Beyond the Limited Images (BLI): The 7th GI Endoscopy Atlas

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Editors
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Over a decade that the image enhancement has been introduced into diagnostic GI Endoscopy. The concept of using the specific wavelength to facilitate the observation in the change of inflammation and neoplasm is very helpful to characterize those lesions in real time. However, the tradeoff of this technology is the limitation in illumination. Until recently, the new concept of using laser as the light source so called “Blue Laser Image (BLI)” is very intriguing since the specific wavelength can be selected while reserving the level of illumination. In addition, the new technique allows the additional enhancement in the illumination power of the white light to better see the object from a far distance.

BLI is a break-through technology that allows endoscopist to examine epithelial lesion without the barrier of darkness when compared with the previous technology. In this atlas the readers will experience in the usefulness of BLI/BLI-bright in many pathological GI lesions including upper tract, lower tract, and small bowel. Moreover, the additional sections on ERCP and EUS are the important parts of educating material in this atlas as they provide many standard and advanced beneficial uses of these therapeutic armamentariums.

We hope that readers will enjoy and gain the knowledge in GI endoscopy by spending time through this latest atlas.

Sincerely,

Prof. Rungsun Rerknimitr, M.D.
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January 20, 2015
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A 45-year-old Thai female without underlying disease presented with iron deficiency anemia. EGD revealed multiple cherry-red areas with small radiating vessels predominately at the gastric fundus and greater curvature size 0.3-0.5 cm. Some of them were oozing blood (Figures 1-3). Argon plasma coagulation (APC) therapy was successfully achieved hemostasis.
Diagnosis:
Gastric angiodysplasia

Discussion:
Angiodysplastic lesions are widely used to describe abnormal vascular lesions at the mucosa of gastrointestinal (GI) tract. The pathogenesis starts from submucosal veins get obstructed and subsequently superficial vessels become dilated and then the communications between small veins, venules and capillaries develop as this can be defined as arteriovenous fistulas or malformation.\textsuperscript{1,2} Endoscopically, irregular, flat, reddish mucosal lesions and sometimes with active blood oozing is the typical finding of angiodysplasia.

Angiodysplasias of the upper GI tract are commonly found in the stomach and duodenum and cause as 4-7% of upper GI bleeding.\textsuperscript{3} Clinical presentations range from occult GI bleeding to massive GI bleeding. Initially, endoscopic therapy is the mainstay for bleeding cessation, especially APC, but re-bleeding after endoscopic therapy can occur in 20-25\%.\textsuperscript{3}

References
Case 2

A 68-year-old female presented with dyspepsia for 6 months. EGD was performed and showed 3 gastric polyps with marked erythematous and swelling mucosa. The largest polyp was 3 cm. in diameter and had small ulcer on top (Figure 1). Blue laser endoscopy showed dilated gastric pit and dense capillary loops (Figure 2). Polypectomy was performed at the largest polyp. Histopathological result was compatible with hyperplastic polyp (Figure 3).

**Figure 1** White light endoscopy showed gastric polyp sized 3 cm. with small ulceration on top.

**Figure 2** Blue laser endoscopy showed dilated pit and dense vascular loop of capillary. Note, small ulceration appeared as white area similar to white light endoscopy.

**Figure 3** Presented marked foveolar hyperplasia, dilated tortuous glands, and edematous stroma.
Diagnosis:
Hyperplastic polyp

Discussion:

Hyperplastic polyp develops from inflammatory proliferation of the gastric foveolar cells. It is associated with \textit{H. pylori} infection and autoimmune gastritis.\textsuperscript{1} Hyperplastic polyp can occur anywhere in the stomach but frequently present at the antrum. Endoscopic features show smooth, dome-shaped and multiple polyps.\textsuperscript{2} Their sizes are generally measured up to 1 cm. in diameter.\textsuperscript{3} However, large hyperplastic polyp can be found and may become lobulated or pedunculated leading to pyloric obstruction.\textsuperscript{1} Moreover, surface epithelium can be eroded and causes GI bleeding.\textsuperscript{3} Potential risk of cancer increases in a polyp larger than 1 cm, therefore hyperplastic polyps sized more than 1 cm. should be completely excised.\textsuperscript{1}

References

Case

Sayamon Kimtrakool, M.D.
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A 64-year-old Thai male presented with early satiety and weight loss (6 kgs in 2 months). Physical examination showed cachexia with mild anemia. Laboratory showed a low hemoglobin level compatible with iron deficiency anemia. EGD revealed retention of food content in the stomach that contained enlarged gastric folds. The gastric mucosa appeared friable and the stomach was not fully distended after air inflation (Figures 1-2). The antrum and duodenum looked normal. Histopathological report showed eccentrically located nuclei compressed by intracytoplasmic mucins with positive for mucin and AE 1/3 (Figure 4). These were compatible with poorly differentiated (signet-rig) adenocarcinoma.

Figures 1 and 2 EGD showed enlarged gastric folds with poor compliance to air insufflation.

Figure 3 EGD revealed thickened gastric folds at the body with normal antrum (antral sparing).
Diagnosis:

Gastric linitis plastica

Discussion:

Gastric linitis plastica is usually caused by diffuse infiltrative adenocarcinoma, typically in a signet-ring type.\(^1\) The endoscopy mostly show thickened gastric folds with poor compliance on air insufflation. The gastric submucosa is frequently infiltrated by malignant cells with edematous mucosa that contains no malignant cells.\(^2\) Thus superficial biopsy alone sometimes may fail to make a diagnosis. To increase diagnostic yield, multiple forceps biopsies at the same site or sonography-guided needle biopsy is recommended.\(^3\) The differential diagnosis of thickened gastric folds included malignancy (eg. lymphoma, adenocarcinoma), infection (eg. tuberculosis, histoplasmosis), amyloidosis, Menetrier’s disease or Zollinger-Ellison syndrome.\(^2\)

References


\(^1\) Ref. 1
\(^2\) Ref. 2
\(^3\) Ref. 3
A 56-year-old Thai female presented with early satiety for 6 months. She had no weight reduction and denied nausea/vomiting. Physical examination was normal. EGD revealed distortion of gastric anatomy and twisted gastric fold but the scope can pass through the second part of duodenum (Figures 1-2). Barium swallowing showed that the position of EGJ is lying above the left hemi-diaphragm (Figure 3) with reverse position of the greater curvature of stomach (Figure 4).
Diagnosis:

Sliding hiatal hernia with chronic gastric volvulus (organo-axial type)

Discussion:

Gastric volvulus is a rare clinical entity and truly unknown the incidence or prevalence. The clinical presentation is ranging from acute abdominal pain to chronic abdominal discomfort depend on the rapidity of onset and the degree of twisting. Acute gastric volvulus is classically characterized by Borchardt’s triad of epigastric pain, vomiting and inability to pass a nasogastric (NG) tube. However, chronic gastric volvulus usually present with mild and non-specific symptom such as abdominal distension after meals, uncharacteristic epigastric pain or dysphagia.

Gastric volvulus divided into primary and secondary etiology. Primary gastric volvulus may arise from laxity of perigastric ligaments result in abnormal rotation of the stomach. Secondary gastric volvulus is usually related with diaphragmatic hernias, hiatal hernias or eventration of the diaphragm. Type of gastric volvulus is classified to the axis rotation of the stomach, including organo-axial and mesentero-axial. Organo-axial volvulus occurs along the luminal long axis which is the most common type (60%) and associated with diaphragmatic defects, whereas mesentero-axial volvulus occurs a plane perpendicular to the luminal axis and less common related to diaphragmatic defects.

The endoscopic appearance is the difficulty of intubating the stomach, distortion of gastric anatomy, and twisted gastric folds. However, the standard test to diagnose this entity is still relied on barium meal since the relationship of all stomach landmarks to the surrounding structures can be observed much better than an observation by endoscopy.

References

A 60-year-old man underwent EGD due to chronic reflux symptom. EGD revealed a 0.5 cm. nodule at the gastroesophageal junction with dilated gastric pit. Bright blue laser imaging (BLI-Brt) showed additional information on dilated and tortuous vessels (Figures 1-2). These findings were compatible with hyperplastic polyp of the cardia.

**Diagnosis:**
Hyperplastic polyp at the gastroesophageal junction

**Discussion:**
Hyperplastic polyp of the gastroesophageal junction (EGJ), also called inflammatory esophagogastric polyp, are uncommon lesions characterized by hyperplastic epithelium (foveolar-type, squamous, or both) with variable amounts of inflamed stroma and endoscopic finding showed a nodule or polyp with/without erythematous mucosa. It often occurred in combination with gastroesophageal reflux disease (GERD) without gastric pathology.\(^1\)\(^2\) It may represent an exaggerated mucosal response to esophageal injury, including reflux associated ulceration and repair, protracted vomiting, Crohn’s disease, post-sclerotherapy for varices, heterotopic gastric mucosa, embedded pills,
infection, and anastomotic related ulcer. This hyperplastic polyp of the stomach is associated with atrophic gastritis with intestinal metaplasia, and Helicobacter pylori-associated gastritis which may regress after treatment. In contrast, hyperplastic polyp of the esophagus has a significantly higher rate of multilayered epithelium and associated with ultrashort (<1 cm.) or short segment (1 to 3 cm.) Barrett’s esophagus.\textsuperscript{3,4} Hyperplastic polyp of the EGJ has minimal malignant potential, therefore endoscopic removal is not necessary.\textsuperscript{5}

References

A 60-year-old man with the history of gastric cancer had undergone partial gastrectomy with gastrojejunostomy (Billroth II) 40 years ago. He presented with chronic dyspepsia and iron deficiency anemia for 4 months. EGD was performed and showed a large polypoid mass at the body with another ulcerative mass at the anastomosis (Figures 1-2). Histopathology showed crowded tumor cells, arranging in solid sheets and complex glands. Those cells pose pleomorphic nuclei with visible nucleoli (Figures 3-4).
Diagnosis:
Moderately differentiated adenocarcinoma of stomach after partial gastrectomy

Discussion:
Gastric adenocarcinoma is the third leading cause of cancer related death in both sexes worldwide.\(^1\) It has been associated with risk factors such as low socioeconomic status, cigarette smoking, nitrites, chronic gastritis, \textit{H.pylori} infection and previous gastrectomy.\(^2\) The patient who underwent gastrectomy had greatest risk of gastric cancer at 15 to 20 years after surgery. The relative risk is 1.5 to 3.0, depending on the type of surgery.\(^3,4\) Billroth II may increase risk 4-fold higher than Billroth I.\(^5\) The cancer tends to occur at or near the surgical anastomosis on the gastric side. The proposed mechanisms were hypochlorhydria resulting in gastric bacterial overgrowth, which increased production of nitrites, chronic enterogastric reflux of bile salts and pancreatic enzymes and atrophy of the remaining fundic mucosa secondary to low levels of antral hormones such as gastrin.\(^6\)

References
A case of 65-year-old Thai male who presented with progressive dysphagia for two months. He was a diagnosed case of hepatocellular carcinoma and chronic hepatitis B who underwent liver transplant two years ago and band ligation for esophageal varices a month prior to surgery. MRI of the abdomen taken 2 months before consult revealed tumor thrombus at the portal vein with porto-systemic collaterals and multiple lymph nodes at the peri-aortic and caval area, no esophageal mass seen (Figure 1). EGD was done that showed a solitary polypoid mass approximately 4 cm. size with some necrosis on its surface, biopsy was taken. Three medium size varices, an ulcer and tissue scar from previous ligation were also noted (Figure 2). Histopathology revealed poorly differentiated carcinoma on H&E staining, positive for hepatocyte immunostaining and negative CK56 (Figure 3).

**Figure 1** Evidence of tumor thrombus on the portal vein and presence of collaterals, multiple aortic and para-caval lymph nodes, no esophageal mass seen.

**Figure 2** Polypoid mass with columns of esophageal varices and an ulcer A. Under White light Endoscopy B. Under BLI Bright (BLI-Bright).
Diagnosis:

Esophageal metastasis from hepatocellular carcinoma

Discussion:

Hepatocellular carcinoma is the most common primary malignant tumor of the liver. They usually metastasized to the lungs and regional lymph nodes as well as adrenal glands.\(^1\) Gastrointestinal tract involvement only occurs about 0.5-2% and the esophagus is the least to be metastasized accounting for less than 0.4%.\(^2\)

There are two different hypotheses concerning the way HCC metastasizes to the esophagus: either by direct invasion of the gastrointestinal tract via continuation between the serosal side of a liver tumor and the esophagus, or by the hematogenous spread of tumor emboli infiltrating via the portal vein system and being disseminated by hepatofugal portal blood flow to the esophagus.\(^3\)

In this patient, the presence of tumor thrombus in the portal vein along with reversal of blood flow form portal hypertension can possibly support that the spread of tumor emboli to the esophagus.

Doubling time for HCC is about 119 +/- 96 days (mean SD) and it becomes short at 48 +/- 8 days in the presence of HBV.\(^4\) This can likely explain why the tumor was not appreciated on the MRI taken two months prior to the endoscopy. This maybe a recent tumor implant that has a rapid growth rate.


References


A 70-year-old Thai male with an underlying of polycytemia vera presented with melena for 1 day. EGD revealed isolated enlarged tortuous vein located at the fundus and greater curvature with white nipple sign but no evidence of esophageal varices (Figures 1 and 2). Histoacryl 0.5 ml. combined with lipiodol 0.8 ml. was injected at the white nipple sign for hemostasis. A CT scan of the upper abdomen was done for a work up of the cause of isolated gastric varices. It showed embolization material at the gastric fundus and the greater curvature without the evidence of splenic vein thrombosis (Figure 3).
Diagnosis:

Bleeding isolated gastric varices (IGV1) without the evidence of splenic vein thrombosis

Discussion:

Isolated gastric varices (IGV) means the presence of gastric varices in the absence of esophageal varices. IGVs are classified into 2 groups; IGV1 and IGV2. IGV1 locates at the gastric fundus whereas IGV2 locates at the gastric antrum, body or pylorus. Splenic vein thrombosis is one of the most common causes of IGV.

Management of acute gastric variceal bleeding should be done with an endoscopic variceal obliteration by using tissue adhesives such as cyanoacrylate. Cyanoacrylate injection is effective for bleeding control with some reports of complication. Endoscopic sclerotherapy or endoscopic band ligation (EVL) are not recommended as the first choice for gastric varices treatment.

References

A 53-year-old female presented with dyspepsia, early satiety, anorexia and significant weight loss for 3 months. She had unremarkable medical history and no family history of cancer. EGD was done and revealed circumferential ulcerative mass at the gastric body with antral sparing. The scope can easily pass through the pylorus but the mucosa was friable with contact bleeding (Figure 1). Biopsy was done at the lesion and the pathology revealed diffusely malignant round cell neoplasm compatible with aggressive NHL (Figure 2). Immunohistochemistry stain was positive for CD 20 but negative for CD3 (Figure 3). These results confirmed the diagnosis of diffuse large B-cell lymphoma (DLBCL). Her bone marrow study was normal.
Diagnosis:
Primary gastric diffuse large B-cell lymphoma (DLBCL)

Discussion:
Gastrointestinal tract is the most common extranodal site involved by lymphoma accounting for 5-20%.¹ The stomach is the most commonly affected organ, accounting for 60-70% of GI lymphomas. The other common GI lymphoma are small intestine lymphomas (20-35%) and colorectal lymphomas (5-10%).² The histological types of gastric lymphomas are mainly represented by MALT lymphoma, a form of indolent disease, followed by the diffuse large B cell lymphoma (DLBCL), a type of aggressive malignancy.² The endoscopic pattern of gastric DLBCL are ulcerations (with single or multiple ulcerations) specially located at the gastric body or at the fundus, diffuse infiltrative, and polypoid mass.¹² Endoscopic findings that achieved statistical significance in favor of lymphoma than gastric adenocarcinoma are extensive disease involving the whole stomach, proximal stomach involvement, extension of tumor into duodenum and presence of volcano crater-like ulcers on polypoid lesions.³

References
A 70-year-old male presented with hematemesis and EGD showed a large gastric ulcer at the lesser curvature with non bleeding visible vessel. Bipolar coaptation was done to achieve hemostasis. Two days later, recurrent hematemesis developed and re-EGD revealed a spurting vessel from the same ulcer (Figure 1). After diluted adrenaline injection for a temporary hemostasis, Histoacryl 0.5 ml. plus lipiodol 0.8 ml. was injected directly into the visible vessel (Figure 2). No bleeding recurred but mild abdominal pain developed. A week later, follow-up EGD showed a large gastric ulcer with surrounded necrotic mucosa without active bleeding or visible vessel resembling partial gastric infraction (Figure 3). CT scan of the abdomen assured that no full thickness infarction was present. Thus conservative treatment continued and patient was discharged safely within 2 weeks.

Figure 1 EGD showed a large gastric ulcer with spurting visible vessel at the lesser curvature.

Figure 2 Histoacryl plus lipiodol was injected into the visible vessel and hemostasis was achieved.

Figure 3 A week later after Histoacryl injection, EGD showed a large gastric ulcer with some necrotic areas resembling submucosal gastric infraction.
Diagnosis:
Submucosal gastric infarction developed after Histoacryl injection for bleeding gastric ulcer

Discussion:
Endoscopic therapy is the first line treatment for bleeding peptic ulcers. Combination therapy with epinephrine injection is suggested in high risk ulcer.\(^1\) Endoscopic hemostasis using Histoacryl was first proposed in 1984 by Gotlib et al., for variceal bleeding. Since then, it has become an effective therapeutic option for gastroesophageal varices and a useful alternative modality for bleeding ulcer.\(^2\) The principal mechanism of its action is a chain polymerization reaction, triggered by contact between the cyanoacrylate and blood, which transforms its original liquid into solid substance.\(^3\) The efficacy of Histoacryl injection for bleeding peptic ulcer is 92% for an initial hemostasis, 83% for permanent hemostasis and rebleeding rate is only 18%.\(^4\) However, in spite of its high hemostatic efficacy, several undesired side effects associated with this procedure limit its popularity. Side effects include dysphagia, retrosternal pain, pyrexia, rebleeding due to detachment of the solidified compound, sepsis, mediastinitis and systemic embolization such as branches of celiac axis cause multiple infarction of liver, gallbladder, pancreas, spleen and stomach.\(^3\) Therefore Histoacryl injection should be considered as the last choice for the treatment of bleeding peptic ulcers.\(^3,5\)

References
A 65-year-old Thai female without underlying disease presented to our GI clinic due to reflux symptoms. EGD under standard white light revealed indented Z line at the EG junction (Figure 1). After switching to Blue Laser Imaging (BLI), this lesion was clearly seen with ridge pattern of mucosal crack (an arrow in Figure 2). Moreover, under the high magnifying BLI, it demonstrated the better detail of ridge pattern vascularity, whereas the standard white light failed to do so (Figures 3 and 4).

Figures 1 and 2 EGD under conventional white light endoscopy showed minimal erythematous mucosa at the EG junction (Left) the improved visualization of ridge pattern was better seen under Blue Laser Imaging (BLI) (Right; arrow).

Figures 3 and 4 Under high magnification, white light endoscopy showed only lazed vascular pattern (Left) whereas BLI better demonstrated dilated and tortuous vessels of the ridge pattern at the EG junction (Right; arrow).
Diagnosis:

Minimal change esophageal reflux disease (MERD)

Discussion:

Minimal change esophageal reflux disease (MERD) is determined by white light endoscopy in the presence of whitish cloudiness of distal esophageal mucosa obscuring blood vessels or erythema area with the absence of clear demarcation. However, the conventional white light endoscopy may be suboptimum for MERD detection and many of those being misdiagnosed as NERD (Non-erosive reflux disease). In the era of digital chromoendoscopy, MERD can be diagnosed better without magnification by using the criteria of triangular indentation, ridge or villiform patterns at the GE junction, increased number and dilated intrapapillary capillary loops (IPCLs), and punctuate erythema at the distal esophagus with 77% sensitivity, 83% specificity, 55% NPV, 93% PPV and 79% accuracy. However with the additional magnification, the previous studies did not show the better diagnostic value for MERD.

