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

**Diagnosis:**
- Gastric antral vascular ectasia (GAVE)

**Discussion:**
- Gastric antral vascular ectasia (GAVE), also named

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**Figure 1-2:** Flat, erythematous punctuate lesions at antrum and pylorus.
watermelon stomach, is a rare entity, but is found in about 4% of all non-variceal upper gastrointestinal bleeding¹. The pathogenesis of GAVE remains poorly understood. Although GAVE is usually found in patients with severe co-morbidities like liver cirrhosis, it is also found in autoimmune connective tissue diseases, chronic renal failure and bone marrow transplantation. It has become clear, however, that portal hypertension does not play an important role in the development of GAVE. This is supported by findings that there is no significant correlation between the degree of vascular ectasia (mean mucosal capillary cross-sectional area) with the degree of portal hypertension and lack of response to measures reducing portal pressures (beta-blockade, TIPS)²,³.

It is vitally important to distinguish between GAVE and portal hypertensive gastropathy. There are distinct entities that require different treatments. Whereas GAVE is most commonly limited to the antrum, portal hypertensive gastropathy (PHG) predominantly causes changes of the mucosa in the fundus and corpus⁴. GAVE patients have more severe liver disease, greater blood loss, lower serum gastrin levels and a higher incidence of previous sclerotherapy⁵. Biopsy was the best way of distinguishing between GAVE and PHG. Microvascular thrombi, vascular ectasia, spindle cell proliferation and fibrohyalinosis in antral biopsies have all been shown to be significantly more associated with GAVE than PHG. Visible columns of red tortuous ectatic vessels along the longitudinal folds of the antrum are pathognomonic endoscopic findings for GAVE⁶. The typical lesion is limited to the antrum.

References
A 62-year-old woman presented with progressive jaundice and acute cholangitis. She had no history of recurrent epitaxis and no episode of GI bleeding. Physical examination showed icteric sclera with multiple telangiectases at the lower lip (Figure 1). Computed tomography of the abdomen showed portal AVM with mild intrahepatic duct dilatation secondary to vascular compression (Figure 2-3). She underwent ERCP that found choledocholithiasis with CBD dilatation and extraluminal compression. EGD revealed multiple angiodysplasias of gastric antrum, body, and duodenal bulb (Figure 3-8).
Figure 3: Multiple angiodysplasias in gastric antrum

Figure 4: Angiodysplasias at lesser curvature of gastric body

Figure 5: FICE image station 0

Figure 6: FICE image station 5

Figure 7: FICE image station 7

Figure 8: FICE image station 8
Diagnosis:

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

Discussion:

Hereditary hemorrhagic telangiectasia (HHT), inherited as an autosomal dominant trait, affects approximately 1 in 5,000 people\textsuperscript{1-4}. The spectrum of disease extends beyond the telangiectasia/AVM. This disease is diagnosed by the Curacao criteria which is based on the presence of at least three of four main clinical features: nose bleeding history, mucocutaneous telangiectasia, visceral involvement (pulmonary, cerebral, hepatic and spinal arteriovenous malformation), and affected first degree relative\textsuperscript{1-4}. Liver involvement consists of extensive intrahepatic vascular malformation associated with blood shunting (arteriovenous, arteriportal and/or portovenous), which leads to significant systemic and hepatobiliary abnormalities. The prevalence of hepatic involvement in HHT was 8-31% in many retrospective studies\textsuperscript{1-3}. The three most common initial clinical presentations are high-output heart failure, portal hypertension, and biliary disease. Biliary involvement characterized by right upper quadrant pain, cholestasis with or without cholangitis. Imaging studies demonstrates biliary stricture or obstruction from vascular impression, and/or bile cysts\textsuperscript{14}.

References

Case 3

Nuttaporn Norrasetwanich, M.D.
Rapat Pittayanon, M.D.
Naruemon Wisedopas-Klaikeaw, M.D.
Rungsun Rerknimitr, M.D.

