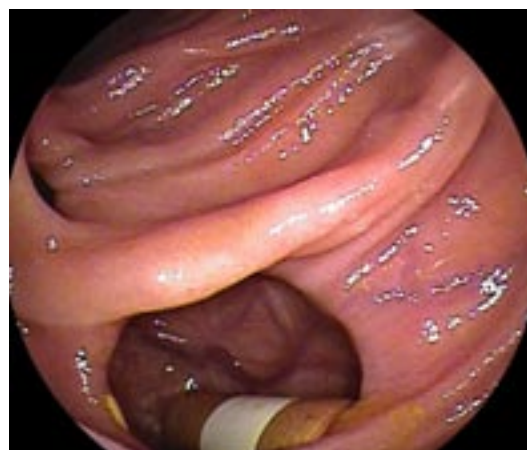
■ Figure 1: Capsule endoscopic image showed the bottom of jejunal diverticulum



■ Figures 2-3: Multiple huge jejunal diverticuli



■ Figure 4: Dilated and congested vessel found in many diverticuli



■ Figure 5: Capsule retention was found in one diverticulum

Diagnosis:

A capsule retention in jejunal diverticulum

Discussion:

Capsule retention is defined as a failure to pass the wireless capsule from the alimentary tract after 2 weeks of capsule ingestion. The contraindications to capsule endoscopy from factors or conditions that may increase the likelihood of capsule retention. These include known intestinal or colonic strictures, and/or ongoing small-bowel obstruction¹. Capsule retention

occurs at a rate of 0.75% worldwide, and has been shown to cause intestinal obstruction and perforation in a handful number of patients². The presence of diverticulum during wireless capsule endoscopy has been previously reported in the literature as part of a series of capsule-related complications. Nevertheless, their presence has not translated into an increased risk of capsule retention³. One study reported small-bowel diverticula associated with a regional transit abnormality (ie, failure of capsule passage from a focal region in the GI tract), but no associated complications were recorded.

References

1. Giday SA, Pickett-Blakely OE, Buscaglia JM, Mullin GE. Capsule retention in a patient with small-bowel diverticulosis. *Gastrointest Endosc* 2009;69:384-6.
2. Barkin JS, Friedman S. Wireless capsule endoscopy requiring surgical intervention: the world's experience. *Am J Gastroenterol* 2002;97:S298.
3. Sears DM, Avots-Avotins A, Culp K, et al. Frequency and clinical outcome of capsule retention during capsule endoscopy for GI bleeding of obscure origin. *Gastrointest Endosc* 2004;60:822-7.



Case 4

Kriangsak Charoensuk, M.D.

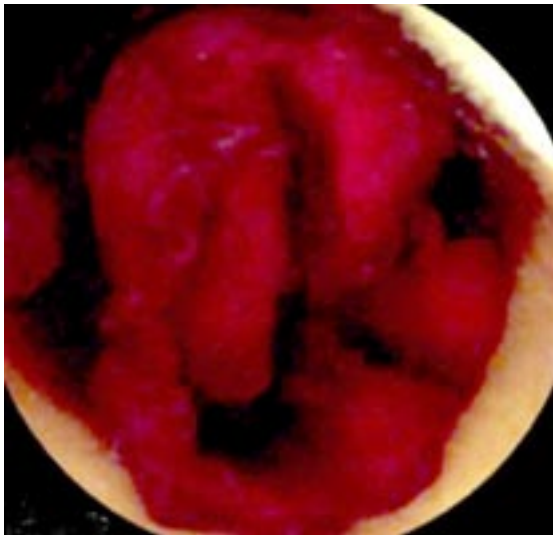
Satimai Aniwon, M.D.

Vichai Viriyautsahakul, M.D.

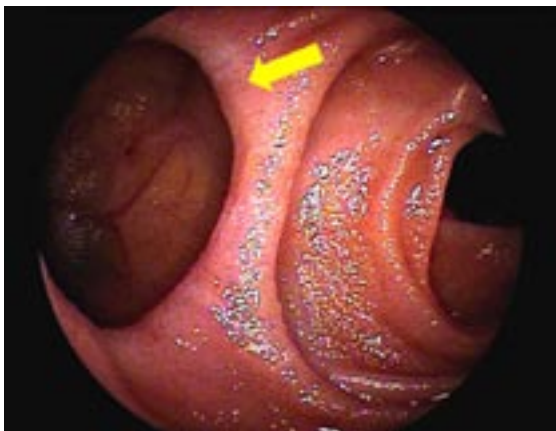
Rungsun Rerknimitr, M.D.

A 62-year-old woman presented with overt obscure gastrointestinal bleeding. Video capsule endoscopy (VCE) was carried out after non diagnostic EGD and colonoscopy. VCE showed only fresh blood and blood clot without identifiable bleeding cause (Figure 1).

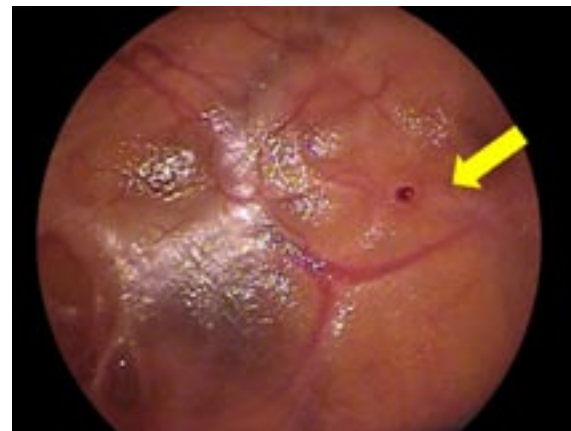
Then double balloon endoscopy (DBE) was performed via the oral route. DBE showed a large diverticulum with non-bleeding visible vessel at proximal part of jejunum (Figure 2-3).



■ Figure 1: Fresh blood without identifiable bleeding cause by VCE image



■ Figure 2: A large jejunal diverticulum



■ Figure 3: Non-bleeding visible vessel at the bottom of diverticulum

Diagnosis:

Jejunal diverticular bleeding with non-bleeding visible vessel

Discussion:

Jejunal diverticulum, first described by Sir Astley Cooper in 1807, is a rare lesion of the small intestine seen in 2-2.3% of small-bowel contrast studies and 1.3-4.6% of autopsy studies. Most jejunal diverticulum is asymptomatic, but the diverticulum could develop complication such as diverticulitis with/without bowel perforation, intestinal obstruction, and bleeding. In the past jejunal diverticular bleeding required surgical treatment. In contrast to VCE that provides only diagnosis, DBE can offer a precise diagnosis and therapy. In this case blood clot can be irrigated and direct endoscopic therapies including coaptation, clipping, injection can be performed¹⁻⁵. In this case, a hemoclip was applied and able to achieve hemostasis.

References

1. Chen TH, Chiu CT, Lin WP, et al. Application of double-balloon enteroscopy in jejunal diverticular bleeding. *World J Gastroenterol* 2010;16:5616-20.
2. Longo WE, Vernava AM. 3rd Clinical implications of jejunoileal diverticular disease. *Dis Colon Rectum* 1992;35:381-8.
3. Rodriguez HE, Ziauddin MF, Quiros ED, et al. Jejunal diverticulosis and gastrointestinal bleeding. *J Clin Gastroenterol* 2001;33:412-4.
4. Woods K, Williams E, Melvin W, et al. Acquired jejunoileal diverticulosis and its complications: a review of the literature. *Am Surg* 2008;74:849-54.
5. Yen HH, Chen YY, Yang CW, et al. The clinical significance of jejunal diverticular disease diagnosed by double-balloon enteroscopy for obscure gastrointestinal bleeding. *Dig Dis Sci* 2010;55: 3473-8.



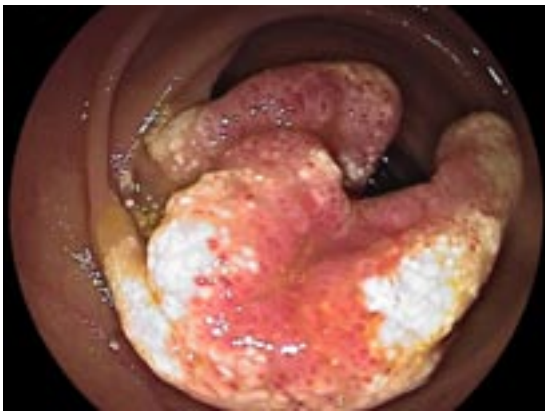
Case 5

Tanassanee Soontornmanokul, M.D.

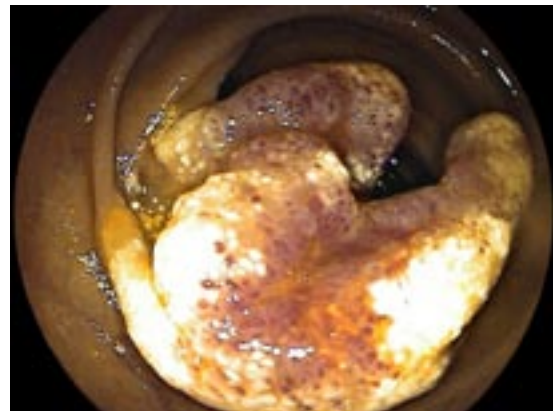
Rungsun Rerknimitr, M.D.

A previously healthy 24-year-old man presented with iron deficiency anemia. DBE was performed to the proximal jejunum. It revealed a large polypoid mass, 7x8

cm in diameter with multiple lymphangiectatic macules on top of the surface (Figure 1-4). Endoscopic resection was done and tissue was sent for histological examination.



■ Figure 1: Large polypoid mass with lymphangiectasia on top



■ Figure 2: FICE image station 6



■ Figure 3: Endoscopic resection



■ Figure 4: Post successful endoscopic resection

Diagnosis:

Primary lymphangioma of jejunum

Discussion:

Sporadic lymphangiectasias are commonly found throughout the small bowel and are considered to be non pathologic. Pathologic lymphangiectasias of the small bowel include primary lymphangiectasia, secondary lymphangiectasia and lymphaticovenous malformations¹. They can lead to distinct symptoms including mid-gastrointestinal bleeding, abdominal pain and protein-losing enteropathy². Lymphangiomas are unusual benign tumors of the small bowel comprising only 3%³. They are malformations of sequestered lymphatic tissue that fail to communicate with the normal lymphatic system. Subsequent cellular proliferation and accumulation of fluid account for the cystic nature of these lesions². Typically, endoscopic finding is an elevated polypoid tumor, yellowish-white to tan. The surface is smooth, often with white specks, and could be impressed by touching lightly with biopsy forceps⁴. Endoscopic examination revealed satellite lesions not detected radiologically⁴. The histopathology showed thin-walled cystic masses with a smooth gray, pink, tan, or yellow external surface. On cut section, they vary in appearance and may contain large

macroscopic interconnecting cysts (often referred to as a cystic hygroma or cystic lymphangioma) or microscopic cysts (cavernous lymphangioma). The cysts may contain chylous, serous, hemorrhagic, or mixed fluid⁵. Traditionally, surgery is the treatment of choice. However, endoscopic resection can be performed in certain patients including this one.

References

1. Safatle-Ribeiro AV, Iriya K, Couto DS, et al. Secondary lymphangiectasia of the small bowel: utility of double balloon enteroscopy for diagnosis and management. *Dig Dis* 2008;26:383-6.
2. Barquist ES, Apple SK, Jensen DM, et al. Jejunal lymphangioma. An unusual cause of chronic gastrointestinal bleeding. *Dig Dis Sci* 1997;42:1179-83.
3. Hsu SJ, Chang YT, Chang MC, et al. Bleeding jejunal lymphangioma diagnosed by double-balloon enteroscopy. *Endoscopy* 2007;39 Suppl 1:E5-6.
4. Shigematsu A, Iida M, Hatanaka M, et al. Endoscopic diagnosis of lymphangioma of the small intestine. *Am J Gastroenterol* 1988;83:1289-93.
5. Levy AD, Cantisani V, Miettinen M. Abdominal lymphangiomas: imaging features with pathologic correlation. *AJR Am J Roentgenol* 2004;182:1485-91.



Case 6

Kriangsak Charoensuk, M.D.

Satimai Aniwon, M.D.

Vichai Viriyautsahakul, M.D.

Rungsun Rerknimitr, M.D.

A 68-year-old woman presented with chronic watery diarrhea for a month. She had history of chronic NSAIDs use. EGD and colonoscopy showed normal endoscopic finding. DBE revealed multiple discrete round clean base ulcers, varying in size along jejunal

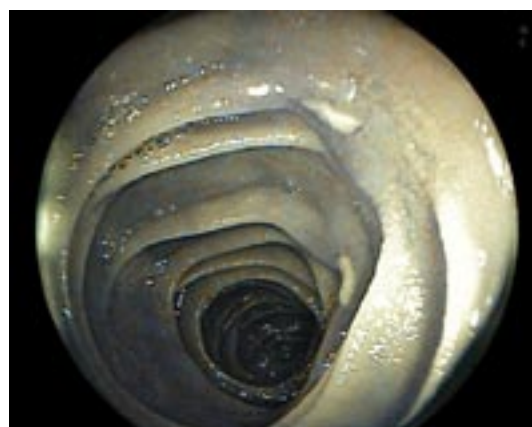
area with intervening normal small bowel mucosa (Figure 1-4). Biopsy was taken from those lesions. Histology was compatible with organizing ulcer. There was no evidence of vasculitis or granuloma.



■ Figures 1-2: Multiple clean base ulcers, varying in size in jejunum



■ Figure 3: Well-defined round clean base ulcer



■ Figure 4: FICE image station 4 demonstrated non-inflamed intervening mucosa

Diagnosis:

NSAID induced small bowel ulcers

Discussion:

Non-steroidal anti-inflammatory drugs (NSAIDs) are some of the most commonly used medications worldwide. The most common side effects are ulcers in the digestive tract including stomach, small bowel, and colon. NSAIDs-induced small bowel injury is a topic that deserves a special attention since the advent of capsule endoscopy and balloon assisted endoscopy. Two third of the patients with NSAID-induced enteropathy are asymptomatic. The clinical presentations are iron deficiency anemia, gastrointestinal bleeding, hypoalbuminemia, vitamin B12 or bile acid malabsorption, diarrhea, and acute abdominal pain. Serious complications especially massive bleeding, stricture and perforation may also develop. Capsule endoscopy is the preferred non-invasive method to visualize lesions. With the more invasive in nature, double-balloon endoscopy can provide therapeutic advantages similar to therapeutic endoscopy in upper GI bleeding¹⁻⁵.

References

1. Higuchi K, Umegaki E, Watanabe T, et al. Present status and strategy of NSAIDs-induced small bowel injury. *J Gastroenterol* 2009;44:879-88.
2. Matsumoto T, Kudo T, Esaki M, et al. Prevalence of non-steroidal anti-inflammatory drug-induced enteropathy determined by double-balloon endoscopy: a Japanese multicenter study. *Scand J Gastroenterol* 2008;43:490-6.
3. Park SC, Chun HJ, Kang CD, et al. Prevention and management of non-steroidal anti-inflammatory drugs-induced small intestinal injury. *World J Gastroenterol* 2011 14;17:4647-53.
4. Tacheci I, Kopacova M, Rejchrt S, et al. Non-steroidal anti-inflammatory drug induced injury to the small intestine. *Acta Medica (Hradec Kralove)* 2010;53:3-11.
5. Yen HH, Chen YY, Soon MS, et al. The role of double-balloon endoscopy in NSAID enteropathy. *Gastrointest Endosc* 2006;64:1035-6.



Case 7

Kriangsak Charoensuk, M.D.

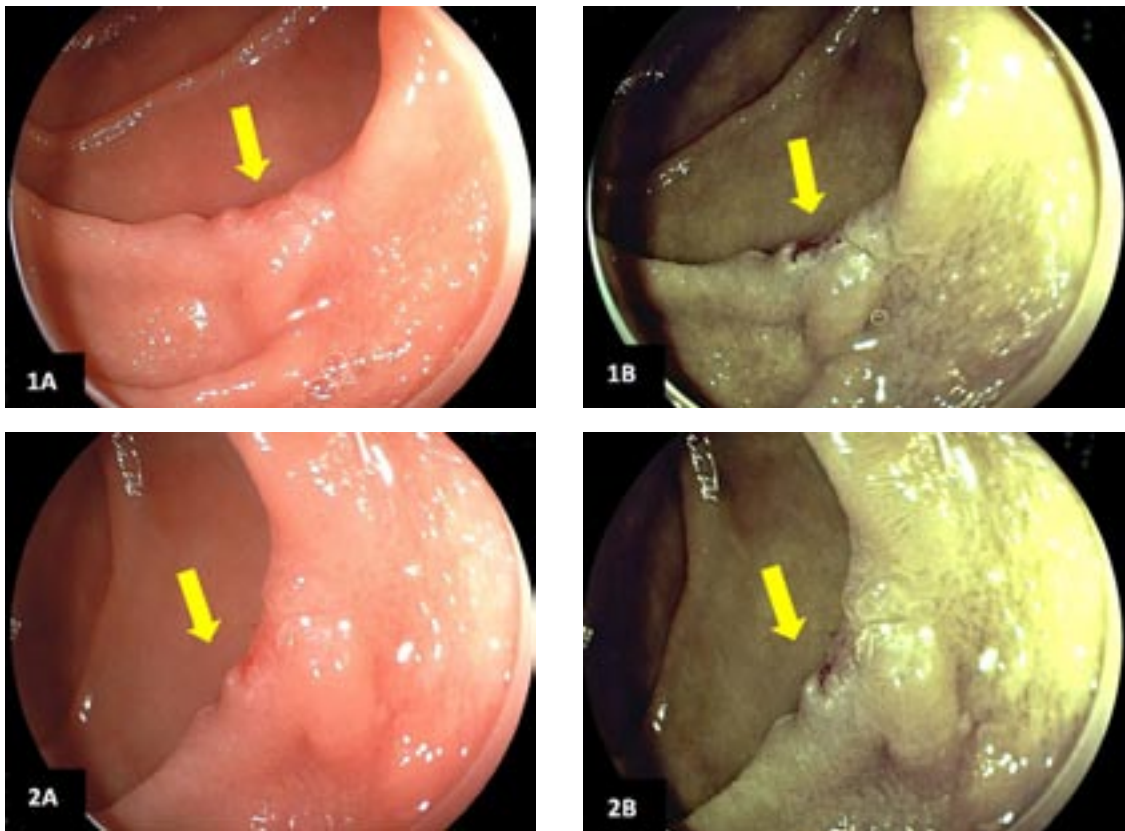
Phonthep Angsuwatcharakon, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

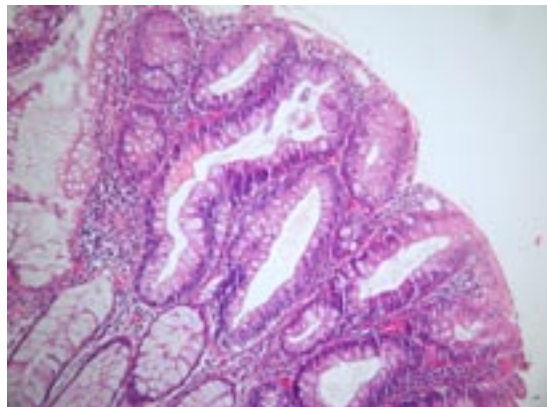
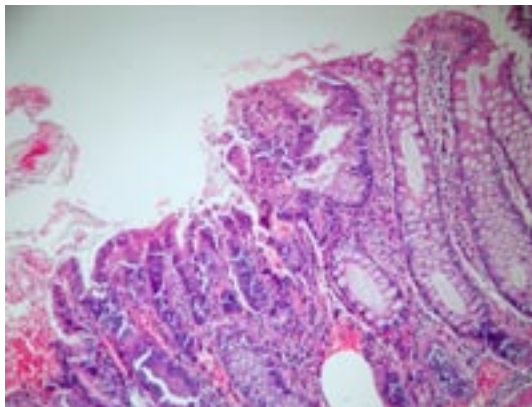
Rungsun Rerknimitr, M.D.

A 68-year-old man presented with epigastric pain for 2 weeks. EGD showed a depressed lesion (0-IIc) in the second part of duodenum (Figure 1A and 2A). Flexible Intelligent Color Enhancement (FICE)

demonstrated increased vascular pattern (Figure 1B and 2B). Biopsy from the lesion showed tubular structure of crypts and glands which lined by low grade dysplastic epithelium (Figure 3-4).



■ Figure 1-2: Depressed lesion in the second part of duodenum (0-IIc) (1A and 2A: white light, 1B, and 2B: FICE station 3)



■ Figure 3-4: Tubular structure of crypts and glands which lined by low grade dysplastic epithelium

Diagnosis:

Low grade tubular adenoma of duodenum

Discussion:

Duodenal polyps were reported in 0.3–4.6% of patients attending for upper gastrointestinal endoscopy and their main histology is adenoma. They often have been discovered incidentally and usually asymptomatic¹⁻⁴. Duodenal adenomas have malignant potential in a similar fashion to colonic adenomas. The risk of carcinoma is greater in ampullary adenomas compared with non-ampullary adenoma, and increases with the size of adenoma. Non-ampullary adenoma that arise in patients without a known polyposis syndrome has a lower risk for malignant transformation than that of patients in familial polyposis^{2,4}. The gross morphology was usually sessile or flat type rather than pedunculated type and depressed lesion^{2,4}. Because the morphological features plus their tendency to grow along folds, duodenal adenomas can be more difficult to detect than colonic adenomas and may be missed by the untrained eye. In this case, FICE may be of help by detecting abnormal vasculature of the depressed lesion. Management of these polyps depends on symptoms, histopathology and endoscopic features. Endoscopic resection is a well established technique for the

treatment many precancerous lesions such as early cancer in esophagus, stomach, and colon. The evidence base for endoscopic resection of duodenal adenomas is limited, but it provides a promising result. Endoscopic mucosal resection (EMR) and endoscopic submucosal dissection (ESD) have been reported as comparable with the chance for complete removal ranges from 79–100%¹⁻⁴.

References

1. Basford PJ, Bhandari P. Endoscopic management of nonampullary duodenal polyps. *Therap Adv Gastroenterol* 2012;5:127-38.
2. Culver EL, McIntyre AS. Sporadic duodenal polyps: classification, investigation, and management. *Endoscopy* 2011;43:144-55.
3. Jepsen JM, Persson M, Jakobsen NO, et al. Prospective study of prevalence and endoscopic and histopathologic characteristics of duodenal polyps in patients submitted to upper endoscopy. *Scand J Gastroenterol* 1994;29:483-7.
4. Kim HK, Chung WC, Lee BI, et al. Efficacy and long-term outcome of endoscopic treatment of sporadic nonampullary duodenal adenoma. *Gut Liver* 2010;4:373-7.

Case 8

Kanita Chattrasophon, M.D.

Rungsun Rerknimitr, M.D.

An 83-year-old man presented with epigastric pain and melena. He has been taking aspirin for 8 years due to chronic atrial fibrillation. EGD revealed a deformity pylorus, healing duodenal ulcer at duodenal bulb and duodenal stenosis at D1-D2 junction (Figure 1).

Therefore, Nasogastroscopy with duodenal dilation was performed successfully by a wire-guided balloon dilator. After dilation, a chronic duodenal ulcer was found at stricture site (Figure 2).



■ Figure 1: Duodenal stricture



■ Figure 2: Post dilation of duodenal stricture

Diagnosis:

Duodenal stricture

Discussion:

Aspirin and NSAIDs are associated with peptic ulcer with or without stricture, diaphragmatic disease of intestine¹, and protein losing enteropathy². Location of stricture starts from antro-pyloric area to postbulbar

duodenal segments. The common site is duodenum pylorus and both duodenum and pylorus. The stricture pattern is usually either a short segment (2-3 mm in length) or web-like circumferential narrowing. Ulceration at the rim of stricture can be found in 30% of the cases³, and this gastric outlet obstruction can be effectively treated by balloon stricturoplasty with good safety^{3,4}.

References

1. Puri AS, Monga R, Garg S, et al. Diaphragm disease of duodenum following longterm NSAIDs use: endoscopic management. *Indian J Gastroenterol* 2004;23:189-90.
2. Higuchi K, Umegaki E, Watanabe T, et al. "Present status and strategy of NSAIDs-induced small bowel injury". *J Gastroenterol* 2009;44:879-88.
3. Noor MT, Dixit P, Kochhar R, et al. NSAIDs-related pyloroduodenal obstruction and its endoscopic management. *Diagn Ther Endosc* 2011;967957.
4. Kochhar R and Kochhar S. "Endoscopic balloon dilation for benign gastric outlet obstruction in adults". *World J Gastrointest Endosc* 2010;2:29-35.



Case 9

Kriangsak Charoensuk, M.D.

Satimai Aniwan, M.D.

Vichai Viriyautsahakul, M.D.

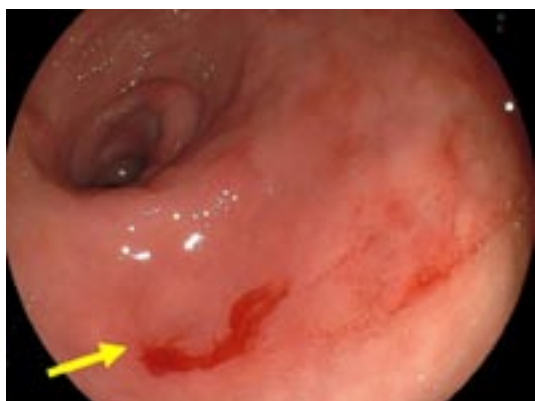
Rungsun Rerknimitr, M.D.

A 68-year-old man presented with maroon stool for 2 days. He had an underlying of chronic renal failure. Physical examination showed pale conjunctiva. He underwent EGD and colonoscopy. The results were unremarkable (Figure 1). Video capsule endoscopy (VCE)

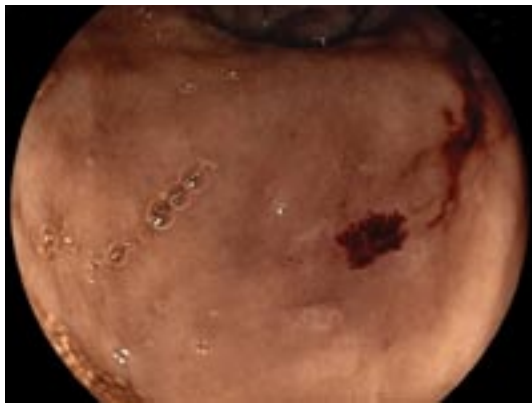
was then carried out but failed to identify the cause of bleeding. His bleeding persisted. DBE was performed later. DBE revealed bleeding angiodysplasias at duodenum (Figure 2-6). The hemostasis was achieved by argon plasma coagulation.



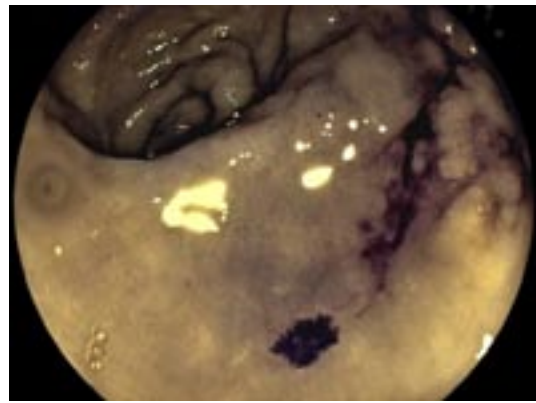
■ Figure 1: Capsule endoscopy image from duodenal region was normal



■ Figure 2-3: bleeding angiodysplasia (white light images)



■ Figure 4: FICE station 2 Figure



■ Figure 5: FICE station 4



■ Figure 6: FICE station 6

Diagnosis:

Bleeding angiodysplasia of duodenum

Discussion:

Angiodysplasias are the most frequent vascular lesions of the gastrointestinal tract and they lead to a significant mortality. One third of obscure gastrointestinal bleeding causes have been related to small bowel angioectasia¹. The cause of angiodysplasia is unknown and the natural history is poorly understood²⁻⁴. Many lesions were thought to arise from a degenerative process associated with aging, local vascular anomalies, and tissue hypoxia. Identifying the culprit lesion can be

difficult because of their multiple locations in appearance and the small size of active bleeding site. Small bowel VCE has a significant advantage for its non-invasive in nature. It provided a much higher sensitivity when compared to angiography³. However, therapeutic advantage is better achieved with DBE⁴.

References

1. Foutch PG. Angiodysplasia of the gastrointestinal tract. *Am J Gastroenterol* 1993;88:807-18.
2. Chen LH, Cao HJ, Chen WG, et al. [Double balloon endoscopy in diagnosis of patients with obscure gastrointestinal bleeding]. *Zhejiang Da Xue Xue Bao Yi Xue Ban* 2012;41:99-104.
3. Eickhoff A, Enderle MD, Hartmann D, et al. Effectiveness and Safety of PRECISE APC for the treatment of bleeding gastrointestinal angiodysplasia--a retrospective evaluation. *Z Gastroenterol* 2011;49:195-200.
4. Samaha E, Rahmi G, Landi B, et al. Long-term outcome of patients treated with double balloon enteroscopy for small bowel vascular lesions. *Am J Gastroenterol* 2012;107:240-6.

Case 10

Kriangsak Charoensuk, M.D.

Linda Pantongrag-Brown, M.D.

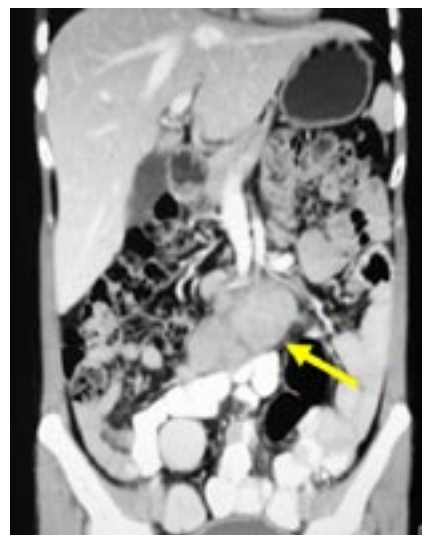
Naruemon Wisedopas-Klaikeaw, M.D.

Rungsun Rerknimitr, M.D.

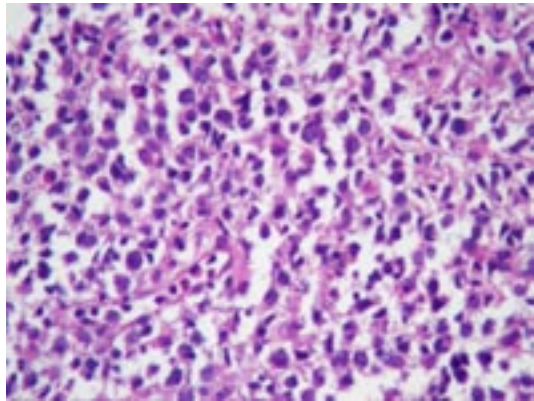
A 37-year-old woman presented with diarrhea, abdominal pain and significant weight loss. She had been diagnosed with IgA nephropathy for 5 years prior to this admission. Physical examination showed hepatosplenomegaly and generalized edema. Laboratory results revealed severe hypoalbuminemia without evidence of significant proteinuria. Computed tomography of the abdomen showed a long segment of thickened

wall of the small bowel with multiple aortocaval, para-aortic and mesenteric lymphadenopathy (Figure 1-2). EGD found a flattened and scalloping mucosa along duodenal region (Figure 3-4). Duodenal and lymph node biopsy revealed diffuse infiltration with medium to large atypical lymphoid cells, of which, they were positive for CD20 and negative for CD3, and Cyclin D1. (Figure 5-6).