References
A 77-year-old Thai male with the underlying of severe Alzheimer’s disease and inadequate oral feeding was on scheduled for a percutaneous endoscopic gastrostomy (PEG) tube placement. He had no history of prior abdominal surgery. He denied the use of antiplatelet and anticoagulant. The physical examination was unremarkable.

**Diagnosis:**

Percutaneous endoscopic gastrostomy (PEG) tube placement

**Discussion:**

Percutaneous endoscopic gastrostomy (PEG) is one of the enteral feeding access by using an endoscopic assisted percutaneously placing a tube into the stomach. Among all techniques, PEG-pull technique is the most commonly used technique\(^1\) and was chosen in this patient. At the beginning, EGD is performed to insufflate the stomach. Then, finger pressure applies at the transillumination point for locating the incision area which should be around 1 inch below left costal margin. The skin is marked and sterilized with aseptic agents. After that, lidocaine is injected by a long needle from the skin through the abdominal wall until presenting in the endoscopic view to confirm the depth of the puncture site. Subsequently a 0.6-0.8 cm. skin incision was done at the marked point and a Trocar catheter was inserted through the incision into the stomach. The needle is replaced by a looped guide-wire (Figure 1) which is ensnared and pulled out through the mouth all together during endoscopy withdrawal. The end of PEG tube is fixed with the looped guide-wire and then pulled back into the mouth through the skin incision from the other site. Finally, the bumper will be attached at the internal incision.\(^2\) Re-endoscopy can be performed to confirm the position of bumper (Figure 2).
Figure 1 EGD showed placing the looped guide-wire through the catheter into the stomach.

Figure 2 EGD showed the bumper located at the proper area.

References
A 38-year-old man had a history of melena for 1 week. EGD revealed one clean-based ulcer sized 3 cm. in diameter at the gastric body. After the treatment to eradicate *H. pylori*, a repeat EGD was performed and the ulcer was still persist (Figures 1-4). Histopathology showed tumor cells, arranging in solid sheet and complex glands. Those cells pose intracytoplasmic mucin and pleomorphic nuclei with visible nucleoli as shown in Figures 5-6.

Figures 1-2 Under white light endoscopy, an ulcerative mucosal mass with elevated irregular border was seen in the body of stomach.

Figures 3-4 BLI-bright showed loss of gastric pit with corkscrew patterns of vascular structure of the gastric mucosa.
Diagnosis:
Poorly differentiated signet ring cell gastric carcinoma

Discussion:
Giant gastric ulcer (>3 cm.) accounts for as many as 10% to 24% of all gastric ulcers. These often have more aggressive disease, with a higher incidence of bleeding and higher mortality rates (10% vs. 3%) compared with patients with smaller ulcers.\(^1\) Gastric ulcer that suggestive of malignancy has specific features such as an associated mass lesion, elevated irregular ulcer borders, and abnormal adjacent mucosal folds. With magnifying endoscopy, fine network patterns and loop patterns are mostly associated with well- or moderately-differentiated adenocarcinoma whereas corkscrew patterns or wavy microvessels mostly correspond to the poorly-differentiated adenocarcinoma.\(^2\) Histopathologic finding often shows signet-ring-like cells with intracytoplasmic mucin pooling and many times the mucin poolings push the nuclei to periphery.\(^3\)

References
A 46-years-old male patient presented with chronic epigastric pain radiating to the back. EGD showed hemicircumferential erythema and edematous mucosa located at superomedial site of D1-D2 junction just proximal to the ampulla (Figure 1). Ct scan of the abdomen revealed hemicircumferential thickening of the duodenal wall and edematous parenchyma of the pancreatic head (Figures 2 and 3).

Diagnosis:
Groove pancreatitis
Discussion:

Groove pancreatitis is a segmental chronic pancreatitis that affects the anatomical area between the pancreatic head, the duodenum, and the common bile duct, referred to as the groove area. Clinical features of groove pancreatitis are shown in Table 1. Distinguishing groove pancreatitis from peripancreatic cancer is clinically important. Multiple biopsies through duodenal mucosa are useful to ensure the diagnosis.

Table 1 Clinical features of groove pancreatitis

<table>
<thead>
<tr>
<th>Feature</th>
<th>Clinical presentation</th>
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<tr>
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<td>Males &gt; Females</td>
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<td></td>
<td>History of heavy alcohol usage</td>
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<td></td>
<td>Chronic pancreatitis</td>
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<td></td>
<td>Marked nausea, vomiting, and weight loss</td>
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<td></td>
<td>Post-prandial abdominal pain</td>
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<td></td>
<td>Jaundice is rare</td>
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<td></td>
<td>Duodenal stenosis with edema and inflamed polypoid mucosa</td>
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<td></td>
<td>Sheet-like, hypodense, poorly enhancing mass between pancreatic head and thickened duodenal wall</td>
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<td></td>
<td>Typical anatomic changes of chronic pancreatitis</td>
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<td></td>
<td>Minimal dilation of the common bile duct</td>
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<td>Duodenal luminal narrowing due to wall thickening ± cysts within duodenal wall</td>
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<tr>
<td></td>
<td>Smooth tubular stenosis of common bile duct</td>
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<tr>
<td></td>
<td>Santorini duct usually not identified or obliterated by mass ± penetrating duct sign</td>
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<tr>
<td></td>
<td>Giant duodenal scarred/ulcerated folds with cystic changes</td>
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<td>Brunner gland hyperplasia</td>
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<td>Extensive duodenal submucosal fibrosis</td>
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<td>Myoid proliferation (myoadenomatosis)</td>
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<td></td>
<td>Cystically dilated ducts or pseudocysts</td>
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<td>Dilated ducts with mucoproteins</td>
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References

A 57-year-old woman with underlying chronic kidney disease and on kidney transplantation list underwent EGD due for cancer screening. She was asymptomatic for GI condition. Her physical examination was unremarkable. EGD revealed a 1 cm. solitary erythematous regular border nodule with ulcer on top at the duodenal bulb (Figure 1). Biopsy was done and histopathology showed moderate chronic and acute duodenitis with ulcer. Some areas showed gastric metaplasia and inflammatory polyp without organism (Figure 2).

**Diagnosis:**
Gastric metaplasia with inflammatory polyp

**Discussion:**
Gastric metaplasia (GM) of duodenum is the presence of gastric-type mucus secreting cells or gastric foveolar epithelium at the surface epithelium of duodenum. It represents the process of duodenal inflammation. The prevalences of GM at different areas were 72 to 90% in duodenal ulcer, 40 to 95% in duodenitis, and 0 to 33% in the normal endoscopic appearance of duodenum,
respectively.\textsuperscript{1,2} Gastric metaplasia usually restricted to duodenal bulb and the diagnosis is based on biopsy result. Alcian blue/Periodic acid-Schiff stain can perform to ensure the definite diagnosis. Endoscopic findings are elevated patches, flat erythematous areas, thickening of mucosal fold, focal or diffuse nodularity, and duodenal mass.\textsuperscript{3} The etiology of gastric metaplasia is still controversial. \textit{H.pylori} was previously mentioned as the cause of gastric metaplasia. However, the recent study found that the prevalence of \textit{H.pylori} infection increased in gastric metaplasia with active inflammation but no strong evidence increased in gastric metaplasia without inflammation.\textsuperscript{4} Moreover, successful eradication of \textit{H.pylori} did not lead to any change in the endoscopic appearance.\textsuperscript{5}

References


A 45-year-old female with underlying familial adenomatous polyposis (FAP) had had a total colectomy many years ago. She underwent a surveillance EGD for upper gastrointestinal polyp. EGD revealed multiple flat polyps at the duodenum with white villi compatible with adenomatous polyps (Figures 1 and 2).

**Diagnosis:**
Duodenal adenoma in Familial adenomatous polyposis (FAP) patient.

**Discussion:**
Familial adenomatous polyposis (FAP) related small bowel adenoma can finally developed to full-blown carcinoma. Duodenal adenoma can be found as polypoid, sessile or depressed polyp that contains white villi (detected by white light endoscopy). This suggests the probability of adenoma. Under BLI the white villi can be more appreciated with better contrast over normal mucosa. The risk factors for carcinoma and recommendation for surveillance were addressed by Spigelman.\(^2,3\) (Tables 1, 2)
**Table 1** Spigelman grading of duodenal adenomas in familial adenomatous polyposis

<table>
<thead>
<tr>
<th>Points</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of polyps</td>
<td>1-4</td>
<td>5-20</td>
<td>&gt;20</td>
</tr>
<tr>
<td>Polyp size (mm)</td>
<td>1-4</td>
<td>5-10</td>
<td>&gt;10</td>
</tr>
<tr>
<td>Histology</td>
<td>Tubular</td>
<td>Tubulovillous</td>
<td>Villous</td>
</tr>
<tr>
<td>Dysplasia</td>
<td>Mild</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
</tbody>
</table>

Stage 0 = 0 points; stage I = 1-4 points; stage II = 5-6 points; stage III = 7-8 points; stage IV = 9-12 points

**Table 2** Recommendation for surveillance

<table>
<thead>
<tr>
<th>Spigelman stage</th>
<th>Bulow et al.</th>
<th>Gallagher et al.</th>
<th>Groves et al.</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>5 yearly endoscopy</td>
<td>5 yearly endoscopy</td>
<td>5 yearly endoscopy</td>
</tr>
<tr>
<td>1</td>
<td>5 yearly endoscopy</td>
<td>5 yearly endoscopy</td>
<td>5 yearly endoscopy</td>
</tr>
<tr>
<td>2</td>
<td>3 yearly endoscopy</td>
<td>2-3 yearly endoscopy</td>
<td>3 yearly endoscopy</td>
</tr>
<tr>
<td>3</td>
<td>1-2 yearly endoscopy</td>
<td>1-2 yearly endoscopy</td>
<td>1-2 yearly endoscopy</td>
</tr>
<tr>
<td>4</td>
<td>EUS consider surgery</td>
<td>EUS/CT consider surgery</td>
<td>consider surgery</td>
</tr>
</tbody>
</table>

**References**

A 65-year-old male had presented with cholangitis caused by periampullary adenoma. Subsequently an ampullectomy was performed and he came for a follow-up EGD as scheduled. EGD revealed papillary-like flat polyp at the neo-ampulla (Figure 1). FICE station 1 demonstrated white villi with normal vascular pattern (Figure 2). Biopsy was done and pathological report revealed a remnant of ampullary adenoma.

**Diagnosis:**
A remnant of ampullary adenoma

**Discussion:**
Ampullary adenoma is rare and usually asymptomatic.\(^1\) Primary carcinoma of the duodenum is an uncommon neoplasm. In a review of 117,433 post mortem examinations, only five patients established duodenal carcinoma. Therefore, the incidence of duodenal carcinoma in post mortem setting was only 0.04%.\(^2\) Magnifying endoscopy can provide clear images of fine surface structures on
the mucosal layer, a compact formation of pits and white villi can be seen in the affected ampulla. The microvascular architecture usually shows no abnormalities. The ampullary lesion can be resected by using endoscopic snare polypectomy, however there is a risk of recurrence.3

References
A 56-year-old female with end-stage renal disease (ESRD) secondary due to type 2 diabetic mellitus has been on maintenance hemodialysis for three years. She presented with iron deficiency anemia and positive stool occult blood. EGD was performed and showed multiple erythematous stripes of red tortuous ectatic vessels along the longitudinal rugal folds in the antrum converging toward the pylorus (Figure 1). Blue laser endoscopy showed dilated gastric pit with increased tortuous vasculatures in a uniform pattern (Figures 2-3). Argon plasma coagulation (APC) therapy was applied.

**Diagnosis:**

Gastric antral vascular ectasia (GAVE)
Discussion:

Gastric antral vascular ectasia (GAVE), though a rare disorder, causes up to 4% of non-variceal upper GI bleeding. GAVE is often associated with systemic illnesses, such as cirrhosis of the liver, autoimmune connective tissue disorders, bone marrow transplantation and chronic renal failure.\(^1\)\(^2\) Typical initial presentations range from occult bleeding causing transfusion-dependent chronic iron-deficiency anaemia to severe acute upper gastrointestinal bleeding. In non-cirrhotic patients with GAVE, the autoimmune diseases are most common underlying.\(^3\)

Non-cirrhotic patients more frequently present the typical endoscopic watermelon-, striped-pattern and are mainly represented by middle-aged women whereas the honeycomb-, diffuse-pattern prevails in patients with cirrhosis.\(^3\)\(^4\)

Endoscopic therapy, particularly treatment with argon plasma coagulation (APC), has shown to be as effective as surgery but safer, and endoscopic therapy should be considered as the first-line treatment for patients with GAVE-related bleeding.\(^4\)

References

A 92-year-old Thai female underwent EGD with a nasogastric scope (EG-580NW2, Fujifilm, Tokyo, Japan) during a PEG replacement and EGD revealed a small clean base ulcer opposite to the PEG bumper (Figures 1 and 2).

**Diagnosis:**

PEG bumper related gastric ulcer

**Discussion:**

Pathophysiology of gastric ulcer developed from a bumper is related to the ischemic process from pressure necrosis.¹ In a Japanese series, gastric ulcers were found in 9/92 patients (9.8%) who had PEG, and all of them were found at the posterior wall of the gastric body where the tip of the PEG tube was attached. 7 from 21 patients (33.3%) who had a PEG tube with a long protrusion of the intragastric bumper developed gastric ulcer, whereas only 2 from 71 patients (2.8%) who had a short protrusion of PEG tube developed ulcer.² Therefore, the appropriate length of the PEG tube is an important factor in preventing gastric ulcer.³

*Figures 1 and 2 showed clean base gastric ulcer opposite to the percutaneous endoscopic gastrostomy bumper (arrow).*
References

A 71-year-old man with old CVA was admitted due to hematemesis 6 hours prior to the admission. He underwent a percutaneous endoscopic gastrostomy (PEG) placement 2 days ago. He had stopped clopidrogrel for 7 days before the procedure and it was discontinued until this admission. He had normal vital signs but showed markedly pale conjunctiva. Abdominal examination was unremarkable. There was the presence of melena by per rectal examination. NG tube showed dark red blood content. Two units of packed red cell were transfused. EGD revealed blood oozing from the inner margin of gastrostomy (Figure 1). APC and hemoclipping were done but failed to control the bleeding. Then new balloon-type PEG was applied. Its tamponade effect stopped the bleeding (Figure 2). A follow-up EGD was done 2 days later and revealed no further bleeding from the PEG site (Figure 3).
Diagnosis:
Post PEG bleeding at the puncture site with successful hemostasis by a PEG balloon tamponade

Discussion:
Percutaneous endoscopic gastrostomy (PEG) is defined as a high-risk endoscopic procedure because there is a risk of bleeding in the absence of anticoagulants and antiplatelet agents (2–2.5%). Post PEG bleeding can be divided into immediate (<1 day), early (1-7 days), delayed (>7 days) and the severity of post-PEG bleeding is based on a modification of a grading system as mild, moderate and severe. Mild is defined by clinical or endoscopic evidence of bleeding but decrease in Hb <3 g/dL and no transfusion needed (modified from post sphincterotomy bleeding). The moderate means transfusion required about 4 units or less and neither angiographic intervention nor surgery needed, whereas severe hemorrhage means transfusion of 5 units or more and angiographic or surgical intervention needed. This patient was classified as early bleeding and moderate severity. Generally, severe bleeding is a rare complication that occurred in 0.02% to 0.06% of the cases. Causes of bleeding include esophagitis, gastric pressure ulcers, concomitant peptic ulcer disease and rarely puncture of the gastric wall vessel (usually bleeding comes from gastroepiploic artery or its perforating branches). Risk factors include anticoagulation, antiplatelet therapy, and the presence of an anatomic variation.
PEG balloon type with tamponade effect had been used to stop bleeding by tightening the external bolster against the abdominal wall, thereby tightening the internal bumper against the bleeding vessel, thus it may be tried to stop the bleeding. Compression should be released within 48 hours to prevent mucosal necrosis and pressure ulcer.⁴

References
A 74-year-old male with underlying diabetes mellitus, hypertension, chronic kidney disease and coronary heart disease was admitted due to cellulitis of the leg. Five days after admission, he developed coffee ground emesis and melena. EGD was performed and revealed a 0.5 cm ulcer with blood oozing from visible vessel at the duodenal bulb (Figures 1). Hemostasis was achieved by endoscopic band ligation.

**Diagnosis:**
Duodenal ulcer with blood oozing from visible vessel (Forrest Classification IB)

**Discussion:**
Ulcers are the most common cause of in-hospital upper gastrointestinal bleeding. Independent risk factors for gastrointestinal bleeding during hospitalization included age >60 years, male sex, liver disease, acute renal failure, sepsis, being on a medicine service, prophylactic anticoagulants, and coagulopathy. According to Forrest Classification of Stigmata of Recent Hemorrhage (SRH), active blood oozing is classified as IB classification which has an average re-bleeding rate, surgery rate, and mortality rate at 55%, 35% and 11% respectively (Table 1). Therefore, endoscopic therapy should be provided. Thermal therapy, injection of sclerosant, clipping, and epinephrine injection plus a second modality are the recommended endoscopic treatment for high risk ulcer. Endoscopic band ligation (EBL) is a standard of care for variceal bleeding. However, the efficacy for controlling peptic ulcer bleeding is also proposed but limited in experience.
Table 1  Stigmata of recent hemorrhage SRH and average rates of further bleeding, surgery, and mortality without endoscopic therapy.*

<table>
<thead>
<tr>
<th>Stigmata</th>
<th>Forest Classification</th>
<th>Further bleeding</th>
<th>Surgery</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active bleeding</td>
<td>IA and IB</td>
<td>55%</td>
<td>35%</td>
<td>11%</td>
</tr>
<tr>
<td>Non-bleeding visible vessel</td>
<td>IIA</td>
<td>43%</td>
<td>34%</td>
<td>11%</td>
</tr>
<tr>
<td>Adherent clot</td>
<td>IIB</td>
<td>22%</td>
<td>10%</td>
<td>7%</td>
</tr>
<tr>
<td>Flat pigmented spot</td>
<td>IIC</td>
<td>10%</td>
<td>6%</td>
<td>3%</td>
</tr>
<tr>
<td>Clean base ulcer</td>
<td>III</td>
<td>5%</td>
<td>0.5%</td>
<td>2%</td>
</tr>
</tbody>
</table>

*Modified from reference 2

References
A 44-year-old female presented with intermittent epigastric pain and early satiety for 2 years. EGD revealed gastric nodularity at the body of stomach with an overlying normal mucosa (Figure 1). Blue laser imaging (BLI) revealed dilated gastric pit with normal vascular pattern (Figure 2). Pathology revealed chronic gastritis without atypical cell.

**Diagnosis:**

Nodular gastritis

---

**Figure 1** EGD showed gastric nodularity at the body of stomach with an overlying normal mucosa.

**Figure 2** BLI showed normal background gastric pit and vascular pattern.
Discussion:

There is a strong association between nodular gastritis and *Helicobacter pylori* (*H*.*pylori*) infection.¹ The endoscopic findings include multiple nodular filling defects and prominence of the areae gastricae (mosaic-like pattern) in the gastric body with overlying normal mucosa. This has been termed as “chicken skin” appearance. Moreover, it may represent inflammation or an infiltrating neoplasm. The other differential diagnoses of this abnormal pattern are the following diseases; Menetrier’s disease, lymphoma, Zollinger-Ellison syndrome and mastocytosis.²

References

A 41-year-old Indian woman with an underlying of diffused large B-cell lymphoma and post radiation treatment presented with dyspepsia. The initial EGD showed an atrophic change of the entire gastric mucosa. Gastric biopsy specimen demonstrated intestinal metaplasia. On annual surveillance, the EGD showed diffuse atrophic mucosa (Figure 1). Blue laser imaging (BLI) endoscopy showed scattered lesions containing light-blue crest appearance (Figure 2). Biopsy showed mild chronic atrophic gastritis and gastric intestinal metaplasia (GIM) without dysplasia. Rapid urease test was negative.

Diagnosis:
Atrophic gastritis with gastric intestinal metaplasia
Discussion:

Gastric intestinal metaplasia and dysplasia are among the well-known precancerous lesions of the stomach. Detection of such precancerous lesions could lead to the diagnosis of early gastric cancer and improving the survival.