A 67-year-old woman with underlying of cryptogenic cirrhosis child B underwent an EGD for esophageal varices surveillance. Standard white light EGD and FICE with magnification showed 1 cm whitish plaque, with irregular surface, at the lesser curvature of gastric antrum (Figure 1-6). Biopsy was performed. Histology demonstrated tubular adenomatous change of the gastric glands with focal high grade dysplasia (Figure 7).
Diagnosis:

Adenomatous polyp with focal high grade dysplasia, tubular proliferation

Discussion:

Gastric adenomas are precancerous neoplastic lesion. They are histologically classified as tubular, villous and tubulovillous types. Adenomatous polyps may occur sporadically or in association with FAP. Endoscopically, adenomatous polyps are typically velvety, lobulated solitary (82%), located in the antrum, typically with size less than 2 centimeters (cm) in diameter. These polyps can be circumscribed lesions, pedunculated or sessile. Histology reveals dysplastic epithelium without detectable invasion of the lamina propria. Their prevalence varies widely and is estimated to be 0.5–3.75% in western countries and 9-27% in prevalent areas of gastric carcinoma, such as China and Japan. Sporadic, gastric adenomatous polyps may be considered as one of the possible steps in the development of gastric adenocarcinoma. Both conditions are often found in patients with chronic, atrophic, metaplastic gastritis. In addition, they share a common epidemiological pattern. There is no proven association with H. pylori infection. The larger adenomatous polyp, the greater chance for polyp to contain foci of adenocarcinoma. A synchronous adenocarcinoma in another area of the stomach has been found in up to 30% of patients with an adenomatous polyp, and up to 50% of adenomatous polyps larger than 2 cm in diameter harbor a focus of adenocarcinoma. Neoplastic progression is greater when polyps are larger than 2 cm in diameter and this occurs in 28.5-40% of villous adenomas and 5% of tubular adenomas.

The risk of association between adenomatous polyps and cancer increases with age. The guidelines of the American Society of Gastrointestinal endoscopy (ASGE) recommend that adenomatous gastric polyps are at increased risk for malignant transformation and should be resected completely. Surveillance endoscopy 1 year
after removing adenomatous gastric polyps is reasonable to assess recurrence at the prior excision site, new or previously missed polyps, and/or supervening early carcinoma. If the result of the examination is negative, repeat surveillance endoscopy should be performed not more frequently than at 3- to 5-year intervals. Follow-up after resection of polyps with high-grade dysplasia or early gastric cancer should be individualized.

References

Case 4

Suparat Khemnark, M.D.
Rapat Pittayanon, M.D.
Rungsun Rerknimitr, M.D.

A 76-year-old woman underwent an EGD in order to follow up a gastric ulcer. Endoscopy showed a healed gastric ulcer with a large gastric diverticulum in the antrum (Figure 1-2).

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

**Figure 2**: FICE station 2

**Diagnosis:**
Large gastric diverticulum

**Discussion:**
Gastric diverticulum is rare and commonly incidentally detected from screening EGD. Its prevalence ranges from 0.04% by contrast study radiographs to 0.01%-0.11% by EGD database. Mostly gastric diverticulum are asymptomatic however it may present with a vague sensation of fullness or discomfort in the upper abdomen¹. A gastric diverticulum should be differentiated from a gastroduodenal fistula, or a double-channel pylorus, which is caused by a penetrating ulcer in the distal antrum that directly erodes into the base of the duodenal cap or into the bulb².

**References**
Case 5

Tanassanee Soontornmanokul, M.D.
Rapat Pittayanon, M.D.
Sombat Treeprasertsuk, M.D.
Rungsun Rerknimitr, M.D.