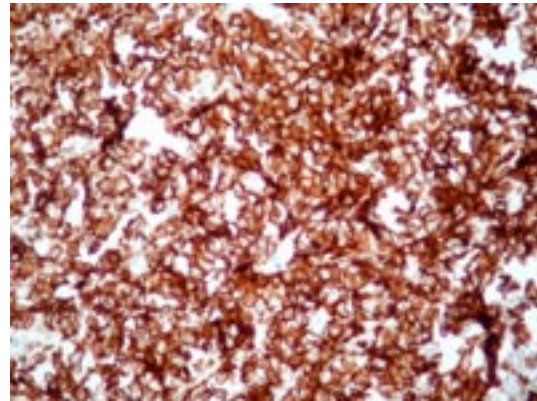
- Figure 1-2: CT scan demonstrated two enlarged intra-abdominal nodes (yellow arrow)



- Figure 3-4: Flattening mucosa (scallop-like) at duodenum; (A) white light image (B) FICE station 4



■ Figure 5: Diffuse medium-sized round cells



■ Figure 6: IH stain was positive for CD20

Diagnosis:

Diffuse large B-cell lymphoma of small intestine

Discussion:

Lymphoma is one of the most common gastrointestinal malignancies and frequently involves small bowel. It more commonly represents generalized lymphoma with associated GI involvement than primary lymphoma. The small bowel and the ileocecal region are involved in 8.6% and 7% of cases, respectively¹⁻². Most cases are non-Hodgkin B cell in origin. The symptoms are nonspecific, such as abdominal pain, abdominal mass, bleeding, obstruction, weight loss, chronic diarrhea, and malabsorption syndrome³⁻⁴. The prevalence of malabsorption and intestinal recurrence are high in enteropathy-associated T cell lymphoma³⁻⁴. The diagnostic evaluation of a suspected lymphoma of the small intestine includes computed tomography (CT), contrast radiography, conventional endoscopy, and capsule endoscopy. CT and/or contrast radiography are usually the initial diagnostic modalities¹⁻³. The distal ileum is classically the most common site of small bowel B-cell lymphoma. Small bowel B-cell lymphoma

manifestations include circumferential bulky mass, and a long segment of bowel wall thickening, associated with regional lymphadenopathy¹⁻⁴.

References

1. Ghai S, Pattison J, O'Malley ME, et al. Primary gastrointestinal lymphoma: spectrum of imaging findings with pathologic correlation. *Radiographics* 2007;27:1371-88.
2. Koch P, del Valle F, Berdel WE, et al. Primary gastrointestinal non-Hodgkin's lymphoma: I. Anatomic and histologic distribution, clinical features, and survival data of 371 patients registered in the German Multicenter Study GIT NHL 01/92. *J Clin Oncol* 2001;19:3861-73.
3. Wan W, Wang J, Jing HM, et al. [Analysis of clinical characteristics and prognostic factors of 110 cases with primary gastrointestinal tract non-Hodgkin's lymphoma]. *Zhonghua Xue Ye Xue Za Zhi* 2011; 32:652-5.
4. Ehrlich AN, Stalder G, Geller W, et al. Gastrointestinal manifestations of malignant lymphoma. *Gastroenterology* 1968;54:1115-21.

Case 11

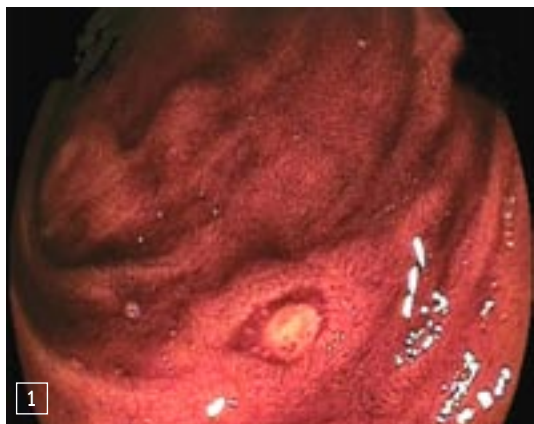
Tanassanee Soontornmanokul, M.D.

Satimai Aniwan, M.D.

Rungsun Rerknimitr, M.D.

A 50-year-old woman presented with hematochezia for 2 days. She had been well until 3 weeks prior to admission; she had a low grade fever with lower quadrant abdominal pain. Later, she developed hematochezia. She had no history of NSAIDs used. The urgent ileo-colonoscopy was performed and revealed the multiple oval, discrete deep ulcers without active bleeding at the terminal ileum. On the next day, she underwent double balloon enteroscopy (DBE) due to

the ongoing bleeding per rectum. DBE showed multiple deep ulcers with blood oozing at the distal jejunum and upper ileum (Figure 1-2). The ileal biopsy demonstrated active ileitis with fibrinous exudates. Neither malignancy nor organism was identified. Modified AFB stain and PCR for tuberculosis were negative. Crohn's enterocolitis with severe bleeding was diagnosed.



■ Figure 1-2: Multiple deep ulcers with oozing at jejunum and terminal ileum

The patient was prescribed with intravenous antibiotic and dexamethasone for 3 days, however the bleeding continued, she required 3 units of blood transfusion. Finally, a 5 mg per kg of infliximab was infused intravenously, and the bleeding stopped on the next day. Induction with the combination of

corticosteroid and infliximab was given at week 0 and 2, and then followed by the maintenance therapy with azathioprine. Two months later, the follow up ileo-colonoscopy revealed normal endoscopic appearance of the terminal ileum (Figure 3-4).



■ Figure 3-4: Two months follow-up ileo-colonoscopy showed normal mucosa in terminal ileum

Diagnosis:

Severe small bowel bleeding in Crohn's disease

Discussion:

Acute lower gastrointestinal bleeding is a rare complication of Crohn's disease (CD)¹. Endoscopic treatment, surgery and arterial embolization have been used to control the massive gastrointestinal hemorrhage, however, the management for severe gastrointestinal bleeding remains a great challenge. Because severe mucosal lesions such as colonic ulcers are the major cause of bleeding, achieving mucosal healing is the therapeutic goal. There are several case reports of using infliximab (anti-TNF agents) to control lower gastrointestinal bleeding in Crohn's disease^{1, 2}. According to IBD database of KCMH and Samitivej hospitals during 2005 and 2010, seven patients with severe GI bleeding in Crohn's disease were prescribed with infliximab 5 mg per

kg to control severe bleeding. Six of them stopped bleeding in one day after infliximab was infused. In our opinion, infliximab therapy may be an effective agent for medical hemostasis in a CD patient with failure to localize the bleeding site before considering surgery³.

References

1. Tsujikawa T, Nezu R, Andoh A, et al. Infliximab as a possible treatment for the hemorrhagic type of Crohn's disease. *J Gastroenterol* 2004;39:284-7.
2. Meyer MM, Levine EJ. Acute hemorrhagic Crohn's disease controlled with infliximab. *Inflamm Bowel Dis* 2009;15:1456-7.
3. Aniwat S, Eakpongpaitsit S, Imraporn B, et al. Infliximab stopped severe gastrointestinal bleeding in Crohn's disease. *World J Gastroenterol* 2012;18:2730-4.



Case 12

Kittiyod Poovorawan, M.D.

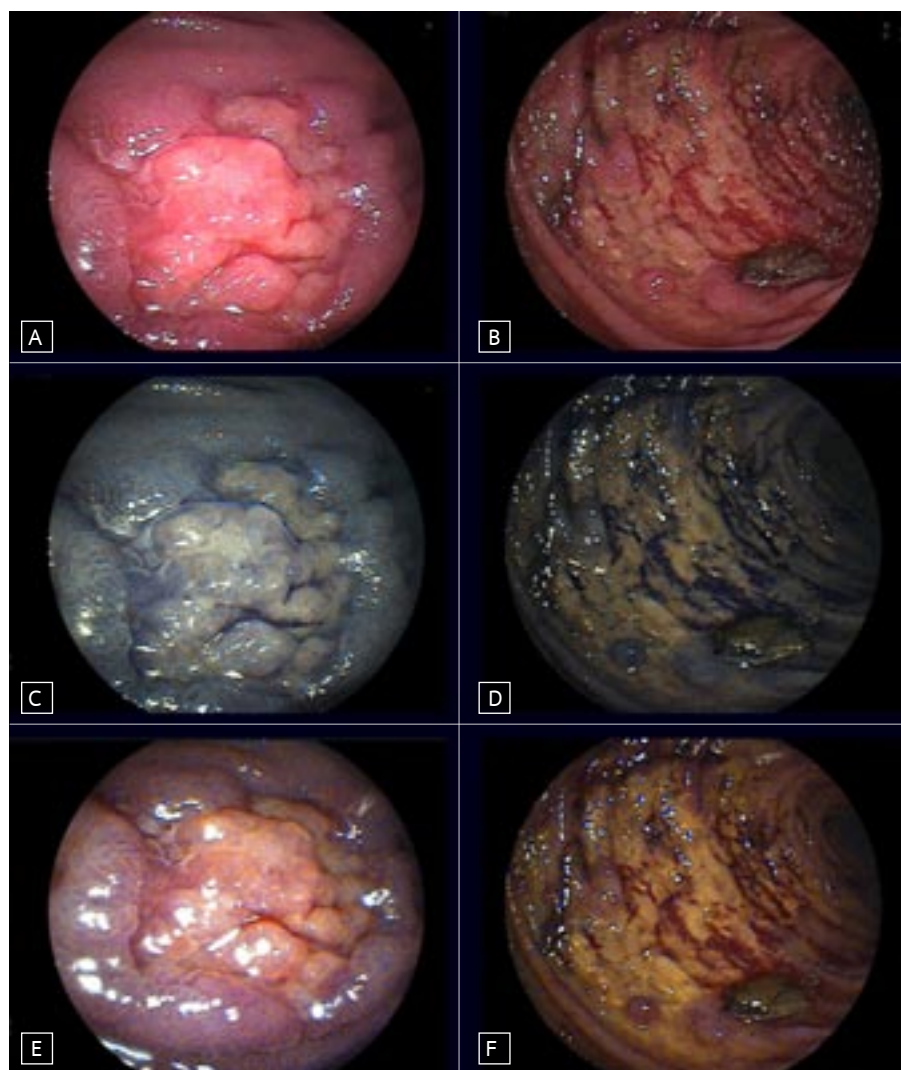
Satimai Aniwan, M.D.

Vichai Viriyautsahakul, M.D.

Rungsun Rerknimitr, M.D.

A 47-year-old man with advanced hilar cholangiocarcinoma presented with melena and anemia for 2 days. Both EGD and colonoscopy failed identify the bleeding cause. Video capsule endoscopy (VCE) was carried out but the capsule failed to pass the pylorus because the enlarged left lobe of the liver compressed

the gastric outlet. DBE was performed and revealed multiple infiltrative ulcers with necrotic tissues in the jejunum. Some ulcers had active blood oozing (Figure 1 A-F). Biopsy from the ulcers confirmed the presence of jejunal metastasis of cholangiocarcinoma.



■ Figure 1: Infiltrative ulcers at jejunum with blood oozing demonstrated by white light (A,B), FICE station 4 (C,D), FICE station 6 (E,F).

Diagnosis:

Cholangiocarcinoma with jejunal metastasis

Discussion:

Mid-gastrointestinal bleeding is the bleeding that originated from the area between the papilla and ileocecal valve¹. Small intestinal bleeding can develop from various causes such as vascular abnormality, ulcers, diverticulum, and neoplasm². Previous reports of jejunal metastasis were mostly from lung cancer and malignant melanoma³. To date, there has been only one case report of metastatic cholangiocarcinoma to jejunum⁴.

References

1. Ell C, May A. Mid-gastrointestinal bleeding: capsule endoscopy and push-and-pull enteroscopy give rise to a new medical term. *Endoscopy* 2006;38:73-5.
2. Xin L, Liao Z, Jiang YP, Li ZS. Indications, detectability, positive findings, total enteroscopy, and complications of diagnostic double-balloon endoscopy: a systematic review of data over the first decade of use. *Gastrointest Endosc* 2011;74:563-70.
3. Garwood RA, Sawyer MD, Ledesma EJ, et al. A case and review of bowel perforation secondary to metastatic lung cancer. *Am Surg* 2005;71:110-6.
4. Hayashi H, Tani T, Tajima H, et al. Small intestinal metastasis from intrahepatic cholangiocarcinoma: report of a case. *Surg Today* 2011;41:859-64.



Case 13

Kittiyod Poovorawan, M.D.

Satimai Aniwan, M.D.

Vichai Viriyautsahakul, M.D.

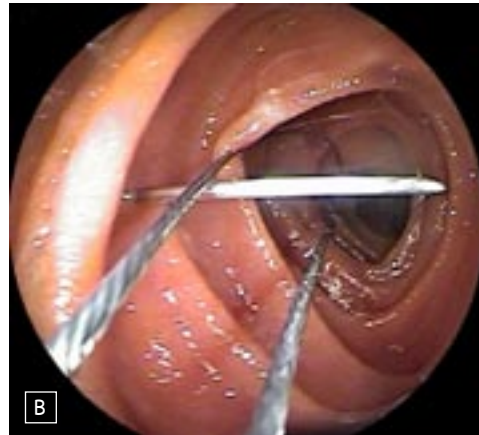
Rungsun Rerknimitr, M.D.

An 87-year-old woman presented with progressive weight loss and early satiety. CT scan of the abdomen demonstrated marked dilatation of stomach, first, and second part of duodenum with abrupt narrowing of the third part of duodenum. Sagittal view reconstruction image revealed that the angle between superior

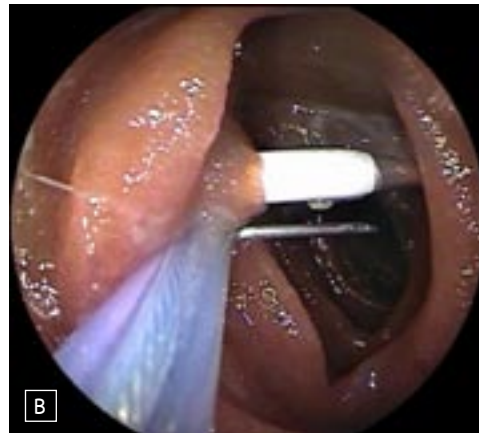
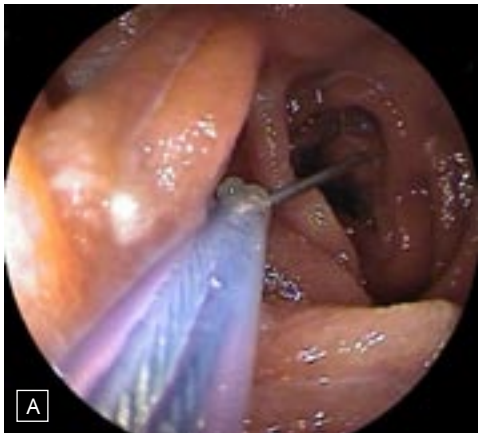
mesenteric artery (SMA) and aorta was only 11 degrees which compatible with SMA syndrome (Figure 1). The feeding was bypassed with direct endoscopic percutaneous endoscopic jejunostomy (PEJ) via double balloon endoscopy (Figure 2-4). Her weight and symptom gradually improved after jejunal feeding.



- Figure 1: CT scan findings : A. Narrowing of duodenal portion between aorta and superior mesenteric artery (thick arrow) with mark dilatation of proximal duodenum and stomach (thin arrow), B. (SMA) and aorta angle was 11 degrees and compatible with SMA syndrome.



■ Figure 2: Direct endoscopic percutaneous jejunostomy: A) Needle punctured through proximal jejunum after illumination guidance, B) A snare was applied over the needle maintain the needle position.



■ Figure 3: A) A snare was applied at the needle and fixed. B) A stylet was punctured parallel to the fixed needle.



■ Figure 4: Endoscopic findings: A) The snare was switch to capture the catheter sheath and thread, B) A thread was pulled out and jejunostomy tube was placed by the pull technique.

Diagnosis:

PEJ placement for superior mesenteric artery (SMA) syndrome.

Discussion:

Superior mesenteric artery syndrome is characterized by compression of the third portion of duodenum due to narrowing space between the superior mesenteric artery and aorta from the decrease of intervening mesenteric fat pad. An aorto-mesenteric artery angle less than 25° is the standard degree for the diagnosis of SMA syndrome¹. The main component of therapy is nutritional support. Enteral feeding distal to the obstruction such as jejunal tube or jejunostomy is

preferred. This case demonstrated the technique for jejunal feeding tube placement by percutaneous endoscopic jejunostomy (PEJ). Leakage, plugging, tube fracture, migration and knotting are possible complications².

References

1. Unal B, Aktaş A, Kemal G, et al. Superior mesenteric artery syndrome: CT and ultrasonography findings. *Diagn Interv Radiol* 2005;11:90.
2. Frenz MB, van Heel D, Siuda G, et al. Unusual malfunction of percutaneous endoscopic jejunostomy feeding tubes in patients with intestinal dysmotility. *Endoscopy* 2004;36:234-5.



Case 14

Tanassanee Soontornmanokul, M.D.

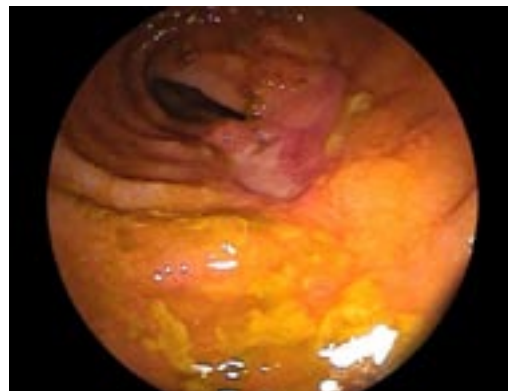
Satimai Aniwon, M.D.

Vichai Viriyautsahakul, M.D.

Rungsun Rerknimitr, M.D.

A healthy 55-year-old man presented with chronic watery diarrhea with significant weight loss for a year. He had no history of NSAIDs use. The first colonoscopy revealed multiple small clean base ulcers near ileocecal valve and terminal ileum. There was no granuloma seen from the biopsy. He was treated as tuberculosis of the GI tract for 2 months. Subsequently, his hemoglobin dropped from 13 g/dl to 9 g/dl in a

month without any visible gastrointestinal bleeding. The follow-up colonoscopy revealed worsening changes of the ileal ulcers. The histological examination showed acute organizing ulcer with negative PCR for tuberculosis. Therefore, double balloon enteroscopy was performed and revealed multiple large deep ulcers with whitish exudate and inflamed surrounding mucosa along jejunum and ileum (Figure 1-6).



■ Figure 1-2: Multiple discrete large deep ulcers with whitish exudates in the jejunum



■ Figure 3-4: Multiple discrete large deep ulcers with whitish exudates in the jejunum



■ Figure 5: FICE image station 5



■ Figure 6: FICE image station 6

Diagnosis:

Small bowel Crohn's disease

Discussion:

It is estimated that 10-30% of patients with Crohn's disease (CD) have small bowel SB involvement¹. At present, DBE is one of the emerging diagnostic tools for diagnosis and management of small bowel Crohn's disease, especially when the conventional studies (ileocolonoscopy and radiographic imaging) have been inconclusive. Mensink et al. found that in spite of the negative result of upper GI endoscopy in patients with Crohn's disease, DBE showed the active lesion in the small bowel, and finally altered the clinical management of their patients². Comparing to the video capsule endoscopy (VCE) that may get stuck in a patient with small bowel stricture, DBE has no risk for this situation and it may even use a rescue when VCE get stuck³. The overall diagnostic yield of DBE in CD is about 50-70%, according to the indications for the procedure, and the yield is highest when the indication is to detect stricture^{2,4,5}.

References

1. Steinhartd HJ, Loeschke K, Kasper H, et al. European Cooperative Crohn's Disease Study (ECCDS): clinical features and natural history. *Digestion* 1985;31:97-108.
2. Mensink PB, Groenen MJ, van Buuren HR, et al. Double-balloon enteroscopy in Crohn's disease patients suspected of small bowel activity: findings and clinical impact. *J Gastroenterol* 2009;44:271-6.
3. Mehdizadeh S, Han NJ, Cheng DW, et al. Success rate of retrograde double-balloon enteroscopy. *Gastrointest Endosc* 2007;65:633-9.
4. Manes G, Imbesi V, Ardizzone S, et al. Use of double-balloon enteroscopy in the management of patients with Crohn's disease: feasibility and diagnostic yield in a high-volume centre for inflammatory bowel disease. *Surg Endosc* 2009;23:2790-5.
5. Cazzato IA, Cammarota G, Nista EC, et al. Diagnostic and therapeutic impact of double-balloon enteroscopy (DBE) in a series of 100 patients with suspected small bowel diseases. *Dig Liver Dis* 2007;39:483-7.

Case 15

Suparat Khemnark, M.D.

Satimai Aniwon, M.D.

Vichai Viriyautsahakul, M.D.

Rungsun Rerknimitr, M.D.

A 73-year-old woman presented with maroon stool. She had been diagnosed as overlap syndrome and she was treated with an oral corticosteroid. One month earlier, she was admitted at the intensive care unit due to severe pneumonia with respiratory failure. During hospitalization, she developed maroon stool and required 4 units of blood transfusion. EGD was normal and colonoscopy showed only fresh blood from the terminal ileum. Then, DBE was carried out. A deep oval

ulcer with a large non-bleeding visible vessel at proximal jejunum was found (Figure 1-2). Due to renal failure and high risk for vascular insufficiency from her underlying disease, antegrade embolization with Histoacryl injection was performed. Technically, the 0.5 ml of the Histoacryl glue is mixed with 0.8 ml Lipiodol contrast medium, and injected in a bolus directly into the vessel (Figure 3-4). There was no recurrent bleeding and no distant emboli reported.



■ Figure 1-2: A deep oval ulcer with a large non-bleeding visible vessel



■ Figure 3: Histoacryl was injected directly into the visible vessel



■ Figure 4: After Histoacryl injection, mucosa was discolored

Diagnosis:

Small bowel ulcer with non-bleeding visible vessel

Discussion:

Obscure gastrointestinal bleeding (OGIB) is defined as persistent or recurrent bleeding from the gastrointestinal (GI) tract after negative evaluations with upper and lower endoscopies. One important characteristic of OGIB is that it is almost always recurrent. It accounts for approximately 5% of all gastrointestinal bleeding¹. OGIB can be divided broadly into obscure overt and obscure occult bleeding. Overt OGIB is characterized by the presence of clinically perceptible bleeding, that is, melena or hematochezia. The majority of OGIB cases are caused by lesions in the small bowel. The most common causes of small bowel bleeding are vascular ectasia, tumors, ulcerative diseases, and Meckel's diverticula². Up to 12% of patients with OGIB are found to have small bowel ulcerations. Therapeutic endoscopic techniques in small bowel bleeding are similar to therapeutic techniques in upper GI bleeding i.e.

bipolar coaptation, hemoclippping, and argon plasma coagulation³. Due to the difficulty of hemoclip deployment in the DBE scope for this patient and the requirement of more stabilize hemostatic method than just diluted epinephrine injection, in this case Histoacryl injection was selected as the therapeutic technique of choice.

References

1. Raju GS., Gerson L, Das A, Lewis B and American Gastroenterological Association. American Gastroenterological Association (AGA) Institute technical review on obscure gastrointestinal bleeding. *Gastroenterology* 2007;133:1697-717.
2. Kovacs TO. Small bowel bleeding. *Curr Treat Options Gastroenterol* 2005;8:31-8.
3. Dulic-Lakovic E, Dulic M, Hubner D, et al. Bleeding Dieulafoy lesions of the small bowel: a systematic study on the epidemiology and efficacy of enteroscopic treatment. *Gastrointest Endosc* 2011; 74:573-80.



Case 16

Pornphan Thienchanachaiya, M.D.

Wiriyaporn Rittidid, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

Rungsun Rerknimitr, M.D.

A 63-year-old man presented with dyspepsia. EGD was performed and found a single duodenal polyp in the bulb, measuring about 6 mm in diameter (Figure 1). Biopsy was performed. Pathological report revealed gastric metaplasia with *Helicobacter pylori*. Six months

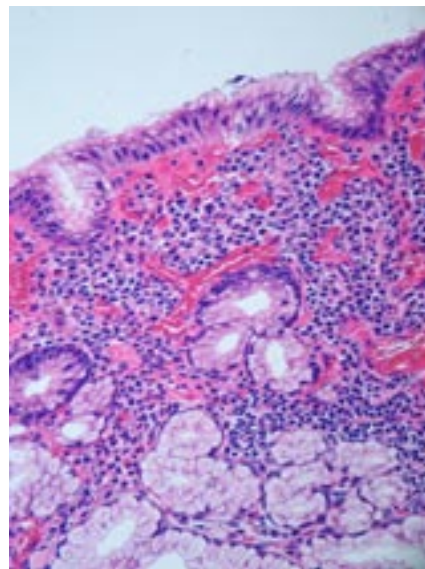
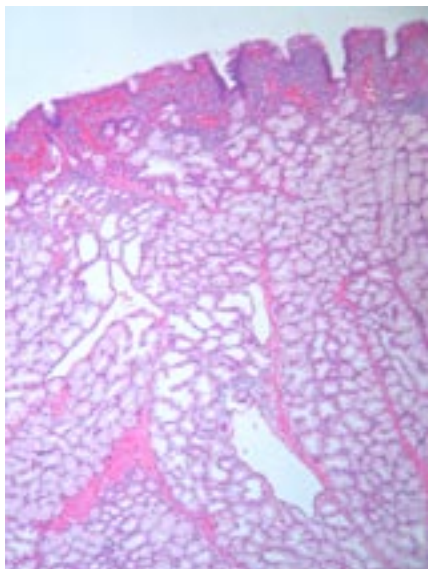
later, follow-up EGD with FICE system showed large duodenal polyp measuring about 10 mm in diameter (Figure 2-4). Endoscopic mucosal resection (EMR) was performed. Microscopic examination showed Brunner gland hyperplasia (Figure 5-6).



■ Figure 1: A duodenal polyp with erosion on top at duodenal bulb, size 6 mm in diameter (yellow arrow).



■ Figure 2-4: A duodenal polyp with erosion on top at duodenal bulb, size 6 mm in diameter (yellow arrow).



■ Figure 5-6: Microscopic examination showed a large polyp and containing structure was compatible with Brunner gland hyperplasia

Diagnosis:

Brunner gland hyperplasia

Discussion:

Brunner gland hyperplasia is not common finding in duodenal bulb. The etiology and pathogenesis of these polyps are uncertain. *Helicobacter pylori* may play a role in pathogenesis. The majority of patients are asymptomatic. Typical endoscopic findings are often diffuse, sessile, and multiple lesions smaller than 10 mm in diameter. Furthermore, Brunner gland adenoma is usually rare and typically a pedunculated polyp sizing about 1-2 cm in diameter¹.

Brunner glands are mucosal and submucosal alkaline secreting glands that hypothesized their mucinous secretion buffer the acidic chyme from the stomach entering to the duodenum. Therefore, the highest concentration of Brunner glands occurs in the first part of the duodenum, gradually decreasing in number in the second and the third portions. Typically cells of Brunner gland are eosinophilic with clear cytoplasm and contain basally oriented nuclei².

Histology of Brunner gland hyperplasia reveals a well-circumscribed submucosal lesion with normal

Brunner glands, glandular lobulation and intervening bands of paucicellular fibrous tissue³.

Treatments depend on size of lesions and the symptoms of patients. Endoscopic polypectomy is considered for large, solitary, or suspected lesions to aid definitive diagnosis and prevent complications from polyp growth. Most lesions are entirely benign and low recurrence rate after polypectomy^{1,3}. There is no evidence to support the endoscopic surveillance of patients with Brunner gland polyp¹.

References

1. Culver EL, McIntyre AS. Sporadic duodenal polyps: classification, investigation, and management. *Endoscopy* 2011;43:144-55.
2. Patel ND, Levy AD, Mehrotra AK, et al. Brunner's gland hyperplasia and hamartoma: imaging features with clinicopathologic correlation. *AJR Am J Roentgenol* 2006;187:715-22.
3. Rocco A, Borriello P, Compare D, et al. Large Brunner's gland adenoma: case report and literature review. *World J Gastroenterol* 2006; 12:1966-8.

Case 17

Nuttaporn Norrasetwanich, M.D.

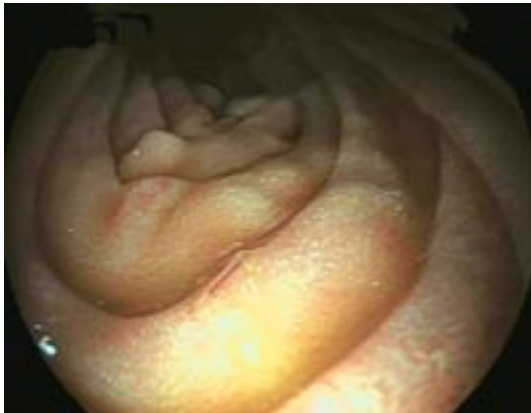
Nopavut Geratikornsupuk, M.D.

Rapat Pittayanon, M.D.

Rungsun Rerknimitr, M.D.

A 30-year-old man presented with intermittent painless melena and anemia for 1 month. He also complained of weakness and fatigue. Laboratory data showed typical iron-deficiency anemia with microcytic hypochromic erythrocytes. He underwent EGD to investigate for the cause of iron deficiency anemia. It

demonstrated a reddish worm moved actively in the second part of duodenum with adjacent multiple erosions (Figure 1-3). The worm was removed endoscopically with forceps and the hookworm was identified (Figure 4).



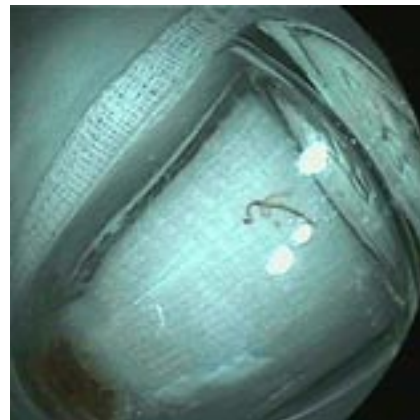
■ Figure 1: White light endoscopy



■ Figure 2: FICE station 2



■ Figure 3: FICE station 4



■ Figure 4

Diagnosis:

Hookworm infestation of the small intestine

Discussion:

Hookworm is distributed everywhere in the world, especially in warm and moist place. Oro-fecal contamination is much more common than the penetration of skin. *Ancylostoma duodenale* and *Necator americanus* are widespread among humans and distinguished from each other by the morphological differences of their mouth capsules, bursa and spicules. They usually live in the upper part of the small intestine, with relatively few in the duodenum¹. The major pathology of hookworm infestation, however, results from intestinal blood loss as a result of adult parasite invasion and attachment to the mucosa and submucosa of the small intestine. Hookworm disease occurs when the blood loss exceeds the nutritional reserves of the host, thus resulting in iron-deficiency anemia. The presence of more than 40 adult worms in the small intestine is estimated to justify the host hemoglobin concentrations to be below 11 g/dL. The chronic protein loss from

heavy hookworm infestation can result in hypoproteinemia and anasarca². Nevertheless, when a round worm is found in the duodenum during upper gastrointestinal endoscopy, differential diagnosis is necessary to determine the final diagnosis and treatment. In this case we used FICE system to enhance the superficial mucosal layer and to discriminate the parasite from other structures³.

References

1. Kato T, Kamoi R, Iida M, et al. Endoscopic diagnosis of hookworm disease of the duodenum. *J Clin Gastroenterol* 1997;24:100-2.
2. Bethony J, Brooker S, Albonico M, et al. Soil-transmitted helminth infections: ascariasis, trichuriasis, and hookworm. *Lancet* 2006;367:1521-32.
3. Fedeli P, Gasbarrini A, Cammarota G, et al. Spectral endoscopic imaging: the multiband system for enhancing the endoscopic surface visualization. *J Clin Gastroenterol* 2011;45:6-15.



Case 18

Nuttaporn Norrasetwanich, M.D.