Annual endoscopy appears justified in all GIM patients with at least one of these conditions: (1) GIM extension >20% of mucosal surface; (2) presence of incomplete type GIM; (3) first-degree relative of gastric cancer patients; and (4) smokers. In the remaining GIM patients, a less intensive (2-3 years) surveillance could be proposed.

While performing a surveillance, image enhanced endoscopy is useful to obtain clear visualization of both the microvasculature and microstructure of the gastric surface to precisely diagnose early malignancies. Blue laser imaging endoscopy (BLI) which produce a bright and high resolution images can detect gastric malignant lesions surrounded by gastric intestinal metaplasia. Most differentiated gastric malignant lesions are visualized by BLI as a brown area while the surrounding gastric intestinal metaplasia is green.

References
A 70-year-old male with multiple myeloma IgG lambda stage 2, type 2 diabetes mellitus and hypertension presented with food regurgitation after a big meal and weight loss for 3 kgs in 6 months. EGD showed an outpouching esophageal wall at 30 cm. from the incisor compressing normal esophageal lumen (Figure 1). An endoscope could pass into the stomach without resistance. Barium swallowing (Figures 2 and 3) and esophageal manometry (Figure 4) were done. Esophageal manometry showed normal resting LES pressure with adequate LES relaxation but a frequent failed peristalsis was present. These finding were compatible with an ineffective esophageal motility. The patient and his family declined to persue for any further treatment.
Figures 2 and 3 An out-pouching lesion at the right lateroposterior aspect of mid esophagus (arrow).

Figure 4 Esophageal manometry showed normal resting LES pressure with adequate LES relaxation but frequent failed peristalsis was found (double arrow head). These were compatible with an ineffective esophageal motility.
Diagnosis:

Midesophageal diverticulum, pulsion type with an ineffective esophageal motility.

Discussion:

Esophageal diverticula are rare, with the prevalence of 0.06-4%, and generally they can be classified according to their location (pharyngoesophageal or Zenker’s, midesophageus or epiphrenic area) or their pathogenesis (pulsion or traction) or their morphology (true or false diverticulum). By their incidence, the most common type is Zenker’s diverticulum. Midesophageal diverticulum comprises less than 30% of all cases and traditionally considered traction diverticula secondary to mediastinal inflammatory reaction. But they could be pulsion diverticula and usually associate with esophageal motility disorder. Regurgitation and dysphagia are the most common complaints of these patients. Other symptoms include heartburn, chest pain, weight loss, halitosis, food impaction and aspiration. Endoscopy and barium swallowing study are the mandatory tests to help for diagnosis of esophageal diverticulum. Perhaps esophageal manometry may add on for the underlying motility disorder. The recommended therapy depends on the size of diverticulum and potential esophageal motility disorder.

References

A 51-year-old male with a history of familial adenomatous polyposis (FAP) and post total colectomy. He underwent a sided-viewing endoscopy as a surveillance for ampullary adenoma. It revealed a prominent size of ampulla with surrounding dense white villi (Figures 1-2). Biopsy was done and pathology showed a polyp lined by low-grade dysplastic epithelium, arranging in tubular shape (Figure 3).

**Figure 1** White light endoscopy showed a prominent sized ampulla with dense white villi (circle).

**Figure 2** FICE station 4 showed dense white villi at the ampulla (circle).

**Figure 3** Histopathology showed a polyp line by low-grade dysplastic epithelium.
Diagnosis:

Ampullary adenoma in familial adenomatous polyposis (FAP) patient

Discussion:

Almost all patients with FAP developed upper gastrointestinal polyps (gastric and duodenal polyps). Of these, two-thirds of the patients, the duodenal adenomas occurred at the periampullary and/or ampullary region.¹ These lesions have a potential for malignant transformation. However not all polyps and ampullas require removal, only the large lesion and the lesion with high grade dysplasia are deemed for removal. Therefore histological evaluation is the essential component for decision making. Recently, the novel instruments such as digital chromoendoscopy may give a real time diagnosis of adenomatous histology. Pittayanon et al. observed that the dense white villi seem to be the strongest support for the diagnosis of ampullary and non-ampullary adenoma. However, there may be insufficient data for high-grade dysplasia differentiation by this method.²

In this case, the patient was diagnosed as ampullary low-grade dysplasia. Therefore, endoscopic surveillance was continued instead of proceeding to ampullectomy.

References

A 71-year-old woman presented with dyspepsia. EGD showed a central depressed lesion at the gastric incisura (Figure 1). On the BLI-bright study, there was an area of dilated and tortuous microvascular with central absence of microsurface pattern (Figure 2). This met the criteria for a cancerous lesion. The lesion was biopsied and its histopathological result was compatible with gastric intestinal metaplasia (GIM) and focal high grade dysplasia (Figure 3). Subsequently, endoscopic submucosal dissection (ESD) was performed (Figures 4-5). The final histopathological result of the en bloc specimen demonstrated a well differentiated adenocarcinoma (Figure 6) with free resection margin.
Histopathological study of the biopsied specimen showed gastric intestinal metaplasia with a focal high grade dysplasia.

Figure 3

An EGD showed submucosa at the margin of lesion during endoscopic submucosal dissection at the incisura.

Figure 4

A 4 cm. en bloc resected specimen.

Figure 5

Histopathological result of the en bloc specimen showed well differentiated adenocarcinoma.

Figure 6

Diagnosis:
Well differentiated adenocarcinoma of the stomach

Discussion:
Early gastric cancer is defined as a lesion confined to mucosal or submucosal layer without the presence of lymph node metastases. Once completely resected, it has a good prognosis because of its low incidence of lymph node metastasis and low recurrent rate.1
Endoscopic submucosal dissection (ESD) can be used as a definitive therapy of pre-malignant and early stage malignant lesions in the digestive tract including gastric high grade dysplasia. It is performed by three steps:

1. the submucosa is injected with fluid to elevate the lesion from the muscle layer,
2. the surrounding mucosa of the lesion is circumferential cut, and
3. the connective tissue of the submucosa beneath the lesion is dissected.

ESD provides an en bloc specimen size larger than 20 mm, leading to a precise histological diagnosis and lower rate of recurrence. However, performing an ESD requires a skilful endoscopist and a long procedural time. Moreover, ESD has a significant rate of perforation at 4.3%. A meta-analysis of ESD in early gastric cancer showed the success rate in en bloc resection at 92%, histologically complete resection rate at 82% and recurrent rate at 0.8%.

References
A 62-year-old male presented with progressive mechanical dysphagia and significant weight loss for 2 months. He denied odynophagia or coughing after swallowing. Transnasal EGD was performed and revealed a circumferential ulcerative and friable mass at mid esophagus (32-36 cm from nostril) causing esophageal obstruction (Figure 1). A fistula was seen above the true esophageal lumen (Figure 2). Transnasal EGD was passed through the regular tract and showed normal distal esophagus, stomach, and duodenum. A green guidewire was inserted via transnasal EGD (Figure 2) and nasogastric tube was placed. Biopsy was taken from the mass and pathology read as moderately-differentiated squamous cell carcinoma. Computer tomography of the thorax exhibited asymmetrical circumferential wall thickening of mid esophagus measured 6 cm in length with bilateral bronchoesophageal fistula (Figure 3). There were also multiple subcentimeter bilateral paratracheal nodes.
Diagnosis:

Squamous cell carcinoma of esophagus with evidence of bronchoesophageal fistula

Discussion:

Fistulization between airway and esophagus is an uncommon finding resulting from both benign and malignant etiologies. The most common benign cause is the complication of endotracheal or tracheostomy tubes.\(^1\) Infection such as tuberculosis or CMV can result in fistula, especially in immunocompromised host.\(^2\) Malignancies can occur from either esophageal or tracheal/bronchial cancer. Tracheo/bronchoesophageal fistula (TBEF) can be found in approximately 5-10% of esophageal cancers.\(^3\) Typical clinical manifestation is uncontrolled coughing after swallowing (Ono’s sign).\(^1\) The other clinical presentations are recurrent chest infection, hemoptysis, chest pain, and odynophagia.\(^1\) Chest imaging and endoscopy help to confirm the diagnosis and can precisely locate the fistula opening.

References

A 27-year-old male was diagnosed as Crohn’s disease for 10 years. He had a history of perianal fistula and underwent total colectomy due to spontaneous colonic perforation. He recently developed watery diarrhea with 6 bowel movements per day. Double balloon enteroscopy (DBE) was performed via anal route. DBE showed multiple well-demarcated deep clean based ulcers starting from the anastomosis up to another 20 cm. (Figures 1-3). The intervening mucosa appeared normal. Biopsy was done from the ulcers and showed moderately active erosive ileitis with small granuloma. AFB stain and PCR for tuberculosis were negative.
Diagnosis:
Active ileitis from Crohn’s disease

Discussion:
Chronic inflammation from Crohn’s disease (CD) can involve entire gastrointestinal tract. Most common affected area is ileo-colonic (50%), followed by colonic disease alone (30%). About 30% of patients can present with only small bowel involvement, most of the lesions are confined to the terminal ileum. Nevertheless, inflammation of the proximal small bowel can occur in 10%. Clinical manifestations are obscure GI bleeding, unexplained anemia, and chronic diarrhea. Previously, the diagnosis of small bowel CD was a big challenge due to the limitation of endoscope. Nowadays, new modalities such as wireless capsule endoscopy, double-balloon enteroscopy have emerged to facilitate the diagnosis of small bowel CD. Endoscopic findings in small bowel CD are quite similar to colonic CD such as apthous ulcer, longitudinal ulcer, and cobblestone mucosa. Two main complications are stricture and fistula. For small bowel stricture, it is more common than colonic stricture and it is usually arisen at the surgical anastomotic site.

Figures 1, 2 Well-demarcated deep clean based ileal ulcers with intervening normal ileal mucosa.

Figure 3 FICE image showed ulcers with indented vasculatures of the ulcer border representing active inflammation.
References
Case 2

Suppakorn Malikhao, M.D.
Satimai Aniwan, M.D.
Vichai Viriyautsahakul, M.D.
Rungsun Rerknimitr, M.D.

A 65-year-old Thai male with hepatitis B cirrhosis, was admitted due to hematochezia. Physical examination revealed large splenomegaly with ascites. EGD showed large esophageal varices without red color sign and colonoscopy showed large rectal varices without bleeding stigmata. During admission, his bleeding persisted. Therefore small bowel varices were suspected. Capsule endoscopy was carried out and it showed adherence blood clot at the proximal small bowel (Figure 1). Subsequently, he underwent double balloon endoscopy. Double balloon endoscopy demonstrated distended jejunal varices with adherent clot in the proximal part of jejunum (Figure 2). Cyanoacrylate glue injection was done (Figures 3, 4). A computed tomography of abdomen was done to evaluate portal hypertension, a huge splenomegaly and multiple engorged small bowel varices were detected (Figures 5, 6). Later, transjugular intrahepatic portosystemic shunt (TIPS) was performed as a definitive treatment. He had no experience of rebleeding after 3 months follow-up.
Diagnosis:
Ruptured jejunal varices

Discussion:
Ectopic varices are dilated portosystemic collateral veins located at unusual sites other than the gastroesophageal region. The incidence of bleeding from ectopic varices is between 1 to 5%. Patients with extrahepatic portal hypertension have the highest incidence of bleeding about 20-30%. The most common sites are duodenum, jejunum, ileum, colon, and rectum. Small intestinal varices imply the development of varices in the jejunum and ileum. The absence of esophageal varices does not exclude the presence of small bowel varices, because one-third of patients with ectopic varices do not have esophageal varices.

Figures 3, 4 Cyanoacrylate glue was injected into the jejunal varices.

Figures 5, 6 CT abdomen showed a huge spleen and multiple engorged small bowel varices.
A triad of portal hypertension, hematochezia without hematemesis, and previous abdominal surgery characterizes small intestinal varices.\textsuperscript{1,3} Diagnosis is often difficult. Capsule endoscopy demonstrated small intestinal varices in 8.1\% of patients with portal hypertension. Double balloon endoscopy has both diagnostic and therapeutic potential, hence it is better than capsule endoscopy if small bowel varices are suspected.\textsuperscript{1} Treatment with cyanoacrylate for duodenal varices is one of the best options and it can achieve obliteration and disappearance of the varices. TIPS, embolization, and surgery have also been used when failed endoscopic treatment.\textsuperscript{1,2}

References

A 9-year-old Thai boy presented with occult obscure gastrointestinal bleeding. Physical examination showed multiple, protuberant dark blue vascular nodules, size 0.5-2 cm. at all extremities and tongue (Figures 1-3). Double balloon enteroscopy (DBE) showed multiple purplish subepithelial blebs; vary in size 0.5-1.0 cm. along jejunum and ileum. The large bleb was identified with active bleeding (Figure 4). Argon plasma coagulation (APC) was applied to achieve homeostasis (Figure 5).
Diagnosis:

Blue rubber bleb nevus syndrome (BRBNS)

Discussion:

Blue rubber bleb nevus syndrome (BRBNS) is a rare disorder. Most cases are sporadic but autosomal dominant inheritance has been reported. Most patients have normal life span. No malignant transformation potential has been reported. Prognosis depends on the extent of visceral organ involvement. BRBNS is characterized by hemangiomas of the skin, other viscera, and GI tract. The most common presentation of BRBNS is gastrointestinal bleeding. Lesions are most commonly found in the small intestine and distal large bowel. They are typically discrete mucosal nodules with a central bluish nipple, although they may be flat, macular, or polypoid.
The aim of treatment is to manage its complications mainly bleeding. Excision of the gut hemangioma and segmental resection of the involved gut have been performed for life-threatening hemorrhage. However, surgery is problematic because of the development of additional lesions later in life. Consequently, endoscopic management, such as electrocoagulation or laser photocoagulation, is preferred. APC is safe and effective with shallow coagulation (not more than 3 mm. in depth). The risk of perforation is much reduced when compared to other contact-type thermocoagulating devices, but the penetration of APC energy should be set to provide an adequate depth of coagulation over the lesions which are, indeed, venous malformations at the submucosal level.

References
A 58-year-old Thai male with chronic alcoholic pancreatitis was referred to our hospital due to massive overt obscure gastrointestinal bleeding. Three months prior admission, he had recurrent history of melena and epigastrium pain. He had normal EGD and colonoscopy. Capsule endoscopy showed blood clots in the proximal small bowel.

During hospitalization, he passed maroon stool and developed hypovolemic shock. After blood transfusion, a side-viewing duodenoscopy revealed bleeding per the ampulla of Vater (Figure 1). Computed tomography showed chronic calcific pancreatitis and a splenic artery aneurysm (Figure 2). Subsequently, he underwent an angiography with a successful selective embolization with metallic coils (Figures 3, 4).

Figure 1 Bleeding per the ampulla of Vater with normal ampullary surface.

Figure 2 Computed tomography showed calcific parenchyma of the pancreas and a splenic artery aneurysm (arrow).
Diagnosis:

Hemosuccus pancreaticus from splenic artery pseudoaneurysm

Discussion:

Hemosuccus pancreaticus is a special presentation of gastrointestinal bleeding from the duodenal papilla through the pancreatic duct. It is usually complicated with chronic pancreatitis, but it is difficult to diagnose because of the intermittent gastrointestinal bleeding. Hemosuccus pancreaticus is usually caused by ruptured pseudoaneurysm of the branch of peripancreatic arcade coming into the pancreatic duct, or into the pseudocyst communicating with the pancreatic duct. Splenic artery and its branches are commonly the origin (45%), followed by gastroduodenal artery (17%) and pancreaticoduodenal artery (16%).

The clinical presentations are upper gastrointestinal bleeding and epigastric pain resulting from the elevation of pressure in the pancreatic duct by blood clots. Jaundice is sometimes presented because of the reflux of blood to the bile duct.

An angiogram with embolization or surgery is the main treatment. Coil embolization is the most frequently described technique. The indications for surgery are persistent shock, failure of embolization and rebleeding after embolization. There are many surgical procedures including pancreatectomy and splenectomy, intracystic ligation of the blood vessels, aneurysm ligation, and bypass surgery with graft.
References


Case 5

Panida Piyachaturawat, M.D.
Satimai Aniwan, M.D.
Rungsun Rerknimitr, M.D.

A 24-year-old male had a history of right hemicolecotomy due to colonic obstruction. Surgical pathology diagnosed as Crohn’s disease. He had no symptom after surgical resection. One month later, a routine endoscopic evaluation to surveillance for postoperative recurrence was performed. Ileocolonoscopy showed inflamed ulcers at the neoterminal terminal ileum with mild ileitis. Under BLI, inflamed area was shown as the dark green color and ulceration was shown as the white color (Figures 1-6). Pathology showed acute organizing ulcer. The final diagnosis was the recurrence of Crohn’s disease.

Figures 1-4 Ulcers at the neoterminal ileum (figures1-2) with inflamed mucosa under BLI (figures 3-4).
Diagnosis:

Postoperative recurrence of Crohn’s disease

Discussion:

An endoscopic surveillance in patients with Crohn’s disease (CD) who underwent ileocolic resection, often shows preanastomotic ileal ulceration before CD symptoms re-appear. Two third of patients with Crohn’s disease had ulceration of the anastomotic area after three months, but only 33% of patients had symptoms indicating relapse.¹

Rutgeert’s et al developed an endoscopic scoring system for predicting the symptom recurrence in postoperative patients with Crohn’s disease.² The Endoscopic recurrence scoring system classifies the grades of inflammation at the ileum into 5 groups; (i0) normal (i1) ≤5 aphthous lesions (i2) >5 lesions with normal intervening mucosa (i3) diffused aphthous ileitis (i4) diffused inflammation with large ulcers, nodules and/or narrowing. The i0-i1 groups are defined as low risk with clinical recurrent rate as <10% in 10 years. Patient in the i2 group has 20% chance of recurrence. Whereas patients in the i3-i4 groups are defined as high risk group which have 50-100% chance of recurrence in the 5 years.²

References

An 89-year-old female was diagnosed superior mesenteric artery syndrome 2 years ago. Percutaneous endoscopic jejunostomy (PEJ) was done for nutritional support. Her weight increased and her symptoms gradually improved. She was well tolerated with an oral intake. Then PEJ tube was removed, however, the jejunocutaneous fistula did not heal. Therefore, push enteroscopy for a closure of nonhealing jejunocutaneous fistula was done. After guidewire insertion via cutaneous opening, a jejunocutaneous fistula was identified (Figures 1-2). Endoscopic clipping of closure fistula was performed (Figure 3).

Figures 1-2 Push enteroscopy showed guidewire passing through jejunocutaneous fistula (arrow).

Figure 3 Hemoclips for a closure nonhealing jejunocutaneous fistula.
Diagnosis:
Closure of nonhealing jejuncutaneous fistula following jejunostomy removal

Discussion:
The use of percutaneous endoscopic gastrotomy or jejunostomy has been increasing, and has become a safe and effective technique for enteral feeding. Persistent gastrocutaneous fistula after gastrostomy tube removal is a common sequel of long-term use. Several techniques have been proposed to closure gastrocutaneous fistula endoscopically. Endoscopic hemoclip is widely available and it is simple to those who familiar with this technique. There were several case reports showed completely healed fistula by endoscopic clips.\textsuperscript{1-3}

References
A 61-year-old male presented with a history of intermittent melena for 3 months. EGD and colonoscopy were normal. Double balloon endoscopy via oral route was performed and revealed subepithelial mass 2 cm. in diameter at the fourth part of duodenum. The superficial mucosa was intact (Figures 1 and 2). He underwent small bowel resection. Histopathological result demonstrated spindle cell tumors with scattered hyalinization and old hemorrhage, GISTs with degenerative change was diagnosed (Figure 3).
Diagnosis:
Gastrointestinal stromal tumor (GIST) of the duodenum

Discussion:
Gastrointestinal stromal tumor (GIST) is intraabdominal mesenchymal tumor originating from the gastrointestinal pacemaker cells with the prevalence of 129 per million for all GISTs groups which account for 1-3% of all gastrointestinal tumors. GISTs can be found along the upper gastrointestinal tract and the most common site is the stomach (60-70%). Duodenal GIST is uncommon and accounts for only 4.5% of all GISTs. It has a low risk of malignant progression. Endoscopic findings usually present as incidental submucosal lesions covered by normal mucosa (subtle, smooth intraluminal protrusions) but can be present as overt gastrointestinal bleeding, abdominal pain, nausea, and vomiting. Tumors larger than 5 cm. are associated with an increased risk for malignancy. The standard treatment is preferably a local resection and pancreatoduodenectomy is recommended for tumors with aggressive behaviours.

