Part 2

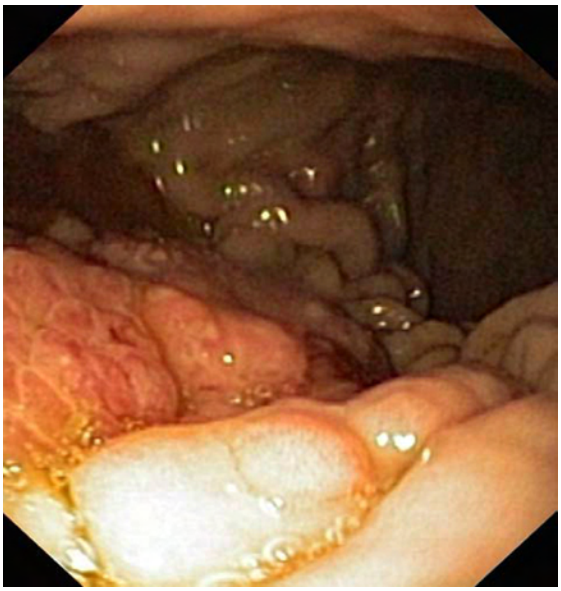
Stomach

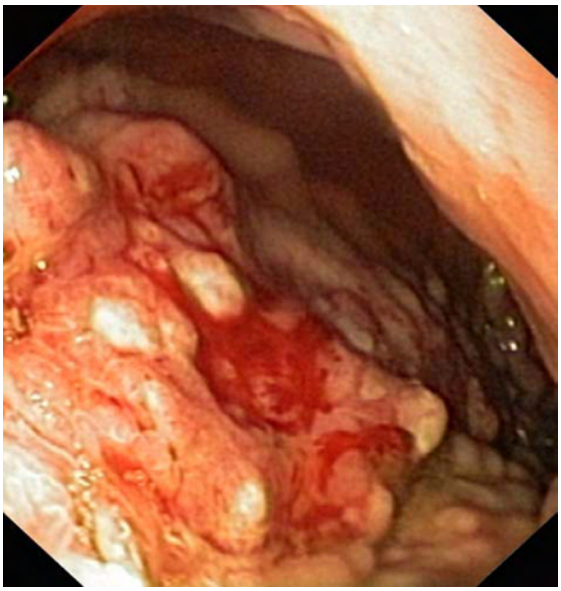
Case 1

Boonlert Imraporn, MD.

Rungsun Rerknimitr, MD.

A 50-year-old man with a history of advanced-stage malignant pancreatic neuroendocrine neoplasm of the pancreas presented with hematemesis for one day. He did not complain of abdominal pain. His vital signs were normal. Esophagogastroscopy was done as shown.





In this case, EGD showed a large mass with friability protruding trough the posterior wall and the greater curvature of the stomach. The normal gastric rugae pattern was loss. This was caused confirmed by histology as **local invasion of malignant pancreatic neuroendocrine tumor.**

Differential diagnoses:

Gastric adenocarcinoma, gastric lymphoma, and other metastatic cancer.

Discussion:

Pancreatic neuroendocrine tumors are rare and represent only 1-2 percents of all pancreatic tumors[1]. The World Health Organization (WHO) classifies this tumor into three types including well differentiated neuroendocrine tumor, well differentiated neuroendocrine cancer and poorly differentiated neuroendocrine carcinoma[2]. In addition, the tumor is defined as functioning or non-functioning tumor according to hormonal production. Generally the functioning tumor as well as well differentiated tumor has better prognosis[3]. Surgery is still the main treatment for these patients.

References

1. Dixon E, Pasieka JL. Functioning and nonfunctioning neuroendocrine tumors of the pancreas. Current opinion in oncology 2007;19:30-5.
2. Papotti M, Volante M, Bongiovanni M, Pecchioni C, Bussolati G. [Diagnosis of (neuro) endocrine tumors: a brief practical review of new classifications and new markers]. Pathologica 2000;92:562-8.
3. Kouvaraki MA, Shapiro SE, Cote GJ, et al. Management of pancreatic endocrine tumors in multiple endocrine neoplasia type 1. World journal of surgery 2006;30:643-53.