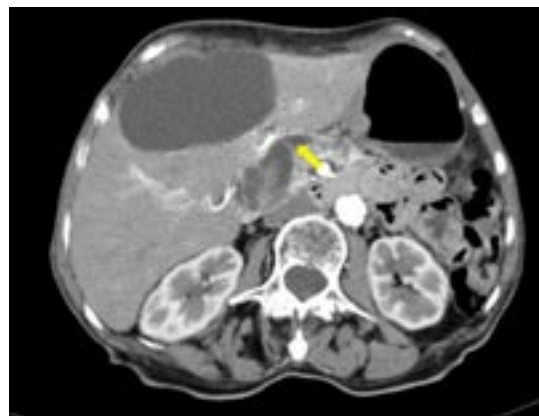
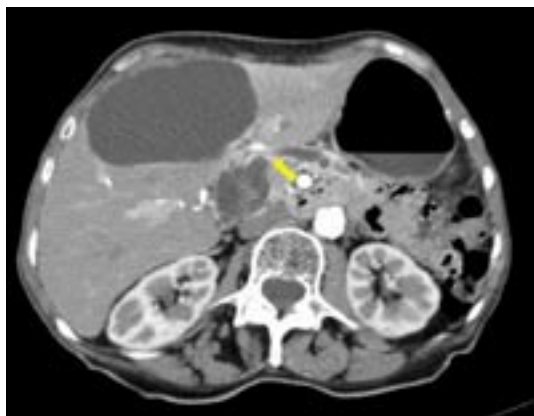
Phonthep Angsuwatcharakon, M.D.

Linda Pantongrag-Brown, M.D.

Rungsun Rerknimitr, M.D.

A 77-year-old woman, underlying breast cancer, underwent computed tomography (CT) of whole abdomen for staging. It demonstrated a 2.4 x 3.7 cm cystic lesion with fine internal septation, and minimal soft-tissue at the pancreatic head. This cystic lesion appeared to connect to the main pancreatic duct, associated with mild dilatation of upstream pancreatic duct. In addition, there were several simple liver cysts

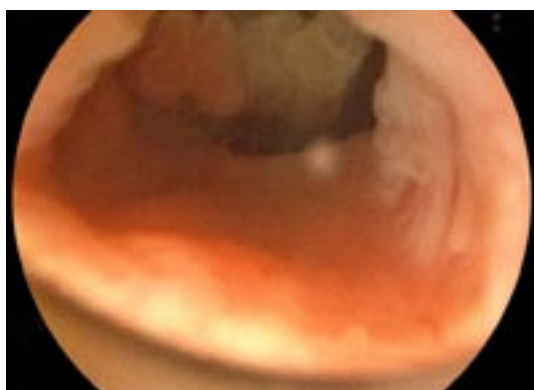
(Figure 1-2). The side-view duodenoscopy was carried out. It revealed patulous pancreatic orifice with mucin plug (Figure 3), giving the “fish-eye appearance”, and small papillary projections inside the pancreatic duct (fish egg) (Figure 4-7). Main duct type of intraductal papillary mucinous neoplasm (IPMN) is the most likely diagnosis.



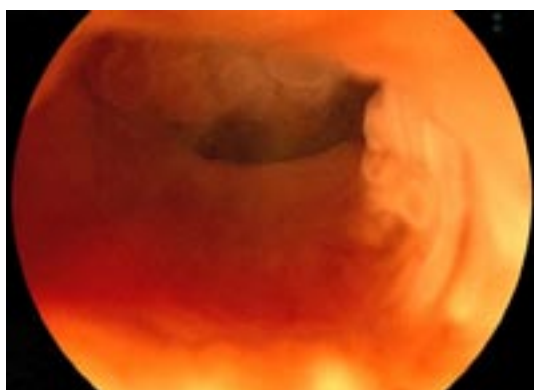
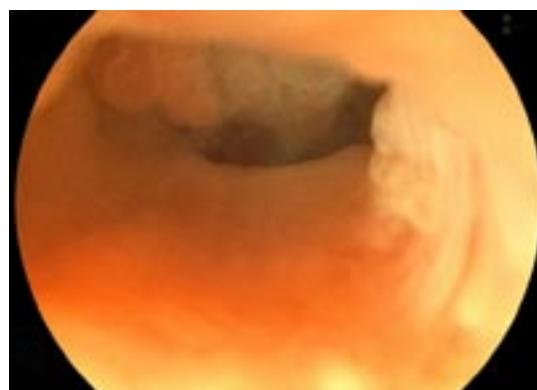
■ Figure 1-2: CT of whole abdomen shows a cystic lesion with fine internal septation and minimal soft-tissue at the pancreatic head. The lesion seems to connect with the main pancreatic duct (arrow)



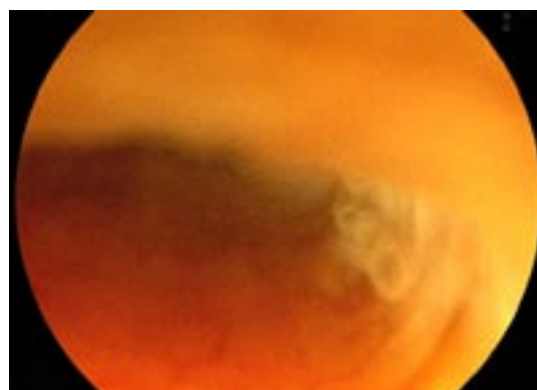
■ Figure 3: Side-view duodenoscopy revealed patulous pancreatic orifice with mucin plug (fish eye)



■ Figure 4-5: Close-up white light imaging revealed “figh egg” apperance of the tumor



■ Figure 6: Close-up FICE (station 2) imaging revealed “figh egg” apperance of the tumor



■ Figure 7: Close-up FICE (station 6) imaging revealed “figh egg” apperance of the tumor

Diagnosis:

Main duct type of intraductal papillary mucinous neoplasm of the pancreas

Discussion:

IPMNs are mucinous lesions that arise from the epithelial lining of the main pancreatic duct or its side branches. They are characterized by neoplastic, mucin-secreting, papillary cells projecting from the pancreatic ductal surface. IPMNs range from premalignant lesions with low-grade dysplasia to invasive malignancy. Clinically, patients may present with recurrent abdominal pain, nausea, or vomiting from secondary pancreatitis. However, IPMNs are most commonly asymptomatic and

discovered incidentally on routine imaging. Diagnosis of IPMNs with multi-detector computed tomography (MDCT) and magnetic resonance imaging (MRI) is frequently used, but still has limitation in distinguishing main duct from branch duct type of IPMNs and in differentiating the broad spectrum of pancreatic cystic lesions. Endoscopic evaluation of these lesions provides additional imaging, serology, and histological data to aid in the identification of IPMNs and to determine treatment course¹.

Yen TH, et al. experienced 109 patients with intraductal papillary mucinous tumors (IPMTs). Ultrasonography (US) contributed significantly to the detection of IPMTs. The characteristic findings

obtained by US/endoscopic ultrasonography (EUS) were dilatation of the main duct, grape-like clusters of dilated branch ducts, a solid tumor or mural nodule, and a mucus echo. Duodenoscopy showed enlargement of the papilla, widely opened orifice of the papilla, and mucin excretion from the orifice (fish eye). All these three endoscopic findings were obtained together in 68 of the 109 patients. A pancreatogram revealed diffuse dilatation of the main duct and/or cystically dilated branch ducts, without stenosis or obstruction, in all patients. A filling defect due to polypoid lesion or mucin in the pancreatic ducts could also be seen in all patients. Peroral transpapillary pancreatoscopy (POPS)

revealed papillary tumors (fish-egg-like appearance), granular mucosa, or mucin².

References

1. Turner B, Brugge W. Diagnostic and therapeutic endoscopic approaches to intraductal papillary mucinous neoplasm. *World J Gastrointest Surg* 2010;2:337-41.
2. Yamao K, Nakamura T, Suzuki T, et al. Endoscopic diagnosis and staging of mucinous cystic neoplasms and intraductal papillary-mucinous tumors. *J Hepatobiliary Pancreat Surg* 2003;10:142-6.



Case 19

Piyapan Prueksapanich, M.D.

Satimai Aniwon, M.D.

Vichai Viriyautsahakul, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

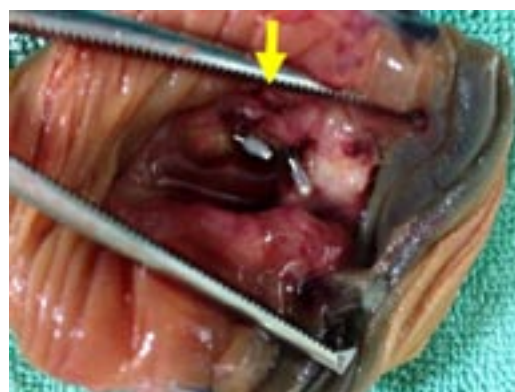
Rungsun Rerknimitr, M.D.

A 78-year-old woman presented with hematochezia and anemic symptoms. EGD reported as unremarkable exam and colonoscopy showed fresh blood in the terminal ileum. She required 5 units of blood transfusion. Overt obscure gastrointestinal bleeding was entertained. Double balloon enteroscopy revealed ulcerative submucosal mass sized 3 cm in diameter with active blood spurting at proximal jejunum

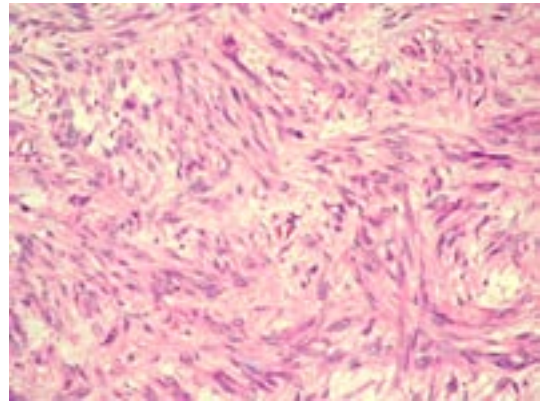
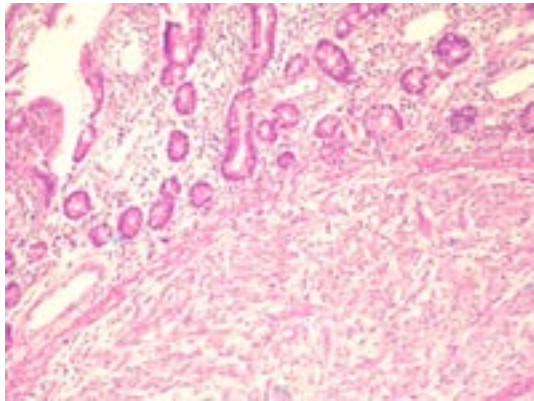
(Figure 1-2). Hemoclipping failed to achieve hemostasis. India ink was tattooed into the wall of small bowel nearby the lesion. Emergency laparotomy with wedge resection of jejunal mass with end-to-end anastomosis was performed (Figure 3-4). Pathological result showed spindle cells tumor compatible with gastrointestinal stromal tumor (GIST) (Figure 5-6).



■ Figure 1-2: Ulcerative submucosal mass with active bleeding at proximal jejunum



■ Figure 3-4: Jejunum mass 3 cm in diameter



■ Figures 5-6: Spindle cells tumor involved in mucosa, submucosa and muscularis propria

Diagnosis:

Bleeding Gastrointestinal Stromal Tumor (GIST) at jejunum

Discussion:

Double balloon enteroscopy was developed by Yamamoto, et al. and provided entire small bowel examination as well as endoscopic intervention. The sensitivity and specificity of a DBE in the diagnosis of small intestinal lesions responsible for intestinal bleeding in patients with obscure gastrointestinal hemorrhage were 92.7% and 96.4%, respectively¹.

Gastrointestinal stromal tumor (GIST) is the most common mesenchymal tumor of the intestinal

tract. Small intestine is the second most common site after stomach. GIST may present with abdominal pain, palpable mass, intestinal obstruction or bleeding. Surgery is the mainstay of treatment².

References

1. Shu T, Keigo M, Yukie Y, et al. Diagnostic yield of double-balloon endoscopy in patients with obscure GI bleeding. *Gastrointest Endosc* 2008; 68:683-91.
2. Shilpa G, Stanley WA, Chandrajit PR. Small intestine gastrointestinal stromal tumors. *Curr Opin Gastroenterol* 2012;28:113-23.



Part 4

Colon

Case 1

Suparat Khemnark, M.D.

Sombat Treeprasertsuk, M.D.

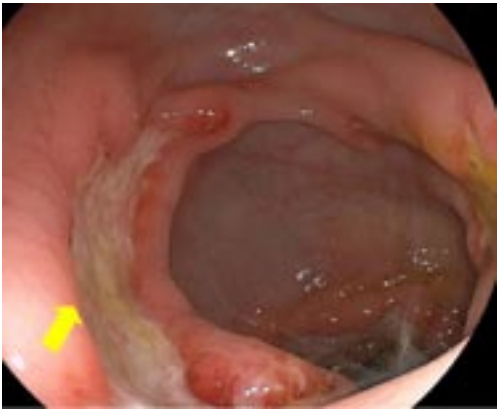
Linda Pantongrag-Brown, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

Rungsun Rerknimitr, M.D.

A 21-year-old monk presented with chronic diarrhea and significant weight loss for 8 months. Stool exam revealed numerous RBC and WBC. Stool test for acid fast bacilli was negative. Colonoscopy showed multiple large transverse ulcers with exudates from the ascending colon to descending colon (Figure 1-2). Barium enema revealed multiple segments of mucosal irregularity and ulceration of the entire colon, with extension to involve ileocecal valve and terminal ileum (Figure 3-4). Chest

radiograph showed reticulonodular infiltration of the right lung, predominantly on the upper lobe (Figure 5). Sputum for acid fast bacilli stain was positive. Computed tomography of the abdomen revealed multiple segments of thickened bowel wall, involving sigmoid colon up to cecum and terminal ileum (Figure 6-7). The degree of wall thickening is more on the right side. Several necrotic mesenteric nodes were present (Figure 8-9).



■ Figure 1: Descending colon (Transverse ulcer)



■ Figure 2: Large ulcer with exudates



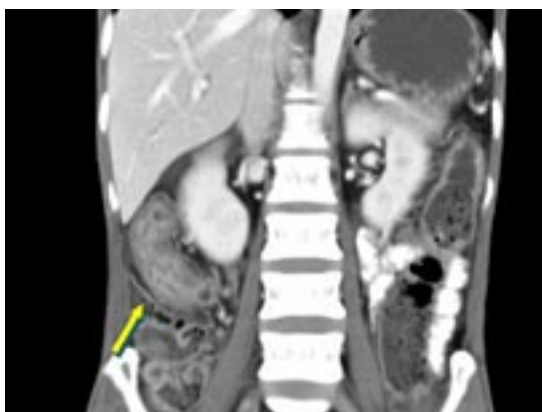
■ Figure 3: Barium enema showed mucosal irregularity of transverse and ascending colon



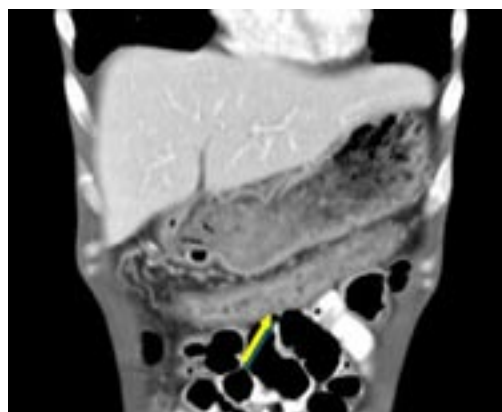
■ Figure 4: BE showed mucosal irregularity of cecum and IC valve



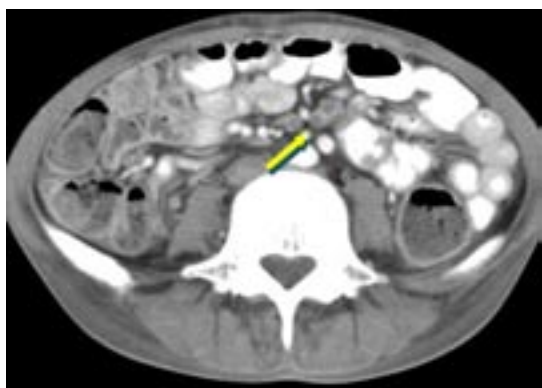
■ Figure 5: Chest X-ray showed reticulonodular infiltration of the right upper lung



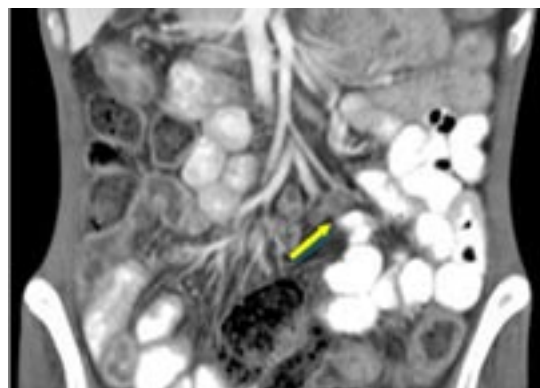
■ Figure 6: CT scan of the abdomen thickened wall of the ascending colon (yellow arrow)



■ Figure 7: Demonstrating thickened wall of the transverse colon (yellow arrow)



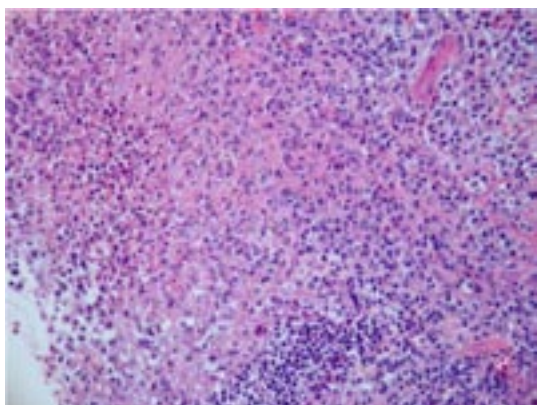
■ Figure 8: Necrotic mesenteric nodes (yellow arrow)



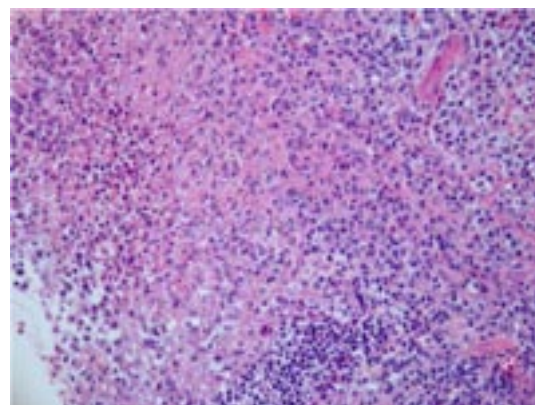
■ Figure 9: Necrotic mesenteric nodes

Colon biopsy showed numerous acute and chronic inflammatory cells infiltrating the lamina propria, forming vague granulomas with ulcerated surface. The remaining mucosa displayed cryptitis and crypt

abscesses. These crypts were lined by reactive epithelium (Figure 10-11). Tissue for acid fast bacilli stain and PCR for tuberculosis were negative.



■ Figure 10: Numerous inflammatory cells infiltrating the lamina propria, and ulcerated surface



■ Figure 11: Vague granulomas

Diagnosis:

Disseminated mycobacterium tuberculosis

Discussion:

Digestive system is one of the sites for extrapulmonary tuberculosis. The digestive system is involved in 66% of patients with abdominal tuberculosis¹. The ileocecal region is the most commonly affected within the gastrointestinal tract followed by the colon. In colonic involvement, the cecum and ascending colon are most commonly affected, followed by the transverse and descending colon². Autopsies of patients with pulmonary tuberculosis before the era of effective treatment demonstrated intestinal involvement in 55-90% of fatal cases³. Direct infection from the wall of the gut is highly possible after drinking unpasteurized milk or swallowing a large number of bacilli from the pulmonary cavity. Reactivation from the body within few years after hematogenous spreading is also a possible mode².

Tripathi and Amarapurkar studied 110 cases of TB in the GI tract to identify the morphologic spectrum of the disease. The most common clinical presentations were abdominal pain (82.7%), fever (58.2%), weight loss

(53.6%), and diarrhea (29.1%). Concurrent involvement of the ileum, cecum and ascending colon were seen most frequently in 56 cases (50.9%). The next most common site of involvement was the terminal ileum alone, in 43 cases (39.1%)⁴. Common colonoscopic findings were ulcers (70%), nodules (56%), deformed cecum and ileocecal valve (40%), strictures (23%), polypoid lesions (14%), and fibrous bands forming mucosal bridges (7%)⁵.

References

1. Bhansali SK. Abdominal tuberculosis. Experiences with 300 cases. *Am J Gastroenterol* 1977;67:324-37.
2. Abraham P, Fersosh PM. Tuberculosis of gastrointestinal tract. *Ind J Tub* 2002;39:251-6.
3. Stolte M, Sticht T, Eidt S, et al. Colitis Tuberculosis. *Endoscopy* 1994;26:659-65.
4. Tripathi PB, Amarapurkar AD. Morphological spectrum of gastrointestinal tuberculosis. *Trop Gastroenterol* 2009;30:35-9.
5. Alvares JF, Devarbhavi H, Makhija P, et al. Clinical, colonoscopic, and histological profile of colonic tuberculosis in a tertiary hospital. *Endoscopy* 2005;37:351-6.



Case 2

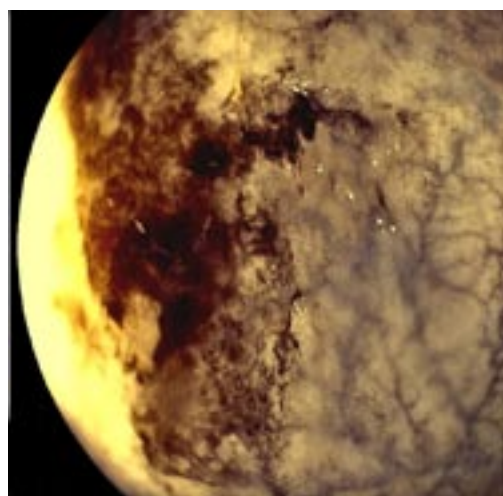
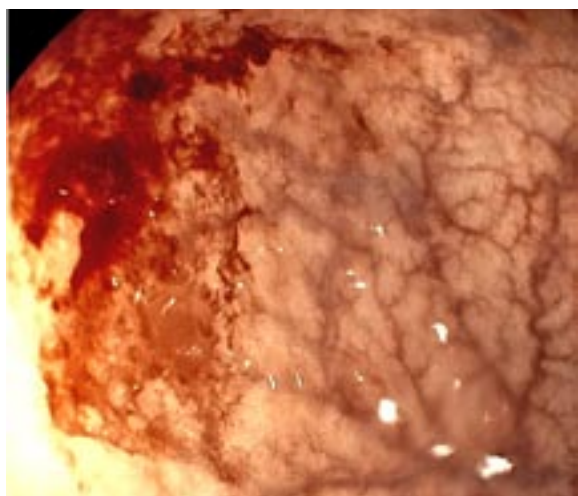
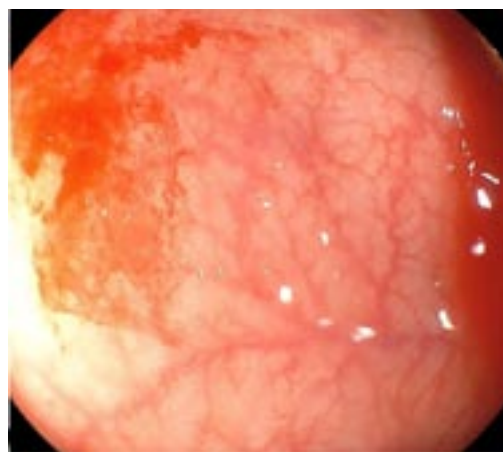
Tanassanee Soontornmanokul, M.D.

Satimai Aniwan, M.D.

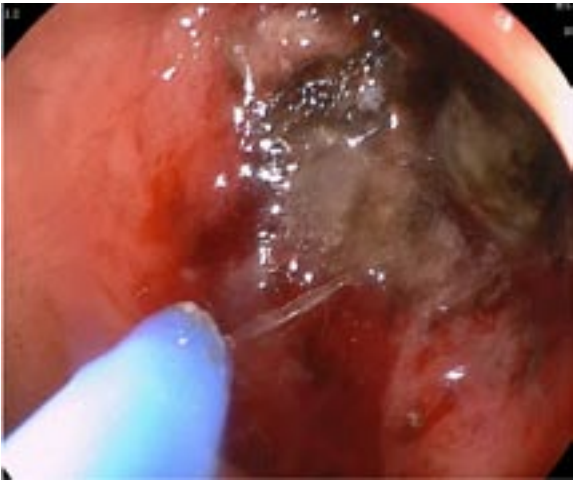
Rungsun Rerknimitr, M.D.

A 67-year-old woman presented with intermittent hematochezia for a year. She had been diagnosed with a locally advanced cervical cancer and

had undergone external radiation for 5 years. She underwent a sigmoidoscopy. Endoscopic findings showed (Figure 1-5).



■ Figure 1-4: Demonstrating neovascularization with bleeding secondary to radiation injury



■ Figure 5: Demonstrating an argon plasma coagulation to ablate the abnormal neovascularization that caused bleeding

Diagnosis:

Radiation induced colitis

Discussion:

Radiation induced proctocolitis is a well-known complication of pelvic irradiation, usually develops within the first year after radiotherapy¹. Rectal bleeding is the most common presenting symptom, and may lead to iron deficiency anemia requiring blood transfusions². Chronic radiation exposure results in varying degrees of neovascular formation over the intestinal mucosa. Generally, the endoscopy shows diffuse hemorrhagic or hyperemic mucosa with telangiectasias. Occasionally there may be circumferential ulcers with a relatively sharp proximal and distal demarcation³. Although most cases of bleeding radiation induced proctocolitis resolve spontaneously, the management may be difficult in severe cases. Argon plasma coagulation (APC) appears to be a simple, safe, and effective to manage this condition, and generally accepted as the treatment of choice⁴. Recent prospective series showed that the success rate

was 98.5% after a median of the 2 treatment sessions, and there were no significant side effects⁵.

References

1. Babb RR. Radiation proctitis: a review. *Am J Gastroenterol* 1996;91:1309-11.
2. Gilinsky NH, Burns DG, Barbezat GO, et al. The natural history of radiation-induced proctosigmoiditis: an analysis of 88 patients. *Q J Med* 1983;52:40-53.
3. Kountouras J, Zavos C. Recent advances in the management of radiation colitis. *World J Gastroenterol* 2008;14:7289-301.
4. Silva RA, Correia AJ, Dias LM, et al. Argon plasma coagulation therapy for hemorrhagic radiation proctosigmoiditis. *Gastrointest Endosc* 1999;50:221-4.
5. Sato Y, Takayama T, Sagawa T, et al. Argon plasma coagulation treatment of hemorrhagic radiation proctopathy: the optimal settings for application and long-term outcome. *Gastrointest Endosc* 2011;73:543-9.

Case 3

Kanita Chattrasophon, M.D.

Satimai Aniwan, M.D.

Rungsun Rerknimitr, M.D.

A 48-year-old woman presented with bloody diarrhea and polyarthralgia. She has been diagnosed with Crohn's disease (CD) for 10 years. She was treated with 6-mercaptopurine. One month prior to admission, she

developed bloody diarrhea and polyarthralgia. Colonoscopy showed circumferential ulceration with severe inflammation (Figure 1-2) causing a narrow colonic lumen at 5 cm from the anal verge (Figure 3-4).



■ Figure 1-2: Circumferential ulceration with severe inflammation



■ Figure 3-4: Severe mucosal inflammation causing luminal stricture

Diagnosis:

Active Crohn's disease with rectal stricture

Discussion:

Non-fistulizing perianal lesion, including ulcerations, stricture and anal carcinoma are observed in Crohn's disease. Stricture occurs as consequence of chronic inflammation or fistula can be found in anus (34%) and rectum (50%)¹. The presentations are bloody diarrhea, constipation, perineal pain, and fecal incontinence. Although these patients have usually a moderate degree of stenosis, they are asymptomatic. However, in a case with severe stenosis, the clinically intestinal obstruction usually presents. There are 2 types of rectal and anal stricture for CD. Rectal stricture type 1, inflammatory stenosis results from anal spasm can be

relaxed and opened easily under anesthesia, but in the rectal stricture type 2, the stricture develops from scarring tissue^{2,3}.

References

1. Robert T. Lewis, David J, et al. Anorectal Crohn's disease. *Surg Clin N Am* 2010;90:83-97.
2. Bouguen G, Siproudhis L, Bretagne JF, et al. Nonfistulizing perianal Crohn's disease: Clinical Features, epidemiology, and treatment. *Inflamm Bowel Dis* 2010;16:1431-42.
3. Bouguen G, Trouilloud K, Siproudhis L, et al. Long-term outcome of non-fistulizing (ulceration, stricture) perianal Crohn's disease in patients treated with infliximab. *Aliment Pharmacol Ther* 2009;30:749-56.



Case 4

Kanita Chattrasophon, M.D.

Satimai Aniwan, M.D.

Rungsun Rerknimitr, M.D.

A 25-year-old man presented with chronic diarrhea and fistula in ano for 6 months. Physical examination showed an opening of perianal fistula with discharge (Figure 1). He underwent colonoscopy. Colonoscopy showed an internal opening of fistula near anal canal (Figure 2) and a deep ulcer in the rectum (Figure 3) with inflammatory pseudopolyp (Figure 4).

Biopsy showed moderate lymphoplasmacytic infiltration in edematous lamina propria and mildly distorted dilated crypts lined by hyperplastic epithelium consistent with inflammatory pseudopolyp. Biopsy tissues were negative for acid fast bacilli and no inclusion body was found. PCR for *Mycobacterium tuberculosis* was also negative.



■ Figure 1: Opening of the fistula in ano



■ Figure 2: Opening of fistula in the anal canal



■ Figure 3: Deep rectal ulcer



■ Figure 4: Inflammatory pseudopolyp

Diagnosis:

Active Crohn's disease with perianal fistula

Discussion:

Perianal fistula is an abnormal connection between the anal canal and skin. Incidence in Crohn's population varies from 17-43%¹. Fistula occurred in 35% over time. Of those fistulas, 54% are perianal types, 24% are enteroenteric fistulas, 9% are rectovaginal fistulas and 13% are miscellaneous fistulas such as enterocutaneous, enterovesical, and intraabdominal fistulas². Perianal fistula could be as an initial presentation of Crohn's disease and preceding intestinal symptoms for years. The symptoms are persistent anal pain, painful defecation, and perianal purulent discharge. Fistulas are classified in 2 types.

1) Simple fistula is defined as a fistula located below the dentate line, single external opening, painless

with no rectovaginal fistula and no anorectal stricture.

2) complex fistula is defined as a fistula located above the dentate line with multiple openings, and/or with abscess formation, and/or with rectovaginal fistula, and/or with anorectal stricture³.

References

1. Schwartz DA, Pemberton JH, Sandborn WJ. Diagnosis and treatment of perianal fistulas in Crohn disease. *Ann Intern Med* 2001;135:906-18.
2. Schwartz DA, Lofus EV Jr, Tremaine WJ, et al. The natural history of fistulizing Crohn's disease in Olmsted County, Minnesota. *Gastroenterology* 2002;122:875-80.
3. Sandborn WJ, Fazio VW, Feagan BG, et al. AGA technical review on perianal Crohn's disease. *Gastroenterology* 2003;125:1508-30.



Case 5

Kittiyod Poovorawan, M.D.

Satimai Aniwan, M.D.

Rungsun Rerknimitr, M.D.

A 70-year-old woman presented with recurrent hematochezia. She had been diagnosed with dilated cardiomyopathy. She underwent flexible sigmoidoscopy. The endoscopic findings showed a visible vessel surrounded with normal mucosa in the rectum at 5 cm

from the anal-verge (Figure 1). After an injection of diluted epinephrine, Argon plasma coagulator (APC) was applied at the lesion. The pulsatile bleeding occurred during applied APC. Hemostasis was achieved successfully (Figure 2-4).



■ Figure 1: A visible vessel surrounded with normal mucosa in the rectum.



■ Figure 2: APC was applied.



■ Figure 3: The pulsatile bleeding developed during applied APC



■ Figure 4: Post APC

Diagnosis:

Rectal Dieulafoy's lesion

Discussion:

Rectal Dieulafoy's is an unusual source of rectal bleeding. Clinical course can be either intermittent or massive rectal bleeding¹. Majority of Dieulafoy's lesion occur in a lesser curvature of stomach within 6 cm of the gastroesophageal junction². Several effective endoscopic treatment of rectal Dieulafoy's had been reported such as combination of epinephrine injection and coagulation therapy, application of a Hemoclip, and APC³.