References
An 88-year-old female presented with coffee ground vomitus for 1 day. Three months prior to admission, she had undergone a Whipple operation due to periampullary malignancy. She also had an underlying disease of chronic atrial fibrillation therefore daily aspirin had been given. Push enteroscopy revealed multiple well-defined clean base ulcers at the anastomotic site (Figures 1 and 2) and its adjacent bowel (Figure 3). The area of small bowel beyond these ulcers appeared normal (Figure 4). *Helicobacter pylori* test was negative.

**Figures 1 and 2** Multiple clean base ulcers at the anastomotic site.

**Figures 3 and 4** Multiple well-defined clean base ulcers with normal edge at the proximal jejunum adjacent to the anastomotic site (left) normal mucosa distally to the lesions (right).
Diagnosis:
Marginal ulcers following Whipple operation

Discussion:
Whipple operation, or pancreaticoduodenectomy, is the surgical treatment of choice for periampullary and pancreatic cancers. Early surgical complications are leakage of the intestinal-pancreatic anastomosis, hemorrhage, pancreatic fistulas, and intra-abdominal abscess.\(^1\) Late complications after procedure include marginal ulcer, reflux esophagitis, diabetes mellitus, and biliary stricture.\(^2\) Pancreatectomy is one of the procedures in Whipple operation, resulting in loss of pancreatic bicarbonate secretion to neutralize the acid and may result in marginal ulcer at the anastomosis. The incidence of the marginal ulcer was 5%.\(^3\) Although pylorus-preserving pancreaticoduodenectomy had been initially performed to prevent marginal ulcer, there was no obvious evidence in the reduction of its incidence.\(^4\) Currently, marginal ulcer can be controlled much more effectively with proton pump inhibitor and/or H.pylori eradication.\(^2\)

References
Case 9

Puth Muangpaisarn, M.D.
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A 21-year-old male with known Peutz-Jeghers syndrome presented with chronic iron deficiency anemia. EGD and colonoscopy were normal. A push enteroscopy was performed. White light imaging revealed two pedunculated polyps with a coarse lobulated surface and grooves 1.0 cm. in diameter at the third part of duodenum and the proximal jejunum (Figures 1 and 2). With magnified BLI-bright, it demonstrated the lobular surface and dilated villi (Figure 3). Polypectomy with hot snare was done. Histopathological results confirmed hamartomatous polyp (Figure 4).

Figures 1 and 2 White light endoscopy showed pedunculated polyps with lobulated surface and grooves at the 3rd part of duodenum extending to the proximal jejunum.

Figure 3 Magnified BLI-bright demonstrated a hamartomatous polyp with lobular surface and dilated villi.
Diagnosis:

Hamartomatous polyp of the small bowel

Discussion:

Peutz-Jeghers syndrome (PJS) is one of the hamartomatous polyposis syndromes that is autosomal dominant disorder and associated with an increased risk for colorectal and other malignancies. The most common site of polyp is jejunum, following by ileum, colon, and stomach. Predominant endoscopic appearance is often pedunculated that can cause clinical symptoms such as intussusceptions, obstruction, and GI bleeding. Its polyps have typical histological features, consisting of a branched, tree-like smooth muscle core (arborization) covered with normal epithelium. The consistent extra-gastrointestinal feature is the mucocutaneous hyperpigmentedated macules, typically presenting on the lip, buccal mucosa, and periorbital area. Patients with PJS are at 70% risk for GI cancers including colon cancer.

References
A 72-year-old Thai male presented with fever, abdominal pain, nausea and vomiting for 3 days. He had underlying of hypertension. Physical examinations appeared unremarkable. Laboratory showed leucocytosis. Serum amylase, lipase and liver function test were within normal limit. CT of the abdomen demonstrated thickening wall with mucosal enhancement of the proximal jejunum (Figures 1 and 2). The mesenteric artery and mesenteric vein were patent. Anterograde double balloon endoscopy (DBE) was done. DBE found a well demarcated area of a segmental circumferential violeceous and edematous mucosa with superficial ulceration at the proximal jejunum (Figures 3 and 4). DBE showed the sharp demarcated area between the normal mucosa and the lesion (Figure 5). Non-occlusive mesenteric ischemia was suspected. Biopsy was obtained. Pathology confirmed the diagnosis of small bowel ischemia. His symptoms improved with conservative treatment within two weeks. His symptoms did not recur during a 9-month follow up.
Figures 3-6 DBE showed a well demarcated area (Figure 3) between a normal area and a segmental circumferential violeceous and edematous mucosa with superficial ulceration at the proximal jejunum (Figures 4, 5). In contrast, the bowel distal to the lesion showed normal mucosa (Figure 6).
Diagnosis:
Acute non-occlusive mesenteric ischemia causing segmental jejunal infarction

Discussion:
The causes of acute mesenteric ischemia are SMA thrombosis, SMA embolus and non-occlusive mesenteric ischemia (NOMI). Each type of mesenteric ischemia creates a high risk of mesenteric infarction and death if diagnosis and treatment are delayed. The severity of bowel injury is varying from transient bowel dysfunction to transmural necrosis that leads to multiorgan failure. This condition portends to high morbidity and mortality. CT angiography is the mainstay of diagnosis. However, in some difficult cases with ambiguous information, endoscopy might have a role to support the diagnosis. A well demarcated border of lesion is the characteristic of endoscopic finding in acute mesenteric ischemia. The other endoscopic features of intestinal ischemia include edema, pale mucosa, hemorrhage, erosion, ulcer and dark bluish necrotic tissue with segmental and circumferential involvement.

References
A 40-year-old male presented with chronic watery diarrhea and generalized edema for 12 months. Laboratory tests showed severe hypoalbuminemia and iron deficiency anemia. Anti-HIV was non-reactive. Stool examinations revealed no WBC or parasites. Stool cultures for bacterial pathogen and parasites were negative. Stool for AFB were also negative. Thyroid function test was normal. The technetium 99 m-labeled human serum albumin (HSA) scintigraphy was normal. A colonoscopy with random biopsy was normal. Subsequently, patient was underwent antegrade double balloon endoscopy (DBE). DBE revealed diffuse flat villi and scalloping appearance from the fourth part of duodenum to the proximal jejunum (Figures 1-4). Biopsy was done. Histopathology showed mild inflammatory cells infiltration with blunting of the villi, no organisms or granuloma seen. A diagnosis was malabsorptive enteropathy, most likely from parasitic infestation. His symptoms improved with antiparasitic treatment.
Diagnosis:
Malabsorptive enteropathy most likely parasitic infestation

Discussion:
A scalloping mucosa or mosaic pattern on enteroscopy is the characteristic of mucosal injury and resulting in atrophy. Celiac disease is the most common causes. However, it could be seen in any other conditions including infection, inflammation, and malignancy such as giardiasis, tropical sprue, HIV-related disease, Crohn’s disease, and lymphoma.\textsuperscript{1,2}

References

Figures 1-4 DBE demonstrated diffuse flat villi with scalloping appearance from the fourth part of duodenum to the proximal jejunum.
An 81-year-old male presented with massive gastrointestinal bleeding and hypovolemic shock. He had an underlying of coronary artery disease. He had been taking aspirin and clopidogrel. After fluid resuscitation and blood transfusion, EGD was unremarkable. He underwent an emergency angiography. There was no extravasation of angiographic contrast. Colonoscopy showed a fresh blood came from terminal ileum without identifiable bleeding cause. Subsequently, antegrade double balloon endoscopy was done. DBE revealed a jejunal ulcer with non-bleeding visible vessel (Figures 1-2). The endoscopic therapy with adrenalin injection followed by hemoclipping was applied. There was no rebleeding during 1 year follow-up.
Diagnosis:
Jejunal ulcer with non-bleeding visible vessel

Discussion:
The common cause of overt gastrointestinal bleeding is NASIDs induced ulcer.\(^1\) Fifteen percent of the ulcers located in the jejunum.\(^2\) In overt ongoing obscure gastrointestinal bleeding, DBE is the preferred investigation to identify the cause of bleeding. Moreover, DBE can offer a prompt endoscopic treatment.\(^1\) Furthermore, the time to perform DBE is also important. A recent study showed that urgent DBE (within 72 hours) provided a higher diagnostic yield than that of non-urgent DBE (70% vs. 30%, p<0.05).\(^1\)

References
A 22-year-old female presented with chronic watery diarrhea, generalized edema, polyarthalgia, and significant weight loss for 2 months. Physical examination showed oral ulcers, hepatosplenomegaly, ascites, and pitting edema of both legs. Laboratory test showed hypoalbuminemia. Stool examination was unremarkable and negative for parasite. Antinuclear antibody was positive with a titer of 1:160. $^{99m}$TC albumin scan showed increased radiodensity in the distal part of small bowel (Figure 1). CT scan of the whole abdomen demonstrated swelling and thickening of bowel wall at the 4th part of duodenum and the distal ileum (Figure 2). Double balloon endoscopy was performed. DBE revealed subepithelial hemorrhage with mosaic pattern of stomach and edematous duodenal mucosa (Figures 3-4). Jejunal examination showed multiple transverse ulcers with exudates on top and loss of the normal villi (Figure 5). Pathological results revealed the inflammatory cells infiltration in the mucosa without malignant cell or granuloma. Diagnosis was systemic lupus erythematosus with protein losing enteropathy. After corticosteroid was given intravenously, her symptoms improved.
Figure 2 CT scan showed swelling and thickening mucosa of duodenum fourth part (arrow) and ileum (star).

Figure 3 The gastric mucosa showed diffuse swelling with subepithelial hemorrhage in mosaic pattern.

Figure 4 Swelling mucosa at the fourth part of the duodenum.
Diagnosis:

Systemic lupus erythematosus with protein losing enteropathy

Discussion:

Protein losing enteropathy (PLE) is an uncommon condition presented by profound edema and severe hypoalbuminemia resulting from excessive loss of serum protein from gastrointestinal tract. The clinical manifestations such as pitting edema and ascites are similar to nephrotic syndrome but urine protein is normal. Diarrhea can be found in 50% of cases. Etiologies can be classified to erosive gastrointestinal disorders, non-erosive gastrointestinal disorders, and disorders involving increased central venous pressure or mesenteric lymphatic obstruction. The confirmed diagnosis of PLE is Tc-99m scintigraphy or fecal alpha-1 antitrypsin clearance test. Endoscopic findings depend on the primary disease. In PLE associated with systemic lupus erythematosus, endoscopic findings are nonspecific but can be seen as intestinal wall edema or ulcer. Additionally, 10% of patients have normal endoscopic finding.

Reference

A 53-year-old male presented with bowel habit change and significant weight loss for 3 months. Physical examination revealed an ill-defined mass at right lower abdomen. His stool examination was normal. Colonoscopy was performed and revealed multiple friable polypoid masses with erosions and white debris on top at the cecum (Figures 1 and 2). Blue laser imaging endoscopy (BLI) showed mucosal inflammation (Figure 3). Ileocecal valve was markedly swelling and causing narrowing of the lumen precluding the passage of a colonoscope into the terminal ileum; however, the remaining colon was normal. Biopsy was done at the mass. Histopathology showed foci of granulomatous inflammation with focal caseous necrosis, and aggregated epitheloid histiocytes (Figure 3). Tissue AFB and PCR-TB were negative.
Colonoscopic findings showed a polypoid cecal mass.

Figure 2 White debris on top of polypoid cecal mass.

Blue laser imaging endoscopy (BLI-bright) showed slightly brown mucosa comparing with the lighter background. The surface pattern and vasculature were normal.

Diagnosis:
Caseous granulomatous colitis, most likely TB colitis

Discussion:
Gastrointestinal tract is one of the common tuberculosis-infected organs, although it is not as commonly seen as pulmonary tuberculosis. Less than one-fourth of patients with gastrointestinal TB have concomitant pulmonary TB.\(^1\) TB colitis can present with different endoscopic characteristics: ulcerative lesion (60%), hypertrophic lesion or mass-like lesion (10%), or ulcero-hypertrophic lesion (30%).\(^2\)

Pattern of distribution is useful for diagnosis. Most cases showed segmental involvement and the most common involvement is at the ileocecal area. Entire colonic involvement is uncommon.\(^2\)

Additive clues for suspicious TB colitis are

Histopathology demonstrated caseous granulomatous inflammation.

Figure 4
patulous IC valve, circumferential ulcers, dirty based ulcer, and rarely anorectal involvement. The differential diagnosis are Crohn’s disease, lymphoma, Bechet’s disease, and amebic colitis. Caseation from histology provides very high specificity (100%) and PPV (100%) for TB diagnosis.

References
A 38-year-old male presented with chronic watery diarrhea and significant weight loss for 1 year. Physical examination was normal. Anti-HIV test was non-reactive. Stool examination showed numerous white blood cells. Stool concentration for parasite, stool for AFB and stool culture were also negative. Colonoscopy was performed and revealed extensive longitudinal deep clean base ulcers along colon with intervening normal mucosa. (Figures 1 and 2). Endoscopic ultrasound demonstrated white bracket comprises the enlarged mucosa, muscularis propria and marks thickened submucosal layer of the ascending colon (Figure 3). Biopsy was obtained. Histological report showed active and chronic colitis with vague granuloma. The tissue was negative for AFB and PCR-TB. The diagnosis was colonic Crohn’s disease.
Diagnosis:

Colonic Crohn’s disease

Discussion:

Crohn’s disease (CD) is a chronic inflammatory bowel disorder. Diagnosis by clinical, endoscopic findings, pathological findings characterized by focal, asymmetric, transmural, and granulomatous inflammation affecting gastrointestinal (GI) tract.\(^1,2\) Colonoscopic and ileoscopic findings showed redness, friability, edema or granular appearance with results in a cobblestone appearance then progresses to ulcers which punctate pattern, longitudinal pattern or extensive pattern and repeated ulceration causes pseudopolyps.\(^3\) Endoscopic ultrasound (EUS) allows clear and reliable differentiation of the various layers of the gastrointestinal wall (GI). EUS can be used to differentiate patients with active CD from UC. A recent study showed that a new forward-viewing radial-array echoendoscope can be used to confirm the significantly thicker colonic wall in CD than that of in UC. In addition, EUS can demonstrate that CD had a characteristic fusion of all 5 layers.\(^4\)

References

A 51-year-old male was admitted to receive an autologous stem cell transplantation. Three days after transplantation, he developed mucous bloody diarrhea and fever. Stool examination for WBC and stool for C. difficile toxin were negative. His condition did not improve with an empiric antibiotic. Subsequently, he underwent colonoscopy with random biopsy. His first colonoscopy appeared normal. Two days following procedure, he developed hematochezia. The second colonoscopy revealed post-biopsy ulcers with visible vessel (Figure 1). Endoscopic clipping was applied to prevent future recurrent bleeding (Figure 2).

**Diagnosis:**

Post-biopsy colonic bleeding
Discussion:

Colonic biopsy and polypectomy can result in bleeding complication. Bleeding divided to immediate and delayed (up to 30 days) bleeding. Post-biopsy bleeding significantly increased in patients taking aspirin, and having conditions associated with primary or secondary platelet dysfunction. Post-biopsy colonic bleeding can be controlled by endoscopic therapy. The technique for hemostasis depends upon the severity of bleeding and individual endoscopist preference. Hemoclipping is one of the effective methods to achieve hemostasis and to prevent recurrent bleeding.

References
An 81-year-old female was admitted to the intensive care unit because of septic shock. She had underlying diseases of chronic kidney disease and hypertension. Two days after hospitalization, she was diagnosed with non-occlusive ischemic colitis. Her symptoms improved with conservative treatment. Two months later, she developed massive hematochezia. Subsequently, she underwent colonoscopy. Colonoscopy showed longitudinal ulcers (single-stripe sign) with granular mucosa, and decreased haustrations. Colonic stricture was found at the rectosigmoid colon and this precluded the passage of the standard colonoscope (Figures 1-3). By replacing with the DBE therapeutic scope (EN-450T5, 9.4 mm. outer diameter and 2.8 mm. accessory channel) the scope was able to pass the stricture, within 60 cm. reach, an active bleeding vessel was detected. Endoscopic therapy with adrenaline injection and bipolar coaptation was successfully performed.

Figure 1 Decreased haustrations of the colon with stricture.

Figure 2 Longitudinal ulcers (single-stripe sign).

Figure 3 Inflamed mucosa with active bleeding at the rectosigmoid colon beyond the stricture.
Diagnosis:
  Chronic ischemic colitis with stricture and bleeding visible vessel

Discussion:
  Ischemic colitis is the second most common cause of lower gastrointestinal bleeding, accounting for approximately 50-60% of all gastrointestinal ischemic episodes.\(^1\) The findings of colonoscopy depend on the stage and severity of ischemia. In the early stages of ischemia, pale, friable or edematous mucosa alone with petechial hemorrhages, scattered erosion, segmental erythema, with or without ulcerations and bleeding, may be observed. The chronic stage of ischemia is characterized by stricture, decreased haustrations, and mucosal granularity.\(^2\) The stricture usually precludes the passage of the standard colonoscope and thus a smaller diameter DBE scope may be applied to reach the upstream colon and to find the target lesion.

References
A 68-year-old female was referred to colonoscopy due to positive stool occult blood test and anemia. She had no experience of visible gastrointestinal bleeding. Colonoscopy revealed multiple flat vascular lesions with enhanced coloration in blue laser imaging (BLI) at the cecum (Figures 1 and 2). Otherwise all of the colon and the rectum examinations were normal.

Diagnosis:
Angiodysplasia of the colon

Discussion:
Angiodysplasia is the most common vascular malformations in gastrointestinal tract.\(^1\) It is defined as the abnormal, ecstatic, dilated, tortuous and usually small (less than 10 mm.) blood vessels visualized within the mucosal and submucosal layers.\(^2\) Angiodysplasia can be found frequently in colon, especially in cecum and ascending colon.\(^3\) About one-third of colonic angiodysplasia has multiple lesions.\(^3\) There are several conditions associated with angiodysplasia such as aortic stenosis (Heyde’s syndrome), Von Willebrand disease, and chronic kidney disease.\(^3\) Angiodysplasia causes 3% to 15% of LGIB cases.\(^4\)
The clinical manifestations of bleeding can be chronic blood loss or active bleeding. According to the ASGE guideline, argon plasma coagulation (APC) may be the preferred method to treat bleeding angiodysplasia. Whereas, endoscopic treatment of non-bleeding lesions is not recommended due to low prevalence of bleeding development.

References
A 57-year-old male underwent a screening colonoscopy. Endoscopic findings revealed sessile polyp 1.5 cm. in diameter with surface erosion at the sigmoid colon (Figure 1). Under BLI exam, it demonstrated dark brown mucosa with tortuous vessels with some areas of disrupted vessels and absent of normal surface pattern (Figures 2 and 3). It was compatible with NICE classification type 3. Endoscopic biopsy was performed. The pathological result showed a well differentiated adenocarcinoma.

**Diagnosis:**

Well differentiated adenocarcinoma of the colon
Discussion:

Chromoendoscopy with magnification has been developed for enhancing visualization of the microvasculature, as well as the architecture of the mucosal surface. This helps to predict pathological results of the colonic polyps, which may lead to reduce in the number of unnecessary biopsies performed and the number of endoscopic removal of non-neoplastic lesions. One of the common uses endoscopic criteria to distinguish between non-neoplastic and neoplastic lesions is the NICE classification. It is a simple category classification consisting of types 1-3 and these are based on the three characteristics: (i) lesion color; (ii) microvascular architecture; and (iii) surface pattern. Each type can predict their histopathology and be helpful to specify the management. NICE type 1 and NICE type 2 can be distinguished with an accuracy of 89%. The accuracy for differentiation between NICE type 2 and NICE type 3 was 84.3%. NICE type 3 provides a high accuracy to diagnose carcinoma.