A 52-year-old man with underlying disease of compensated alcoholic cirrhosis underwent upper endoscopic examination for esophageal varices surveillance. It revealed the snake-skin mosaic pattern with flat hemorrhagic spots in gastric fundus, which was compatible with severe portal hypertensive gastropathy (PHG). White light endoscopy (WLE) and Flexible Spectral Imaging Color Enhancement (FICE) system (station 2 and 3) without magnification was applied to examine the lesions. Findings are shown in Figure 1-3.

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

Severe portal hypertensive gastropathy

Discussion:

Portal hypertensive gastropathy (PHG) is characterized by typical gastric mucosal lesions associated with portal hypertension. Typical location is in gastric fundus and upper body of the stomach although it can present in entire gastric mucosa or even in other part of gastrointestinal tract, including the small bowel or the colon. PHG may mimic with diffuse form of gastric antral vascular ectasia (GAVE). It is usually asymptomatic but, when symptomatic, it most frequently causes chronic gastrointestinal blood loss and iron deficiency anemia. PHG may present with hematemesis and/or melena, as an uncommon cause of acute gastrointestinal bleeding (GIB) in patients with cirrhosis. Diagnosis of acute GIB from PHG is established when active bleeding from gastropathy lesions or non-removable clots overlying these lesions is observed or when there is PHG without other cause of GI bleeding can be demonstrated. Non-selective beta-blockers have been shown to decrease bleeding from both acute and chronic forms of bleeding from PHG.

References

Case 6

Suparat Khemnark, M.D.
Rapat Pittayanon, M.D.
Linda Pantongrag-Brown, M.D.
Sombat Treeprasertsuk, M.D.
Rungsun Rerknimitr, M.D.

A 61-year-old man, diagnosed as cirrhosis with hepatoma from hepatitis B virus, underwent an EGD for esophageal varices surveillance. EGD with Flexible Spectral Imaging Color Enhancement (FICE) system was performed. It revealed large gastric varices at the cardia without recent bleeding stigmata (Figure 1-2). CT scan of the abdomen showed an enhancing tortuous, tubular structure on gastric fundus mucosa which was consistent with gastric varices (Figure 3).

Diagnosis:

Gastric varices

Discussion:

Gastric varices are dilated submucosal veins within the wall of the stomach. Sarin et al. has classified gastric varices into four anatomical types; 2 types of gastroesophageal varices and 2 types of isolated gastric varices. Type 1 gastroesophageal varices (EGV) which
involve lesser curvature of the stomach are the most common type. Type 2 gastroesophageal varices located on greater curvature are associated with higher mortality rate and can bleed easily. Type 1; isolated gastric varices involve only gastric fundus; have a high incidence of bleeding. Type 2; isolated gastric varices; are mainly ectopic.

Splenomegaly, portal venous thrombosis, platelet count <135,000/mm³, and albumin <3.5 g/dl are independent predictors of large EGV in hepatocellular carcinoma patients. Most of these predictors are related to the complications of portal hypertension.

On CT scan, varices appear round, tubular, or serpentine structures that are smooth with homogeneous attenuation, and enhance with contrast material to the same degree as adjacent vessels.

References
Case 7

Suparat Khemnark, M.D.
Rapat Pittayanon, M.D.
Sombat Treeprasertsuk, M.D.
Rungsun Rerknimitr, M.D.

A 65-year-old woman had been fed via balloon-type percutaneous gastrostomy tube for 2 years due to acute stroke and bed-ridden status. Over the last month, she suffered from peri-stoma leakage causing irritation on skin after gastrostomy tube was exchanged. Two weeks later, she came back to the hospital and the physician increased water volume to the balloon. Probably by gravity the PEG tube accidentally migrated down and left with only a short distance of feeding end near the skin (Figure 1). EGD found migration of balloon gastrostomy tube to duodenum. Balloon was deflated and removed. Gastric ulcer under the area of compression was found (Figure 2-3). A new PEG (non-balloon type) placement was done.