References

1. Chen YY, Yen HH. Massive bleeding from a rectal dieulafoy lesion: combined multidetector-row CT diagnosis and endoscopic therapy. *Surg Laparosc Endosc Percutan Tech* 2008;18:398-9.
2. Veldhuyzen van Zanten SJ, Bartelsman JF, et al. Recurrent massive haematemesis from Dieulafoy vascular malformations--a review of 101 cases. *Gut* 1986;27:213-22.
3. Apiratpracha W, Ho JK, Powell JJ, Yoshida EM. Acute lower gastrointestinal bleeding from a dieulafoy lesion proximal to the anorectal junction post-orthotopic liver transplant. *World J Gastroenterol* 2006;12:7547-8.



Case 6

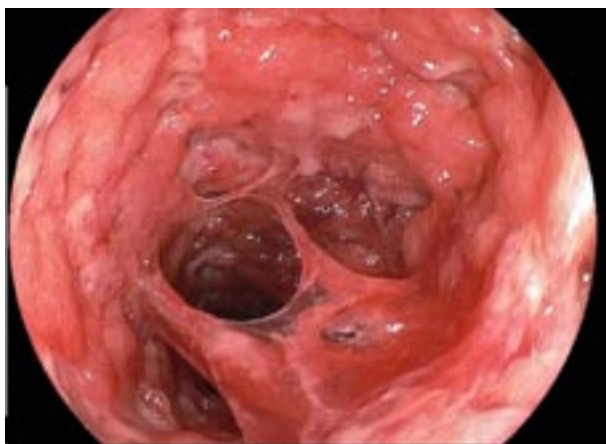
Kittiyod Poovorawan, M.D.

Satimai Aniwan, M.D.

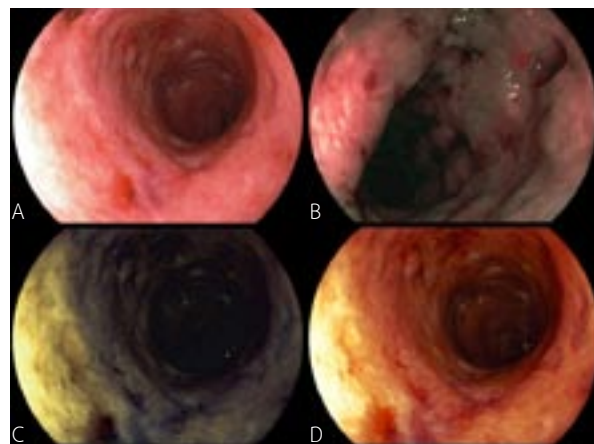
Rungsun Rerknimitr, M.D.

A 59-year-old woman presented with mucous bloody diarrhea for a year. She had no fever and no weight loss. Stool examination, stool culture, and stool test for *C.difficile* toxin assay were negative. Subsequently, she underwent colonoscopy. Colonoscopy revealed diffuse

erythema, friable mucosa with ulceration and mucosal bridge of the entire colon (Figure 1-2). The terminal ileum was normal. Biopsy showed chronic colitis consistent with ulcerative colitis. Her clinical conditions improved significantly after corticosteroids treatment.



■ Figure 1: Diffuse erythema, friable mucosa with ulceration, and mucosal bridge of the entire colon



■ Figure 2: Diffuse erythema, friability of the mucosa with multiple ulcers under white light image (A), Under FICE station 8 (B), Under FICE station 4 (C), Under FICE station 6 (D)

Diagnosis:

Active severe ulcerative colitis

Discussion:

Ulcerative colitis (UC) is immunologically mediated disease characterized by chronic colonic mucosal inflammation. Mucosal bridging, characterized by bridging of regenerative mucosal tissue from one wall to an adjacent wall, occasionally found in UC¹. This condition carries no malignant potential and can be found in other colitis conditions including ischemic colitis, infective colitis, and colonic tuberculosis².

References

1. Van Moerkercke W, Deboever G, Lambrecht G, et al. Severe bridging fibrosis of the colon in a man with inflammatory bowel disease. *Endoscopy* 2007;39 Suppl 1:E294.
2. Gupta G, Nijhawan S, Chander S, et al. Colonic mucosal bridging in ulcerative colitis. *Indian J Gastroenterol* 2012;31:39.

Case 7

Nuttaporn Norrasetwanich, M.D.

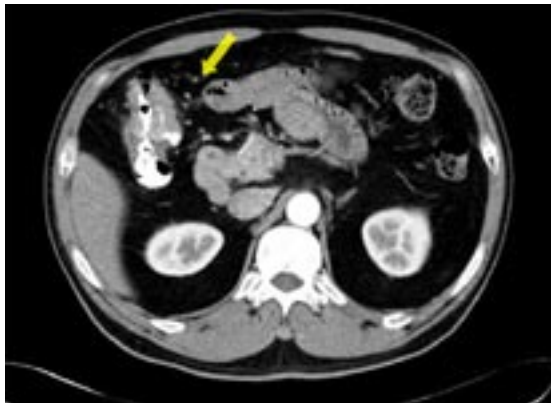
Linda Pantongrag-Brown, M.D.

Naruemon Wisedopas-Klaikeaw M.D.

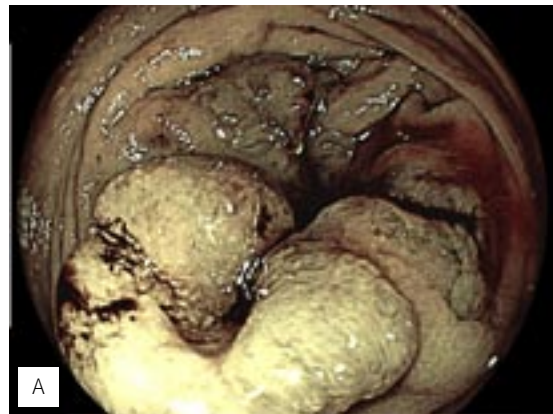
Rungsun Rerknimitr, M.D.

A 52-year-old man presented with bowel habit change. He had bloody diarrhea and anemia for a month. He lost weight about 5 kgs. Computed tomography of the abdomen demonstrated circumferential thickened wall of the hepatic flexure of colon, about 5.4 cm in length, associated with minimal pericolonic fat stranding, suggestive of carcinoma (T3) (Figure 1). Colonoscopy revealed a circumferential

ulcerative mass with friable mucosa and easily contacts bleeding at transverse colon. The colonic lumen was narrowed but scope could pass through this lesion (Figure 2-3). Biopsy of the lesion showed complex neoplastic sheets with occasional glandular formation associated with desmoplastic reaction. Those lining cells contained markedly pleomorphic nuclei. The diagnosis was well-differentiated adenocarcinoma (Figure 4).



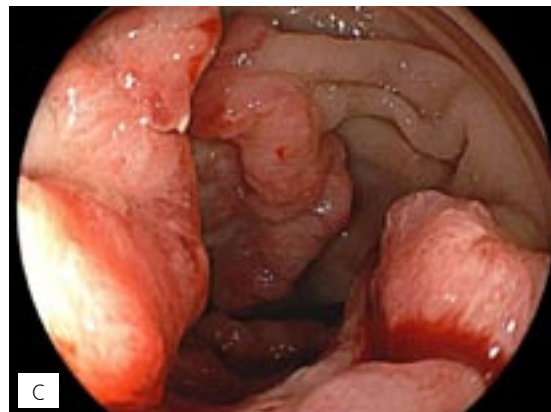
■ Figure 1: Circumferential wall thickening of the hepatic flexure



A

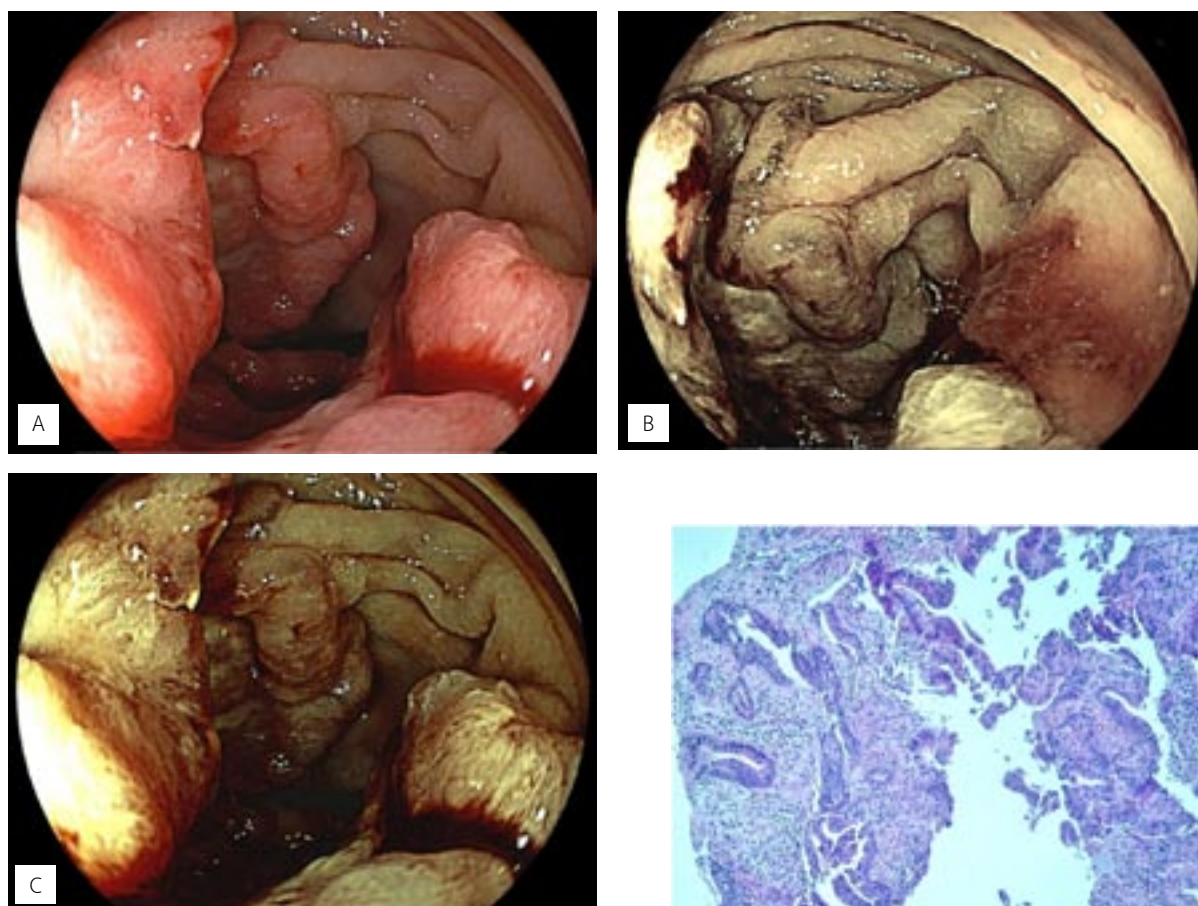


B



C

■ Figure 2: White light image (A) , FICE station 0 (B) and FICE station 8 (C)



■ Figure 3: White light image (A), FICE station 0 (B) and FICE station 1 (C)

■ Figure 4: Well-differentiated adenocarcinoma

Diagnosis:

Colon cancer (well-differentiated adenocarcinoma)

Discussion:

Colon cancer is the second leading cause of cancer death in men and the third leading cause in women in the United States. Since the 1980s, there has been a persistent trend in the increasing percentage of right-sided colon cancers, and decreasing percentage of left-sided and sigmoid colon cancers¹.

During embryologic development, the right colon (cecum, ascending colon, proximal two-thirds of the transverse colon) arises from the midgut and the left

colon (distal one-third of the transverse colon, descending and sigmoid colon, rectum) arises from the hindgut. This difference is reflected in the dual blood supply. Right-sided colon cancers (RCCs) are typically bulky, exophytic, polypoid lesions projecting into the lumen and causing significant anemia. Left-sided colon cancers (LCCs) are infiltrating, constricting lesions encircling the lumen, often leading to obstruction². Accumulating evidence suggests that the risk of colorectal cancer is different for proximal and distal tumors^{2,3}. White light endoscopy is generally enough to detect the circumferential colon cancer, however FICE may be able to depict additional abnormal vasculatures related to neoplasm⁴.

References

1. Meguid RA, Slidell MB, et al. Is there a difference in survival between right-versus left-sided colon cancers? *Ann Surg Oncol* 2008;15:2388-94.
2. Benedix F, Kube R, Meyer F, et al. Comparison of 17,641 patients with right- and left-sided colon cancer: differences in epidemiology, perioperative course, histology, and survival. *Dis Colon Rectum* 2010;53:57-64.
3. Iacopetta B. Are there two sides to colorectal cancer? *Int J Cancer* 2002;101:403-8.
4. Liu YX, Huang LY, Bian XP, et al. Fuji Intelligent Chromo Endoscopy and staining technique for the diagnosis of colon tumor. *Chin Med J (Engl)* 2008;121:977-82.



Case 8

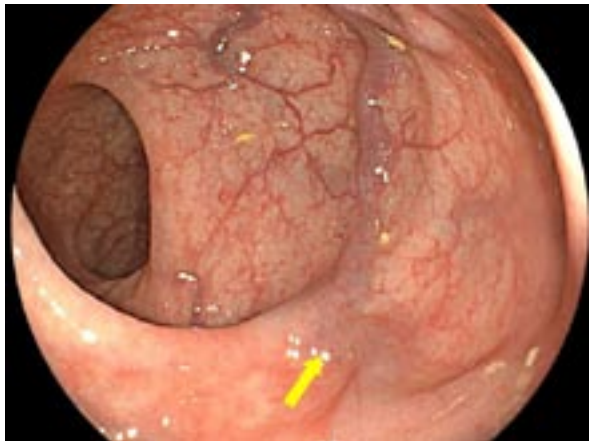
Tanassanee Soontornmanokul, M.D.

Satimai Aniwan, M.D.

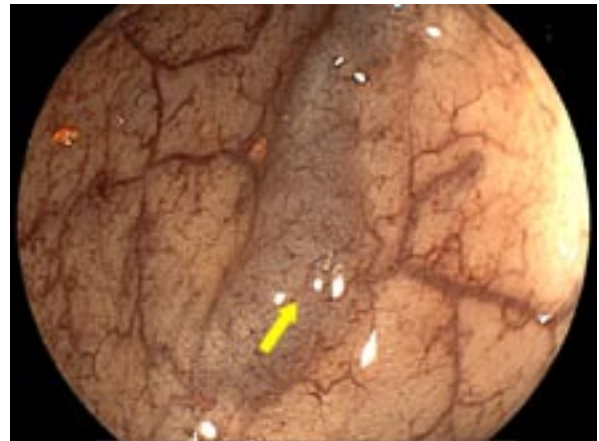
Rungsun Rerknimitr, M.D.

A 56-year-old man with compensated HBV cirrhosis underwent colonoscopy for an indication of iron deficiency anemia. He had no previous history of visible gastrointestinal bleeding. EGD revealed severe portal

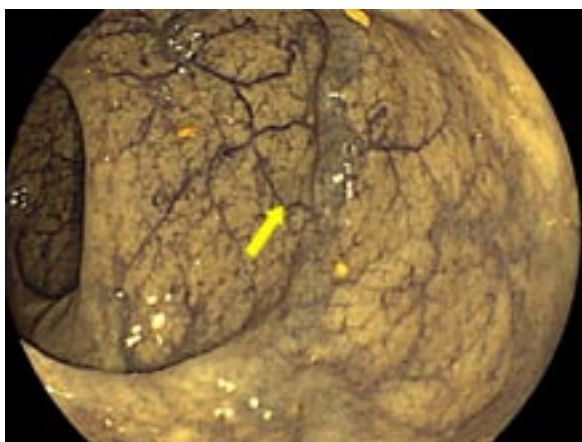
hypertensive gastropathy, esophageal, and gastric varices without any stigmata of recent bleeding. Colonoscopic findings showed a tortuous dilated vein extending from the anus to lower rectum without red color sign (Figure 1-4).



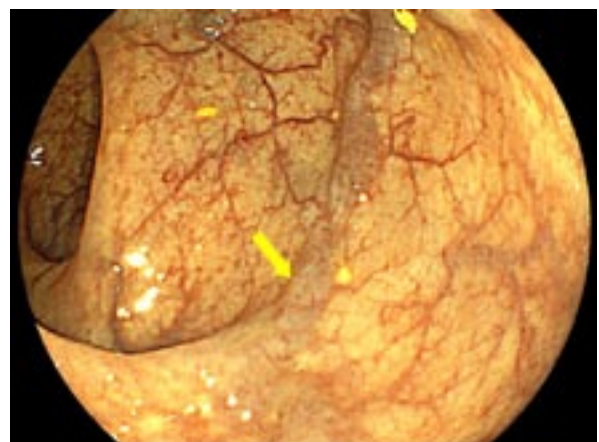
■ Figure 1: White light image



■ Figure 2: FICE station 4



■ Figure 3: FICE station 2



■ Figure 4: FICE station 6

Diagnosis:

Non-bleeding rectal varices

Discussion:

Rectal varices are collateral vessels that connect the superior hemorrhoidal veins (inferior mesenteric and portal circulation) with the middle and inferior hemorrhoidal veins (pudendal vein and systemic circulation). The prevalence of anorectal varices in patients with portal hypertension was 40-77%¹ with minimal risk of significant bleeding. However, bleeding from rectal varix could be fatal². Rectal varices often coexist with hemorrhoids and must be distinguished from each other by their endoscopic characteristics, i.e. varices usually extend from the anal canal into the rectum, whereas hemorrhoids are confined to the anal canal². At present, there are still no evidence-based guidelines on the management of rectal varices. A case series of successful endoscopic band ligation and endoscopic injection sclerotherapy with N-butyl-2-cyanoacrylate of bleeding rectal varices were reported^{2,3},

but the results were controversial and needed further controlled prospective studies. Recently, Weilert F, et al. reported the using EUS-guided therapy with embolization coil and glue injection to control bleeding rectal varices⁴.

References

1. Chawla Y, Dilawari JB. Anorectal varices--their frequency in cirrhotic and non-cirrhotic portal hypertension. *Gut* 1991;32:309-11.
2. Coelho-Prabhu N, Baron TH, Kamath PS. Endoscopic band ligation of rectal varices: a case series. *Endoscopy* 2010;42:173-6.
3. Ryu SH, Moon JS, Kim I, et al. Endoscopic injection sclerotherapy with N-butyl-2-cyanoacrylate in a patient with massive rectal variceal bleeding: a case report. *Gastrointest Endosc* 2005; 62:632-5.
4. Weilert F, Shah JN, Marson FP, et al. EUS-guided coil and glue for bleeding rectal varix. *Gastrointest Endosc*. 2011 Dec 13. [Epub ahead of print]



Case 9

Tanassanee Soontornmanokul, M.D.

Satimai Aniwan, M.D.

Linda Pantongrag-Brown, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

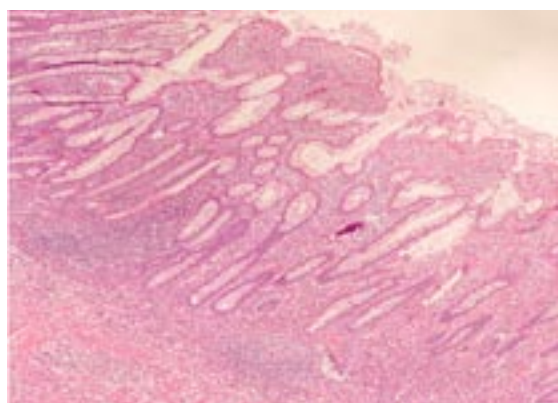
Rungsun Rerknimitr, M.D.

A 46-year-old man with a history of bowel resection secondary to severe Crohn's disease, presented with recurrent RLQ abdominal pain and palpable mass. CT whole abdomen showed circumferential wall thickening of the ileocecal region without lymphadenopathy (Figure 1-2). A recurrent

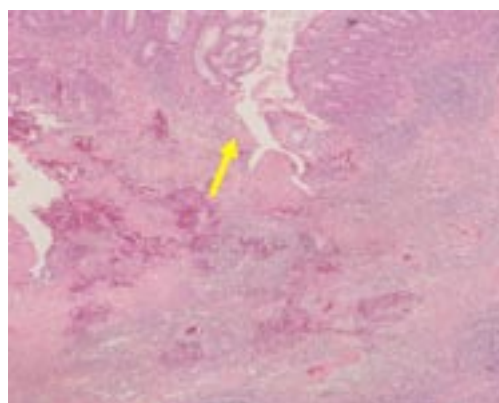
Crohn's disease with ileocolic obstruction was clinically entertained. Right hemicolectomy with ileocelectomy was performed. Histopathology showed chronic colitis with crypt distortion and crypt atrophy without any organism (Figure 3-4).



■ Figure 1-2: Circumferential wall thickening of the ileocecal region (yellow arrow)



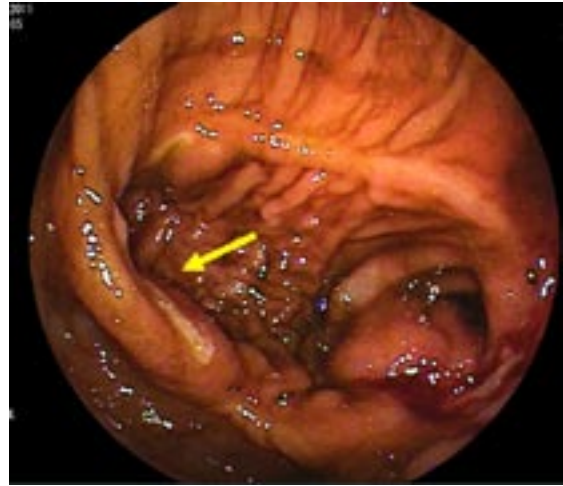
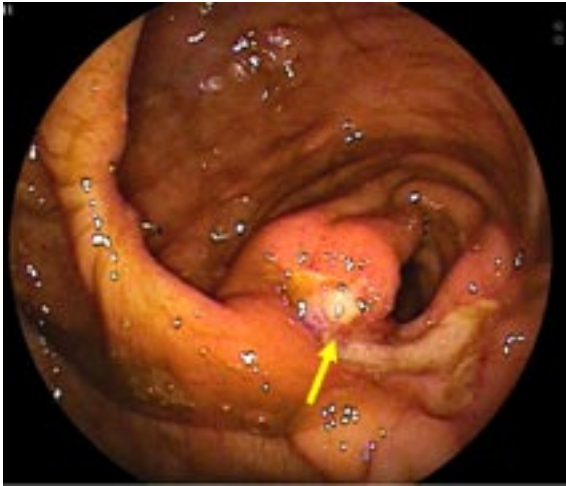
■ Figure 3: Crypt distortion and crypt atrophy



■ Figure 4: Fissure ulcers with transmural colitis

Post operation, the patient was treated with corticosteroids and azathioprine. A follow-up colonoscopy at 4 months later revealed a large

anastomotic ulcer with normal mucosa at the neo-terminal ileum (Figure 5-6).



■ Figure 5-6: A large clean based ulcer along ileocolonic anastomosis with normal neo-terminal ileum

Diagnosis:

Post-operative recurrent Crohn's disease with anastomotic ulcer

Discussion:

About 80% of patients with Crohn's disease required surgery at some stage of the disease. Post-operative recurrence was almost unavoidable in the absence of treatment. The rate of recurrence is about 65-90% within 12 months, and 80- 100% within 3 years after the operation¹. An ileocolonoscopy is the gold standard for diagnosis of post-operative recurrence; therefore, it should be performed within the first year after surgery. Prophylactic treatment is recommended after small intestinal resection. A meta-analysis of the four controlled trials has shown that thiopurines were more effective than mesalazine or imidazole for

preventing both clinical and endoscopic recurrence at 1-2 years². Prophylaxis is recommended to be started within two weeks after surgery, and should be continued for at least 2 years.

References

1. Van Assche G, Dignass A, Reinisch W, et al. The second European evidence-based Consensus on the diagnosis and management of Crohn's disease: Special situations. *J Crohns Colitis* 2010;4:63-101.
2. Peyrin-Biroulet L, Deltenre P, Ardizzone S, et al. Azathioprine and 6-mercaptopurine for the prevention of postoperative recurrence in Crohn's disease: a meta-analysis. *Am J Gastroenterol* 2009;104:2089-96.



Case 10

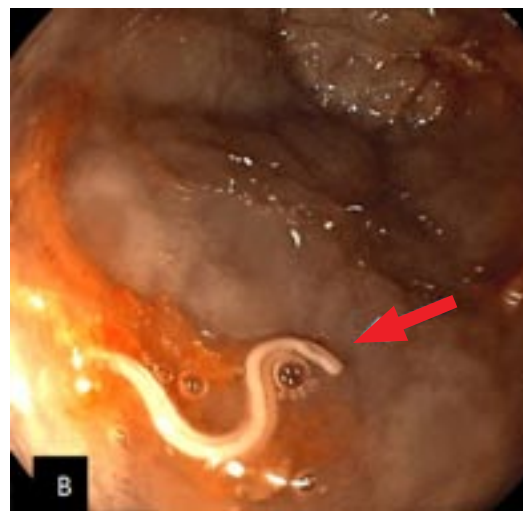
Pornphan Thienchanachaiya, M.D.

Phonthep Angsuwatcharakon, M.D.

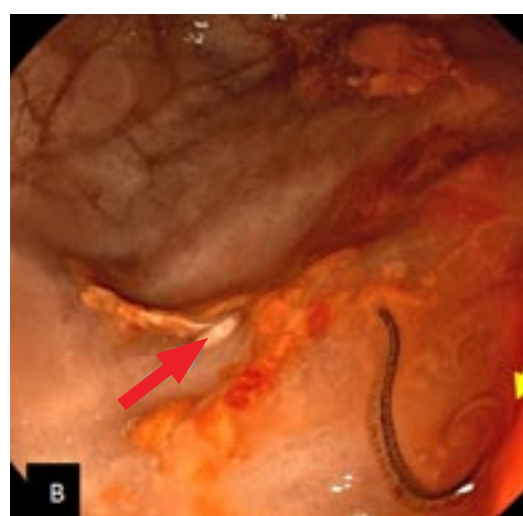
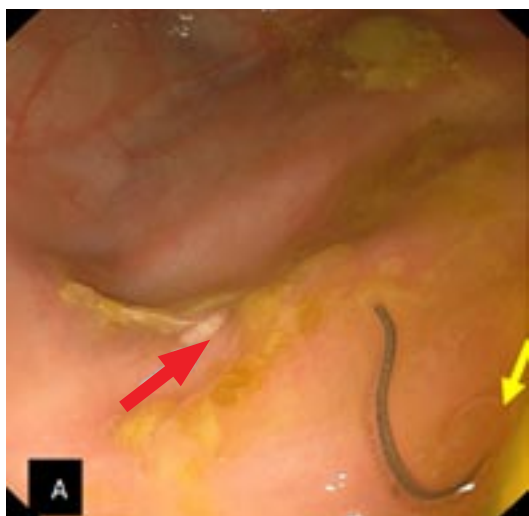
Rungsun Rerknimitr, M.D.

A 19-year-old woman presented with chronic watery diarrhea and significant weight lost for 6 months. She had been in excellent health until 6 months ago. Stool ova and parasite examinations revealed no

pathogen. Colonoscopy was performed and showed multiple small whitish mobile thin worms with threadlike anterior half, coiled and straight posterior end and penetrating mucosa of cecum (Figure 1-6).



■ Figure 1-2: A whitish whipworm with obtuse posterior end in cecum (red arrow); was recognized to be female worm under white light mode (A) and FICE station 2 (B).



■ Figure 3-4: Two whipworms, one at red arrow showed straight posterior end, to be female whipworm and another one at yellow arrow showed coiled posterior end, to be male whipworm under white light mode (A) and under FICE station 2 (B).



■ Figure 5-6: Regularly beaded round cells (stichocytes) form the stichosome (red arrow) in male whipworm under white light mode (A) and under FICE station 2 (B).

Diagnosis:

Trichuris trichiura infestation

Discussion:

Trichuris trichiura (whipworm) infestation is an endemic in tropical and temperate countries, including Southeast Asia. Most patients are asymptomatic, especially if less than 10 worms or if only males are present, whereas, infestation with larger numbers of worms may cause abdominal pain, diarrhea, weight loss, and anemia¹. Heavy colonic infection causes syndrome named *Trichuris* dysentery syndrome. Those patients mainly children presented with mucoid diarrhea, rectal bleeding, rectal prolapsed, iron deficiency anemia and clubbing of fingers².

Whipworm is transmitted by feco-oral route and inhabits the human cecum and proximal large bowel. The adult worms have a thin, tapered anterior region. The female worm is 30 to 50 mm in length, has an uncoiled posterior extremity and lays 3,000 to 20,000 eggs per day. The male is slightly smaller, and has a coiled caudal extremity with a copulatory spicule². At the esophageal part of whipworms, there are stichocytes made of number of stichosomes and stichocytes exhibit exocrine granules that contain a variety of excretory and secretory products that may alter host cell physiology to allow the worm to establish parasitism in the host³.

Diagnosis of trichuriasis is by the demonstration of brown, barrel-shaped ova in feces. However, in some patients, stool examination could not show ova while colonoscopy could demonstrate whipworm infestation^{1,4}. Colonoscopy usually demonstrates the mobile whitish worm; 30-50 mm in length with threadlike anterior end, which penetration in the mucosa. The worms are most common found in cecum. Surrounding colonic mucosa usually appeared edematous and erythematous but ulceration was not common¹.

References

1. Chandra B, Long JD. Diagnosis of *Trichuris trichiura* (whipworm) by colonoscopic extraction. *J Clin Gastroenterol* 1998;27:152-3.
2. Khuroo MS, Khuroo NS. *Trichuris* dysentery syndrome: a common cause of chronic iron deficiency anemia in adults in an endemic area (with videos). *Gastrointest Endosc* 2010;71:200-4.
3. Taguchi H, Yamamoto H, Miyata T, et al. In vivo diagnosis of whipworm (*Trichuris trichiura*) with high-definition magnifying colonoscope (with video). *Gastrointest Endosc* 2008;68:376-7; discussion 7.
4. Chang CW, Chang WH, Shih SC, et al. Accidental diagnosis of *Trichuris trichiura* by colonoscopy. *Gastrointest Endosc* 2008;68:154.

Case 11

Kanita Chattrasophon, M.D.

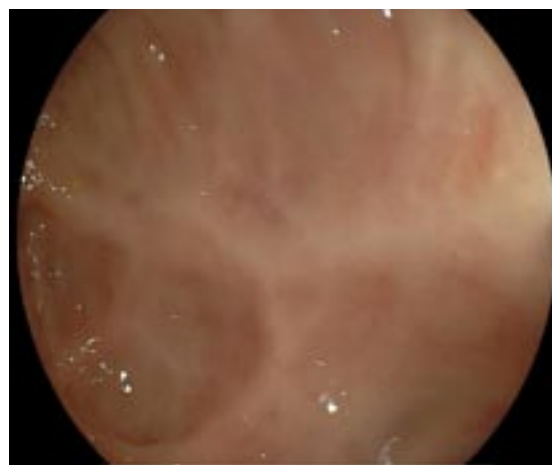
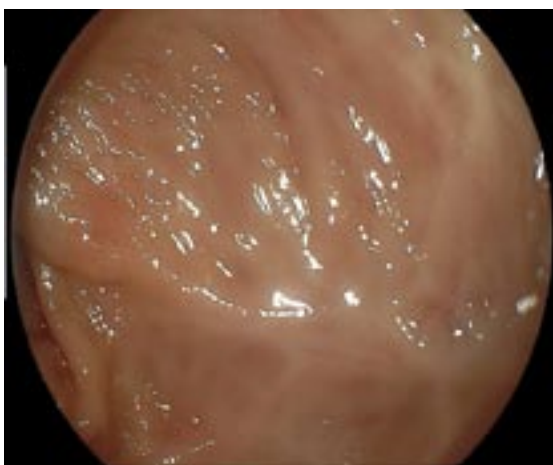
Satimai Aniwon, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

Rungsun Rerknimitr, M.D.