References

An 88-year-old female presented with iron deficiency anemia. She underwent a colonoscopy. Colonoscopy revealed a 1.5 cm. polyp lp (Paris classification) with friable mucosa and small erosion (Figure 1). Under BLI, a high density of capillary network with lack of uniformity (Sano’s classification type IIIa). The polyp also showed the irregular arrangement of pit pattern (Kudo’s classification type V) (Figure 2). Snare polypectomy was done. Histopathology diagnosis was intramucosal adenocarcinoma.
Diagnosis:
Malignant colonic polyp with intramucosal adenocarcinoma

Discussion:
Colon cancer is a common malignant tumor and its incidence rate is increasing in recent years. The key point is to improve the survival rate is to have diagnosis and treatment at the early stage. Digital chromoendoscopy like NBI, FICE, and BLI can mainly detect the forms of pit pattern and capillary pattern and it helps in characterization of the lesions that could not be characterized with an ordinary endoscopy. However digital chromoendoscopy does not improve the detection rates of colorectal neoplasia. Thus it can effectively differentiate of neoplastic and non-neoplastic lesions. Its sensitivity and specificity for the diagnosis of colonic neoplasm are 91.5% and 91.7%, respectively.

References
A 53-year-old male underwent a colonoscopy for colorectal cancer screening. Colonoscopy showed a sessile polyp sized 0.5 cm. at the sigmoid colon. BLI showed vascular pattern with meshed capillary vessels surrounded with mucosal gland (Sano’s classification type II). Pit pattern showed tubular pit (Kudo’s classification type IIIs). Based on the NICE classification, its color was darker relative to the background and its vessels were thick brown. Surface pattern revealed tubular white structure surrounded by brown vessels. Therefore it was compatible with NICE classification type II (Figures 1-2). Polypectomy was done. Histopathological result demonstrated dilated tubular crypts lined by low grade dysplastic epithelium (Figure 3). Tubular adenoma was diagnosed.

*Figures 1 and 2* A sessile polyp with meshed capillary vessels surround tubular pit pattern under BLI-bright.
Diagnosis:
Tubular adenoma

Discussion:
Adenomatous polyps have been found 20-40% of screening colonoscopies in people older than 50 years\(^1\) and sub-classified by their histological appearance as tubular, villous, or tubulo-villous adenomas. Tubular adenoma is the most common histological subtype, constituting approximately 65-80% of all adenomatous polyps with histological appearance of branched tubular glands. Tubular adenomas are most often pedunculated polyps and generally less atypia than villous adenomas which are more commonly sessile polyps. Polyps with high-grade dysplasia are more likely to harbor cancer.\(^2\)

References
A 58-year-old male underwent a colonoscopy surveillance after polypectomy for many years. Colonoscopy revealed two polyps. A polyp with Paris classification II measured as 1.0 cm. was found in the rectum (Figure 1). BLI showed high density of branching capillary vessel (Sano’s classification type IIIa) and gyrus-like pit pattern (Kudo’s classification type IV) which compatible with NICE classification type 2 (Figures 1 and 2). Polyp removal was done. Pathological result confirmed as a tubular adenoma.

Figures 1 and 2 Sessile polyps with high density of capillary vessels and gyrus-like pit pattern under BLI-bright.
Diagnosis:
Tubular adenoma

Discussion:
There are four types of colonic polyps: adenomatous, hyperplastic, harmartomatous and inflammatory. In addition to these histologic features, polyps are generally described as being either sessile or pedunculate.\(^1\) Sessile adenomas are larger in size, and they have more granular or nodular surface and more irregular or vague margin than hyperplastic polyps.\(^2\) Adenomatous polyps are considered precursors for colorectal cancer. The risk of malignancy increases with the size of the polyp and the degree of villous component.\(^3\)

References
A 67-year-old female underwent a screening colonoscopy. She had a family history of first degree relative of colon cancer. Colonoscopy revealed a 1.2 cm Ip polyp (Paris classification) in the ascending colon (Figure 1). Under BLI-bright showed white branched structure of pit pattern (Kudo’s classification type IV) surrounded by irregular brown vessels (Sano’s classification type IIIa) (Figure 2). Snare polypectomy with prophylactic hemoclip was performed (Figure 3). Pathology report confirmed as a tubular adenoma with high grade dysplasia.

Diagnosis:
Tubular adenoma with high grade dysplasia
Discussion:

Polyps of the colon are mucosal lesions which project into the lumen of the bowel. According to autopsy studies, colonic polyps occur in more than 30% of people over the age of 60. Approximately 70-80% of resected polyps are adenomatous. Adenomatous lesions have a well-documented relationship to colorectal cancer. Endoscopic findings show round, oval, honeycomb-like pit pattern. Pit pattern may be elongated and large diameter compatible with adenomatous polyp classified by Kudo. The overall accuracy of magnified endoscopy in predicting adenomatous histology was 93.4%. The negative predictive value for diagnosing adenomatous histology in the diminutive rectosigmoid polyps was 93.3%. Mostly, the polyps can be removed during colonoscopy using snare polypectomy and/or electrocautery techniques.

References

Case 11

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A 55-year-old male visited to our hospital for colorectal cancer screening program. Colonoscopy was performed and showed a 1 cm. sessile polyp (Paris classification Is) with abrupt cessation of capillaries at the margin of the polyp (Figure 1). Under BLI and magnified BLI-bright revealed wavy pits surrounded by brown vessels (Figures 2 and 3). Endoscopic mucosal resection was done. Histopathology diagnosed serrated sessile adenoma.

Figure 1 WLI showed a sessile polyp with suddenly abrupt cessation of capillaries at the margin of polyp.

Figures 2-3 BLI and magnified BLI-bright demonstrated wavy pits surrounded by brown vessels.
Diagnosis:
Serrated sessile adenoma

Discussion:
Serrated sessile adenoma/polyp (SSA/P) is one of serrated lesions of the colon characterized histologically by a saw-tooth appearance of the crypt epithelium. Most common location of SSA/P is proximal colon. Endoscopic features are almost always sessile or flat lesion. Its color is typically similar to the surrounding mucosa or may be pale mucosa. Sometime it can present as prominent folds. Additionally, mucous cap, peripheral rim of debris or bubbles, or obscuring submucosal vessels can be commonly seen in SSA/P comparing to adenoma.

References
A 34-year-old female was diagnosed with extensive ulcerative colitis (UC) 2 years ago. Her symptoms partially improved with oral mesalamine 3 g/day. She had occasional streak of blood with stool. She underwent a colonoscopy for endoscopic assessment. Colonoscopy showed severe spontaneous bleeding and ulceration at the rectum. Under BLI, the bleeding and inflamed area in the dark brown color and the ulcers in the white area (Figures 1-2). The whitish round crypts and villous mucosa structure were detected in BLI-bright with magnification (Figures 3-4). The other part of colon presented with multiple pseudopolyps (Figures 5-6). Biopsy from inflamed area was obtained. Histopathology confirmed as active UC.
Diagnosis:
Severe proctitis of ulcerative colitis

Discussion:
Ulcerative colitis usually shows the loss of vascular pattern, hyperemia of mucosa, mucosal edema, friable with easily contact bleeding mucosa, coalescence of small ulcers resulting in large ulcers and pseudopolyps that can defined severity of disease by Mayo score endoscopic findings; normal or inactive disease (grade 0) defined as normal endoscopy, mild disease (grade 1) defined as erythema, decreased vascular pattern, mild friability, moderate disease (grade 2) defined as marked erythema, absent vascular pattern, friability, erosions, and severe disease (grade 3) defined as spontaneous bleeding, ulceration.\(^1\) A previous study of applied NBI for the assessment of ulcerative colitis severity. Under NBI magnification, active UC was shown as round crypts, villous or granular mucosal surface.\(^2\)

References

Figures 5-6 Multiple pseudopolyps with normal surface similar to the adjacent colonic mucosa demonstrated by magnified BLI-bright.
A 69-year-old female presented with bowel habit change, iron deficiency anemia, and significant weight loss for 2 months. A colonoscopy revealed a circumferential mass measure as 6 cm. in size at the ascending colon with narrowing lumen. A colonoscope was not able to pass through the lesion (Figure 1). Under BLI-bright examination, it showed dark green color of the irregular colonic mucosa along with elongated, tortuous vessels which were compatible with NICE classification type 3 (Figure 2). Histopathological results confirmed as an invasive adenocarcinoma (Figure 3).
Diagnosis:
Invasive adenocarcinoma of the colon

Discussion:
An advanced endoscopic procedure removing early colorectal cancer such as endoscopic submucosal dissection (ESD) can be a better replacement than an invasive surgery. However, a deep invaded submucosal cancer occasionally has lymph node metastases. Therefore ESD could not be performed on this lesion. Thus, an accurate endoscopic diagnosis can determine the proper therapeutic approach. A novel blue laser imaging system (BLI) has been shown to be useful for the diagnosis of invasive colorectal cancers. The overall diagnostic accuracy for the differentiation between non-neoplastic and neoplastic lesion was 99.3%. In the view of diagnostic accuracy for deeply invaded submucosal cancer, BLI provided 94.3% in accuracy.

References
Case 14

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A 34-year-old homosexual male presented with constipation and scanty bleeding per rectum during defecation. Colonoscopy in BLI mode was performed and revealed whitish multiple fleshy cauliflower-like lesions with enlarged and elongated loop of capillary vessels (Figures 1-3). Other areas of the colon were normal. Biopsy was done. The pathological result showed a stratified squamous epithelium with papillomatosis and acanthosis (Figure 4). There were dysplasic cells in the epithelium (Figure 5). Condyloma acuminata with anal intraepithelial neoplasia was diagnosed.

Figure 1 White light examination showed whitish fleshy cauliflower-like lesions with edematous mucosa in the anal canal.

Figures 2 and 3 BLI with magnification demonstrated enlarged and elongated loop of capillary pattern without irregularity.
Diagnosis:

Condyloma acuminata with anal intraepithelial neoplasia

Discussion:

Condyloma acuminata or squamous papilloma, caused by human papilloma virus (HPV) is usually common in the oropharynx and the esophagus. Papilloma in the anus and the rectum is an uncommon entity.\(^1\) HPV serotype 16 and 18 are frequently found in dysplasia and malignancy.\(^2\) Anal HPV disease is linked to the immunosuppression caused by HIV infection and is associated with the practice of anal intercourse.\(^2\) Clinical manifestations include rectal bleeding, mucoid discharge, pain, and pruritus ani.\(^2\) The endoscopic feature is shown as a gray or pink fleshy cauliflower-like lesion in the perianal area, the anal canal or the rectum.\(^2\) It can be treated by many methods including excision, cryotherapy, or podophyllin painting.\(^3\) Malignant transformation should be suspected in atypical or pigmented lesion, the lesion that refractory to standard treatment and the detection of dysplastic cells.\(^2\)

References
An asymptomatic 50-year-old male underwent a colonoscopy for colorectal cancer screening. Magnifying white light image showed a 0.3 cm. sessile polyp (Paris classification Is) at the sigmoid colon (Figure 1). Magnifying BLI and BLI-bright demonstrated uniform round pit (Kudo’s classification type I), surrounding by lacy-like pattern of capillary vessels (Sano’s classification type I), which was compatible with NICE classification type 1 (Figure 2). Polyp removal was done with biopsy forceps. Pathological diagnosis was confirmed as hyperplastic polyp (Figure 3).
Diagnosis:

Hyperplastic polyp at the sigmoid colon

Discussion:

Colonoscopy is considered an effective approach for the detection and diagnosis of colorectal neoplasm. Using chromoendoscopy, NBI, FICE, i-Scan, and recently BLI systems which have advantages for the diagnosis of colorectal polyps can enhance on the reading of vascular patterns and pit-like structures, which were termed “surface patterns” at the Japanese consensus symposium. These endoscope systems can predict the histopathological diagnosis and are useful for selecting a therapeutic method.

In the image evaluation of BLI magnification, the diagnostic accuracy of BLI without magnification for differentiating between neoplastic and non-neoplastic polyps <10 mm. in diameter was 95.2%, which was greater than that of white light (83.2%).

References

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A 44-year-old Thai female presented with abdominal pain, distension and constipation for 5 days. Seven years prior admission, she had been diagnosed as colonic Crohn’s disease. Mistakenly, she lost to follow-up because she had no symptoms. She took no medications until this admission. A computed tomography of the abdomen showed markedly dilated bowel containing fecal content of the ascending and transverse colon (proximal to the distinct transitional zone of collapsed sigmoid colon) (Figure 1). There were no signs of stragulation or perforation. Four days after intravenous fluid and NG tube insertion for bowel decompression, she could pass stool with the improvement of distension. She subsequently underwent a sigmoidoscopy. Sigmoidoscopy demonstrated inflamed mucosa at the rectosigmoid colon with multiple discrete deep inflammed ulcers causing luminal stricture. The endoscope was able to pass through the stricture site (Figures 2 and 3). Biopsy was obtained. Histopathology showed acute organizing ulcer with cryptitis but without dysplasia or organism (Figure 4). Intravenous corticosteroids was given. Her symptoms improved after 7 days of medical treatment.
Diagnosis:

Active colonic Crohn’s disease with partial colonic obstruction.

Discussion:

Intestinal stricture is a major complication of Crohn’s disease, occurring in one third of patients after 10 years of disease. The stricture can occur anywhere in the gastrointestinal tract, especially ileocecal valve, ileocolonic anastomosis, duodenum, sigmoid colon, and anal canal. The obstruction occurs because of either luminal narrowing from inflammation or post-inflammation scar tissue. Corticosteroids is the main stay of treatment in inflammatory stricture. However, in case of fibrotic stricture, medical therapy was shown to be ineffective.

References

A 46-year-old female presented with chronic constipation and a history of straining for 5 years. She had an intermittent rectal bleeding. Colonoscopy revealed an erythematous polypoid lesion with white slough and multiple shallow ulcers (Figure 1) at the anterior wall of rectum (10 cm. above the anal verge). Under BLI-bright, the ulcer showed as a white area surrounded with regular vessels in the dark brown mucosa (Figure 2). Biopsy was performed. Histopathological result showed superficial mucosal ulceration, prominent fibromuscular hyperplasia within the lamina propria and crypt elongation with focal dilation (Figure 3).

Diagnosis:
Solitary rectal ulcer syndrome
Discussion:

Solitary rectal ulcer syndrome (SRUS) is a benign disease. It is a misnomer because multiple ulcers can be found in 40% of the patients, while 20% of the patients have a solitary ulcer, and the rest of the lesions differ in shape and size, including hyperemic mucosa, polypoid lesions mimicking rectal cancer. The etiology and pathogenesis are associated with direct trauma, local ischemia and uncoordinated defecation. Clinical features are rectal bleeding, copious mucus discharge, prolonged excessive straining, perineal and abdominal pain, feeling of incomplete defecation and constipation. However, up to 26% of the patients can be asymptomatic. Endoscopy usually discovers the lesions at the anterior wall of rectum and the distance of lesion(s) from anal margin varies from 3 to 10 cm. Biopsy should be taken to exclude other diseases. Key histological features include fibromuscular obliteration of the lamina propria with upward extension from hypertrophic and splayed muscularis mucosae and glandular crypt abnormalities. Several treatment options have been used for SRUS including conservative treatment such as high-fiber diet with additional bulking agents, medical therapy, biofeedback and surgery.
References:


A 75-year-old Thai female underwent a colonoscopy for colorectal-cancer screening. A flat polyp was found. Chromoendoscopy with indigo carmine showed a slightly elevated lesion or laterally spreading tumor (LST) granular type size 2.0 cm. at the descending colon. The surface of LST was composed of superficially homogenous small nodules which was consistent with LST-granular with homogeneous type 0-IIa in Paris classification (Figures 1 and 2). Under BLI and BLI bright, it showed brown color surface with short tubular pit pattern (Kudo’s classification type IIIL) surrounded by regular thick brown vessels (Sano’s classification type II) (Figures 3 and 4). Endoscopic mucosal resection was performed (Figures 5 and 6). Histological diagnosis confirmed the presence of tubular adenoma without malignant transformation (Figure 7).
Figures 3 and 4  Blue laser imaging (BLI) and BLI-bright demonstrated brown color background with short tubular pit pattern (Kudo’s classification type IIII) surrounded by thick brown vessels (Sano’s classification type II).

Figures 5  Successful endoscopic mucosal resection (EMR).

Figure 7  Tubular adenoma with low grade dysplasia without malignant transformation.

**Diagnosis:**
Laterally spreading tumor of the colon granular type

**Discussion:**
Laterally spreading tumor (LST) of the colon was defined as a lesion greater than 10 mm. in diameter with typically extends laterally rather than vertically along the colonic wall and belongs to the class non-polypoid colorectal neoplasia. They were classified into two subtypes as the granular-type (LST-G) which endoscopically consist of numerous nodules having a homogenous color in comparison with the surrounding colonic mucosa and non-granular (LST-NG) which consist of a smooth surface.¹²
Because of their morphological features, these lesions may be missed by standard white light colonoscopy.\textsuperscript{3} LSTs have an increased rate of submucosal invasion. Rates of invasion, particularly for the LST-NG subtype are as high as 30%-40%, whereas the granular subtype is significantly lower (about 5%-10%).\textsuperscript{4} Therefore, endoscopists need to have an awareness of their potential presence and follow-up on them accordingly. The therapeutic strategy for LST-G is endoscopic mucosal piecemeal resection with the area including the large nodule resected first, whereas LST-NG should be removed en bloc with endoscopic submucosal dissection (ESD) because of the higher potential for malignancy, often multifocal and greater difficulty in diagnosing depth of submucosal invasion.\textsuperscript{5}

References
Case 19

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An asymptomatic 72-year-old male underwent colonoscopy for colorectal cancer screening. During white light colonoscopy a polyp Isp (Paris classification) size 1.5 cm. (Figure 1) was detected. Under magnifying BLI and BLI-bright examination, they revealed dendritic pits (Kudo’s classification type IV) surrounding with irregular capillaries pattern (Sano’s classification type IIIA) (Figures 2 and 3). Snare polypectomy was performed. Pathology demonstrated villous adenoma (Figure 4).

Figure 1 White light imaging showed a 1.5 cm. colonic polyp Isp.

Figures 2 and 3 Magnifying BLI and BLI-bright showed irregular capillary network with dendritic pit pattern.
Diagnosis:
Villous adenoma

Discussion:
Blue laser imaging (BLI) colonoscopy has a high diagnostic accuracy for differentiation neoplastic from non-neoplastic polyps and adenomatous from cancerous lesions, (99.3% and 85.0% respectively). Additionally, BLI technique could provide a better (95.2%) accuracy for differentiation small neoplastic from small non-neoplastic polyps (<10 mm.) as compare to white light endoscopy (83.2%). The characteristics of vascular and surface pattern enhancement were divided into three groups by Sano’s classification. Type I was absence meshed capillary pattern which observed in hyperplastic polyp, Type II was regular and uniform capillaries pattern that detected in adenomatous polyps, Type III was irregular and non-uniform meshed-like capillaries pattern which observed in cancerous lesions.

References
An 80-year-old male presented with iron deficiency anemia. Colonoscopy revealed a large pedunculated polyp size 2.5 cm. in diameter in the sigmoid colon. Under magnifying blue laser imaging, it demonstrated an elongated and branched pit pattern surrounded by a regular thick brown vessel which was compatible with NICE classification type II (Figures 1 and 2). The endoloop-assisted snare polypectomy was done (Figures 3-5). Histopathological result showed a tubulovillous adenoma.
The technique of deploying an endoloop involved opening the loop, then maneuvering it around the polyp, and bringing it down to the level of the pedicle. After securing the endoloop in good position around the pedicle, endoloop was tightened and then released from the sheath.

The snare was manipulated around the polyp and tightened around the stalk, above the closed endoloop, and snare polypectomy can be performed.