![Figure 1: Shortening of gastrostomy tube](image1)

![Figure 2: PEG migration](image2)

![Figure 3: Compression ulcer](image3)
Diagnosis:
Gastrostomy tube migration

Discussion:
PEG tube placement is associated with several complications. The migration of the balloon into the pylorus, duodenum or proximal jejunum can cause various symptoms including abdominal pain, recurrent vomiting and increased leakage around the stoma, jaundice, pancreatitis, and gastrointestinal obstruction\(^1\-\(^3\).

Upper gastrointestinal study can confirm the diagnosis. In the case of balloon type PEG tube, deflating the balloon and pulling the tube back should be done in order to relieve the symptom\(^1\).

References
A 56-year-old man presented with bilious, post-pandrial vomiting for 1 month. He had lost his weight, about 30 kg in a few months. Physical examination revealed hyperactive bowel sound with succussion splash at the epigastrium. Epigastric mass was palpable. Thus, gastric outlet obstruction was diagnosed. CT of the whole abdomen demonstrated a circumferential mass with multiple small calcifications involving the antrum and pylorus, causing gastric outlet obstruction (Figure 1-2). EGD revealed a large circumferential ulcerative mass, containing necrotic tissue at the antrum of the stomach, which was easily bled with contact. Scope could not pass through the pylorus due to complete obstruction by the antral mass (Figure 3-8). Biopsy specimen showed patchy infiltration of tumor cells possessing pleomorphic nuclei with vacuolated cytoplasm. Nuclei were paced at periphery. The tumor cells were surrounded with mild mucinous lake and inflammatory background. Focal necrosis and mitoses were also observed. The diagnosis was poorly-differentiated adenocarcinoma with signet-ring cell appearance (Figure 9-10).
The 5th Atlas for GI Endoscopy (FICE)
Diagnosis:
Gastric adenocarcinoma (poorly-differentiated adenocarcinoma with signet-ring cell appearance)

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

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

References
A 77-year-old female, presented with intermittent melena for 3 months. Her physical examination showed cervical lymphadenopathy. EGD with FICE system was done and found a huge exophytic mass at gastric antrum as shown in Figure 1-4. Biopsy was done and the pathological report revealed moderately-differentiated adenocarcinoma (Figure 5).
Diagnosis:
Adenocarcinoma of stomach

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

EGD is the currently the procedure of choice for the diagnosis of gastric cancer. Tissue diagnosis and anatomic localization of the primary tumor are best obtained by EGD. Distinct irregular mucosal surface and vascular patterns have been found to correlate with the presence of dysplasia and carcinoma. About 90% to 95% of cancerous (malignant) tumors of the stomach are adenocarcinomas. Endoscopic findings of a gastric adenocarcinoma are in variety appearances such as mass (exophytic mass, circumferential mass) or depressed mucosal lesion (ulcerated mucosa).

References
A 35-year-old woman presented with chronic intermittent dyspepsia for 2 years. Her symptom was not respond by PPI therapy. EGD with 50x to 100x magnified FICE was performed. It revealed light blue crest (LBC) which is a fine, blue-white line on the crest of epithelial surface (Figure 1A), large long crest (LLC) which is a combination of linear dark and light areas (Figure 1B), and villous pattern (VP) which is a raised area of villi above the gastric mucosal surface (Figure 1C). Biopsies for gastric intestinal metaplasia (GIM) diagnosis from many abnormal areas were done (Figure 2).

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

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

Discussion:
GIM is a well known premalignant lesion for gastric cancer\(^1\). From the current studies, the accuracy of digital chromoendoscopy (esp. NBI) for GIM diagnosis is varying from 78%-98% by using light blue crest (LBC) criteria\(^2,4\). Rerknimitr et al. proposed the other two findings for GIM diagnosis which are light long crest (LLC) and villous pattern (VP). However LLC and VP showed low sensitivity (17% and 29%, respectively) with good specificity (95% and 97%, respectively)\(^4\).

Reference