A 65-year-old woman presented with chronic mucous bloody diarrhea. She had been diagnosed as extensive ulcerative colitis. She was treated with corticosteroids as an induction to a remission. Azathioprine has been given as a maintenance therapy.

Follow-up colonoscopy showed mucosal atrophy, fibrotic scar, and the loss of normal haustral folds, resulting in shortening of the colon and a decrease in luminal diameter (Figure 1-2).



■ Figure 1-2: Mucosal atrophy, fibrotic scar, and the loss of normal haustral folds

Diagnosis:

Chronic inflammation in long standing ulcerative colitis causing a burn out colitis

Discussion:

According to the international organization of IBD, the definition of mucosal healing was absence of friability, blood, erosions, and ulcers in all segment of gut mucosa or disappearance of normal vascular pattern. Remission could be successful in several drugs such as 5-aminosalicylates, steroids, and infliximab¹. Data from

many studies suggested that mucosal healing associated with the better outcomes and predicted the possibility of a long-term remission in ulcerative colitis², especially decreasing the risk of relapse. Forty percent of patients who achieved mucosal healing with oral and rectal steroids did not relapse during 1 year of follow-up compared to 18% of those who did not achieve mucosal healing³. Moreover, in a large population-based study reported that UC patients who achieved mucosal healing at 1 year had a lower risk of colectomy at 5 years⁴.

References

1. Lichtenstein GR, Rutgeerts P. Importance of mucosal healing in ulcerative colitis. *Inflamm Bowel Dis* 2010;16:338-46.
2. Rutgeerts P, Vermeire S, Van Assche G, et al. Mucosal healing in inflammatory bowel disease: impossible ideal or therapeutic target? *Gut* 2007;56:453-5.
3. Wright R, Truelove SR. Serial rectal biopsy in ulcerative colitis during the course of a controlled therapeutic trial of various diets. *Am J Dig Dis* 1966;11:847-57.
4. Froslic KF, Jahnsen J, Moum BA, et al. Mucosal healing in inflammatory bowel disease: results from a Norwegian population-based cohort. *Gastroenterology* 2007;133:412-22.



Case 12

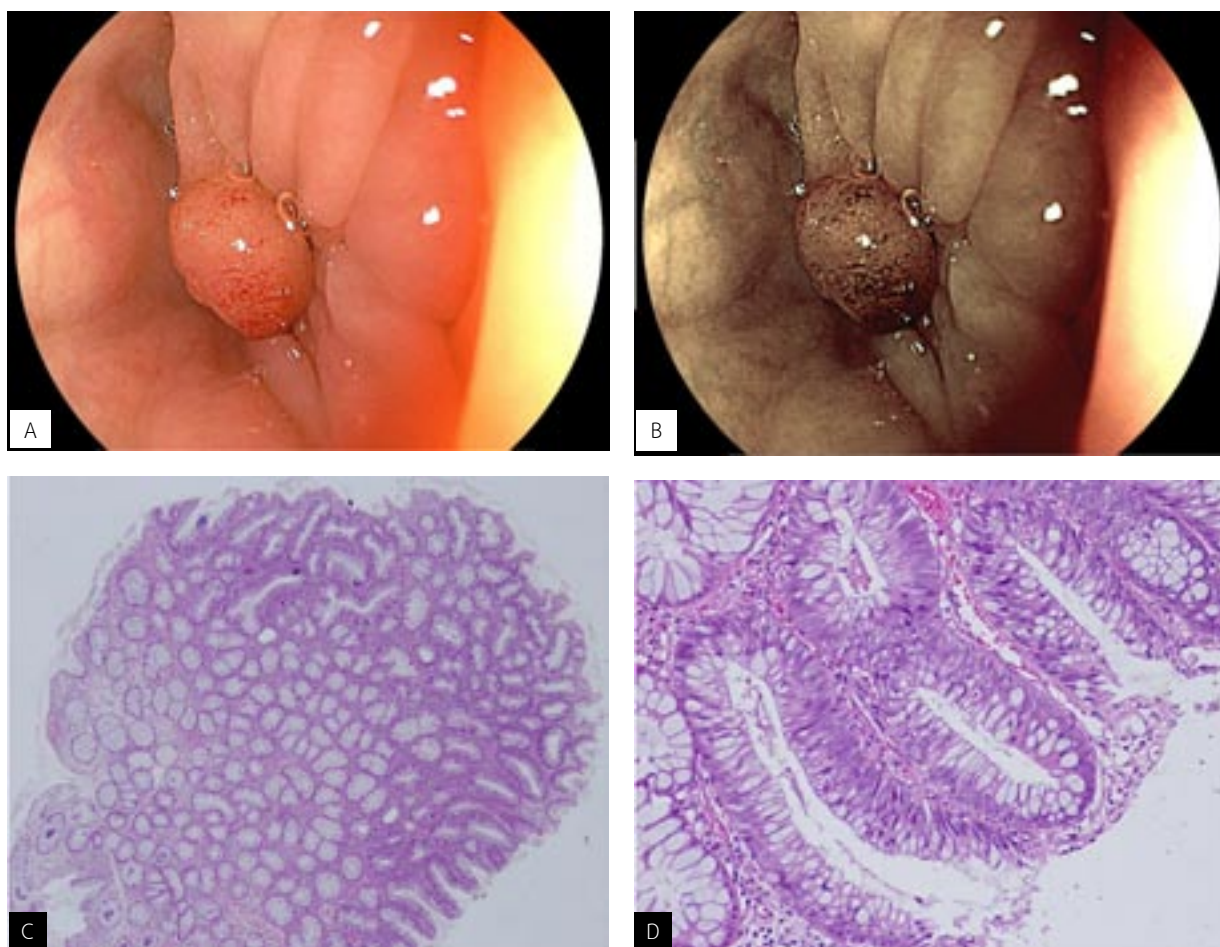
Tanassanee Soontornmanokul, M.D.

Satimai Aniwai, M.D.

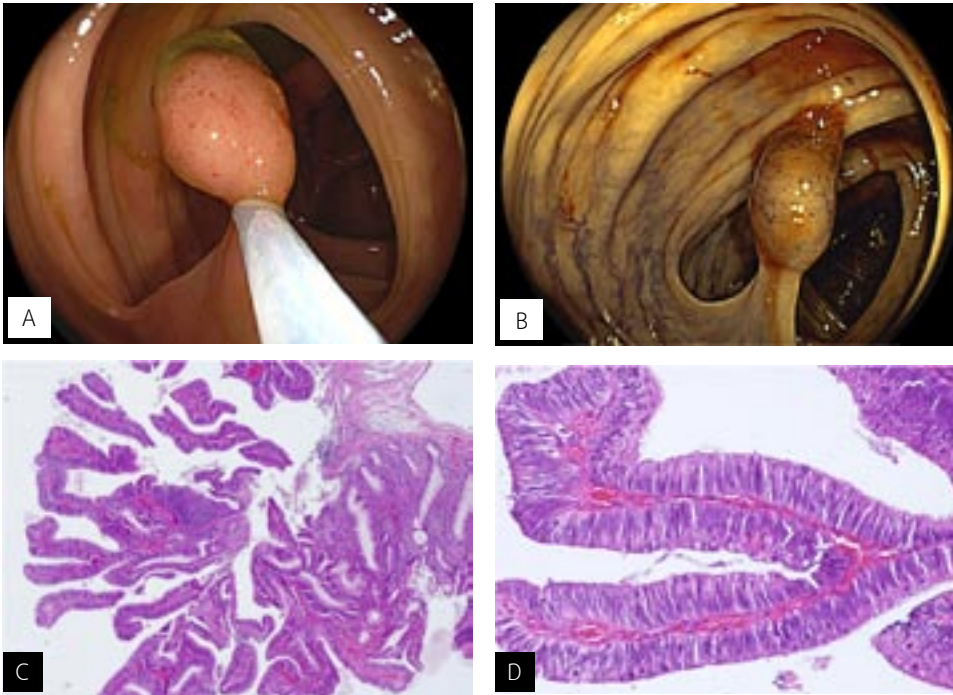
Rungsun Rerknimitr, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

Asymptomatic 55-year-old man underwent colonoscopy for colorectal cancer screening (Figure 1-2).



■ Figure 1: White light image (A) and FICE station 0 (B) revealed one sessile colonic polyp at sigmoid colon. With FICE, a large tubular pits pattern (Kudo classification III L) and meshed capillary vessels surrounded mucosal glands (Sano classification II) were displayed. Histopathological examination showed tubular adenoma with low grade dysplasia. (C, D)



■ Figure 2: White light image (A) and FICE station 4 (B) revealed one pedunculated colonic polyp at transverse colon. With FICE, a branch-like pits pattern (Kudo classification IV) and meshed capillary vessels surrounded mucosal glands (Sano classification II). Histological examination shows papillary growth pattern of colonic crypt, compatible with tubulovillous adenoma.

Diagnosis:

Colonic tubular adenoma and tubulovillous adenoma

Discussion:

Most of colon cancers originate within previously benign adenomas. Endoscopic discrimination of small adenomas from non-neoplastic polyps is essential, because polypectomy of adenomas can reduce the risk of subsequent colorectal cancer. However, the diagnostic accuracy of conventional endoscopy for colonic polyps that smaller than 5 mm in size was not satisfactory¹. FICE (Flexible Spectral Imaging Color enhancement) developed by Fujifilm Corporation, Tokyo, Japan reported as a new non-dye diagnostic tool for the differentiation of neoplastic polyp from non-neoplastic one^{1, 2}. Yoshida, et al. showed that FICE magnification correlated well with the histopathological diagnoses. When compared the FICE reading results with Narrow Band imaging (NBI) reading results, they found that the sensitivity and specificity of the two methods

were comparable (77.7 vs. 63.6% and 100% vs. 99%, respectively)³. Therefore FICE magnification for colorectal tumors can be used as a diagnostic tool to predict histology and it is useful to avoid an unnecessary polypectomy.

References

1. Togashi K, Osawa H, Koinuma K, et al. A comparison of conventional endoscopy, chromoendoscopy, and the optimal-band imaging system for the differentiation of neoplastic and non-neoplastic colonic polyps. *Gastrointest Endosc* 2009;69 (3 Pt 2):734-41.
2. Yoshida N, Naito Y, Inada Y, et al. The detection of surface patterns by flexible spectral imaging color enhancement without magnification for diagnosis of colorectal polyps. *Int J Colorectal Dis* 2012;27:605-11.
3. Yoshida N, Naito Y, Kugai M, et al. Efficacy of magnifying endoscopy with flexible spectral imaging color enhancement in the diagnosis of colorectal tumors. *J Gastroenterol* 2011;46:65-72.

Case 13

Suparat Khemnark, M.D.

Rungsun Rerknimitr, M.D.

A 60-year-old man underwent colonoscopy for colorectal cancer screening. Colonoscopy found a lateral spreading tumor (2 cm in diameter). Biopsy revealed

tubular adenoma with focal high grade dysplasia (Figure 1-3). Then colonoscopy with endoscopic mucosal resection (EMR) was performed (Figure 4).



■ Figure 1-3: Lateral Spreading Tumor with FICE demonstrate type IIIIL pit pattern with mesh capillary Sano pattern II



■ Figure 4: Endoscopic mucosal resection technique (A) Initial injection of a saline solution into the submucosal layer to lift the lesion. (B) Snare resection was performed to resect the entire lesion (C) Post EMR

Diagnosis:

EMR (Endoscopic Mucosal Resection) in one piece for a 2 cm lateral spreading tumor (non-granular type)

Discussion:

Lateral Spreading Tumors (LSTs) of the colorectum are defined as lesions greater than 10 mm in

diameter with a low vertical axis that typically extend laterally and circumferentially rather than vertically along the colonic wall laterally along the luminal wall¹. The frequency of invasive carcinoma is lower than that of polypoid lesions of similar size.

These lesions can be subdivided into two subtypes based on endoscopic macroscopic findings:

LST-G (granular) type with even or uneven nodules on the surface and LST-NG (non-granular) type with a smooth surface. The previously reported that LST-G with large nodules or depressions tend to invade the submucosal layer², recently a trial reported that LST-NG type has a higher potential for malignancy than LST-G type³.

LSTs are usually removed by endoscopic mucosal resection (EMR) but larger tumors may require piecemeal resection². However, LSTs with deep submucosal invasion should not be treated by EMR because of the high risk of lymph node metastasis.

References

1. Kudo S. Endoscopic mucosal resection of flat and depressed types of colorectal cancer. *Endoscopy* 1993;25:455-61.
2. Saito Y, Fujii T, Kondo H. Endoscopic treatment for laterally spreading tumors in the colon. *Endoscopy* 2001;33:682-6.
3. Uraoka T, Saito Y, Matsuda T. Endoscopic indications for endoscopic mucosal resection of laterally spreading tumours in the colorectum. *Gut* 2006;55:1592-7.



Case 14

Kriangsak Charoensuk, M.D.

Phonthep Angsuwatcharakon, M.D.

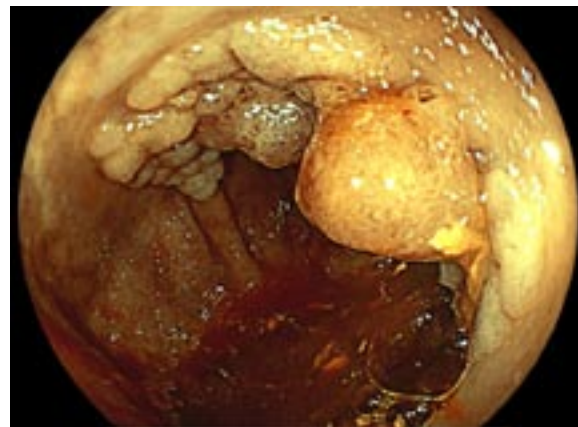
Rungsun Rerknimitr, M.D.

A 65-year-old man has been diagnosed adenocarcinoma of sigmoid colon and he underwent sigmoidectomy. A colonoscopy for colorectal cancer surveillance was performed 6 months later. Colonoscopy showed a granular lateral spreading lesion

(Paris classification 0-IIa) at cecum, 5 cm in diameter (Figure 1-2). “Underwater” endoscopic mucosal resection (UEMR) without submucosal injection was performed (Figure 3-4). Pathological finding revealed tubular adenoma with focal high grade dysplasia.



■ Figure 1: A granular lateral spreading tumor (LST) (Paris classification 0-IIa)



■ Figure 2: Pit pattern was Kudo's classification IIIIL and vascular pattern was Sano's classification II. (FICE station 1)



■ Figure 3: Underwater EMR with snare without submucosal saline injection



■ Figure 4: After underwater EMR

Diagnosis:

“Underwater” endoscopic mucosal resection (UEMR) for granular lateral spreading tubular adenoma with focal high grade dysplasia

Discussion:

Endoscopic mucosal resection (EMR) is a well-established method for resecting sessile colon polyps. Submucosal injection has been considered an integral part of the EMR technique¹. Now there has been a report about a newly developed method of water immersion (UEMR) that eliminates submucosal injection. UEMR enables complete removal of large sessile colorectal polyps without submucosal injection. A Prospective, observational study in 60 consecutive patients referred for resection of large sessile colorectal polyps were treated with UEMR². Complete resection was successful in all patients without early complication. There was no perforation or postpolypectomy syndrome. Delayed

bleeding occurred in 3 patients and was managed conservatively. The technique was safe in a large patient cohort, and the early recurrence rate appears low. Use of a water interface for UEMR has potential advantages that deserve further study^{2,3}.

References

1. Wood NJ. Endoscopy: no need for submucosal injection with ‘underwater’ EMR of large sessile colorectal polyps. *Nat Rev Gastroenterol Hepatol* 2012;9:188.
2. Binmoeller KF, Weilert F, Shah J, Bhat Y, Kane S. “Underwater” EMR without submucosal injection for large sessile colorectal polyps (with video). *Gastrointest Endosc* 2012;75:1086-91.
3. Othman MO, Wallace MB. Endoscopic mucosal resection (EMR) and endoscopic submucosal dissection (ESD) in 2011, a Western perspective. *Clin Res Hepatol Gastroenterol* 2011;35:288-94.



Case 15

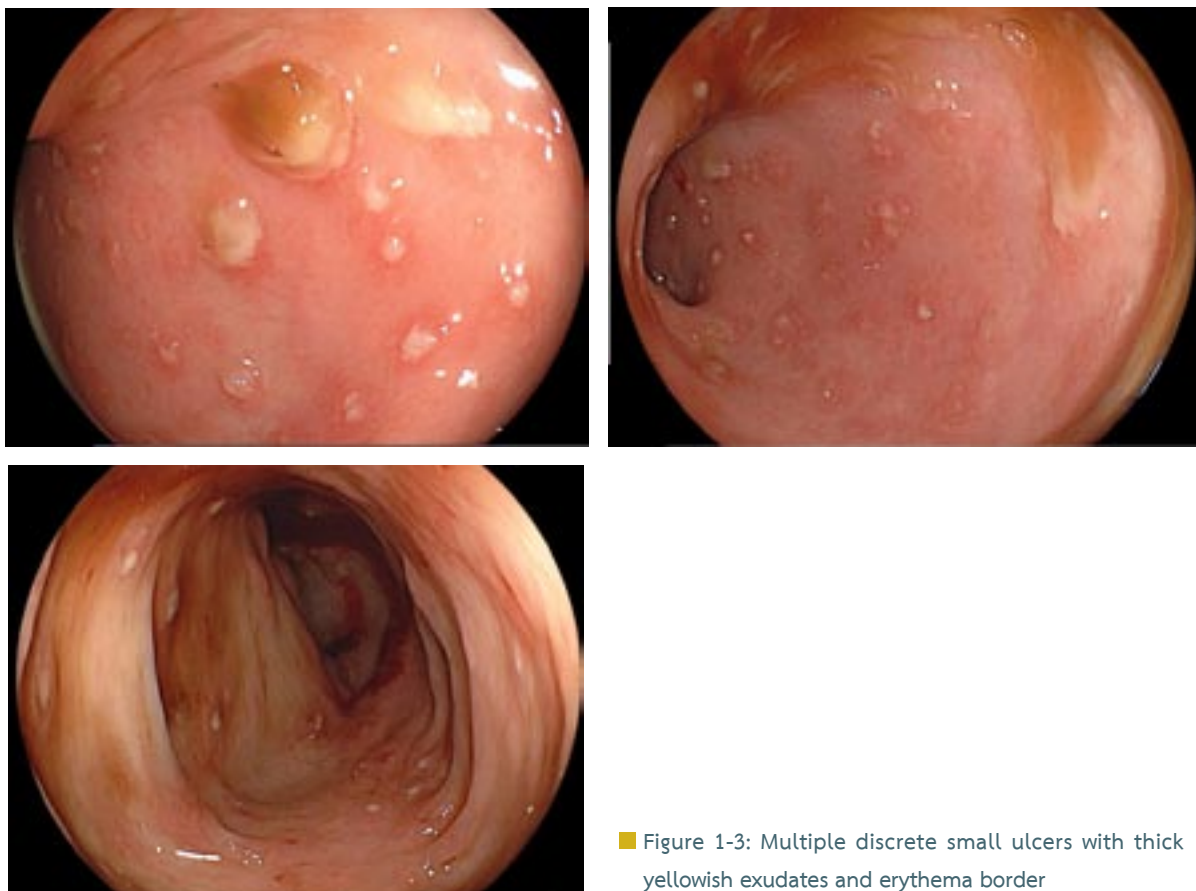
Nuttaporn Norrasetwanich, M.D.

Satimai Aniwon, M.D.

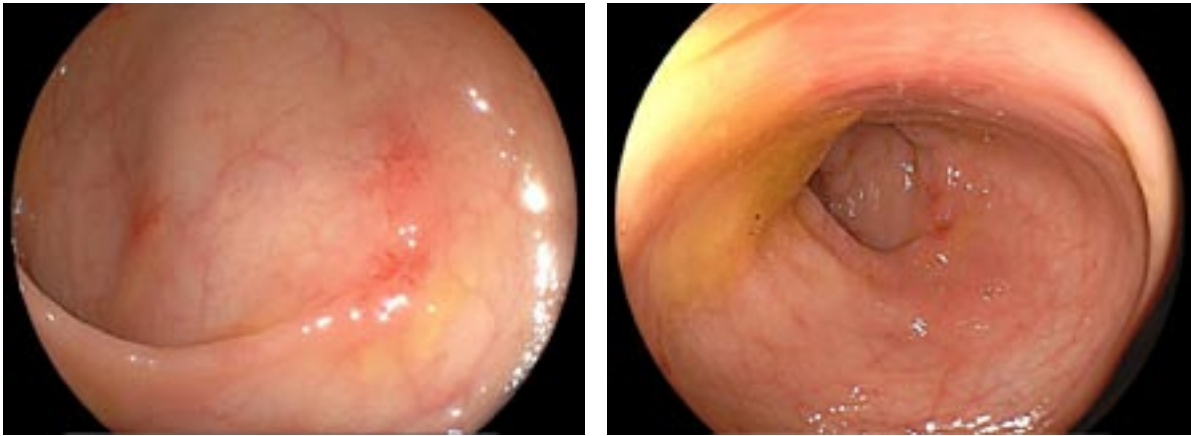
Rungsun Rerknimitr, M.D.

A 66-year-old man with no previous medical illness presented with a 3-day history of bloody diarrhea and lower abdominal pain. One day prior to admission, he developed rectal bleeding. His stool examination demonstrated *Entameba histolytica* cysts. Sigmoidoscopy showed multiple discrete small ulcers with thick yellowish exudates and erythema rim. There was normal intervening mucosa along the rectosigmoid

colon (Figure 1-3). Colonic biopsy revealed multiple foci of erosion with acute and chronic inflammatory infiltration in the lamina propria. No organism was seen in the submitted tissue. He was treated with 10-day metronidazole. Complete colonoscopy was performed 2 weeks later and it showed a markedly improvement of the lesions (Figure 4-5).



■ Figure 1-3: Multiple discrete small ulcers with thick yellowish exudates and erythema border



■ Figure 4-5: Follow-up colonoscopy

Diagnosis:

Amebic colitis

Discussion:

E. histolytica can infect people of both genders and all ages; however, populations at risk may vary with geographic location, host susceptibility, and differences in organism virulence. The simple life cycle of *E. histolytica* begins when infectious cysts are ingested in fecally contaminated food or water. After ingestion and passage through the stomach, the organism excysts and emerges in the large intestine as an active trophozoite. Trophozoites multiply by simple division and encyst as they move further down the large bowel. Cysts are then expelled with the feces and may remain viable in a moist environment for weeks to months¹.

Amebic colitis may occur days to years after initial infection and is characterized classically by abdominal pain and bloody diarrhea. Watery or mucus-containing diarrhea, constipation, and tenesmus may also occur. Complications of intestinal disease include stricture, rectovaginal fistulas, formation of an annular intraluminal mass (ameboma), bowel obstruction, perianal skin ulceration, toxic megacolon, perforation, peritonitis, shock, and death¹.

Colonoscopy is useful for the diagnosis of amebic colitis but is not required if stool antigen detection or PCR is positive. Amebic colitis can appear as punctuate hemorrhagic areas or small ulcers (up to centimeters in diameter) with exudative centers and hyperemic borders. The cecum and ascending colon are affected most commonly, although in severe disease the entire colon may be involved. In addition, early in the infection process, endoscopy results may be entirely normal. As disease progression occurs, mucosa may become hyperemic due to inflammatory changes, and pseudomembranes can occur, resembling inflammatory bowel disease. Aspirates content from colonic ulcers should be examined immediately microscopically for motile trophozoites².

References

1. Pritt BS, Clark CG. Amebiasis. *Mayo Clin Proc* 2008;83:1154-9.
2. Bercu TE, Petri WA, Behm JW. Amebic colitis: new insights into pathogenesis and treatment. *Curr Gastroenterol Rep* 2007;9:429-33.

Case 16

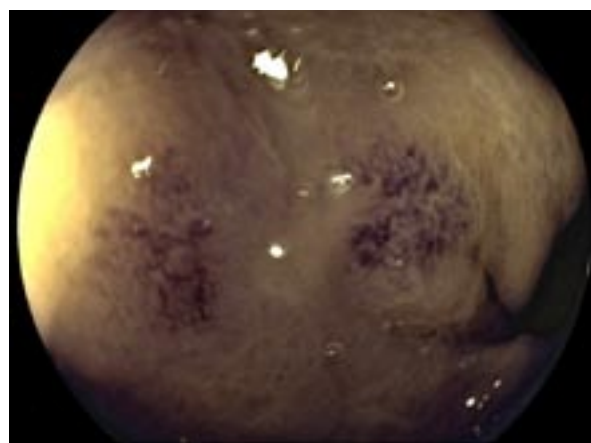
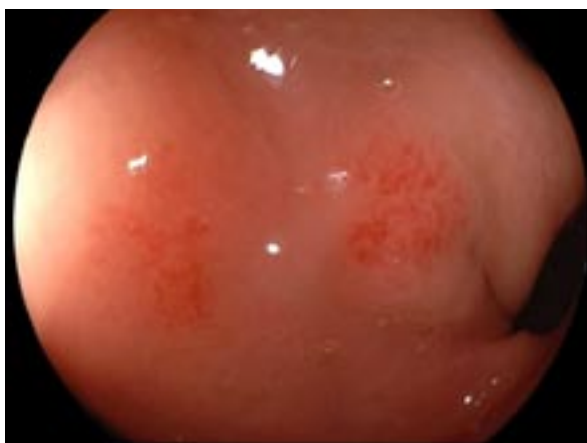
Nuttaporn Norrasetwanich, M.D.

Satimai Aniwon, M.D.

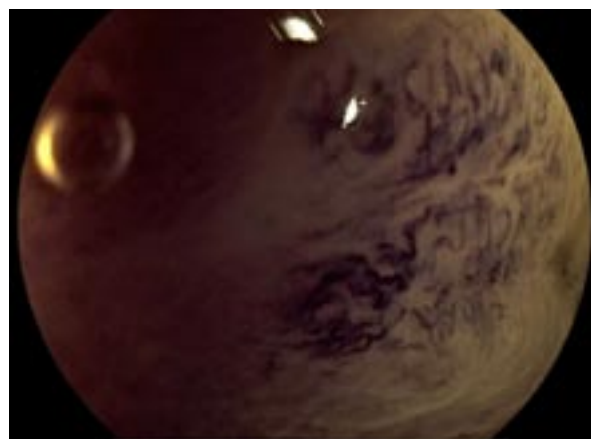
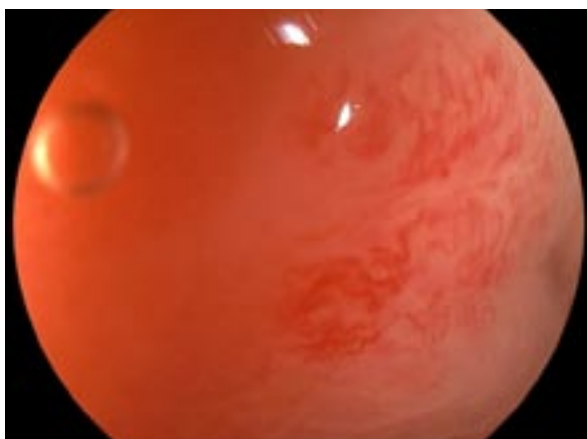
Rungsun Rerknimitr, M.D.

A 66-year-old woman presented with intermittent bloody stool passage for 3 days. She had no abdominal pain. She had been diagnosed with cervical cancer 1 year previously and treated with pelvic

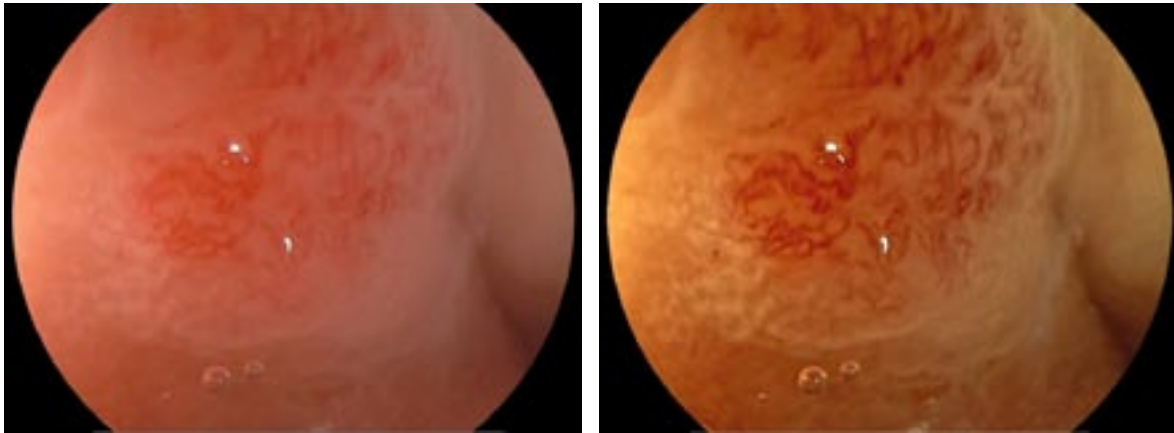
radiotherapy. Colonoscopy revealed multiple twisted submucosal telangiectasia at rectum approximately 10 cm from the anal verge (Figure 1-6).



■ Figure 1-2: Demonstrated neo-vascularized pattern under white light and FICE station 4



■ Figure 3-4: A closer look of neo-vascularization under white light and FICE station 4



■ Figure 5-6: A 100 times magnifications under white light and FICE station 6

Diagnosis:

Radiation proctitis

Discussion:

Irradiation of the pelvic floor due to cervical or prostate cancer leads in about 5–20% of patients to the development of post-radiation rectal telangiectasias. These start to occur and bleed usually several months after radiation injury; in some patients bleeding starts as late as 2 years following irradiation¹. Radiation-induced mucosal damage results in endothelial dysfunction, microvascular injury with intimal fibrosis, and fibrin thrombi of small arteries and arterioles leading to ischemia, fibrosis and the development of neovascular lesions. The lesions can be better depicted under FICE in this patient.

Chronic radiation proctitis resolves spontaneously in many cases, but in some can lead to persistent rectal bleeding and iron deficiency anemia requiring blood transfusion. Treatment remains

unsatisfactory. Medical measures, including formalin application, topical sucralfate, 5-amino salicylic acid enemas, short chain fatty acids, and antioxidants such as vitamin E and pentoxifylline have been used with limited success. Surgical management is associated with high morbidity and mortality². The currently preferred endoscopic method is argon plasma coagulation which provides controlled, superficial, non-contact coagulation of all existing abnormal vessels. It is crucial to apply coagulation very carefully in order to avoid the creation of deeper ulceration in a fragile ischemic mucosa¹.

References

1. Regula J, Wronska E, Pachlewski J. Vascular lesions of the gastrointestinal tract. *Best Pract Res Clin Gastroenterol* 2008;22:313-28.
2. Rustagi T, Mashimo H. Endoscopic management of chronic radiation proctitis. *World J Gastroenterol* 2011;17:4554-62.



Case 17

Nuttaporn Norrasetwanich, M.D.

Satimai Aniwan, M.D.

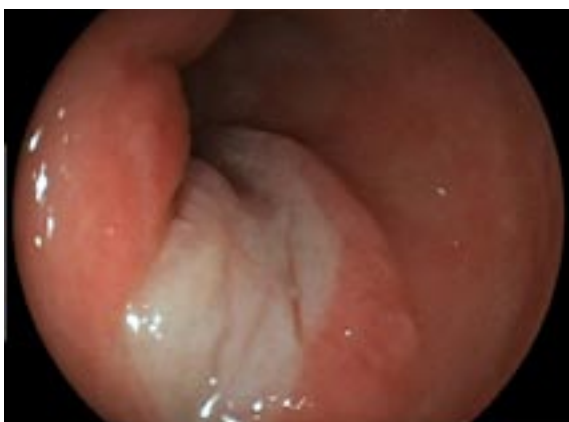
Rungsun Rerknimitr, M.D.