Diagnosis:
Endoloop-assisted polypectomy for a large tubulovillous adenoma

Discussion:
Currently, endoscopic polypectomy is the standard procedure for polyp removal, as it is the least invasive and effective in reducing the incidence of colorectal cancer. However, it is not a risk-free procedure and its complications are bleeding (0.3-6.1%), perforation (0.5%) and post-polypectomy syndrome (0.5%). The incidence of post-polypectomy bleeding is more often after the removal of large pedunculated polyps due to the presence of a large blood vessel within the stalk. To minimize the risk of bleeding after resection of large pedunculated
polyps, several preventive endoscopic techniques have been used such as epinephrine injection, hemoclip and endoloop (detachable snare). The endoloop procedure was originally developed by Hachisu in 1991. The principle technique of deploying an endoloop is tightening of a loop around the polyp stalk for completely stops the blood flow in the stalk, act like a tourniquet. After securing the endoloop in a good position around the stalk, the snare cautery polypectomy is performed above the closed endoloop. The endoloop-aided snare resection is more effective than snare polypectomy and as effective as prophylactic clip in prevention post polypectomy bleeding in large pedunculated colonic polyps.

References
A 67-year-old man had been diagnosed as colon cancer with liver metastasis. He had undergone a total colectomy with wedge resection at the segment VI and VII of liver, followed by systemic chemotherapy since last year. He presented with painless progressive jaundice for 1 month. The computed tomography demonstrated the intra-ductal tumor continuing from the previous resection base. The tumor spread into common bile duct (CBD), right and left hepatic ducts resulting in biliary dilatation. ERCP (Figure 1) showed a 4-cm-long stricture at the hilum, Bismuth type IIIA, causing upstream dilatation. The specimen of intraductal biopsy revealed as metastatic colonic adenocarcinoma (positive CDX2, CK7 and CK20). For palliative biliary drainage, bilateral self-expandable metallic stents (SEMS) were inserted across the stricture in a parallel fashion (Figure 2).
Diagnosis:
Intrabiliary metastasis of colon cancer

Discussion:

Intrabiliary growth from metastatic colorectal carcinoma is an unusual finding and can clinically mimic cholangiocarcinoma.\textsuperscript{1-3} The intrabiliary tumor growth can be either intraluminal or intraepithelial extensions.\textsuperscript{4} In case of hepatic metastasis by colon cancer, the incidence of intrabiliary growth is higher than non-colorectal cancer (10.6\% vs. 0.7\%).\textsuperscript{1} The main duct has a slightly higher incidence of tumor involvement than minor ducts (56\% and 44\%, respectively), and the involvement of major duct is more likely to produce obstructive jaundice.\textsuperscript{1} The patients with preoperative jaundice and radiographic evidence of an intrabiliary filling defect are considered to be unresectable, and the five-year survival is 0\% compared to 33\% for those who are resectable.\textsuperscript{5}

\textbf{Figure 1} Cholangiogram showed a 4 cm. long stricture at the hilum, Bismuth IIIA, causing upstream dilatation. Two guide-wires were inserted into the right and left hepatic ducts before SEMS insertion.

\textbf{Figure 2} Two SEMSs were inserted in a parallel fashion.
References
A 61-year-old man had undergone laparoscopic bilateral adrenalectomy 3 weeks ago. After the procedure, he had persistent discharge from the percutaneous drain about 200 mL/day. The amylase level of fluid was 50,343 mg/dL. Computed tomography of the abdomen showed a 9.1x8.4x7.9 cm. fluid collection, containing multiple air bubbles, between the disconnected pancreatic head and body (Figure 1). ERCP was performed and the pancreatogram revealed a disconnected pancreatic duct together with contrast leakage into the collection at the body of pancreas (Figures 2-3). Although the guide-wire could not bridge the disconnected duct, a single pigtail stent was inserted into proximal pancreatic duct to facilitate the flow of pancreatic fluid and collection into the duodenum. (Figure 4). The output of the external drain decreased dramatically until it could be removed.

Figure 1 Computed tomography revealed a 9.1x8.4x7.9 cm. fluid collection containing multiple air bubbles between the completely disrupted pancreatic body.

Figure 2 Pancreatogram showed pancreatic duct was completely disrupted at the body.
Diagnosis:
Disconnected pancreatic duct syndrome

Discussion:
Disconnected pancreatic duct syndrome (DPDS) is characterized by the disruption of main pancreatic duct, therefore pancreatic secretion cannot drain into the gastrointestinal tract. DPDS is usually occurred after the necrosis of pancreatic parenchyma.\textsuperscript{1} The most common cause of DPDS is severe acute pancreatitis.\textsuperscript{1} Laparoscopic adrenalectomy was reported to cause the injury to the pancreas.\textsuperscript{2} The imaging modalities for diagnosing DPDS are CT scan, nuclear MRI/MRCP, and ERCP.\textsuperscript{3} The management of fluid collection and DPDS is challenging. The endoscopic treatment with internal drainage of fluid collection via transgastric-, transduodenal- or transpapillary routes had the success rate of 61-75%.\textsuperscript{3,4} The endoscopic internal drainage may prevent the development of cutaneous fistula from the percutaneous drainage. The preferred treatment of DPDS is endoscopic (and/or percutaneous rendezvous) stenting, however this may require the successful bridging of the disconnected ducts and only certain patients would respond to the proximal drainage as in our patients.\textsuperscript{7} The distal pancreatectomy is the salvage treatment but it carries a higher morbidity and mortality.
References
A 54-year-old man was referred for an evaluation of progressive abdominal distension. One month prior to this presentation, he developed an attack of acute alcoholic pancreatitis which was resolved under a standard supportive care. Two weeks later, he developed abdominal distension and discomfort. The abdominal CT scan showed pancreatic necrosis at the body of pancreas with a new onset of the large amount of ascites (Figure 1). During admission, octreotide and bowel rest were initiated. A large volume paracentesis (LVP) was performed with ascitic fluid analysis resulted in amylase of 30,018 U/L, albumin of 8,700 mg/L, and white blood cells of 658/μL (PMN 68%). The ascitic fluid cultures were negative. The ERCP showed partial main pancreatic duct disruption with an extravasation of the injected contrast from pancreatic body (Figure 2). A plastic stent was placed across the disrupted duct (Figure 3). Patient was clinically and symptomatically improved without a need of further paracentesis. On a follow-up clinic visit, patient was markedly improved.
Diagnosis:
  Partial main pancreatic duct disruption with pancreatic ascites

Discussion:
  Pancreatic ascites refers to an accumulation of peritoneal fluid in the presence of pancreatic disease and is usually secondary to pancreatic pseudocysts or pancreatic duct leakage. The main causes of pancreatic duct leak are acute pancreatitis, chronic pancreatitis, trauma, post pancreatic procedure, and surgery. The amylase level in ascites greater than 3 times of normal serum level is used to diagnose pancreatic ascites.\(^1\) The ERP is not only helpful to identify the leakage site and size but also can give an opportunity for treatment. In a case with complete pancreatic duct obstruction where ERP could not demonstrate the distal duct, MRP may be helpful to identify the distal duct and to locate the leakage site.\(^2\)

Conservative therapy for pancreatic ascites includes a serial LVP, elemental diet, parenteral nutrition, and somatostatin analogues injection. These interventions are thought to decrease pancreatic secretions and can facilitate the fistula closure, promote serosal apposition and healing. Unfortunately, this approach has a high failure rate, up to 40-60%. Risk factors for failure of this conservative therapy include the severity of pancreatic duct disease and the degree of biochemical abnormalities on admission (low serum sodium and serum albumin levels and a higher ratio of ascitic fluid protein to total serum protein levels).\(^3\)

Figure 3 A plastic stent was successfully placed across the disrupted site.
The main objective of the intervention for fistula closure is to promote the flow of pancreatic fluids back into the digestive tract. These procedures can be either endoscopic or surgical options encompassing a wide variety of procedures. Endoscopic transpapillary stenting is a non-invasive treatment for either partial or side branch pancreatic duct disruption. It provides a higher success rate, and has lower morbidity than surgical and conservative approaches. The factors that determine the successfulness of transpapillary stenting are site and severity of leakage.3,4

References
A 57-year-old man had been diagnosed with benign biliary stricture from chronic pancreatitis for 10 years. He presented with acute cholangitis for 2 days. Endoscopic retrograde cholangiography (ERC) (Figure 1) showed a 3 cm. smooth tapering stricture of the distal common bile duct (CBD) with the upstream duct dilation. Endoscopic retrograde pancreatography (ERP) via minor duodenal papilla (Figure 2) revealed a 2.5 cm. stricture at the proximal pancreatic duct with dilated main pancreatic duct compatible with chronic pancreatitis. A fully covered self-expandable metallic stent (FCSEMS) was inserted in CBD and a plastic stent was inserted in main pancreatic duct (Figure 3).

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**Figure 1** Cholangiogram via major duodenal papilla revealed a distal CBD stricture, 3 cm. in the length, this caused upstream biliary dilatation.

**Figure 2** Pancreatogram via minor duodenal papilla revealed proximal pancreatic duct stricture, 2.5 cm. in the length, with markedly dilated main pancreatic duct and dilated pancreatic side branches.
Diagnosis:
Distal CBD stricture and pancreatic duct stricture from chronic pancreatitis in an underlying pancreas divisum

Discussion:
The distal CBD stricture is commonly caused by chronic pancreatitis especially in the advanced stage. The clinical presentations vary from incidental finding to jaundice and cholangitis. Jaundice is typically transient but may be recurrent whereas cholangitis occurs up to 10% of patients which the presentation may range from subclinical episodes or overt septicemia. Pancreas divisum is the most common congenital variation of pancreatic duct formation and has prevalence of 4-10% in general population. Although the majority of pancreas divisum are asymptomatic, the prevalence of pancreas divisum is higher in idiopathic pancreatitis than general population. However, there is no clear association between pancreas divisum and chronic pancreatitis. The diagnosis of pancreas divisum can be made either by ERCP, MRCP, or EUS. The treatment options for pancreas divisum are minor papilla sphincterotomy, minor papilla dilatation, stent insertion, or surgery after fail conservative treatment. Endoscopic treatment for biliary stricture caused by chronic pancreatitis is a less invasive measure when compare with surgery. Multiple plastic stent placement for 1 year with scheduled exchange every 3 months was recommended by European Society of Gastrointestinal Endoscopy (ESGE). FCSEMS can be inserted without stent exchange up to 10-12 months and provide stricture resolution rate of 80%. However stent migration occurred up to 19% at 12 month-period.

Figure 3 Pancreatic stent across minor duodenal papilla (arrow) together with metallic stent in CBD (arrow head) demonstrated “the crossed duct sign”, which represented pancreas divisum.
References

An 86-year-old woman presented with progressive painless jaundice for 1 month. Physical examination revealed palpable gallbladder. Liver function test showed markedly elevated serum bilirubin (18.2 mg/dl). The computed tomography of the abdomen showed an enhancing soft tissue lesion occupying at the periampullary region with upstream bile duct dilation (Figure 1). The side-viewing duodenoscopy found a large ampullary mass. Endoscopic retrograde cholangiopancreatography (ERCP) revealed distal CBD stricture, 2 cm. in the length, causing upstream biliary dilatation (Figure 2A). A self-expandable metallic stent was placed across the stricture (Figure 2B). The histopathology of biopsy specimen from the ampullary mass revealed moderately differentiated adenocarcinoma. Because of her age, we did not offer her for any further treatment.

Figure 1 Computed tomography of the abdomen revealed an enhancing soft tissue mass at the peri-ampullary area (arrow) causing biliary obstruction.
Diagnosis:
Ampullary adenocarcinoma

Discussion:
Ampullary tumors are rare, with an approximate 5% incidence of all gastrointestinal neoplasms. Primary malignant ampullary tumors are adenocarcinoma, lymphoma, neuroendocrine, and signet ring cell carcinoma. Metastatic neoplasms include malignant melanoma, hypernephroma, and lymphoma. Of these malignant tumors, adenocarcinoma is the most common malignant ampullary lesion. Side-viewing duodenoscopy and ERCP are important tools for an evaluation of all ampullary tumors including size, margin and extent of intraductal growth. There are several endoscopic appearances of ampullary tumors, endoscopic assessment alone sometimes may not be sufficient for diagnosis. Tissue sampling is always required to establish the diagnosis. However, obtaining biopsy after endoscopic sphinctertomy may be difficult to interprete due to coagulated and necrotic tissue. Endoscopic ultrasonography are helpful in tumor (T) and nodal (N) staging. The overall accuracy of T and N staging are 56-91% and 50-81%, respectively.
References

A 65-year-old man had had a liver transplant from hepatitis B-related cirrhosis, severe portal hypertension evidenced by CT abdomen (Figure 1), and he later developed post-liver transplant biliary stricture. A fully covered metallic stent was placed for stricture treatment for 9 months. He was scheduled for stent removal in this admission. The cholangiogram revealed the inplaced metallic and a filling defect above the stent, compatible with CBD stone. The metallic stent was successfully removed by biopsy forceps. The stone could not be extracted either by sweeping balloon or basket. Then, the CRE balloon (8-10 mm.) was applied for papillary dilatation. After balloon deflation, there was active bleeding per ampulla. The cholangiogram showed the worm-like filling defects at distal common bile duct (Figure 2), compatible with choledochal varices. Therefore, a fully covered metallic stent was inserted across the major duodenal papilla to stop the bleeding (Figure 3). After stent deployment, the bleeding completely stopped. Octreotide was administrated for the additional 3 days, the patient required 1 unit of blood transfusion. His hematocrit was stable and he was discharged home safely.

**Figure 1** CT abdomen showed huge splenomegaly and multiple porto-systemic collateral vessels (arrow).
Diagnosis:
Hemobilia developed from balloon dilation of common duct containing choledochal varices

Discussion:
Choledochal varices are the abnormal venous dilatation at the bile duct wall, most are secondary to portal hypertension. There are two venous plexuses along the extrahepatic bile ducts, the paracholedochal veins of Petren and the epicholedochal venous plexus of Saint. The varices of paracholedochal plexus can cause extrinsic impressions to the bile duct, while the varices of epicholedochal venous plexus can cause the fine, irregular mural changes of the bile duct. The majority of patients are asymptomatic, however the common presentations in symptomatic patients are obstructive jaundice, or bleeding (spontaneous or secondary to endoscopic procedure). The reported procedures that induce severe bleeding are endoscopic stent removal, or bile duct dilatation. The diagnosis of choledochal varices need high clinical suspicious. The typical cholangiogram of choledocal varices shows smooth extrinsic compression on cholangiogram. The endoscopic ultrasound is also helpful for diagnosis because it allows visualization of the relationship between bile duct and the dilated venous collaterals or varices.

Figure 2 Cholangiogram showed worm-like filling defects at distal common bile duct (arrow).

Figure 3 After the placement of a fully covered metallic stent (arrow).
Treatments of choledochal varices include portosystemic shunting and hepaticojejunostomy in case of biliary obstruction. Portosystemic shunting can be achieved by transjugular or surgical approaches. For a patient presenting with choledochal variceal bleeding, endoscopic sclerotherapy, portosystemic shunting\textsuperscript{7}, or insertion of fully covered metallic stenting are reported to achieve successful hemostasis.\textsuperscript{8}

References

A 22-year-old man presented with recurrent jaundice for one year. This time, jaundice recurred for a week but accompanied with fever and abdominal pain for two days. Liver function test revealed elevated transaminases, alkaline phosphatase and direct hyperbilirubinemia. CT scan of the abdomen revealed fusiform dilatation of the common bile duct and left intrahepatic ducts, no stone was noted (Figure 1). Cholangiography showed choledochal cyst type 4A (Todani classification) without filling defect, but bile sludge was extracted during balloon sweeping. Double pig tail 10F was inserted to provide temporary drainage. He was then referred to surgeon for definitive management.
Diagnosis:
Cholangitis, Choledochal cyst type 4A

Discussion:
Choledochal cysts are congenital anomalies of the biliary tract that are manifested by disproportionate dilatations of the biliary system. The incidence of choledochal cysts shows significant geographic variation, being higher in the Asian population and reaching up to 1 in 1000. Choledochal cysts are not familial; female children are affected more commonly than male children. Cases have been described in utero and in older adult patients, but approximately two thirds of patients seek medical attention before the age of 10. The most common type based on Todani and colleagues’ classification is type 1 accounting 80-90%. This case presented with multiple left intrahepatic and common bile duct cysts which was classified as type 4A, the second most common with the incidence of 12-25% based on Gadelhak, et al 20 years single center experience (Figure 3). The cause of choledochal cysts has not been well established. Cysts are composed of a fibrous wall; there may be no epithelial lining or a low columnar epithelium. The infantile form of choledochal cyst disease often presents first month of life and as many as 80% with jaundice and acholic stools. In a 2008 series, adults were more likely to exhibit abdominal pain (97% vs. 63%, P < 0.001), and children were more likely to experience jaundice (71% vs. 25%, P = 0.001). The classic triad of abdominal pain, jaundice, and a palpable abdominal mass is observed in less than 20% of patients. The diagnosis of a choledochal cyst can be established by ultrasonography. In the older patients, percutaneous transhepatic cholangiography or ERCP may help define the anatomic features of the cyst. MRCP and CT scan is less effective than ERCP for detecting minor ductal abnormalities and small choledochoceles in

Figure 3 Cholangiogram revealed saccular dilatation of the common bile duct (arrow) and saccular dilatation of the left intrahepatic duct (arrow head), compatible with Todini type 4A.
Complete excision of the cyst with reconstruction of the extrahepatic biliary tree is the best treatment strategy to avoid long-term complications especially malignant transformation, recurrent cholangitis and gallstones.\textsuperscript{10,11}

References
A 9-year-old boy, with a history of recurrent epigastric pain for 5 years, presented with severe epigastric pain and vomiting for 2 days. The computed tomography showed atrophy of pancreatic parenchyma with a 2x3 cm. pseudocyst at the tail of pancreas. The pancreatogram showed dilated main pancreatic duct, size 0.5 cm., with round-shaped filling defect at the genu of pancreas, compatible with pancreatic duct stone and chronic pancreatitis (Figures 1-2). Unfortunately, the pancreatic stone could not be removed during the first ERCP with partial pancreatic sphincterotomy, and the second attempt of ERP was scheduled in the next 7 days. However, two days after the first ERCP, the patient relieved from the abdominal pain abruptly. The pancreatogram during the second ERP showed the disappearance of previously-seen filling defect in the pancreatic duct (Figure 3).
Diagnosis:

Chronic pancreatitis with spontaneously passed pancreatic duct stone

Discussion:

Chronic pancreatitis is characterized by parenchymal destruction and change in ductal structure.\(^1\) Pancreatic duct stones develop during the natural course of chronic pancreatitis and are observed in up to 90% of the patients.\(^2\) Hypothetically, intraductal hypertension from stone obstruction and parenchymal ischemia cause the abdominal pain.\(^3\) The removal of pancreatic duct stone can alleviate pain and improve pancreatic function by restoring pancreatic duct flow.\(^4,5\) The first-line treatment for pancreatic duct stones is an endoscopic therapy\(^6\) especially those of less than 5 mm. and located in the pancreatic head or body.\(^7\) Approximately 50% of pancreatic stones can be removed effectively by standard techniques, including endoscopic sphincterotomy or stone retrieval with a balloon, basket, and/or forceps alone.\(^5\) The addition of extracorporeal shock wave lithotripsy (ESWL) increases clearance rates up to 60-90% and it is the preferred strategy for the stone(s) that larger than 5 mm. Pancreatic stone removal is more challenging in the presence of pancreatic stricture. However in non-stricture pancreatic duct, the stone could be removed by only sphincterotomy or, theoretically, spontaneous migration can develop after the improvement of post-sphincterotomy papillary edema.
References


Case 9

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A 49-year-old Thai woman presented with recurrent cholangitis. The CT of upper abdomen revealed left intrahepatic duct (IHD) dilatation, multiple stones in IHD and common hepatic duct (CHD), and atrophy of the left lobe of liver (Figure 1). ERCP confirmed markedly dilation of left IHD with numerous round-shaped filling defects in left IHD. Multiple attempts of balloon extraction were done, however the complete stone clearance could not be achieved because of the associated stricture (Figure 2). Then, the surgical team was consulted for left hepatic resection.

Figure 1 CT of upper abdomen showed left IHD dilatation with multiple IHD stones (arrow) and left lobe atrophy (arrow head).