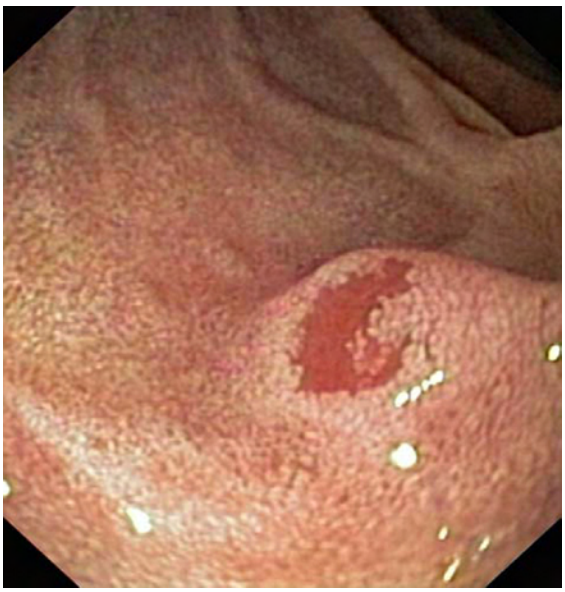
Case 2

Phonthep Angsuwatcharakon, MD.

Rungsun Rerknimitr, MD.

A 69- year-old man presented with chronic dyspepsia.

EGD was done as shown.



The EGD showed a 0.3 cm. nodule with smooth surface and salmon-pink mucosa in the second part of duodenum. Biopsy was taken.

Histology revealed gastric mucosa

The diagnosis is duodenal gastric metaplasia

Disscussion:

Duodenal gastric metaplasia is the presence of gastric epithelium in the the duodenum. It represents mucosal response to excessive injury mainly acid. *Helicobacter pylori* can colonize in this area and cause duodenal ulcer. Risk of ulcer increases around 5-fold with the presence of gastric metaplasia or *H. pylori* and increases up to 50-fold in the presence of both[1].

Reference

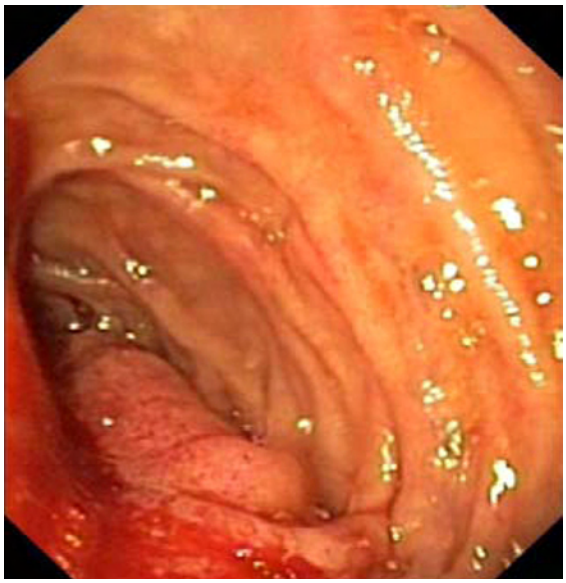
1. Peura DA. Ulcerogenesis: integrating the roles of Helicobacter pylori and acid secretion in duodenal ulcer. Am J Gastroenterol. 1997 Apr;92(4 Suppl):8S-13S; discussion S-6S.

Case 3

Chatporn Kittitrakul, MD.

Rungsun Rerknimitr, MD.

A 59-year-old female patient, presented with hematemesis for 2 hours. Esophagogastro-duodenoscopy was done as shown.



Endoscopic findings:

a large mass with friable mucosa occupied half of the duodenal lumen and extended from post-duodenal bulb to 2nd part of the duodenum

Diagnosis:

Invasion of extrinsic cancer of the duodenum

Differential diagnoses:

Pancreatic cancer invasion, hepatocellular carcinoma invasion, cholangiocarcinoma invasion, gallbladder carcinoma invasion, and gastrointestinal tumor (GIST).