A 20-year-old woman presented with chronic constipation, including excessive straining, and a sense of incomplete evacuation for 4 years. She also had intermittent bloody output per rectum. Colonoscopy revealed a hemi-circumferential anterior wall rectal ulcer size 1.5 cm in diameter at 5 cm from anal verge (Figure

1-4). Biopsy showed benign colonic mucosa with acute organizing ulcer. The colonic glands among the inflammation were distorted and deformed with focal cystic dilatation. Stromal fibrosis was noted. Solitary rectal ulcer was most likely.



■ Figure 1-2: White light endoscopy revealed a shallow ulcer at the anterior wall of the rectum



■ Figure 3-4: White light and FICE station 1 revealed another ulcer with well demarked border

Diagnosis:

Solitary Rectal Ulcer Syndrome (SRUS)

Discussion:

Solitary rectal ulcer syndrome (SRUS) consists of several different clinical pathologic processes. These processes, however, end in a mutual common pathway that is associated with reduced blood perfusion of the rectal mucosa, leading to local ischemia and ulceration. SRUS was described in the early nineteenth century by the French anatomist J. Cruvillier in his report on chronic rectal ulcer¹.

In fact, the name of SRUS is a misnomer, because only 25% to 30% of patients have a solitary ulcer. Certain patients may have multiple ulcers (30%–40%), hyperemic mucosa (15%–20%), or polypoid lesions². Abid S, et al. reviewed 116 patients diagnosed with SRUS histologically, solitary and multiple lesions were presented in 79 (68%) and 33 (28%) patients respectively; ulcerative lesions in 90 (78%), polypoidal in 29 (25%), erythematous patches in 3 (2.5%) and petechial spots in one patient³. The lesions usually locate on the anterior rectal wall, 4 to 10 cm from the anal verge. Lesions range from 0.5 to 6 cm, although most are 1 to 1.5 cm in diameter. Some ulcers have

rolled edges and may bleed, raising concern for a possible malignancy. It is generally a disorder of young adults (third or fourth decade of life), with an incidence of 1 to 3 in 100,000 persons per year. Women are somewhat more prone to develop SRUS than men. Symptoms are nonspecific; rectal bleeding and the passage of mucus are most commonly reported. Straining at stool, feelings of incomplete evacuation, rectal discomfort, and urgency are common². Despite the diverse causes the microscopic changes are analogous, comprising fibromuscular obliteration and disorientation of the muscularis mucosa¹.

References

1. Edden Y, Shih SS, Wexner SD. Solitary Rectal Ulcer Syndrome and Stercoral Ulcers. *Gastroenterol Clin North Am* 2009;38:541-5.
2. Lacy BE, Weiser K. Common anorectal disorders: diagnosis and treatment. *Curr Gastroenterol Rep* 2009;11:413-9.
3. Abid S, Khawaja A, Bhimani SA, et al. The clinical, endoscopic and histological spectrum of the solitary rectal ulcer syndrome: a single-center experience of 116 cases. *BMC Gastroenterol* 2012;12:72.



Case 18

Nuttaporn Norrasetwanich, M.D.

Rapat Pittayanon, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

Rungsun Rerknimitr, M.D.

A 56-year-old man presented with fever and bloody diarrhea for 2 weeks. He had a history of kidney transplantation 2 months ago. He received immunosuppressive drugs; mycophenolate sodium, tacrolimus and prednisolone 10 mg/d. Colonoscopy was performed. It revealed diffuse subepithelial hemorrhage and multiple shallow ulcers extend from sigmoid to terminal ileum (Figure 1-4). Biopsy showed erosive

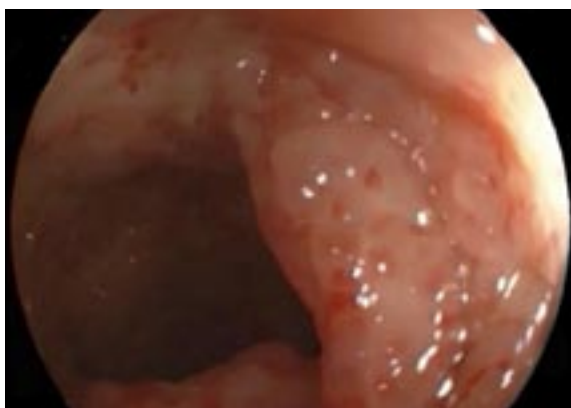
surface and edematous lamina propria. Numerous neutrophils and lymphoplasmacytic cells infiltrated in lamina propria. Many endothelial cells of vessels in lamina propria showed large cells with intranuclear inclusions (Figure 5). The diagnosis was CMV colitis. CMV immunohistological stain was positive in several cells (Figure 6). His serum CMV viral load was 51,800 copies/ml.



■ Figure 1: Diffuse subepithelial hemorrhage of the colon

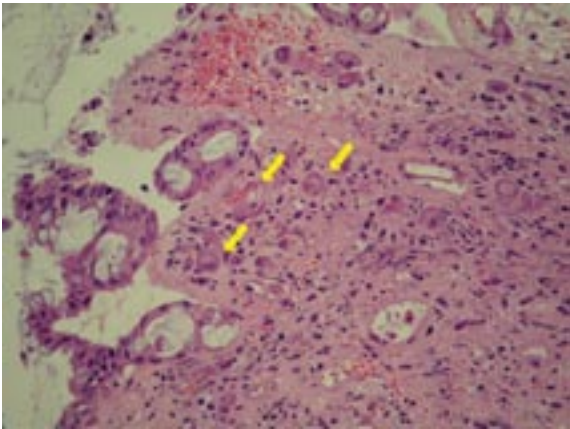


■ Figure 2: Multiple ulcers with edematous rim

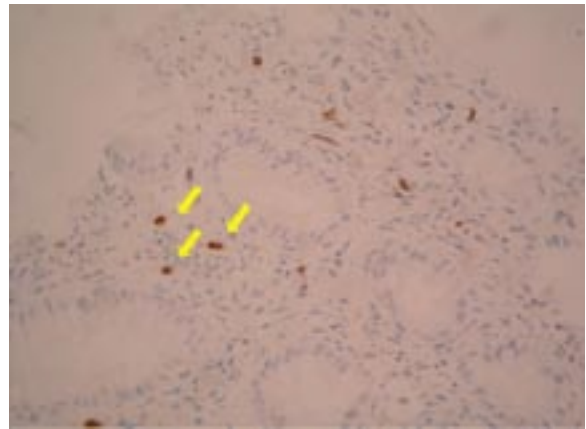


■ Figure 3-4: Severe edematous colonic mucosa with subepithelial hemorrhage





■ Figure 5: CMV infected cell with intranuclear inclusion (yellow arrows)



■ Figure 6: Positive immunohistological stain for CMV (yellow arrows)

Diagnosis:

Cytomegalovirus colitis

Discussion:

CMV remains the single most important pathogen affecting the outcome of solid organ transplantation. CMV has the direct effects of morbidity and mortality related to infection, but also contributes to a multitude of short and long-term indirect effects mediated by its modulation of the immune system. Luminal tract disease is the most common manifestation. Esophagitis and colitis are the most frequently observed luminal syndromes, usually characterized by ulcerative lesions¹.

CMV colitis usually manifests with abdominal pain, persistent small-volume diarrhea, and rectal bleeding. Bloody diarrhea or hematochezia are the most common symptoms in immunocompetent patients with CMV colitis. Although a wide spectrum of findings can

occur, typical endoscopic findings are mild and patchy to include erythematous colonic mucosa with edema and subepithelial hemorrhage. Less commonly endoscopic findings are discrete ulceration surrounded with normal colonic mucosa, colitis with ulceration, and pseudomembrane formation (very rare). The gold standard for diagnosis remains histopathology and immunohistochemical staining for CMV is the best confirmation test².

References

1. Lemonovich TL, Watkins RR. Update on cytomegalovirus infections of the gastrointestinal system in solid organ transplant recipients. *Curr Infect Dis Rep* 2012;14:33-40.
2. You DM, Johnson MD. Cytomegalovirus Infection and the Gastrointestinal Tract. *Curr Gastroenterol Rep* 2012;14:334-42.



Case 19

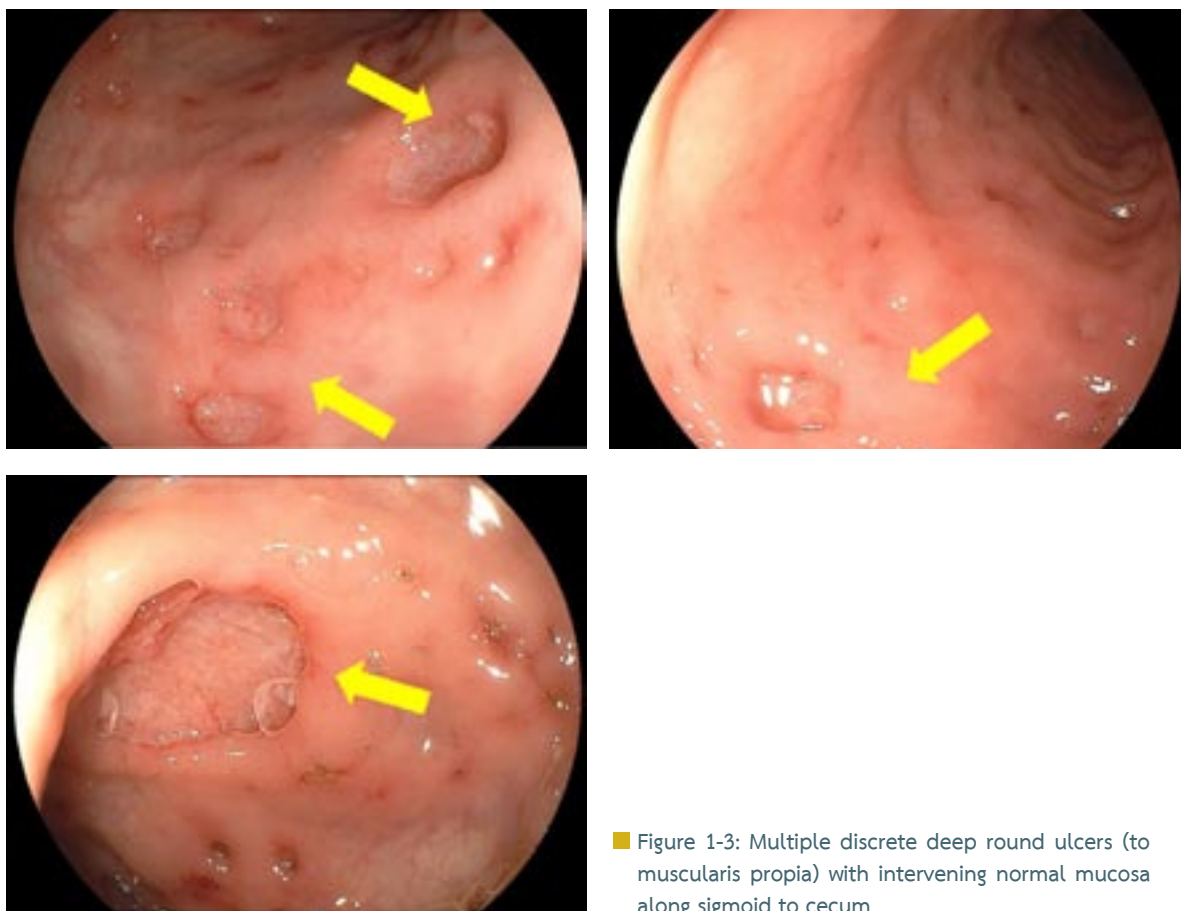
Kriangsak Charoensuk, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

Rungsun Rerknimitr, M.D.

A 45-year-old woman presented with chronic mucous bloody diarrhea, fever and weight loss. She had been previous healthy and no history of NSAIDs use. Physical examination revealed oral ulcers but no genital ulcer. Pathergy test was negative. Colonoscopy showed multiple discrete deep round ulcers with normal

intervening mucosa from sigmoid to cecum (Figure 1-3). Pathological biopsy revealed moderate acute colitis, intact crypt architecture. There were numerous lymphocytes and large number of neutrophilic infiltration. No organism, granuloma nor intranuclear inclusion body seen in lamina propria.



■ Figure 1-3: Multiple discrete deep round ulcers (to muscularis propria) with intervening normal mucosa along sigmoid to cecum

Diagnosis:

Intestinal Behçet's disease

Discussion:

Behçet's disease, BD is an inflammatory disorder of unknown etiology, characterized by recurrent oral aphthous ulcers, genital ulcers, uveitis, and skin lesions¹. Pathophysiologic characteristics of BD are vascular injuries, hyperfunction of neutrophils, and autoimmune responses. Disease occurs along the ancient Silk Road with highest prevalence in Turkey. The onset is typically in the third or fourth decade of life. Involvement of the gastrointestinal tract, central nervous system, and large vessels is less frequent, although it can be life threatening. A prevalence of intestinal BD ranges widely by area from 3 to 16% of all patients with BD². The intestinal lesions of BD occur in two forms: mucosal inflammation and ischemia/infarction. Distinguishing intestinal BD from Crohn's disease or intestinal tuberculosis is difficult, especially in patients with ileocolonic ulcerations, demonstrated by colonoscopic examinations. Lee SK, et al. found that deep round and irregular/geographic-shaped ulcers and focal distributions

are suggestive of Behçet's disease. While the longitudinal ulcers and segmental/diffuse lesions suggest Crohn's disease. Diagnosis of Behçet's disease in the validation set produced sensitivity, specificity, and negative and positive predictive values of 94.3%, 90%, 94.7%, and 89.2%, respectively³. Histopathological finding showed vasculitis of the small veins and venules with inflammatory cells including lymphocytic infiltration.

References

1. Haznedaroglu IC, Ozcebe OI, Dundar SV. Behcet's disease. *N Eng' J Med* 2000;342:588.
2. Cheon JH, Kim ES, Shin SJ, et al. Development and validation of novel diagnostic criteria for intestinal Behcet's disease in Korean patients with ileocolonic ulcers. *Am J Gastroenterology* 2009;104:2492-9.
3. Lee SK, Kim BK, Kim TI, et al. Differential diagnosis of intestinal Behcet's disease and Crohn's disease by colonoscopic findings. *Endoscopy* 2009;41:9-16.



Case 20

Kriangsak Charoensuk, M.D.

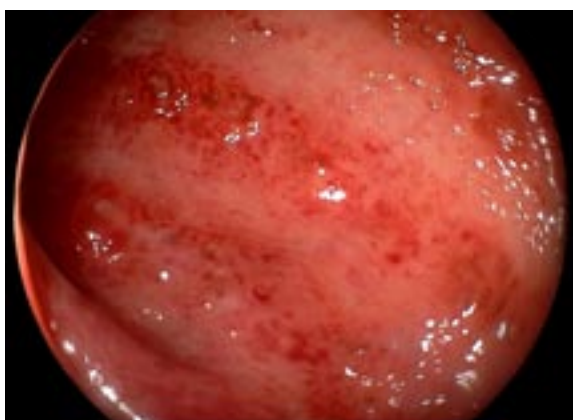
Satimai Aniwan, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

Rungsun Rerknimitr, M.D.

A 46-year-old woman presented with chronic mucous bloody diarrhea. She had been well until a month before, she had mucous bloody diarrhea without fever and weight loss. Colonoscopy found continuous granular and friable mucosa from lower rectum to

descending colon (Figure 1-4). There was some edematous and mild erythema of the mucosal folds at transverse colon. Biopsy showed lymphocyte and plasma cells infiltration at lamina propria with focal cryptitis and crypt abscess compatible with ulcerative colitis.



■ Figure 1-2: Granular and friable mucosa at rectum, sigmoid and descending colon



■ Figure 3: Edematous and mild erythema of mucosa at transverse colon



■ Figure 4: Normal colonic mucosa at ascending colon

Diagnosis:

Ulcerative colitis; granular pattern

Discussion:

Ulcerative colitis (UC) is a chronic disease characterized by diffuse mucosal inflammation limited to the colon. It involves the rectum in about 95% of cases and may extend proximally in a symmetrical, circumferential, and uninterrupted pattern to involve parts or all of the large intestine^{1,2}. The diagnosis of UC is suspected on clinical grounds and supported by the appropriate findings on proctosigmoidoscopy or colonoscopy, biopsy, and by negative stool examination for infectious causes. Endoscopic finding in ulcerative colitis (UC) typically reveals the following: erythema, edema/loss of the usual fine vascular pattern, granularity of the mucosa, friability/spontaneous bleeding,

pseudopolyps, erosions and ulcers³. The granular appearance is manifested by changes in light reflection during colonoscopy. Instead of reflecting light in large patches, the granular mucosa reflects a multitude of small points of light, giving the appearance of “wet sandpaper”^{2,3}.

References

1. Kornbluth A, Sachar DB. Ulcerative colitis practice guidelines in adults: American College Of Gastroenterology, Practice Parameters Committee. *Am J Gastroenterol* 2010;105:501-23.
2. Danese S, Fiocchi C. Ulcerative colitis. *N Engl J Med* 2011;365:1713-25.
3. Waye JD. The role of colonoscopy in the differential diagnosis of inflammatory bowel disease. *Gastrointest Endosc* 1977;23:150-4.



Case 21

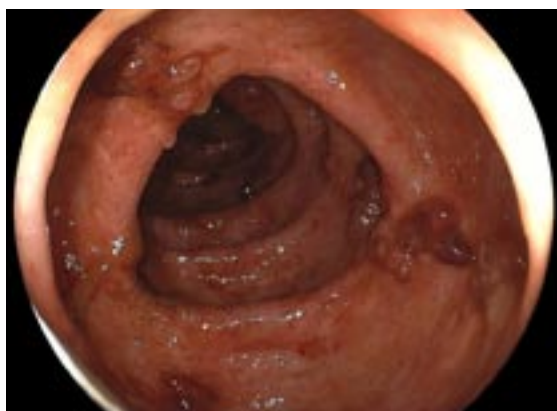
Kriangsak Charoensuk, M.D.

Satimai Aniwon, M.D.

Rungsun Rerknimitr, M.D.

A 33-year-old man presented with bleeding per rectum. He was diagnosed with ulcerative colitis for 3 years. He was in remission with azathioprine. Two weeks earlier, he developed bleeding per rectum and

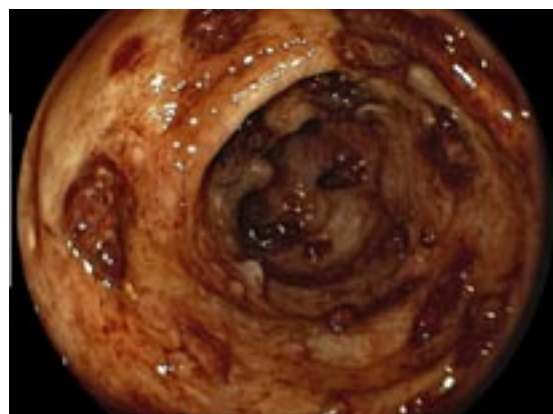
tenesmus. Colonoscopy showed multiple inflamed pseudopolyps along colon (Figure 1-4). Biopsy did not reveal granuloma. Inclusion body and acid fast bacilli were negative.



■ Figure 1-2: Multiple inflammatory pseudopolyps



■ Figure 3: White light image of inflammatory pseudopolyps



■ Figure 4: FICE image station 6 showed distinct pseudopolyps

Diagnosis:

Ulcerative colitis with inflammatory pseudopolyps

Discussion:

Ulcerative colitis, UC is characterized by recurrent episodes of inflammation limited to the mucosal layer of the colon. It almost invariably involves the rectum and may extend in a proximal and continuous involve other portions of the colon¹. There are different endoscopic findings in UC, included pseudopolyps. Pseudopolyps are not specific for UC but

are more common in UC, occurring in approximately 20 percent of cases. They have varying in size and be associated with increased severity and more extensive involvement in UC².

References

1. Danese S, Fiocchi C. Ulcerative colitis. *N Engl J Med* 2011;365:1713-25.
2. Jalan KN, Walker RJ, Sircus W, et al. Pseudopolyposis in ulcerative colitis. *Lancet* 1969;2:555-9.



Case 22

Kessarín Thanapirom, M.D.

Rapat Pittayanon, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

Linda Pantongrag-Brown, M.D.

Rungsun Rerknimitr, M.D.

A 55-year-old man presented with significant weight loss. Computed tomography of the abdomen showed a 5 cm heterogenous mass with mild enhancement at pelvic cavity near the right external iliac artery (Figure 1-2). Tumor resection was performed. Histological findings revealed neuroendocrine tumor. As

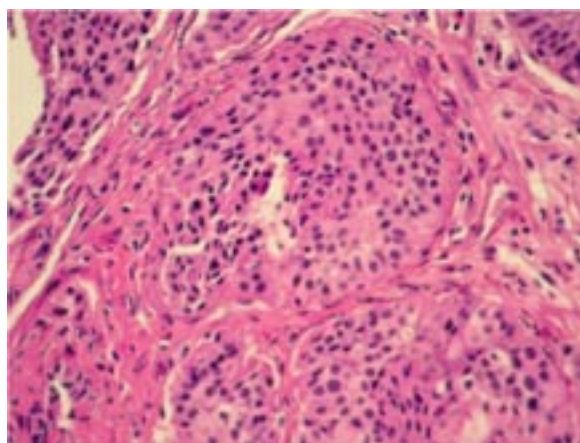
a workup of primary tumor, he underwent colonoscopy. The endoscopic finding showed submucosal mass at lower rectum (Figure 3). Biopsy showed round cell tumor which exhibited nest and sheet pattern. The chromogranin and synaptophysin had been done and stained in the tumor cells (Figure 4).



■ Figure 1-2: Heterogenous enhancing mass at right pelvic cavity near right external iliac artery



■ Figure 3: Submucosal mass at rectum, 2.5 cm in diameter, smooth surface and regular border.



■ Figure 4: Nest and sheet pattern of round cell tumor

Diagnosis:

Neuroendocrine tumor (NETs) of the rectum

Discussion:

Neuroendocrine tumors (NETs) of colon and rectum are derived from enterochromaffin cells of the gut, found throughout the intestinal tract within the crypts of Lieberkühn. The clinical presentation of NETs is non-specific, depending on the site of origin. Half of all rectal NETs were diagnosed incidentally during colonoscopy in patients without specific rectal symptoms. Symptomatic patients presented with lower gastrointestinal bleeding, pelvic/rectal discomfort and bowel habit change. The carcinoid syndrome was rarely seen with rectal NETs¹. The endoscopic appearance of rectal NETs is a nodule with yellowish color; multiple nodular lesions are rare. Risk of metastasis and prognosis of rectal NETs are associated with tumor size, muscular and lymphovascular invasion, of which

endoscopic ultrasound (EUS) is the best modality to assess. Resection is the main treatment of rectal NETs. Endoscopic resection has the role for small nodule less than 10 millimeters in diameter². Various endoscopic resection including endoscopic submucosa dissection (ESD), endoscopic mucosal resection (EMR) have been reported; however the best approach is still debated³.

References

1. Eggenberger JC. Carcinoid and other neuroendocrine tumors of the colon and rectum. *Clinics in colon and rectal surgery* 2011;24:129-34.
2. Lambert R. Gastric and rectal carcinoids. *Endoscopy* 2010;42:661-3.
3. LeeD S, Jeon SW, Park SY, et al. The feasibility of endoscopic submucosal dissection for rectal carcinoid tumors: comparison with endoscopic mucosal resection. *Endoscopy* 2010;42:647-51.



Case 1

Narisorn Lakananurak, M.D.

Pradermchai Kongkam, M.D.

A 71-year-old female patient presented with painless jaundice. Past history was insignificant. Physical examination revealed icteric sclera. CT demonstrated hilar obstruction with bilateral intrahepatic bile duct dilation. Percutaneous biliary drainage (PTBD) was placed to relieve her jaundice. Since the patient was considered resectable then EUS was requested for pathological diagnosis of the hilar lesion.

EUS was performed with a linear EUS probe (Fuji, SU-8000, Japan). It demonstrated a perihilar hypoechoic lymph node measuring 9x9 mm in diameter as shown in Figure 1. EUS-FNA was performed twice with a 22-gauge needle. Bloody tissue was obtained, smeared on glass slides and sent for pathological examination. Cytopathology showed atypical cell suggestive for adenocarcinoma.

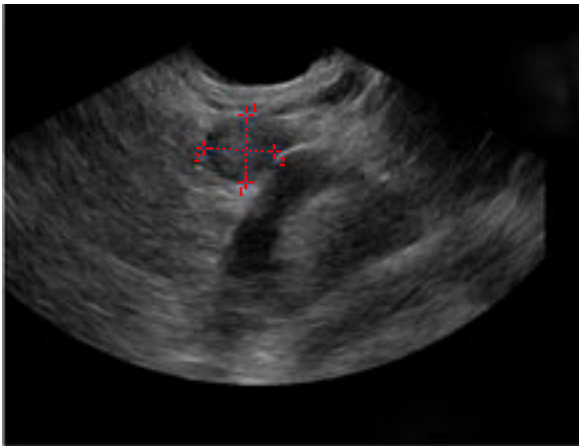
Diagnosis:

Hilar cholangiocarcinoma

Discussion:

Hilar cholangiocarcinoma usually presented in an advanced stage with typical lesion of infiltrating lesion in the bile duct. Nonetheless, identification of hilar mass seems to be difficult due to a nature of periductal infiltration instead of mass lesion. Pathological diagnosis is traditionally obtained by ERCP guided procedures such as brushing, biopsy or cholangioscopy. However, their sensitivity is very limited. EUS is nowadays increasingly used for an evaluation of perihilar cholangiocarcinoma. A recent observational single-centered study reported that the sensitivity of EUS for detection of hilar cholangiocarcinoma was 83% (25/30), however the sensitivity of EUS-FNA for making a pathological diagnosis was only 59%¹. Another retrospective study recruiting 32 patients with hilar lesions reported the sensitivity and specificity rate of EUS-FNA for making a pathological diagnosis were 52% and 100%, respectively².

Although current data from a few studies using EUS and EUS-FNA as a diagnostic tool for hilar cholangiocarcinoma showed a low sensitivity rate however all available studies were small and retrospective in fashion. EUS in this patient nicely demonstrated perihilar hilar lymph node with a positive EUS-FNA for adenocarcinoma. Therefore, EUS may be considered as one of diagnostic tests for a diagnosis of hilar cholangiocarcinoma when other modalities have limitation.



■ Figure 1: Demonstrated a peri-hilar lymph node. Cytopathology from EUS-FNA showed a diagnosis of adenocarcinoma

References

1. Mohamadnejad M, DeWitt JM, Sherman S, et al. Role of EUS for preoperative evaluation of cholangiocarcinoma: a large single-center experience. *Gastrointest Endosc* 2011;73:71-8.
2. Manu NK, Derek MM, Viney W, et al. Role of EUS/ EUS-guided FNA in the management of proximal biliary strictures. *Hepatogastroenterology* 2011;58:1862-5.



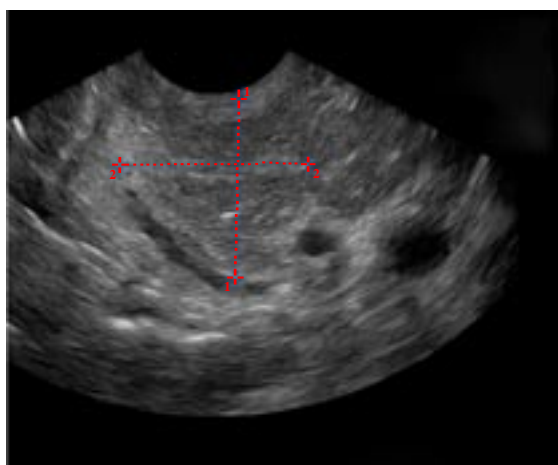
Case 2

Wiriaporn Ridditid, M.D.

Pradermchai Kongkam, M.D.

A 58-year-old male patient presented with painless jaundice for 1 month. He has lost his body weight for 6 kgs without abdominal pain. His past history was significant for subtotal colectomy from colon cancer 9 years ago. Physical examination showed icteric sclera. CT scan of the upper abdomen showed bile duct dilation without any demonstrated cause. ERCP was then performed; it revealed a bulging ampulla and narrowing distal common bile duct with upstream bile duct dilation. A biliary stent was placed across the stricture. EUS was scheduled for an evaluation of distal biliary stricture.

A linear EUS probe (Fuji, SU-8000, Japan) was used for the procedure. Endosonographically, a hypoechoic ampullary mass measuring 31x22 mm in maximal diameter was identified (Figure 1). A biliary stent was endosonographically demonstrated. The tumor was considered potentially resectable and surgical specimen later confirmed as pancreatic neuroendocrine tumor (PNET).



■ **Figure 1:** A hypoechoic ampullary mass demonstrated by a linear EUS probe (Fuji, SU-8000, Japan)

Diagnosis:

PNET presented as periampullary tumor

Discussion:

Peri-ampullary tumor can be overlooked by trans-abdominal imaging studies however EUS can detect ampullary tumor as shown in this case. Several studies confirmed definite utility of EUS for diagnosis of ampullary neoplasm^{1,2}. A recent study demonstrated that EUS can identify cause of bile duct obstruction in 9% of patients presenting with unexplained bile duct obstruction. In comparison with ERCP, EUS however is not significantly superior to ERCP for detecting ampullary tumor³.

References

1. Manta R, Conigliaro R, Castellani D, et al. Linear endoscopic ultrasonography vs. magnetic resonance imaging in ampullary tumors. *World J Gastroenterol* 2010;16:5592-7.
2. Reddymasu SC, Gupta N, Singh S, et al. Pancreato-biliary malignancy diagnosed by endoscopic ultrasonography in absence of a mass lesion on transabdominal imaging: prevalence and predictors. *Dig Dis Sci* 2011;56:1912-6.
3. Chen CH, Tseng LJ, Yang CC, et al. The accuracy of endoscopic ultrasound, endoscopic retrograde cholangiopancreatography, computed tomography, and transabdominal ultrasound in the detection and staging of primary ampullary tumors. *Hepatogastroenterology* 2001;48:1750-3.

Case 3

Wiriyaporn Ridtitid, M.D.

Pradermchai Kongkam, M.D.

A 55-year-old female patient presented with dysphagia. Upper endoscopy revealed an exophytic mass at mid esophagus as shown in Figure 1. Endoscopic mucosal biopsy was performed and confirmed as squamous cell carcinoma. Endoscopically, the lesion was diagnosed as esophageal cancer. EUS was scheduled for staging of this esophageal cancer. It demonstrated a circumferential hypoechoic lesion invading through serosa as shown in Figure 2. This was consistent with T4 esophageal cancer.

Diagnosis:

Esophageal cancer

Discussion:

Accurate pre-treatment staging of esophageal cancer is crucial as it helps to avoid an unnecessary

surgery. EUS is the investigation of choice for this staging. A recent large retrospective study reported an overall accuracy rate of EUS in T staging at 74% with a sensitivity rate for T1, T2, and T3 at 82%, 43% and 83%, respectively¹. This demonstrated that EUS is still an unsatisfactory tool for staging of esophageal cancer particularly in T2. Smith et al retrospectively compared accuracy of EUS based on surgical pathology in patients undergone minimally invasive esophagectomy; from 71 patients, an overall accuracy rate of EUS for pretreatment T staging was 72% with an accuracy rate of T0, T1, T2 and T3 at 80%, 75%, 39% and 88% respectively². Based on results from these studies, advanced staged esophageal cancer by EUS was likely to be accurate and precluded surgery from the only curative treatment.



■ Figure 1: Demonstrated an exophytic mass in the esophagus. This was consistent with esophageal cancer.



■ Figure 2: Demonstrated a hypoechoic lesion surrounding esophagus. The lesion invaded through serosa and was endosonographically staged as T4. The procedure was performed by a radial probe (Fuji, SU-8000, Japan).

References

1. Pech O, Gunter E, Dusemund F, et al. Accuracy of endoscopic ultrasound in preoperative staging of esophageal cancer: results from a referral center for early esophageal cancer. *Endoscopy* 2010;42:456-61.
2. Smith BR, Chang KJ, Lee JG, et al. Staging accuracy of endoscopic ultrasound based on pathologic analysis after minimally invasive esophagectomy. *Am Surg* 2010;76:1228-31.