Figure 2 ERCP demonstrated markedly dilated left IHD with numerous round-shaped filling defects (arrow).
Diagnosis:
Oriental cholangiohepatitis

Discussion:
Oriental cholangiohepatitis was also known as recurrent pyogenic cholangitis, intrahepatic pigmented stone, and Hong Kong disease. The pathological changes were primarily in the bile ducts such as proliferation, inflammatory cells infiltration and fibrous changes at bile ducts wall. Occasionally, the lateral segment of left lobe liver might be shrunken. Chronic infestations at bile ducts such as Clonorchis sinensis, Opisthorchis species, Fasciola hepatica and Ascaris lumbricoides were the most described in pathogenesis of oriental cholangiohepatitis. Chronic infestations result in recurrent cholangitis, bile duct injury, bile stasis and leading to stone formation. The management of oriental cholangiohepatitis is multidisciplinary approach including endoscopists, intervention radiologist, and hepato-biliary surgeon. Hepatic resection is preferred in patients with hepatic segment atrophy, focal disease, or failure of less invasive therapy.

References
Case 10

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Rungsun Rerknimitr, M.D.

A 72-year-old woman presented with progressive painless jaundice for 2 weeks. The CT scan of upper abdomen showed a mass at liver hilum causing bilateral intrahepatic bile ducts dilatation (Figures 1 and 2). ERCP revealed an amorphous filling defect in common hepatic (CHD), left and right intrahepatic ducts (IHDs) with upstream biliary dilatation (Figures 3 and 4). Two uncovered self-expandable metallic stents (SEMSs) were inserted bilaterally into left and right IHDs in a parallel fashion (Figure 5).

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**Figures 1 and 2** CT scan of abdomen showed a mass at liver hilum (arrow) causing bilateral intrahepatic bile duct dilatation.

**Figure 3** An amorphous filling defect in CHD, left and right IHDs with upstream biliary dilatation.
Diagnosis:

Bilateral SEMSs placement in hilar Cholangiocarcinoma, Bismuth type IV

Discussion:

Cholangiocarcinoma is the most common primary malignancy of the biliary tract and classified into intrahepatic or extrahepatic by using the second order of bile ducts as the reference anatomy. Extrahepatic cholangiocarcinoma can be further divided into hilar (Klatskin tumor) and distal cholangiocarcinoma which are located proximal and distal to cystic duct insertion respectively. Three commonly used system to classified hilar Cholangiocarcinoma are the Bismuth-Corlette system, the Memorial Sloan-Kettering Cancer Center, and the TMN system. The Bismuth-Corlette system provides preoperative assessment of local spread and classifies as type I-IV. Type I, tumor is distal to the hepatic duct confluence. Type II, tumor extends to and involve the hepatic duct confluence. Type IIIa and IIIb, tumor involves the common hepatic duct and the right or left hepatic ducts, respectively. Type IV, tumor extends into the confluence and both intrahepatic bile ducts. The goal of palliative stenting in Klatskin tumor is to drain adequate liver volume, at least 50%, irrespective of unilateral, bilateral, or multisegmental stenting. The most suitable stent for this purpose is metallic stent.
References
A 26-year-old Thai woman, with a history of complete excision of choledochal cyst and hepaticojejunostomy anastomosis for 9 years, presented with recurrent cholangitis for 6 months. MRI and MRCP revealed intrahepatic duct dilatation with intrahepatic duct stone. Double balloon enteroscopy (DBE)-assisted ERCP revealed a stricture at the hepaticojejunostomy anastomosis and a stone in the left intrahepatic duct (Figures 1-3). The stone was later removed after balloon dilation of the stricture.

Diagnosis:
DBE assisted-ERCP in hepaticojejunostomy anastomosis
Discussion:

There are many indications for hepaticojejunostomy include choledochal cyst resections, traumatic or iatrogenic bile duct injury, bile duct stricture, biliary fibrosis from chronic pancreatitis and malignant conditions such as cholangiocarcinoma and carcinoma infiltrating bile duct.\(^1\) The incidence of hepaticojejunostomy anastomotic stricture is 4%-10%\(^2\) which can lead to bile stasis and intrahepatic lithiasis.\(^3\) Because re-operation carries a high morbidity, endoscopic or radiological management are preferred. Although ERCP in surgically altered anatomical configurations are technically challenging, DBE-assisted ERCP is a safe and feasible technique to access and manage biliary tract.\(^4\) However, the accessories used in DBE-assisted ERCP is limited by the length and channel diameter of the enteroscope.

References

A 47-year-old male presented with high-graded fever and jaundice for 3 days. He had a past history of intrahepatic cholangiocarcinoma and distal common bile duct obstruction from metastatic lymph node compressing common duct which has been treated with a fully covered self-expanding metallic stent (FCSEMS) and chemotherapy since 1 month ago. Physical examination showed low blood pressure, high body temperature, and moderate jaundice. His abdomen was mildly tender without guarding. Blood chemistry revealed thrombocytopenia, cholesstatic liver function tests, and rising serum level of creatinine. He was diagnosed as septic cholangitis. Transabdominal ultrasonography showed dilated both intrahepatic ducts (IHDs). Side viewing endoscopy showed post-sphincterotomy ampulla without stent seen. A fluoroscopy demonstrated proximal migration of the FCSEMS (Figure 1). A cholangiogram revealed complete obstruction of bilateral IHDs by FCSEMS at the hepatic hilum (Figure 2). The migrated FCSEM was successfully removed by a rat-tooth forceps and a snare. The final cholangiogram showed dilatation of bilateral intrahepatic ducts with resolution of distal common bile duct stricture (Figure 3).
Diagnosis:

Upward migration of biliary metallic stent causing intrahepatic ducts obstruction and acute cholangitis

Discussion:

Stent migration is one of the common complications of FCSEMS with a relative risk of 7.13 when compared with non-covered SEMS (NCSEMS). Similar to other causes of biliary obstruction, patient with stent migration usually present with abdominal pain, jaundice and/or cholangitis. Abdominal imaging is helpful for a diagnosis of this complication.

The meta-analyses of self-expandable metallic stent for palliative treatment of malignant distal biliary obstruction showed that FCSEMS had 61 days longer\(^1\) of stent patency than NCSEMS. However, there was no benefit of stent patency after 6 or 12 months.\(^2\) The FCSEMS had a lower rate of tumor ingrowth but had a higher tumor overgrowth rate and a high migration rate when compare with that of NCSEMS.\(^1,2\) To prevent stent migration, a technique of putting a double pigtail plastic stent through the SEMS lumen as a “stent lock” had been proposed.\(^3\)
References


A 42-year-old Thai female presented with anemic symptom and epigastric pain for 5 months. Physical examination was unremarkable. EGD showed a subepithelial mass approximately 3 cm. at the fundus (Figure 1). Subsequently, EUS was performed. It revealed a heterogenous hypoechoic mass measuring 23x22 mm. in diameter. This mass had regular border, round shape, and originated from the fourth layer of the gastric wall. No perigastric lymph node was seen (Figure 2). EUS guided core needle biopsy was performed with a 19G needle. Histopathology showed neoplastic spindle cells compatible with gastrointestinal stromal tumor.
Diagnosis:
Gastrointestinal stromal tumor (GIST)

Discussion:
EUS is the most reliable test to delineate GI tract wall layers. It is currently the test of choice for diagnosing subepithelial lesions with the highest diagnostic value among other radiological tests. Moreover, EUS-FNA can provide cytopathological diagnosis of subepithelial lesions with the sensitivity, specificity and accuracy rate at 89%, 88%, and 89%, respectively.\(^1\)\(^,\)\(^2\)

In this case, the tumor was eventually diagnosed as GIST based on the results of EUS-FNA and EUS findings. Endosonographically, it is a hypoechoic tumor originated from the 4\(^{th}\) layer (rarely 2\(^{nd}\) layer).\(^3\) The EUS appearance of GIST’s is not specific and can be seen with other mesenchymal tumors. Hence tissue sampling of a suspected GIST by EUS-FNA is essential to establish the diagnosis.\(^4\)

References
Case 2

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A 61-year-old male presented with painless obstructive jaundice and significant weight loss. CT scan of upper abdomen showed an ill-defined mass occupying the hilum of liver causing biliary tract obstruction and carcinomatosis peritonei was also detected. Biliary metallic stents were placed across the hilar stricture by ERCP. Endoscopic ultrasound (EUS) demonstrated omental cake, ascites (Figure 1) and multiple intraabdominal lymphadenopathy (Figure 2). EUS-guided fine needle aspiration was performed from celiac lymph node for tissue diagnosis (Figure 3). Histopathological result showed well-differentiated adenocarcinoma.

Figure 1 Ascites and omental cake were noted.

Figure 2 Previously placed metal stent was in placed. Multiple lymph nodes were identified.

Figure 3 Celiac lymph node showed as a hypoechoic lesion.
Diagnosis:
Advanced stage cholangiocarcinoma

Discussion:
Preoperative diagnosis of hilar cholangiocarcinoma is difficult. Although ultrasound, CT scan, MRCP, and PET scan provide important clues regarding the differential diagnoses and staging, the definitive diagnosis requires tissue sampling. ERCP with brush cytology and intraductal biopsy is the standard approach for tissue diagnosis with high specificity, but the sensitivity was only 20-60%. EUS-FNA is an alternative for tissue diagnosis. The sensitivity of EUS-FNA for diagnosis of the primary tumor was 73% and significantly higher in distal than hilar cholangiocarcinoma. Another approach is EUS-FNA of the lymph nodes. Endoscopic ultrasonographic morphologies of the lymph nodes include long-axis length, roundness, echogenicity, and homogeneity. They are the only features differentiating benign from malignant lymphadenopathy in esophageal, pancreatobiliary, mediastinal, and celiac nodes, unfortunately, its efficacy is low in cholangiocarcinoma.

References
A 43-year-old female presented with dyspepsia for 2 months. EGD revealed a 1-cm. subepithelial mass in the gastric antrum (Figure 1). The patient was referred for EUS (Figures 2-3). EUS showed a heterogenous, hypoechoic lesion originating from the 3rd layer of the stomach measuring 13x13 mm.

*Figure 1* Endoscopic finding of a 1-cm. subepithelial protuding lesion with central umbilication in the gastric antrum.

*Figure 2-3* EUS showed a heterogenous, hypoechoic lesion originating from the 3rd layer of the stomach measuring 13x13 mm.
Diagnosis:

Heterotopic pancreas

Discussion:

Heterotopic pancreas, also known as ectopic pancreas or pancreatic rest, is a pancreatic tissue existing in an organ or tissue distinct from the pancreas. It is usually found in the upper gastrointestinal tract, with >90% of the cases involving the stomach, duodenum or jejunum but can exist at any position in the abdominal cavity.¹ Unusual locations are the colon, spleen or liver.² Most affected patients are asymptomatic although a minority may present with a variety of symptoms such as abdominal pain and distension. Some of them can present with other symptoms including pancreatitis, islet cell tumor, pancreatic carcinoma, and pancreatic cyst.³ The typical endoscopic appearance in the stomach is as a firm round or oval umbilicated subepithelial nodule along the greater curvature situated several centimeters proximal to the pylorus.¹ As it is a subepithelial lesion in origin, it should be distinguished from GIST. Endosonographically, pancreatic rest in the stomach is a hypoechoic, heterogeneous submucosal mass, although the muscularis propria and mucosa may be occasionally involved. This is occasionally difficult to differentiate from GIST based on EUS findings alone. In equivocal case, EUS-FNA is required. Description of ductal structures within the lesion is a distinctive feature, but is observed only in a minority of cases.⁴

References
A 45-year-old female presented with significant weight loss and early satiety for 3 months. Linear EUS was performed. Upper endoscopy revealed nodular, thickened of the gastric wall with poor distention upon maximum air inflation (Figure 1). The lesion started from the area below esophagogastric junction (EGJ) downward to the antrum. Friable mucosa was noted around the antrum, and the scope could pass through the pylorus. Endosonographically, thickening gastric wall was noted, measuring 13 mm. in its maximum thickness (Figure 2). Muscularis propria and muscularis mucosa were measured as 4 and 8 mm. respectively (Figure 3). A round homogenous hypoechoic well-defined lymph node sized 8x7 mm. in diameter was identified (Figure 4). Ascites was not seen. A fine needle aspiration from the gastric wall was performed and the tissues were sent to pathological examination. Histological result showed poorly-differentiated adenocarcinoma.
Diagnosis:
Linitis plastica from poorly-differentiated adenocarcinoma of the stomach

Discussion:
Linitis plastica is a rare form of diffuse infiltrative gastric adenocarcinoma. The characteristic endoscopic feature is marked thickening of the gastric mucosal folds and gastric wall rigidity that fail to distend on insufflation.¹ The most common site is the antral and pyloric regions. Lesion are mainly located in subepithelial layers. Consequently, mucosal biopsy showed negative results for malignancy up to 30%.² EUS is useful for differentiating between benign and malignant entities in the patients with large gastric folds as it can delineate gastric walls layer by layer. Only the thickened deep layers from EUS is considered to be an independent predictive factor of malignancy.³ The recent study from China presented EUS features in biopsy-proven gastric linitis plastica.⁴ About sixty percent of the patients showed that the five sonographic layers had disappeared and had been replaced by hypoechogenic thickening of the gastric wall. In the rest of the patients, the first three sonographic layers were blurred and thickened, and the forth layer was significantly thickened.⁴

References
A 30-years-old male presented with severe epigastric pain radiating to his back for 4 days. He had a history of heavy alcoholic drinking about 120 grams per day for 10 years and also had several episodes of epigastric pain for 2 months. After a conservative treatment of acute pancreatitis, he developed an intractable vomiting. He was diagnosed as acute on top chronic pancreatitis with gastric outlet obstruction, A CT scan of abdomen (Figures 1-2) and esophagogastrroduodenoscopy with push enteroscopy (Figures 3-4) were done for investigating the cause of his vomiting. EUS was performed for an evaluation of local complication of pancreatitis (Figures 5-6). An ill-defined heterogenous hypoechoic lesion measuring 24x25 mm. at the head of pancreas representing a walled-off necrosis with adjacent swollen mucosa of the 2nd part duodenum was discovered.

**Figure 1** Computed tomography of abdomen showed a 5.8x3.8x3.8 cm. rim enhancing fluid collection at pancreatic head, causing pressure effect to the 2nd to 3rd part of duodenum (arrow). There also were multiple non-enhancing cystic portions scattered in entire pancreas, consistent with acute necrotic collections.

**Figure 2** Computed tomography showed a peripancreatic fluid collection at the pancreatic head causing extremely upstream dilatation of proximal duodenum, stomach and esophagus.
Diagnosis:

Acute on top chronic pancreatitis with gastric outlet obstruction due to duodenal mucosal edema and a walled-off pancreatic necrosis.
Discussion:

Severe acute pancreatitis can result in duodenal obstruction from duodenal mucosal edema or external compression due to inflammation and edema of the pancreas, pseudocysts or pancreatic abscess.\(^1,2\) The incidence of duodenal obstruction in hospitalized patients with pancreatitis is 1.2%.\(^3\) In one case series, only 9 of 878 patients of pancreatitis had duodenal obstruction associated with acute pancreatitis. All of them involved with the second or third part of duodenum.\(^1\) Duodenal obstruction associated pancreatitis is usually self-limited and successfully treated with conservative management. However, fibrosing pancreatoduodenitis may occur and requires a surgical intervention.\(^1,3\) Walled-off pancreatic necrosis (WON) which found in 1-9% of the cases. Typically occurred at 4 to 6 weeks after the initial episode of pancreatitis and may lead to pain, fever and chill. CT scan, MRI or ultrasonography can be used for diagnosing such local collection of the pancreas. Endoscopic ultrasonography (EUS) can be applied for both diagnostic and therapeutic intentions and it is so called EUS-guided transgastric necrosectomy.\(^4,5\)

References
A 70-year-old male presented with chronic epigastric pain, bloating and regurgitation for 6 years. Abdominal examination was unremarkable. EGD showed gastritis and enlargement of the major ampulla measuring about 1.5 cm. in diameter at the second part of duodenum (Figure 1). Biopsy was performed. Pathological finding showed papillary adenoma with focal high grade dysplasia. MRI of upper abdomen showed a large major papilla protruding into the duodenal lumen measuring 1.2 cm. in diameter (Figure 2). EUS was performed. It revealed a heterogenous hypoechoic ampullary mass measuring 1.0x2.2 cm. in diameter with pancreatic invasion. Normal CBD and pancreatic duct were noted. No peripancreatic lymphadenopathy were identified (Figure 3). Whipple operation was performed. The surgical histology showed tubulovillous adenoma of the ampullar of Vater with focal malignant change into adenocarcinoma (in situ), free all resected margins, no peripancreatic node metastasis (0/12 nodes) (Figure 4).
Figure 3 A heterogenous hypoechoic ampullary mass measuring 1.0x2.2 cm. in diameter was identified.

Figure 4 Histological findings showed tubulovillous adenoma of ampullar of vater.

Diagnosis:
Ampullary adenocarcinoma in situ

Discussion:
Ampullary tumors comprise less than 1% of malignant gastrointestinal tumors. Endoscopic findings of malignant transformation are the induration or rigidity of the lesion, the presence of ulcerations, lack of elevation after submucosal injection or the presence of a submucosal mass.

Normal papilla by a radial technique shows a hypoechoic, homogeneous thickening, with a crescent moon shape, well demarcated lesion next to the duodenal wall. By a linear technique, the boundaries of the papilla are less clear but opening and the tract of the bile and pancreatic ducts through the papilla can be better observed. It is difficult for EUS to identify a focal malignancy within an ampullary adenoma but invasive carcinoma can be excluded. Ampullary adenocarcinoma shows more hypoechoic and heterogeneous echogenicity than adenoma.
EUS can be used for T and N staging in TNM classification: T1 identified as no interface between ampullary tumor and duodenal wall. T2 identified as interface between ampullary tumor and duodenal wall. T3 identified as invasion of ampullary carcinoma in the periampullary pancreatic tissue less than 2 cm. T4 identified as invasion greater than 2 cm. or to other structures. Positive lymph nodes or “N” criteria include lymph nodes greater than 10 mm. round shape, distinct margins, hypoechoic echogenicity and confirmed by FNA. Meta-analysis showed the sensitivity and the specificity for detecting nodal invasion at 70% and 74%, respectively.

References
A 72-year-old Thai male presented for an annual medical checkup. CT scan of upper abdomen revealed an 8 mm. hypoechoic nodule at the body of pancreas. MRI of upper abdomen showed numerous lobulated cystic lesions scattering along the pancreas with connection to dilated main pancreatic duct (Figure 1). Radial-array EUS was performed. It demonstrated anechoic lesion measuring 19x13 mm. with thin internal septation without mural nodule at the head of pancreas (Figure 2) and another 21 mm. cystic lesion at the tail of pancreas (Figure 3). The main pancreatic duct was diffusely dilated measuring about 5 mm. at the body of pancreas (Figure 4).