Case 4

Wiriyaporn Ridtitid, M.D.

Phonthep Angsuwatcharakon, M.D.

Pradermchai Kongkam, M.D.

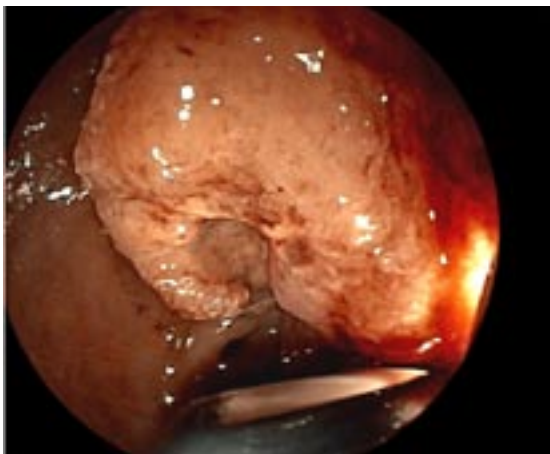
A 62-year-old female patient presented with jaundice, weight loss and prolonged fever for a few months. CT scan revealed a 42x35 mm mass at the uncinate process of the pancreas with tumor invasion to the second part of duodenum. ERCP was scheduled for biliary drainage. Unfortunately, as shown in Figure 1, the ampullary tumor was so large, causing distortion of the duodenum and precluded the cannulation that bile duct. We therefore switched the procedure to EUS guided biliary drainage. EUS was performed with a linear probe (Fuji, SU-8000, Japan). It revealed a hypoechoic ampullary mass measuring 53x50 mm in diameter with dilated bile duct and main pancreatic duct as shown in Figure 2. The bile duct was then punctured with a 19G needle under endosonographic and fluoroscopic guidance as shown in Figure 3 and 4, respectively. Subsequently, a double pigtail plastic stent was successfully placed immediately above the major papilla (Figure 5). Her jaundice was afterward improved without major postprocedural complication.

Technique:

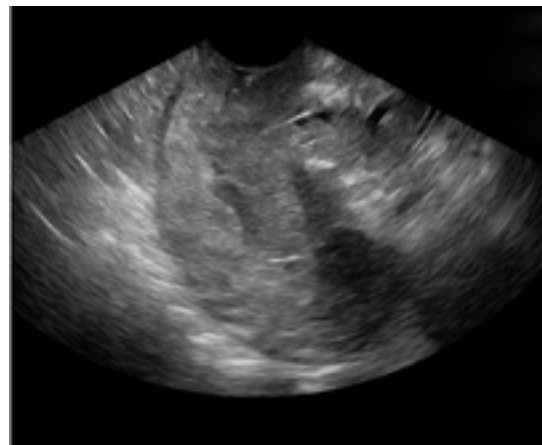
EUS guided choledochoduodenostomy

Discussion:

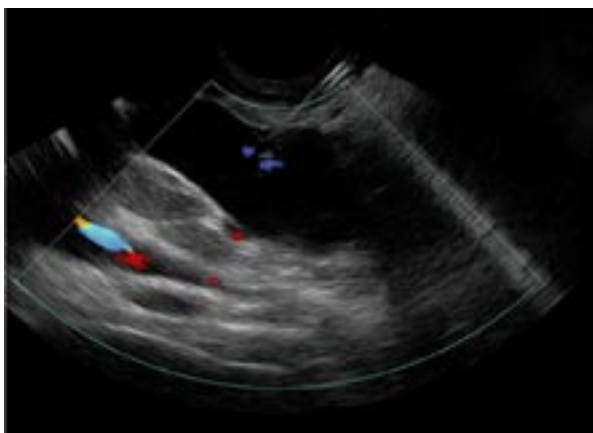
In this case, ERCP failed to access common bile duct through major papilla due to enlarged and distorted ampulla. EUS guided choledochoduodenostomy has been reported as an alternative procedure for biliary drainage. A recent retrospective cohort study from a tertiary referral hospital recruited 95 consecutive patients with failed ERCP over a 4-year period, EUS guided bile duct therapy was successful in 86% with a complication rate of 10%¹. Another prospective randomized study compared EUS guided biliary drainage (n=13) with percutaneous biliary drainage (PTBD) (n=12), all procedures were technically and clinically successful with comparable complication rate (EUS vs. PTBD: 15.3% vs. 25%)².



■ Figure 1: Demonstrated a fungating ampullary tumor



■ Figure 2: Demonstrated a hypoechoic ampullary tumor



■ Figure 3: Demonstrated a guide-wire in the common bile duct. The procedure was performed with a linear EUS probe (Fuji, SU-8000, Japan)



■ Figure 4: Demonstrated a guide-wire in the common bile duct and partial cholangiogram performed by a linear EUS probe (Fuji, SU-8000, Japan)



■ Figure 5: Demonstrated a double pigtail stent placed from bile duct to second part of duodenum. The stent was located above the major papilla

References

1. Shah JN, Marson F, Weilert F, et al. Single-operator, single-session EUS-guided anterograde cholangiopancreatography in failed ERCP or inaccessible papilla. *Gastrointest Endosc* 2012;75:56-64.
2. Artifon EL, Aparicio D, Paione JB, et al. Biliary Drainage in Patients With Unresectable, Malignant Obstruction Where ERCP Fails: Endoscopic Ultrasonography-Guided Choledochoduodenostomy Versus Percutaneous Drainage. *J Clin Gastroenterol* 2012;46:768-74.



Case 5

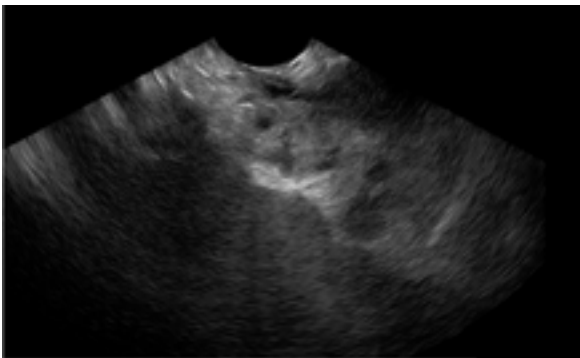
Suparat Khemnark, M.D.

Pradermchai Kongkam, M.D.

A 59-year-old male patient presented with abdominal distention and significant weight loss for 10 kgs in 2 months. Ultrasonography and CT scan of the abdomen showed ascites and peritoneal nodules. Physical examination revealed abdominal distension. Endoscopic ultrasound was scheduled for an evaluation of peritoneal metastasis. The procedure was performed by a linear EUS (Fujifilm, S-8000, Japan). It revealed multiple peritoneal nodules and omental cake as shown in Figure 1. EUS FNA was performed. Aspirated fluid showed straw color fluid. Cytopathology demonstrated mucin and multiple clusters of poorly differentiated adenocarcinoma consistent with signet ring cell carcinoma.

Diagnosis:

Carcinomatosis peritonei diagnosed by FNA of the omental cake



■ Figure 1: Demonstrated an omental cake and lymph nodes in the abdomen

Discussion:

Finding the cell type of carcinomatosis peritonei remains a challenge clinical exercise. Definite management requires a proper diagnosis. Etiologies include hematologic malignancies, metastatic disease, and primary peritoneal tumor. Tissue confirmation is strongly suggested. In general, ascites cytology has low yield as plenty amount of fluid may dilute concentration of cells. Omental cake or intra-abdominal lymph node can be targeted for biopsy by a percutaneous route. However, in some situation, the target lesions located deeply in the abdomen and thus it is quite difficult for the percutaneous approach to obtain tissue. In such circumstances, EUS is an alternative diagnostic procedure to attain the diagnosis. In this case, EUS successfully made the diagnosis without complication. DeWitt et al, demonstrated that EUS guided paracentesis revealed malignancies in 16 from 60 patients (27%)¹. Another study confirmed a high specificity rate with moderate sensitivity rate ².

References

1. DeWitt J, LeBlanc J, McHenry L, et al. Endoscopic ultrasound-guided fine-needle aspiration of ascites. *Clin Gastroenterol Hepatol* 2007;5:609-15.
2. Wardeh R, Lee JG, Gu M. Endoscopic ultrasound-guided paracentesis of ascitic fluid: a morphologic study with ultrasonographic correlation. *Cancer Cytopathol* 2011;119:27-36.

Case 6

Suparat Khemnark, M.D.

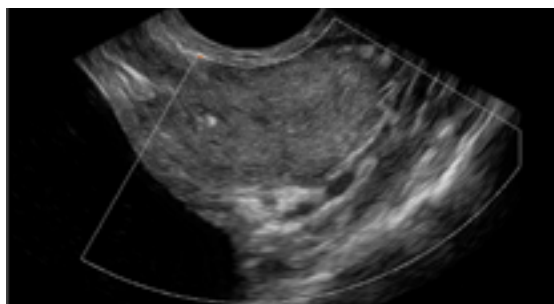
Kittiyod Poovorawan, M.D.

Pradermchai Kongkam, M.D.

A 53-year-old male patient presented with rectal bleeding. He is a candidate for liver transplantation because of decompensated cirrhosis. A month ago, he had active lower GI bleeding and was diagnosed with rectal varices as a cause of bleeding. At that time, 2% ethoxysclerol was injected into the rectal varices. His bleeding ceased for a few weeks. Unfortunately, he again developed another episode of rectal bleeding. Sigmoidoscopy revealed a sub-epithelial lesion in the rectum. There were no residual varices or suspicious bleeding stigmata left. However, it was not confirmed by only the endoscopic view whether this lesion was the varices after sclerotherapy. Endoscopically, the lesion was a sub-epithelial lesion with smooth surface and located in rectum. Endosonographically, a hypoechoic solid lesion with surrounding varices was identified as shown in Figure 1.

Diagnosis:

Post-ethoxysclerol injection rectal varices without stigmata of recent bleeding



■ Figure 1: Demonstrated a hypoechoic mass underneath the sub-epithelial rectal lesion. This was consistent with a post-ethoxysclerol-injection mass in the rectum.

Discussion:

Rectal varices are one of causes for lower GI bleeding in cirrhotic patients. Specific treatments included sclerosing agent injection. In this patient, ethoxysclerol was successfully injected into the rectal varices prior to the current episode of bleeding. It was then doubt that if the endoscopic finding of rectal sub-epithelial lesion explained his rectal bleeding or not. EUS then was very useful in the situation to delineate the nature of lesion. Eventually, the lesion was proven as varices of post sclerotherapy. In a cohort study of EUS for an evaluation of deep rectal varices in 96 patients with cirrhosis, EUS identified deep varices in 51% of patients. Of 83 patients with no rectal varices by endoscopic examination, EUS discovered rectal varices in 47% of them¹. Prior study also confirmed the useful role of rectal EUS for an evaluation of rectal varices in cirrhotic patients².

References

1. Wiechowska-Kozłowska A, Bialek A, Milkiewicz P. Prevalence of ‘deep’ rectal varices in patients with cirrhosis: an EUS-based study. *Liver Int* 2009;29:1202-5.
2. Dhiman RK, Choudhuri G, Saraswat VA, et al. Endoscopic ultrasonographic evaluation of the rectum in cirrhotic portal hypertension. *Gastrointest Endosc* 1993;39:635-40.

Case 7

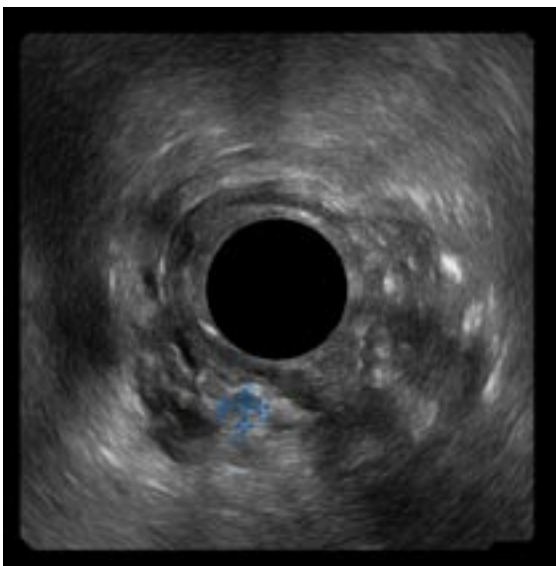
Wiriyaporn Ridditid, M.D.

Pradermchai Kongkam, M.D.

A 71-year-old female patient presented with abdominal pain for 9 months. Over the last 2 months, she developed jaundice. ERCP was scheduled for biliary drainage. Cholangiogram revealed distal biliary stricture and a plastic biliary stent was subsequently placed across the stricture. EUS was scheduled for an evaluation of this indeterminate distal biliary stricture. It was performed with a radial EUS probe (Fuji, SU-8000, Japan). EUS showed a few peri-ductal lymph nodes surrounding distal common bile duct (Figure 1). This raised a suspicion of distal cholangiocarcinoma.

Diagnosis:

Distal cholangiocarcinoma.



■ Figure 1: Demonstrated periductal lymph nodes surrounding distal common bile duct.

Discussion:

In patient with indeterminate biliary stricture diagnosed by standard imagings, EUS plays an important role in order to identify the nature of stricture. A retrospective study published confirmed this fact as it reported a high sensitivity rate of EUS for a diagnosis of malignant biliary stricture in patients with a previous diagnosis of unknown cause biliary stricture by prior ERCP with negative intraductal sampling by ERCP¹. With regard to EUS role in distal cholangiocarcinoma, a large retrospective study demonstrated role of EUS for an evaluation of patients with cholangiocarcinoma. The study recruited 81 patients with cholangiocarcinoma. Of these, 51 patients (63%) had distal cholangiocarcinoma. Tumor detection rate of distal cholangiocarcinoma by EUS was 100%. Of all 81 patient with either hilar or distal cholangiocarcinoma, tumor detection rate of EUS was significantly superior to CT scan (94% vs. 30%; $P < 0.001$)².

References

1. Lee JH, Salem R, Aslanian H, et al. Endoscopic ultrasound and fine-needle aspiration of unexplained bile duct strictures. *Am J Gastroenterol* 2004;99:1069-73.
2. Mohamadnejad M, DeWitt JM, Sherman S, et al. Role of EUS for preoperative evaluation of cholangiocarcinoma: a large single-center experience. *Gastrointest Endosc* 2011;73:71-8.

Case 8

Wiriyaporn Ridditid, M.D.

Pradermchai Kongkam, M.D.

An 85-year-old male patient was recently diagnosed as colon cancer with liver metastasis. In addition to multiple liver masses, CT scan of the abdomen revealed multiple gall stones and mildly dilated common and intra-hepatic bile duct without definite cause. EUS was scheduled for an evaluation of possible common bile duct stone. A linear EUS probe (Fuji, SU-8000, Japan) was used for the procedure. Ultrasonographic images showed a hyperechoic material in common bile duct as shown in Figure 1. This was consistent with common bile duct stone.

Diagnosis:

Choledocholithiasis

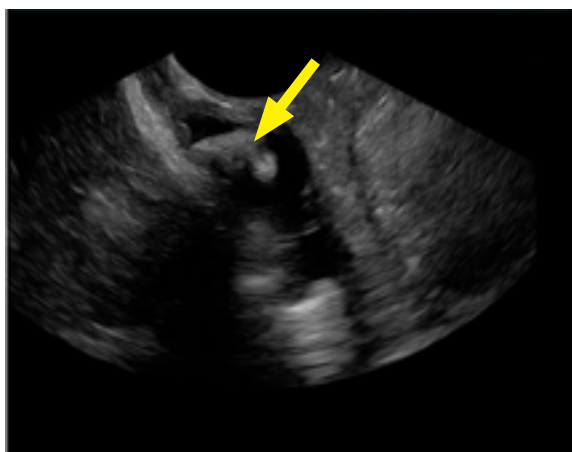
Discussion:

EUS and MRCP seem to be the most appropriate tests in patients with intermediate risk to

have a common bile duct stone as these tests provided high sensitivity rate for stone detection with an acceptably low rate of complication¹. A recent retrospective study demonstrated that EUS for the evaluation of unknown cause of biliary dilation had more diagnostic value in patients with abnormal liver function tests than that in patients with normal tests².

References

1. Aljebreen A, Azzam N, Eloubeidi MA. Prospective study of endoscopic ultrasound performance in suspected choledocholithiasis. *J Gastroenterol Hepatol* 2008;23:741-5.
2. Malik S, Kaushik N, Khalid A, et al. EUS yield in evaluating biliary dilatation in patients with normal serum liver enzymes. *Dig Dis Sci* 2007;52:508-12.



■ Figure 1: Demonstrated a hyperechoic structure with posterior acoustic shadow in distal common bile duct. This was consistent with distal common bile duct stone

Case 9

Tanassanee Soontornmanokul, M.D.

Pradermchai Kongkam, M.D.

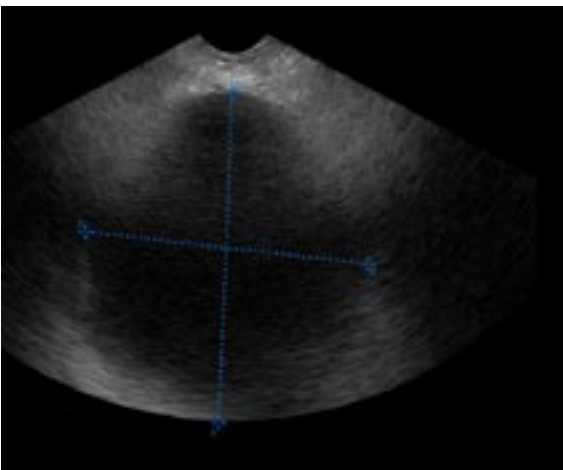
A 67-year-old male patient presented with jaundice and weight loss. CT scan of the abdomen read as a hilar cholangiocarcinoma. Two uncovered metal stents were placed to relieve his jaundice. Three days after the procedure, a markedly distended gall bladder causing a hydrop gallbladder was noted. EUS was called for gallbladder drainage. The procedure was performed with a linear EUS probe (Fujifilm, S-8000, Japan). The gallbladder was markedly distended as shown in Figure 1. Via gastric puncture with a 19G needle was successfully performed as shown in Figure 2. A guide-wire was inserted into the gallbladder and subsequently replaced with a double pigtail plastic stent under EUS and fluoroscopic guidance as shown in Figure 3 and 4. Good cystic fluid was observed (Figure 5). No immediate complication. A week later, the gallbladder became smaller and no post-procedural complication was observed.

Diagnosis:

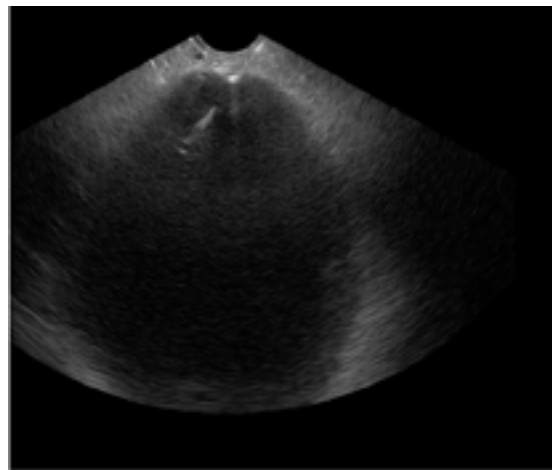
EUS guided gallbladder drainage

Discussion:

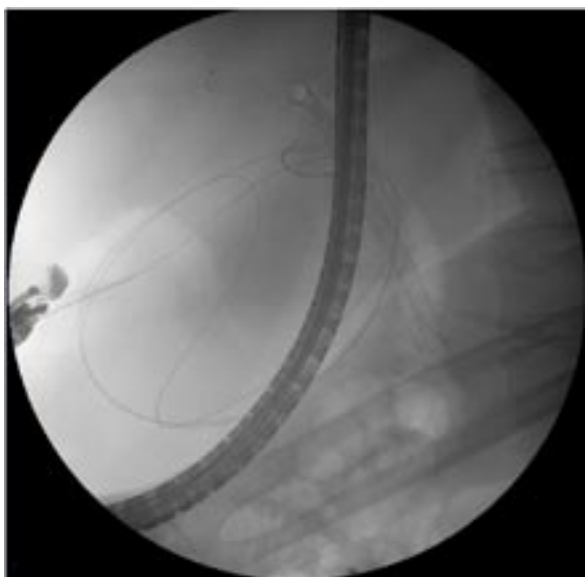
Cystic duct obstruction is the main cause of acute cholecystitis. Drainage of obstructed gall bladder is mandatory for definite treatment. The drainage procedures can be performed by percutaneous, surgical, ERCP or EUS approach. In this patient, the gallbladder was finally successfully drained by EUS guidance¹. A recent prospective randomized controlled trial compared EUS and percutaneous guided biliary drainage in 59 patients with acute, high-risk, or advanced-stage cholecystitis. Technical and clinical success rate were higher than 95% and not significantly different between both arms. Complications rate of EUS and percutaneous approach were similar at 7% and 3%, respectively². Therefore, based on current data and result from this case, EUS should be considered as a standard alternative for gall bladder drainage.



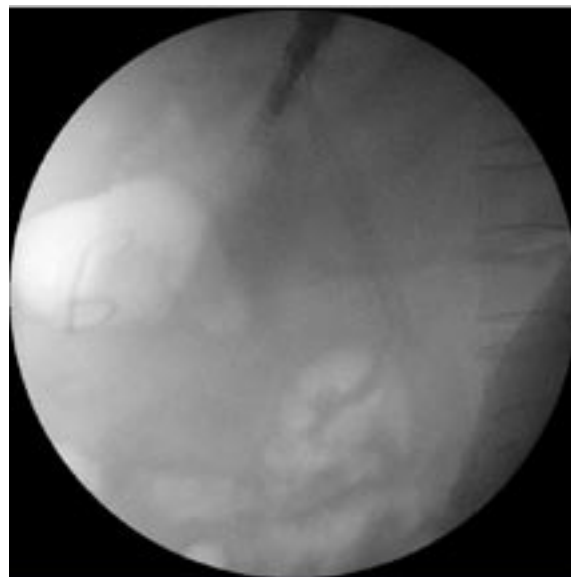
■ Figure 1: Demonstrated a markedly distended gallbladder



■ Figure 2: Showed an EUS-FNA needle in the gallbladder.



■ Figure 3: Demonstrated multiple loops of guide wire was place in the gallbladder



■ Figure 4: Demonstrated a double pigtail plastic stent bridging between stomach and gallbladder. Noted two metal stents for biliary drainage were in place



■ Figure 5: Demonstrated an external part of a double pigtail plastic stent in the stomach

References

1. Subtil JC, Betes M, Munoz-Navas M. Gallbladder drainage guided by endoscopic ultrasound. *World J Gastrointest Endosc* 2010;2:203-9.
2. Jang JW, Lee SS, Song TJ, et al. Endoscopic ultrasound-guided transmural and percutaneous transhepatic gallbladder drainage are comparable for acute cholecystitis. *Gastroenterology* 2012; 142:805-11.

Case 10

Suparat Khemnark, M.D.

Tanassanee Soontornmanokul, M.D.

Pradermchai Kongkam, M.D.

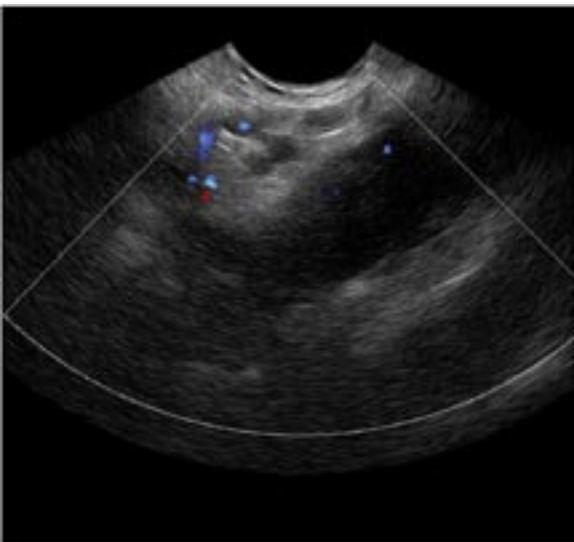
A 34-year-old female patient presented with chronic abdominal pain from chronic pancreatitis. Several conventional methods to control her chronic abdominal pain had failed. EUS guided celiac plexus neurolysis was requested to decrease her longstanding intractable pain. The procedure was performed with a a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan). The scope was placed just above the origin of celiac vessel as demonstrated in Figure 1. Then 0.25% bupivacaine and 98% alcohol was injected to this area. Figure 2 demonstrated the same area after alcohol injection was successfully performed. No immediate complication after procedure. Two weeks later, her pain decreased significantly.

Technique:

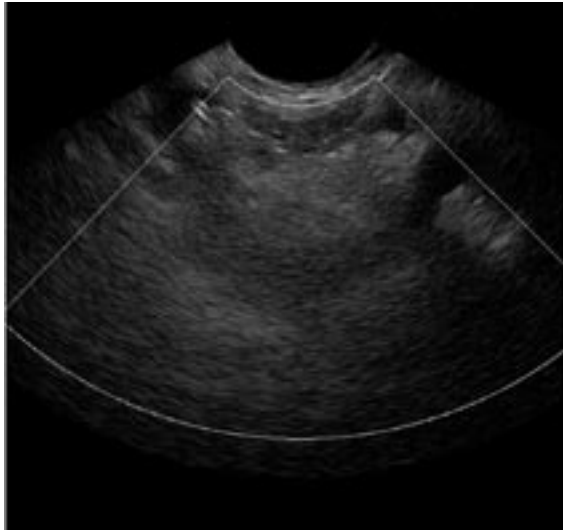
Celiac plexus neurolysis

Discussion:

EUS guided celiac plexus block is a conventional options to control the pain from chronic pancreatitis with an efficacy rate at 51%. In contrast, EUS guided celiac plexus neurolysis with alcohol injection instead of steroid injection is more preferred to diminish the pain from pancreatic cancer with an efficacy rate of 73%¹. Recently celiac plexus neurolysis with alcohol injection has been adopted to use in chronic pancreatitis patients with pain and the technique showed a higher rate of pain improvement than celiac plexus block from steroid².



■ Figure 1: Demonstrated celiac artery originating from abdominal aorta. The procedure was performed with a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan)



■ Figure 2: Demonstrated hyperechoic area above origin of celiac axis from aorta. This was consistent with post injection of alcohol into area around celiac axis. The procedure was performed with a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan)

References

1. Kaufman M, Singh G, Das S, et al. Efficacy of endoscopic ultrasound-guided celiac plexus block and celiac plexus neurolysis for managing abdominal pain associated with chronic pancreatitis and pancreatic cancer. *J Clin Gastroenterol* 2010;44:127-34.
2. Levy MJ, Topazian MD, Wiersema MJ, et al. Initial evaluation of the efficacy and safety of endoscopic ultrasound-guided direct Ganglia neurolysis and block. *Am J Gastroenterol* 2008; 103:98-103.



Case 11

Phonthep Angsuwatcharakon, M.D.

Wiriyaporn Ridditid, M.D.

Pradermchai Kongkam, M.D.

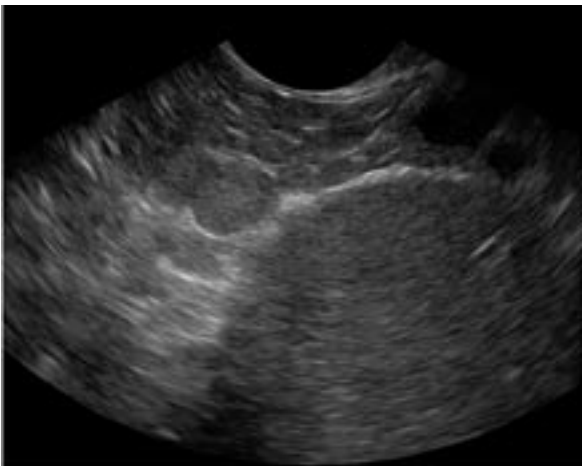
A 42-year-old male patient presented with frequent fainting for 8 months from hypoglycemia. Over the last few weeks, he required frequent hospitalizations with supplemental intravenous glucose to prevent recurrent hypoglycemia. Blood tests were diagnostic for Insulinoma. MDCT scan with pancreatic protocol failed to reveal any mass in the pancreas. EUS was scheduled to search for pancreatic neuroendocrine tumors (PNETs). EUS was performed with a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan). It revealed a homogeneous hypoechoic mass measuring about 9x7 mm in diameter at the tail of pancreas (Figure 1). The mass was adjacent with the spleen. EUS-FNA was performed with a 22G needle (Cook Company) (Figure 2). Tissue was sent for cytopathology. Subsequently, distal pancreatectomy was performed. The final surgical pathology was consistent with Insulinoma. No further hypoglycemic symptoms reported.

Diagnosis:

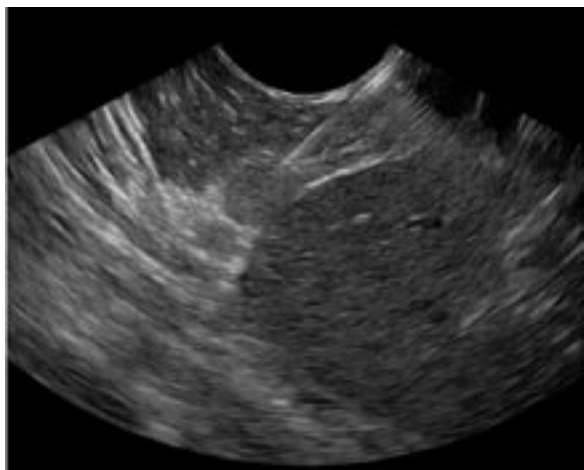
Insulinoma

Discussion:

PNETs can present as either non-functioning PNETs or functioning PNETs. In the former group, presenting symptoms are usually mass-related ones and cross sectional imaging can readily identify the lesion(s). In the other hand, the latter group usually presented with their hormonal symptoms. Therefore, at the time of presentation, size of lesions is usually too to be detected by CT scans. Insulinoma is the most common functioning pancreatic neuroendocrine tumors and has remained to be a clinical challenge. Aniwan, et al, reported that 19 from 109 pancreatic masses detected by EUS were initially missed by CT scan. Another retrospective study compared efficacy of EUS versus CT for detection of Insulinoma, the overall sensitivity of EUS and CT for identifying Insulinoma was 83.3% and 16.7%, respectively².



■ Figure 1: Demonstrated a homogeneous hypoechoic mass measuring about 9x7 mm in diameter at the tail of pancreas. The mass was located adjacent to the spleen. The procedure was performed with a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan)



■ Figure 2: Demonstrated EUS-FNA needle (22G, Cook company) in the middle of the lesion. The procedure was performed with a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan)

References

1. Aniwat S, Kongkam P, Rittitid W, et al. Predicting Factors of Pancreatic Lesions Missed by CT but Diagnosed by EUS. *Gastrointest Endosc* 2011; 73, Suppl:AB251-AB2.
2. Ardengh JC, Rosenbaum P, Ganc AJ, et al. Role of EUS in the preoperative localization of insulinomas compared with spiral CT. *Gastrointest Endosc* 2000;51:552-5.



Case 12

Rungsun Rerknimitr, M.D.

Rapat Pittayanon, M.D.

Pradermchai Kongkam, M.D.