**Diagnosis:**

Branch duct type intraductal papillary mucinous neoplasm (BD-IPMN)
Discussion:

IPMNs are divided into 3 types including main duct type (MD-IPMN), branch duct type and mixed type.\(^1\) EUS provides the sensitivity and the specificity at 86% and 99% respectively for the diagnosis of IPMN.\(^2\) MD-IPMN is demonstrated as diffusely or segmentally dilated main pancreatic duct (MPD) more than 5 mm. in diameter without obstruction.\(^1\) BD-IPMN is shown as a well-defined pancreatic cystic lesion communicating with the MPD.\(^1\) Unfortunately, connection between pancreatic duct and cysts cannot be demonstrated in all. Mural nodule, solid component and MPD larger than 10 mm. are features that suggestive for malignant transformation of IPMN.\(^3\)

References


Figures 3 and 4 EUS showed anechoic lesion with internal septation at the tail of pancreas with connection to dilated main pancreatic duct.
A 60-year-old Thai male presented with recurrent epigastric pain and weight loss for 10 kg in a month. He intermittently passed melena for 3 weeks. Upper endoscopy revealed multiple ulcerative masses with central necrotic area along duodenal C-loop (Figures 1 and 2). EUS demonstrated an irregular heterogeneous hypoechoic lesion measuring 20 mm. in thickness occupying the whole layers of duodenal wall (Figure 3). A perilesional homogeneous hypoechoic round lymph node measuring 12 mm. in diameter was identified (Figure 4). Histological report from mucosal biopsy was compatible poorly differentiated adenocarcinoma.
Diagnosis:
Duodenal adenocarcinoma

Discussion:
EUS has not been widely used for an evaluation of small bowel disease because the limitation of its oblique endoscopic view which makes the passage of the scope in to deep small bowel seeming impossible. Fortunately, the recently designed forward-viewing radial-array echoendoscope makes EUS feasible for the small bowel endosonographic examination. Preoperative evaluation by EUS is useful as it can determine size, echogenicity, invasion depth of the tumor, origin or margin of the lesions and regional lymph nodes metastasis. Typical endosonographic appearances of duodenal carcinoma are heterogeneous hypoechoic lesion with poorly determined margins, and loss of all layers architecture. The accuracy for predicting the malignant duodenal epithelial lesions is 93.5%. EUS currently gains popularity as the reliable preoperative diagnostics tools for local staging of upper GI malignancy.

References
An 86-year-old female presented with acute abdominal pain for 10 days. She denied fever and jaundice. Physical examination was normal. Her liver function test showed mild jaundice and transaminitis. Transabdominal ultrasonography demonstrated multiple small gallstones in the gallbladder. There was a hyperechoic lesion measuring about 6 mm. in diameter with posterior acoustic shadow in CBD (Figure 1). CBD measured as 10 mm. in diameter. Choledocholithiasis was diagnosed. Endoscopic retrograde cholangiography was performed. Cholangiogram showed multiple filling defects in dilated CBD (Figure 2). Standard sphincterotomy was done. Seven pigmented stones were removed with a trapezoid basket and a balloon.

**Figure 1** Hyperechoic lesion measuring 6 mm. in diameter with posterior acoustic shadow was detected in the common bile duct.

**Figure 2** Cholangiogram confirmed multiple filling defects in dilated CBD.
Diagnosis:

Choledocholithiasis

Discussion:

Stones in the bile duct typically originate from the gallbladder. However, they can primarily develop in the bile duct. The clinical presentations vary widely from no symptom to ascending cholangitis or acute pancreatitis.\(^1\) In classical cases, diagnoses can be made by history and physical examination. However, in equivocal cases, radiological tests are required to confirm or exclude the presence of stones in the CBD. Transabdominal ultrasound which seems to be the least invasive test, unfortunately provides the low sensitivity around 30\% whereas ERCP which is the gold standard has the sensitivity and the specificity rate at 94\% and 95\%, respectively.\(^2,3\) To date, EUS with a lower complication rate than ERC has replaced ERC to prove the presence of CBD stones. According to the ASGE guideline, the patients who have symptomatic gallstone with intermediate likelihood of choledocholithiasis based on clinical predictors (e.g. age older than 55 years, abnormal liver biochemical test other than bilirubin) are candidates for an EUS evaluation of CBD stones.\(^4\)

References

A 36-year-old Thai male presented with jaundice for a month. He had a history of right upper quadrant abdominal pain lasting for 2 days before jaundice. He denied fever or chill. Liver function tests showed directed hyperbilirubinemia. CT scan of abdomen showed common bile duct dilatation, measuring 9 mm in diameter without any gallstone or other explainable causes of CBD obstruction. Radial-array EUS revealed a hyperechoic material with posterior acoustic shadow measuring 8 mm in diameter in the dilated CBD (Figure 1). Choledocholithiasis was diagnosed. Consequently, ERC was performed and showed multiple filling defects in dilated CBD (Figure 2). Standard sphincterotomy was done. Stones were removed successfully by balloon extraction.

Figure 1 EUS revealed a hyperechoic material with posterior acoustic shadow measuring 8 mm in diameter in common bile duct.

Figure 2 Cholangiogram showed multiple round filling defects in the CBD, consistent with CBD stones.
Diagnosis:
Choledocho lithiasis

Discussion:

In order to demonstrating a CBD, transabdominal ultrasound is the suboptimal test because of its poor sensitivity (22-55%). Traditionally, ERCP has been accepted as the gold standard tool for diagnosis and treatment but ERCP is associated with significant complication. Thus, in patients with intermediate risk for the presence of common bile duct stone, EUS or MRCP should be used as the first diagnostic tool to avoid the unnecessary risks from ERCP procedure.

Both EUS and MRCP have the sensitivity and the specificity rate around 90% and 99% respectively for the detection of common bile duct stone. However, the stone size is affecting the diagnostic rate of MRCP for choledocholithiasis. The sensitivity of MRCP decreases to 33-71% in the setting of small CBD stones (<6 mm). In contrast, the sensitivity of EUS for diagnosing CBD stones was not affected by the small size of stones (<5 mm).

References
A 59-year-old male presented with acute alcoholic pancreatitis 6 months ago. He had a past history of significant alcoholic drinking. MRI of upper abdomen showed pancreatic parenchymal atrophy, no calcification nor mass were detected. Bile ducts were normal. EUS was performed for an evaluation of chronic pancreatitis. It showed normal main pancreatic duct measuring 2 mm. in diameter without calculi or irregularity (Figure 1). Hyperechoic strandings and foci were seen in the head of pancreas (Figure 2). Atrophy and lobulations were noted in the body and tail of pancreas. Pancreatic mass could not be detected.

**Figure 1** EUS showed main pancreatic duct measuring 2.2 mm. in diameter. Lobulations were seen in pancreatic parenchyma.

**Figure 2** Hyperechoic strandings were seen in the head of pancreas.
Diagnosis:
   Early chronic pancreatitis

Discussion:
Endoscopic ultrasound (EUS) has become a well-accepted tool for the diagnosis of chronic pancreatitis. EUS can detect early stage of chronic pancreatitis that may be missed by other radiomodalities.\(^1\) The most practical diagnostic criteria for the diagnosis of chronic pancreatitis is Rosemont criteria which can be divided into two groups of criteria: parenchymal and ductal criteria.\(^2\) Each subgroup can be classified to major criteria and minor criteria. Parenchymal criteria compose of hyperechoic foci with or without shadowing, lobularity with or without honeycombing, cysts, and stranding. Ductal criteria are main pancreatic duct (MPD) calculi, irregular MPD contour, MPD dilatation, dilate side branches, and hyperechogenic MPD margin. These components are used for an evaluation of chronic pancreatitis (consistent, suggestive, and indeterminate).\(^3,4\) In this patient, EUS findings of hyperechoic stranding foci and lobulations were found and suggestive for chronic pancreatitis.

References
A 59-year-old male with a history of total laryngectomy for pyriform carcinoma presented for chromoendoscopy as a part of esophageal cancer surveillance. Chromoendoscopy with Lugol’s solution revealed an unstained lesion sized 1.5x1 cm. located at 26 cm. from the incisor. EUS was done to evaluate for tumor invasion. EUS showed a heterogeneous hypoechogenic lesion with an invasion to esophageal submucosa, compatible with T1 invasion (Figure 1). Thus endoscopic submucosal dissection was performed. Histopathological result showed complete resection of squamous cell esophageal carcinoma.
Diagnosis:
Early squamous cell carcinoma of esophagus

Discussion:
Accurate TNM staging of esophageal cancer is important as it will determine the specific treatment and prognosis.\(^1\) Cross-sectional imaging including CT scan, MRI and PET scan are suitable for an evaluation of metastatic or advanced stage esophageal cancer, unfortunately these tests are limited in capability to stage an early or locally advanced stage of esophageal cancer as they cannot clearly delineate esophageal wall layer.\(^2\) In contrast, EUS provides the most accurate local staging of esophageal cancer with the sensitivity for tumor invasion (T stage) at 81%-90% [higher in advanced (T4) than early disease (T1)].\(^2\)

References
A 57-years-old male presented with an incidental pancreatic cyst. He had no history of abdominal pain, nausea/vomiting, weight loss, steatorrhea or jaundice. Computed tomography (CT) of abdomen showed a lobulated multiseptated, mixed hypo- and hyperdense solid cystic lesion measuring 6x5 cm. in diameter with central calcification involving the head and neck of pancreas causing dilation (5 mm.) of the upstream main pancreatic duct (Figures 1-2). The pancreatic parenchyma appeared atrophic without calcification. EUS revealed a multi-loculated cysts with honeycomb appearance located at the head to neck of pancreas. It measured 55x68 mm. in maximal diameter. It has both micro and macrocystic appearance and was not communicated with the main pancreatic duct (Figures 3-5). Fluid was aspirated for 1 mL and tested for a String test which resulted as negative. Cystic fluid CEA and amylase levels were reported as low.
Figures 3-4 EUS revealed a multi-loculated pancreatic cysts measuring 55x68 mm. in diameter with honeycomb appearance located from the head through the neck of pancreas.

Figure 5 EUS showed a dilated main pancreatic duct measuring 6 mm. in diameter.

Figures 6-7 T1-weighted magnetic resonance (MR) with gadolinium showed an enhancement of the thin septations that radiate from a central scar. T2-weighted MR showed a homogeneously hyperintensity lesion and dilation of the proximal part of pancreatic duct.
Diagnosis:
Pancreatic serous cystadenoma

Discussion:
Pancreatic serous cystadenoma (SCA) is the most common cystic tumor of the pancreas and it is mostly benign in nature. SCA originates from centro-acinar cells which are lined by a simple, glycogen-rich cuboidal epithelium. It is commonly found in female at age 7th decades. It comprises of multiple small fluid-filled cysts, and can arise in any region of the pancreas. Most patients are asymptomatic but may have non-specific symptoms including abdominal pain, nausea/vomiting and rarely jaundice or weight loss. It is associated with Von Hippel-Lindau syndrome in certain cases. SCA manifests typically as a microcystic or honeycombed lesion, however, 20% of them have macrocystic apperance. The highly suggestive feature on CT scan or MRI of SCA is a focal, welldemarcated lesion with central scar or “sunburst” calcification, which found in only 20% of SCA. EUS typically reveals a lobular multimacro and/or microcystic lesion in the pancreas with posterior acoustic enhancement reminiscent of a honeycomb without connecting to the main pancreatic duct. The cystic fluid appeared thin, clear, non-mucinous and/or bloody appearance. Cystic fluid analysis of CEA level <4 ng/mL has 100% and 93% in the sensitivity and the specificity, respectively.

References
An asymptomatic 76-year-old male presented with widened mediastinum detected during a routine chest X-ray. CT scan of chest showed an irregular enhancing mass measuring 4.6x6.7x16.7 cm. with matted paraesophageal lymph nodes at the prevertebral region of right lung. There were feeding arteries arisen from descending aorta and branches of right pulmonary artery (Figure 1). Primary lung cancer was suspected. EUS examination demonstrated a large well-defined, irregular border, heterogeneous hypoechoic mediastinal mass located at 25-38 cm. deep from the incisor. The mass measured about 6 cm. in diameter (Figure 2). EUS-FNA was performed with a 25G FNA needle. The final cytopathological diagnosis was squamous cell carcinoma of the lung.

Figure 1 An irregular enhancing mass measuring 4.6x6.7x16.7 cm. with matted paraesophageal nodes at the prevertebral region of right lung.

Figure 2 A large well-defined, irregular border, heterogeneous hypoechoic mediastinal mass 6 cm. in diameter located at 25-38 cm. deep from the incisor.
Diagnosis:

Squamous cell lung cancer with mediastinal metastasis

Discussion:

EUS is well suited to evaluate the lesions located in the posterior mediastinum. The lesions could be lung cancer, metastatic cancer, lymphoma, tuberculosis, neurogenic tumors, duplication cysts, mediastinal abscess, and atrial myxoma etc. The overall sensitivity and specificity of EUS-FNA for diagnosing malignant lymph nodes is >90%. The most common primary site for metastatic mediastinal lymph nodes is lung cancer, of which 80% are non-small-cell lung cancers (NSCLC) and the other 20% are small-cell carcinomas.

The recent studies report that EBUS-TBNA is also highly accurate for mediastinal staging of NSCLC. EBUS-TBNA and EUS-FNA appear to be equally successful in sampling the tissue. The subcarinal nodal station, nodes in the right paratracheal regions are more easily accessed by EBUS-TBNA, whereas nodes in the subaortic and left tracheobronchial angle are better sampled by EUS-FNA.

References

A 61-year-old male presented with acute confusion for 1 day. He had had a recent history of transient psychomotor retardation a month ago. Blood test showed a low level of sugar, high insulin level, and normal C-peptide concentration. CT scan of upper abdomen revealed a 1.0x0.6 cm. arterial enhancing lesion at the pancreatic body (Figure 1). EUS revealed a well-defined homogenous slightly hypoechogenic mass measuring 87 mm. in diameter at the neck of pancreas and not compressing the main pancreatic duct (Figure 2). Patient subsequently underwent a successful surgical enucleation. Surgical pathology was consistent with pancreatic neuroendocrine tumor (PNETs); insulinoma.
Diagnosis:

Insulinoma of the pancreas

Discussion:

Previously, gastroenteropancreatic neuroendocrine tumors (GEP-NETs) were rare with an incidence of 0.5% of all neoplasms.\(^1\) However, the incidence has been increased because of the advancement in radiological imaging. PNETs are clinically classified as either non-functioning or functioning PNETs. The functioning PNETs are usually small due to their early presentation from an over production of hormone(s). EUS is very helpful to locate this small tumor.

Potential pitfalls of EUS for the identification of insulinoma are its isoechoic appearance, small size, multiplicity, and pedunculated lesions at the pancreatic tail.\(^2,3\)

References

Case 16

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A 57-year-old Thai female presented with bowel habit change. Colonoscopy revealed a circumferential mass at the sigmoid colon. The scope could pass this narrowing segment (Figure 1). EUS to evaluate the depth of tumor invasion showed an irregular hypoechoic lesion measuring 38x36 mm. in diameter occupying at least half of the circumference of colonic wall (Figure 2). The lesion invaded to muscularis propria layer. Perilesional lymph node measuring 5x6 mm. in diameter was identified (Figure 3). It was staged as at least T3N1Mx by EUS. The patient underwent surgery and histology showed moderately differentiated adenocarcinoma with pericolonic nodes and later hepatic metastasis was confirmed. The final staging was T4N1M1.

Figures 2 and 3 EUS showed an irregular hypoechoic mass invading to muscularis propria with perilesional lymphadenopathy.

Figure 1 Colonoscopy showed a circumferential mass in the sigmoid colon.
Diagnosis:

Advanced sigmoid colon carcinoma (T4N1M1)

Discussion:

EUS is the accurate local staging tool for rectal cancer with the sensitivity and specificity at 80-96% and 75-98%, respectively.\(^1\) Typical EUS findings of colorectal cancer were hypoechoic lesion infiltrating into colonic wall.\(^2\) The current design of EUS with oblique endoscopic view makes the passage of the echoendoscope beyond the rectum seeming difficult. Recently, a new forward-viewing, radial-array echoendoscope is available for local staging of colon cancer beyond the rectum. A recent study showed the superior accuracy of EUS over CT scan for local staging of colon cancer.\(^3\)

References

A 73-year-old Thai male presented with fever and epigastric pain for 5 days. He had a history of acute gallstone pancreatitis 3 weeks ago. Physical examination showed localized upper abdominal distension with cystic-consistency mass without jaundice. CT scan of upper abdomen revealed a large heterogeneous cyst containing air bubbles along the lesser curve of stomach (Figures 1 and 2). He was diagnosed as infected pancreatic pseudocyst endoscopic view during EUS showed a large bulging lesion of the gastric wall (Figure 3). EUS demonstrated a large anechoic cyst measuring 12.2x11.5 cm. in diameter with small part of solid component lesion (Figure 4). EUS-guided drainage of the pseudocyst was performed with a 19G needle, a guide-wire and a cystotome (Figure 5). A cystogastrostomy was created (Figure 6). A covered self-expanding metallic stents with angled flare ends (Nagi stent®, Taewoong Medical, Gyeonggi-do, Korea), 16 mm. in diameter and 30 mm. in length, was inserted. Fluoroscopy confirmed the stent was placed in a proper position (Figures 7 and 8). The distended abdomen became nearly flat at day 2 after the procedure. However, his fever remained elevated at day 4 after the drainage procedure; we therefore decided to perform the direct endoscopic necrosectomy (DEN) for 2 more times at day 6 and 9 (Figures 9 and 10). Minimal amount of turbid fluid was seen and removed during DEN. A serial CT scan of abdomen showed a significant improvement of post drainage pseudocyst (Figures 11 and 12).
Figure 3 EGD showed a huge bulging lesion of gastric wall.

Figure 4 EUS demonstrated a large anechogenic cyst with some solid component.

Figure 5 Fluoroscopic view of a guidewire coiling inside the pancreatic pseudocyst.

Figure 6 The cystotome and diathermy was applied over a guide wire to create a cystogastrostomy tract.
Figures 7 and 8 EGD and fluoroscopy confirmed the proper position of stent (arrow).

Figures 9 and 10 Endoscopic images of the internal wall of pseudocyst during the direct endoscopic necrosectomy (DEN).
Diagnosis:

EUS-guided cystogastrostomy for drainage of pancreatic pseudocyst with direct endoscopic drainage (DEN)

Discussion:

Endoscopic intervention for symptomatic pancreatic pseudocyst drainage comprises of conventional trans-luminal drainage, trans-papillary drainage, and EUS-guided trans-luminal drainage. The clinical outcomes and technical success of these methods are comparable. Nevertheless, the advantages of EUS-guided trans-luminal drainage are the visualized approach, onsite differential diagnosis, ascertaining the nature of a fluid collection, and feasibility to perform the procedure in non-bulging lesion.

The covered self-expanding metallic stents (CSEMS) with angled flare ends (Naqi stent ®, Taewoong Medical, Gyeonji-do, Korea) is specifically designed for EUS-guided trans-luminal pseudocyst drainage. Advantages are the large caliber, long patency, and good visibility under fluoroscopy. However, stent migration can occur in around 15% and they can progress to severe complication including perforation or obstruction.
References
An 87-year-old female presented with fever and epigastric pain for 3 days. She had a past history of pancreatic cancer obstructing common bile duct that treated with a biliary metal stent 8 months ago. Her physical examination showed localized peritonitis at the right upper abdomen along with the positive murphy’s sign. She was diagnosed as acute gangrenous cholecystitis. CT scan of abdomen showed distended gallbladder with irregular wall. Pericholecystic fat stranding was also noted. The stent was in place (Figures 1-2). An emergency percutaneous cholecystostomy was performed. One week after the procedure, her peritonitis resolved and she started an oral intake. An elective cholecystectomy was offered, however the patient and her family declined and they chose to undergo an EUS guided cystogastrostomy instead.

EUS guided cystogastrostomy was performed with a 19-gauge needle (Echotip; Cook). A guidewire was then passed into the gallbladder (Figure 3). The tract was dilated with a needle knife and a 6-French Soehendra dilator. A 7-Fr 5 cm. double pigtail stent was successfully placed from the gallbladder to the stomach under fluoroscopic guidance (Figures 4-5). There was no complication.
**Figure 3** EUS images showed mild gallbladder distention with percutaneous cholecystostomy catheter in place (arrow).

**Figures 4 and 5** Fluoroscopic and endoscopic images of a double pigtail stent bridging between the gallbladder and the stomach.
Diagnosis

EUS-guided cholecystogastrostomy

Discussion

Acute cholecystitis may develop after metallic stent placement in malignant biliary obstruction. The standard management is cholecystectomy. However, since many patients in this setting are critically ill, and not suitable for surgery. An alternative treatment such as a percutaneous cholecystostomy may be offered, however, the discomfort form the external drain is a major concern. Recently, an endoscopic drainage including trans-papillary gallbladder drainage and EUS guided transmural drainage have become a more attractive alternatives. This case confirmed that an EUS-guided gallbladder drainage with a single-step stent placement is feasible and safe.

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BLAZING THROUGH WITH LASER TOWARD A NEW-GENERATION OF ENDOSCOPES.

New-generation endoscopy system with lasers

LASEREO