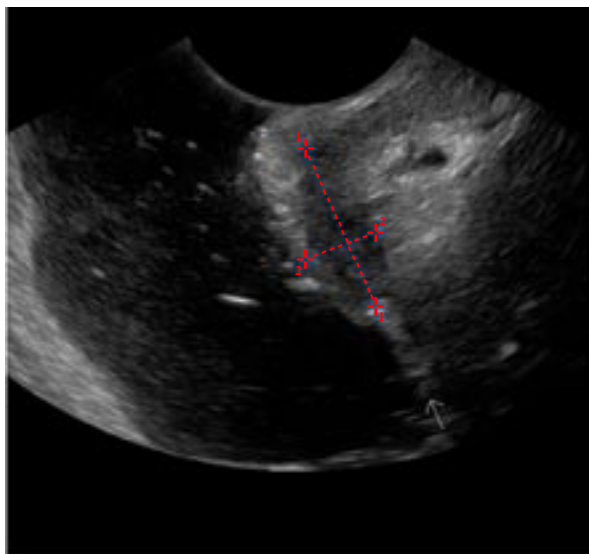
A 56-year-old Thai male patient presented with intermittent hematemesis, hematochezia, and melena 7 months ago. Physical examination revealed markedly pale conjunctiva. Initial hematocrit was at 17%. Upper endoscopy revealed a large gastric ulcer with pigmented spot at the body of stomach. Diluted adrenaline was locally injected into the lesion to control the bleeding. Consequently, bleeding ceased. Two months later, repeat EGD revealed a healed ulcer in the same location. A rapid urease test was positive. Biopsy from the ulcer was done. Unfortunately, the pathology showed well-differentiated gastric adenocarcinoma. The patient was then planned for endoscopic submucosal dissection (ESD) to completely remove the tumor. EUS was then scheduled for pre-ESD evaluation. The procedure was performed by a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan). It revealed a hypoechoic lesion measuring 14x11 mm in diameter invading through muscularis propria (Figure 1-2). It was endosonographically staged as T3N0M0. It confirmed the higher degree of invasion and surgery was more appropriate.

Diagnosis:

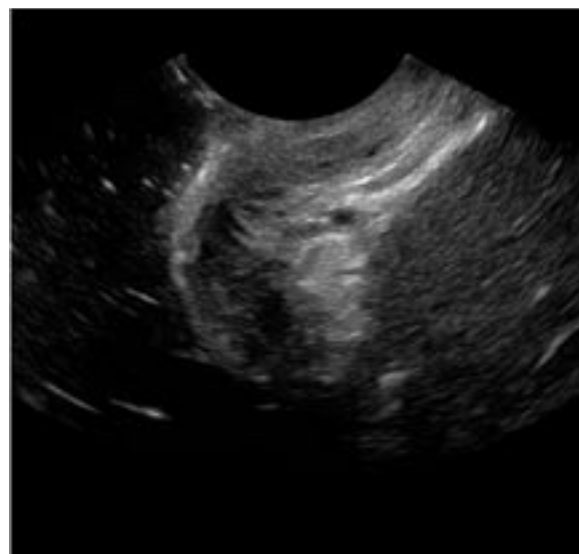
Gastric cancer

Discussion:

Currently, gastric cancer can be locally endoscopically removed by endoscopic submucosal dissection (ESD). This procedure is much less invasive than surgical removal and patients can be discharged within a day or a few days after procedure with a low morbidity rate. However, in general, the procedure should not be performed in any lesions reaching to muscularis propria layer (T2). In this patient, the lesion was endosonographically staged as T3 due to its invasion through muscularis propria layer. It is therefore more appropriate to switch the removal method to surgical removal instead of ESD. A recent systematic review and meta-analysis concluded that pool accuracy rate of EUS for T staging was 75% with a moderate Kappa value (0.52) and T3 is the most accurate stage classified by EUS¹. Power et al, recruited 94 patients with gastric cancer and divided them into low- (T1-2) and high-risk group (T3 and 4) according to EUS T staging. Subsequently, laparoscopy was performed. Of those, 19 patients were identified as occult metastatic disease; 18 of them were in EUS high-risk group whereas only 1 of them was in EUS low-risk group².



■ Figure 1: Demonstrated a hypoechoic lesion invading through muscularis propria. This was endosonographically staged as T3N0M0 gastric cancer. The procedure was performed by a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan).



■ Figure 2: Demonstrated left a hypoechoic lesion being located to left lobe of the liver. The procedure was performed by a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan).

References

1. Cardoso R, Coburn N, Seevaratnam R, et al. A systematic review and meta-analysis of the utility of EUS for preoperative staging for gastric cancer. *Gastric Cancer* 2012;15 Suppl 1:19-26.
2. Power DG, Schattner MA, Gerdes H, et al. Endoscopic ultrasound can improve the selection for laparoscopy in patients with localized gastric cancer. *J Am Coll Surg* 2009;208:173-8.



Case 13

Kanita Chattrasophon, M.D.

Suparat Khemnark, M.D.

Pradermchai Kongkam, M.D.

A 73-year-old male patient presented with chronic epigastric discomfort for 2 months. He had lost his weight for 2 kgs. Physical examination appeared unremarkable. Serum CA 19-9 was 55,854 U/ml. Other blood tests were unremarkable. CT scan revealed a liver mass in left lobe with intrahepatic bile duct dilation and multiple lymph nodes and ascites in the abdomen. EUS was scheduled to obtain the tissue for diagnosis. The procedure was performed with a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan). It revealed a large heterogeneous hypoechoic mass measuring 52x48 mm in diameter in the left lobe along with multiple lymph nodes, and a large omental cake as shown in Figure 1-3, respectively. EUS-FNA was obtained from these 3 areas with a 22G Pro-core needle (Wilson-Cook, Winston

Salem, NC, USA). Cytopathology was consistent with adenocarcinoma.

Diagnosis:

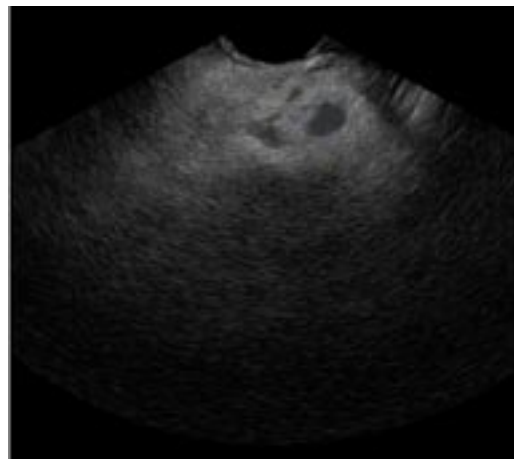
Metastatic intra-hepatic cholangiocarcinoma

Discussion:

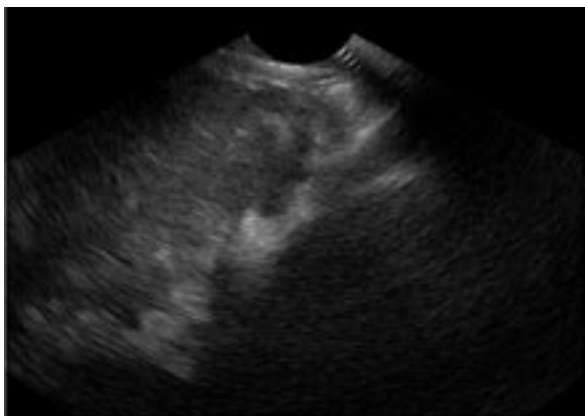
Although laparoscopy is the standard technique to provide tissue diagnosis in this case, selecting the most appropriate target by EUS for the highest sensitivity with the lowest risk is challenging and safer for the patient. A case report recently demonstrated that EUS-FNA can provide a pathological diagnosis of recurrent cholangiocarcinoma by obtaining the tissue from the omental cake¹. A case series recruited 12 patients with unknown cause ascites, EUS and EUS-FNA can identify etiologies of ascites in majority of patients².



■ Figure 1: Demonstrated a heterogeneous hypoechoic liver mass in left lobe of the liver. This figure was obtained by a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan).



■ Figure 2: Demonstrated a round hypoechoic lymph node in the abdomen. This figure was obtained by a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan).



■ Figure 3: Demonstrated omental cake in the abdomen. This figure was obtained by a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan).

References

1. Rial NS, Gilchrist KB, Henderson JT, et al. Endoscopic ultrasound with biopsy of omental mass for cholangiocarcinoma diagnosis in cirrhosis. *World J Gastrointest Endosc* 2011;3:124-8.
2. Rana SS, Bhasin DK, Srinivasan R, et al. Endoscopic ultrasound-guided fine needle aspiration of peritoneal nodules in patients with ascites of unknown cause. *Endoscopy* 2011;43:1010-3.



Case 14

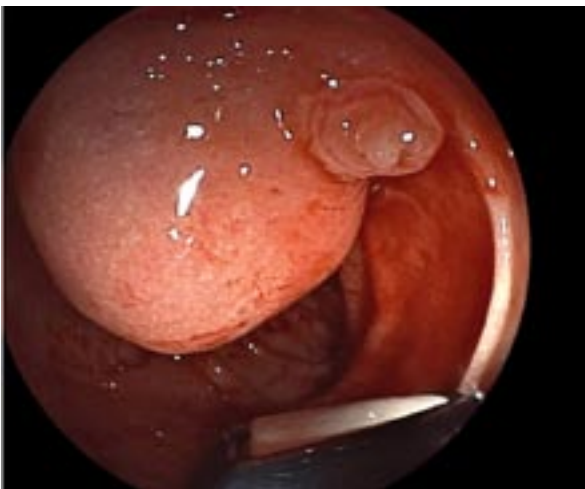
Phonthep Angsuwatcharakon, M.D.

Pradermchai Kongkam, M.D.

A 46-year-old male patient presented with an incidental MRI finding of a 3-cm lobulated mass with central necrosis in 2nd to 3rd part duodenum. He has no significant past medical and surgical history. A side-viewed duodenoscopy showed a sub-epithelial ampulla mass as shown in Figure 1. EUS was scheduled for an evaluation of the mass. EUS was performed with a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan). It revealed a hypoechoic mass measuring about 22x22 mm in diameter as shown in Figure 2. The mass originated from a 4th layer of intestinal wall and was suggestive as a stromal tumor. EUS-FNA was performed with a 22 G needle and pathology with c-KIT (CD117) confirmed as gastrointestinal stromal tumor (GIST).

Diagnosis:

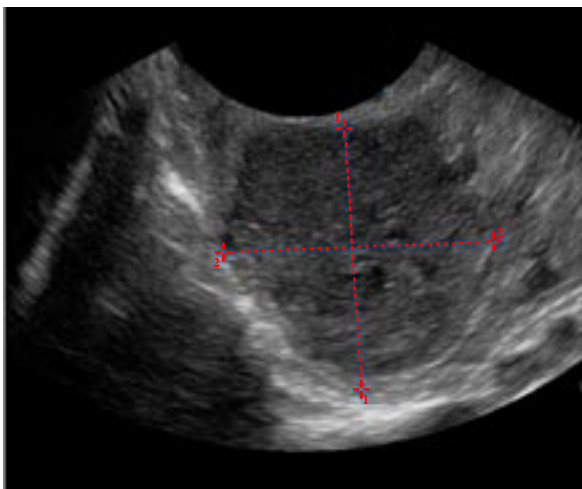
Periampullary GIST



Discussion:

Periampullary tumor is an uncommon tumor¹. Majority of tumors are either adenoma or adenocarcinoma². GIST has been rarely reported as a cause of peri-ampullary tumor³. It can present as a sporadic case or part of neurofibromatosis syndrome⁴. The definite treatment is a surgical resection. Preoperative diagnosis and staging is crucial as this can determine type and invasiveness of surgery. EUS is a proven critical diagnostic test that can provide the preoperative diagnosis as shown in this case. It can endosonographically delineate origin of the lesion and then suggest the most likely diagnosis. Surgical removal methods for such tumors included Whipple's operation, duodenectomy^{5,6}. In some advanced case, preoperative use of Imatinib changed an unresectable tumor to be a resectable one⁷. However, in this case, the tumor was considered resectable, therefore the patient was proceeded directly to surgery without need of neoadjuvant chemotherapy.

■ Figure 1: Demonstrated a peri-ampullary sub-epithelial tumor.



■ Figure 2: Demonstrated a hypoechoic mass originating from 4th layer of duodenal wall. This was suggestive for gastrointestinal tumor (GIST). This picture was obtained by a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan).

References

1. Henson DE, Schwartz AM, Nsouli H, et al. Carcinomas of the pancreas, gallbladder, extrahepatic bile ducts, and ampulla of vater share a field for carcinogenesis: a population-based study. *Arch Pathol Lab Med* 2009;133:67-71.
2. Posner S, Colletti L, Knol J, et al. Safety and long-term efficacy of transduodenal excision for tumors of the ampulla of Vater. *Surgery* 2000; 128:694-701.
3. Filippou DK, Pashalidis N, Skandalakis P, Rizos S. Malignant gastrointestinal stromal tumor of the ampulla of Vater presenting with obstructive jaundice. *J Postgrad Med* 2006;52:204-6.
4. Relles D, Baek J, Witkiewicz A, et al. Periampullary and duodenal neoplasms in neurofibromatosis type 1: two cases and an updated 20-year review of the literature yielding 76 cases. *J Gastrointest Surg* 2010;14:1052-61.
5. Liyanage CA, Abeygunawardhana S, Kumarage S, et al. Duodenum-preserving local excision of a gastrointestinal stromal tumor. *Hepatobiliary Pancreat Dis Int* 2008;7:214-6.
6. Cavallini M, Cecera A, Ciardi A, et al. Small periampullary duodenal gastrointestinal stromal tumor treated by local excision: report of a case. *Tumori* 2005;91:264-6.
7. Park JE, Dong SH, Cho KH, et al. Successful resection of locally advanced gastrointestinal stromal tumor of the ampulla of Vater after treatment with imatinib. *Korean J Gastroenterol* 2010;56:39-44.

Case 15

Pornphan Thienchanachaiya, M.D.

Naruemon Wisedopas-Klaikeaw, M.D.

Pradermchai Kongkam, M.D.

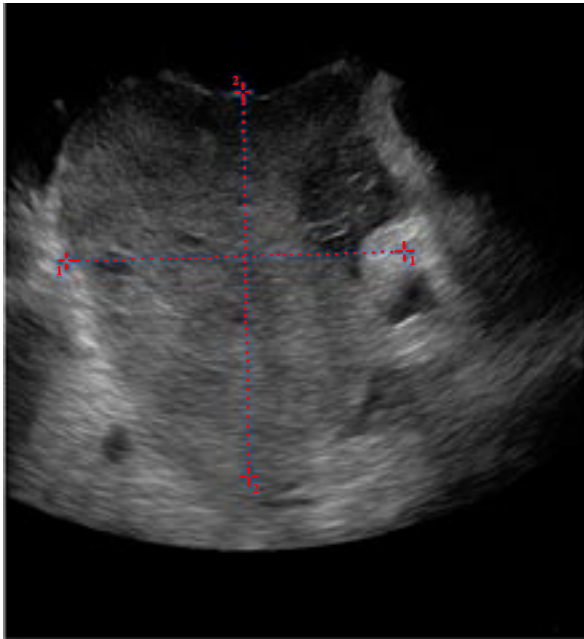
A 73-year-old female patient presented with left upper quadrant abdominal discomfort for a month. She had lost her weight for 2 kgs in the last month. She reported no chest symptoms and denied a history of smoking. Physical examination was unremarkable. CT scan revealed a hypo-density mass measuring about 3 cm in diameter next to the lower part of esophagus. EUS was scheduled for an evaluation of the mass and for tissue diagnosis. The procedure was performed with a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan). It revealed a well-defined border hypoechoic mass measuring about 30x14 mm in diameter (Figure 1). The mass was located adjacent to distal esophagus at 33 cm from the incisor. FNA was performed with 22G needle (Wilson Cook, Winston Salem, NC, USA). Microscopic examination showed a core tissue composed of diffuse proliferation of poorly cohesive small sized mitotically active cells with scant cytoplasm. In addition, frequent nuclear molding and granular salt and pepper chromatin texture were observed. This was consistent with malignant small cell neoplasm of the lung.

Diagnosis:

Small cell lung cancer

Discussion:

Posterior mediastinal mass can be due to chronic inflammation, infection or malignancy. Managements for these different diseases are completely different. Therefore, specific diagnosis is mandatory before an exact plan for the treatment can be established. EUS is the most optimal tool for the evaluation of the lesion in posterior mediastinum and to obtain tissue for diagnosis. Nowadays, mediastinoscopy to gain tissue preoperatively can be avoided by either EUS or endoscopic bronchial ultrasonography (EBUS) as recommended by a recent review¹. A recent large prospective study from Japan reported the accuracy rate of EUS and EBUS at 90% for mediastinal staging of lung cancer. The sensitivity, specificity, and positive and negative predictive values were 71.8%, 100%, 100%, and 86.6%, respectively².



■ Figure 1: Demonstrated a hypoechoic mass measuring 41x36 mm in diameter in posterior mediastinum by a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan).

References

1. Herth FJ. Nonsurgical staging of the mediastinum: EBUS and EUS. *Semin Respir Crit Care Med* 2011; 32:62-8.
2. Ohnishi R, Yasuda I, Kato T, et al. Combined endobronchial and endoscopic ultrasound-guided fine needle aspiration for mediastinal nodal staging of lung cancer. *Endoscopy* 2011;43:1082-9.

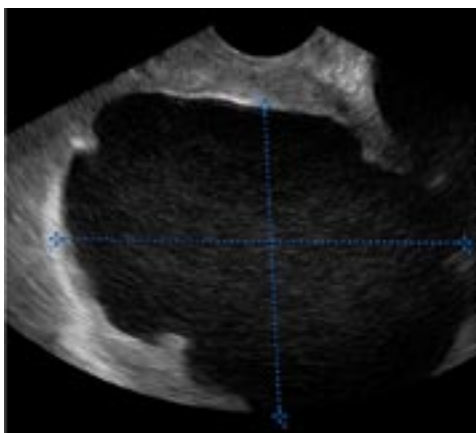


Case 16

Sasipim Sallapant, M.D.

Pradermchai Kongkam, M.D.

A 64-year-old female patient presented with abdominal distension, 20-kg weight loss and abdominal pain for 2 months. Physical examination revealed abdominal distension with a palpable non-tendered epigastric mass. CT scan showed a diffusely enlarged pancreas with multiple small pancreatic cysts and splenic vein thrombosis. EUS was scheduled for an evaluation of the cystic lesion and possible pathological diagnosis. EUS was performed with a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan). It demonstrated a large pancreatic cyst measuring 67x51 mm in diameter as shown in Figure 1. The cyst had mural nodule and thickening wall as shown in Figure 2. EUS FNA was performed with a 22G needle. Aspirated fluid showed straw color. Cystic fluid CEA and amylase was 6,606 ng/ml and 84 U/L, respectively. Cytopathological result was consistent with adenocarcinoma. This cyst was then diagnosed as mucinous cystadenocarcinoma.



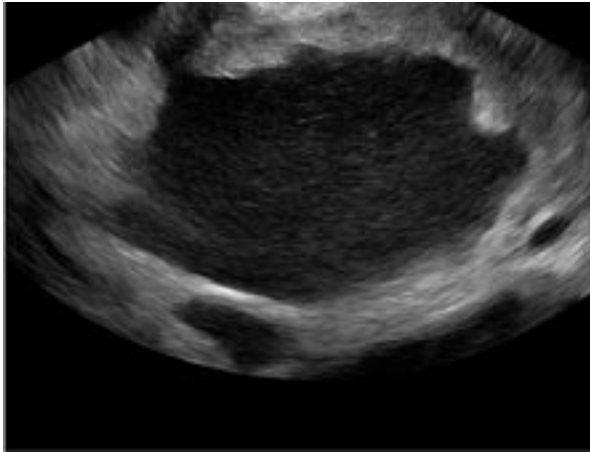
Diagnosis:

Mucinous cystadenocarcinoma

Discussion:

Pancreatic cysts can be simply classified as benign and malignant pancreatic cystic neoplasm. Preoperative diagnosis is very important as benign cysts should not be resected whereas malignant ones should go for resection. Standard imaging alone by CT scan is not accurate enough to differentiate both entities¹. EUS plays an important role for preoperative diagnosis of these cysts as it can provide cystic fluid analysis, cytopathology². Practically, high cystic fluid CEA level is suggestive for malignant cystic neoplasms including malignant cystadenocarcinoma (MCN), and intraductal papillary mucinous neoplasm (IPMN)³. To differentiate between these 2 lesions, high cystic fluid amylase level is suggestive for IPMN whereas low level supports a diagnosis of MCN. In this case, low level of cystic fluid amylase in combination with endosonographic features strongly confirmed a diagnosis of mucinous cystadenoma or cystadenocarcinoma and further cytology was able to pin point it as cystadenocarcinoma.

■ Figure 1: Demonstrated a large pancreatic cyst measuring 67x51 mm in diameter at body of pancreas by a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan).



■ Figure 2: Demonstrated a pancreatic cyst with mural nodule and thickening wall by a Convex Scan Ultrasonic Video Endoscope EG-530UT2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan).

References

1. Hawes RH, Clancy J, Hasan MK. Endoscopic ultrasound-guided fine needle aspiration in cystic pancreatic lesions. *Clin Endosc* 2012;45:128-31.
2. Brugge WR. The use of EUS to diagnose cystic neoplasms of the pancreas. *Gastrointest Endosc* 2009;69:S203-9.
3. Brugge WR, Lewandrowski K, Lee-Lewandrowski E, et al. Diagnosis of pancreatic cystic neoplasms: a report of the cooperative pancreatic cyst study. *Gastroenterology* 2004;126:1330-6.



Case 17

Rapat Pittayanon, M.D.

Pradermchai Kongkam, M.D.

A 55-year-old female patient presented with abdominal distension. CT scan revealed a pelvic mass with ascites. Exploratory laparotomy revealed a pelvic mass which was later removed. Pathology was consistent with neuroendocrine tumor. Subsequently, a colonoscopy was scheduled for an evaluation of possible invasion of the disease. A rectal sub-epithelial mass was identified as shown in Figure 1. Biopsy showed neuroendocrine tumor. EUS was then scheduled for an evaluation of the lesion in the rectum. The procedure was performed with a radial Scan Ultrasonic Video Endoscope EG-530UR2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan). It revealed a homogeneous hypoechoic mass measuring about 20x6 mm in diameter in rectum as shown in Figure 2. The mass originated from 2nd layer of rectal wall. This was consistent with neuroendocrine tumor which was proven from the previous biopsy.

Diagnosis:

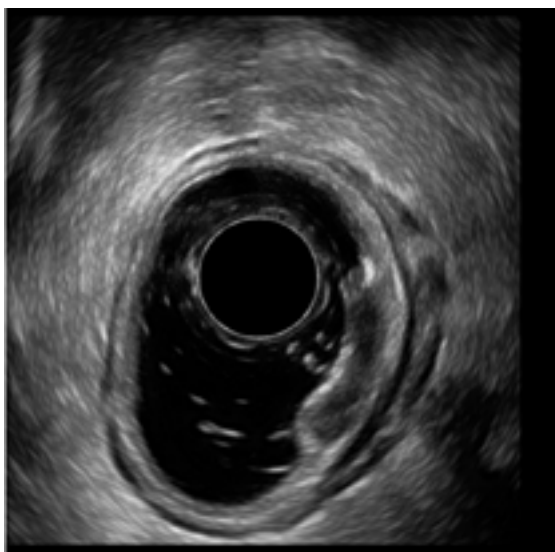
Rectal neuroendocrine tumor



Discussion:

Sub-epithelial lesions in the rectum can develop from various diseases. Neuroendocrine tumor is one of those. Preoperative non-invasive diagnosis is crucial as it can determine for the specific treatment. Mucosal biopsy with jumbo forceps can occasionally provide the definite diagnosis. A recent retrospective studies from 6 referral centers recruited 129 patients with sub-epithelial lesions, all underwent EUS with jumbo biopsy forceps, a definite diagnosis was made by jumbo biopsy forceps use in 76 from 129 patients (58.9%). Forty-five of 129 patients (34.9%) had significant bleeding requiring some form of endoscopic hemostasis¹. EUS is considered an investigation of choice to delineate rectal wall layers and it can guide the most likely diagnosis of sub-epithelial lesions with low complication rate². In this case, the patient already had a mucosal biopsy which showed neuroendocrine tumor. Therefore, EUS was then only a confirmatory test to confirm the presence of neuroendocrine tumor.

■ Figure 1: Demonstrated a rectal sub-epithelial mass with a post-biopsy ulcer on the surface of lesion.



■ Figure 2: Demonstrated a homogeneous hypoechoic mass originating from the 2nd wall layer of rectum. The procedure was performed by a radial scan Ultrasonic Video Endoscope EG-530UR2 (FUJIFILM Corporation, Tokyo, Japan) and Ultrasound Processor SU-8000 (FUJIFILM Corporation, Tokyo, Japan).

References

1. Buscaglia JM, Nagula S, Jayaraman V, et al. Diagnostic yield and safety of jumbo biopsy forceps in patients with subepithelial lesions of the upper and lower GI tract. *Gastrointest Endosc* 2012;75:1147-52.
2. Larghi A, Verna EC, Ricci R, et al. EUS-guided fine-needle tissue acquisition by using a 19-gauge needle in a selected patient population: a prospective study. *Gastrointest Endosc* 2011;74:504-10.



Case 18

Tanassanee Soontornmanokul, M.D.

Phonthep Angsuwatcharakon, M.D.

Linda Pantongrag-Brown, M.D.

Pradermchai Kongkam, M.D.

Rungsun Rerknimitr, M.D.

A 74-year-old woman with advanced stage of colon cancer presented with obstructive jaundice for 3 weeks. CT scan of the upper abdomen (Figure 4-5) shows multiple liver metastases, and matted necrotic lymphadenopathy around the hepatoduodenal ligament. These large nodes encased and possible invaded the distal common bile duct (CBD) resulting in obstruction and dilatation of the CBD and upstream bile ducts.

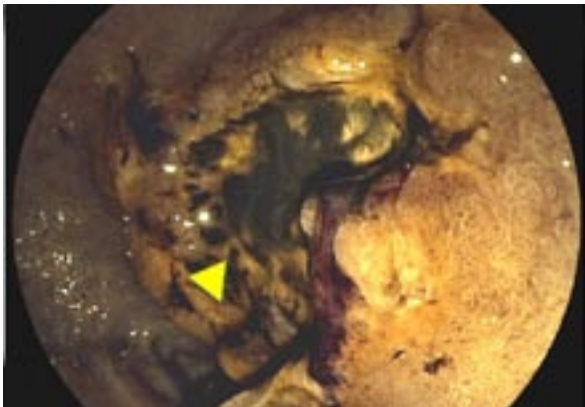
Findings were compatible with liver metastasis from colon cancer. Finally, ERCP was performed and revealed common bile duct obstruction from tumor with possible intraductal growth and D1-D2 junction (Figure 1-3). FICE system demonstrated ulcerative metastatic tumor at the D1-D2 junction. Finally, an uncovered self expandable metallic stent was deployed to bypass the obstruction via the ampulla (Figure 6).



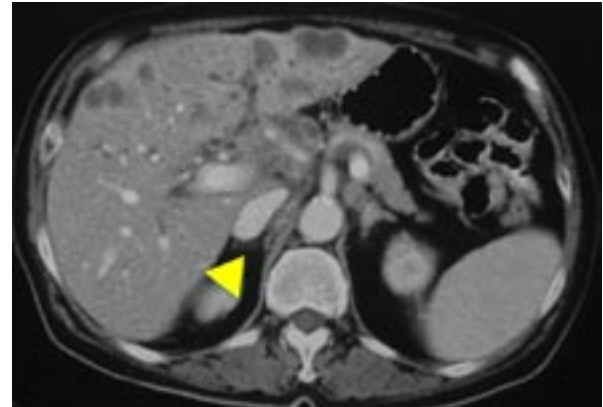
■ Figure 1: White light imaging



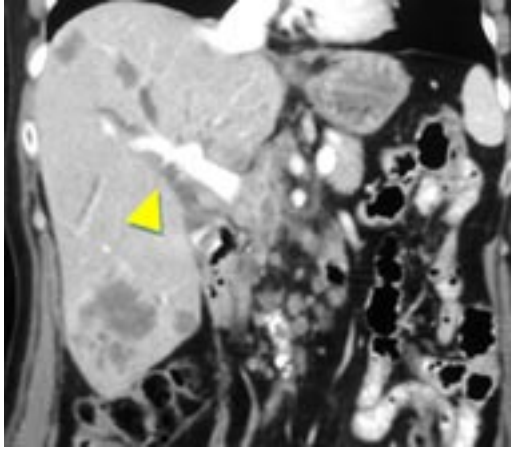
■ Figure 2: FICE station 2



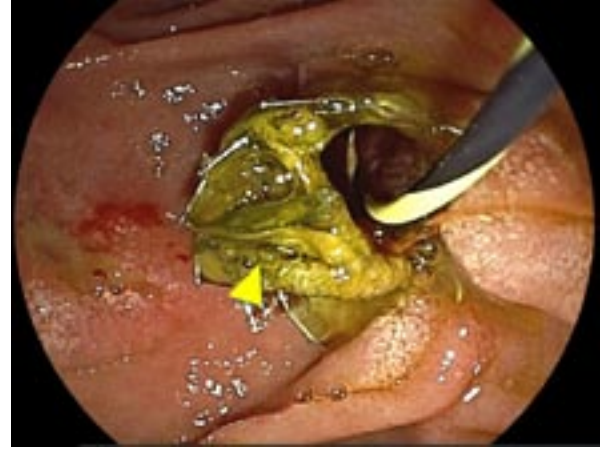
■ Figure 3: FICE station 4



■ Figure 4: FICE station 4



■ Figure 5: CT upper abdomen



■ Figure 6: Uncovered SEMS was inserted to bypass the obstruction.

Diagnosis:

Intraductal metastasis from advanced colonic adenocarcinoma

Discussion:

There are several causes of jaundice in patients with advanced colonic cancer including hepatic parenchymal metastasis, extrinsic compression by hilar adenopathy, tumor infiltration of ductal walls, and intrabiliary filling defects by masses of sloughed cells, mucus, clots, or tumor^{1,2}. Intrinsic involvement of bile ducts by colonic cancer, either by growing primarily within intrahepatic or extrahepatic bile ducts, is an unusual pattern of tumor growth³. It has been proposed that peribiliary capillary plexus communicating either with portal veins or hepatic arteries may be the route of such a particular kind of metastasis. Another possibility is that the tumor directly metastasizes to the bile duct⁴. Pivoski, et al. reported colonic intrabiliary metastases in 14 patients and found that all patients who were imaged had intrahepatic ductal dilatation, but only 2 of them had jaundice and intrabiliary filling defects². Intrabiliary tumor growth may be accompanied with hepatic parenchymal metastasis or, less commonly, the solitary intrabiliary tumor growth on imaging³. Sometimes it is difficult to make the differential diagnosis with the

papillary type of cholangiocarcinoma. A definite diagnosis should be established by careful attention to medical history, and immunostaining of CK7 and CK20. A CK20-positive and CK7-negative pattern is highly characteristic of liver metastases from colorectal cancer⁵.

Recently, imaging color enhancement can be helpful for detecting early neoplasia in the biliary tree, especially when using direct per oral cholangioscopy^{6,7}. However, prospective randomized studies are required.

References

1. Riopel MA, Klimstra DS, Godellas CV, et al. Intrabiliary growth of metastatic colonic adenocarcinoma: a pattern of intrahepatic spread easily confused with primary neoplasia of the biliary tract. *Am J Surg Pathol* 1997;21:1030-6.
2. Pivoski SP, Klimstra DS, Brown KT, et al. Recognition of intrabiliary hepatic metastases from colorectal adenocarcinoma. *HPB Surg* 2000;11:383-90
3. Ghittoni G, Caturelli E, Viera FT. Intrabiliary duct metastasis from colonic adenocarcinoma without liver parenchyma involvement: contrast enhanced ultrasonography detection. *Abdom Imaging* 2010;35:346-8.

4. Kubo M, Sakamoto M, Fukushima N, et al. Less aggressive features of colorectal cancer with liver metastases showing macroscopic intrabiliary extension. *Pathol Int* 2002;52:514-8.
5. Uehara K, Hasegawa H, Ogiso S, et al. Intrabiliary polypoid growth of liver metastasis from colonic adenocarcinoma with minimal invasion of the liver parenchyma. *J Gastroenterol* 2004;39:72-5.
6. Kim HK, Moon JH, Choi HJ, et al. Early bile duct cancer detected by direct peroral cholangioscopy with narrow-band imaging after bile duct stone removal. *Gut Liver* 2011;5:377-9.
7. Itokawa F, Itoi T, Sofuni A. Peroral video-cholangioscopy using narrow band imaging for early bile duct cancer (with video). *J Hepatobiliary Pancreat Sci* 2010;17:731-2.



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