Discussion/comment:

Due to the characteristic of this mass which is friable and easy to develop contact bleeding, the most likely etiology of this lesion is malignancy. Differential diagnoses are periampullary cancer, carcinoma of the pancreas with duodenal invasion and other metastatic tumors. Duodenum is one the most common locations of metastatic lesions in gastrointestinal tract, which can be found up to 44.2%.[1] Primary tumors are colon cancer, lung cancer, gastric cancer, hepatocellular carcinoma, gallbladder cancer, sarcoma, renal cell carcinoma, choriocarcinoma, adrenal cortical cancer, and cancer of unknown primary.

Biopsy from this duodenal mass was done. Presence of non-keratinized squamous cell carcinoma was found, which is consistent with metastatic cancer of the cervix.

Reference

1. Wei SC, Su WC, Chang MC, Chang YT, Wang CY, Wong JM. Incidence, endoscopic morphology and distribution of metastatic lesions in the gastrointestinal tract. J gastroenterol Hepatol 2007;22:827-31.

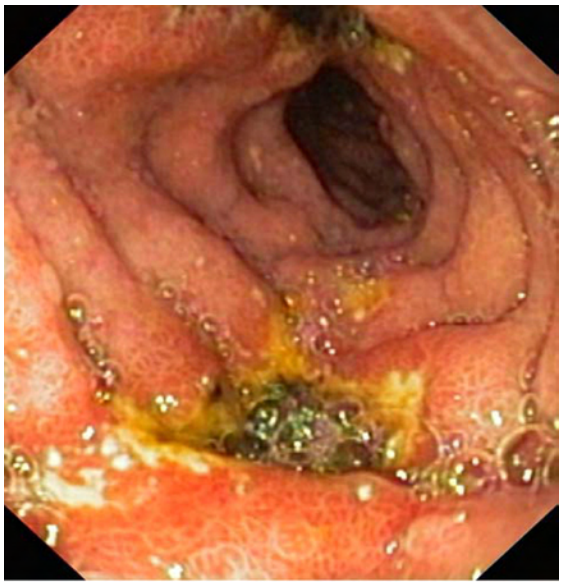
Case 4

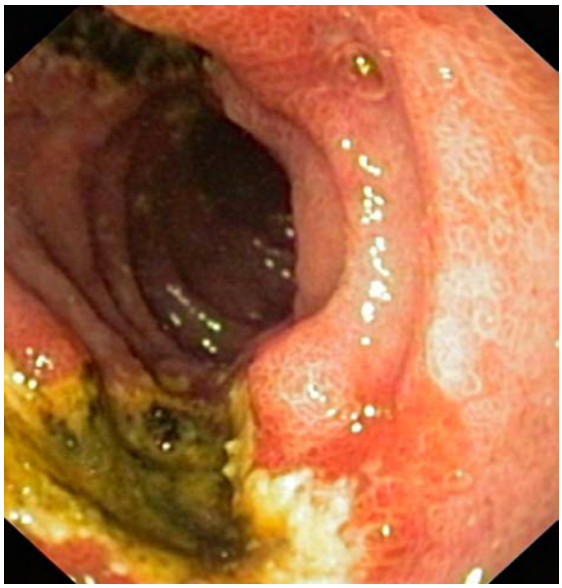
Chatporn Kittitrakul, MD.

Rungsun Rerknimitr, MD.

A 54-year-old male patient, presented with anemia without history of melena. He had a previous diagnosis of alcoholic cirrhosis, Child-Pugh C.

Esophagogastroduodenoscopy was done and shown as figures.





Endoscopic findings:

- Duodenal mucosa: diffusely swelling with mosaic pattern
- Two large clean-based duodenal ulcers in the second part of duodenum, size 3x5 cm.

Diagnosis:

Duodenal ulcers with portal hypertensive duodenopathy.

Discussion:

Causes of anemia in this patient can be explained by the endoscopic findings, duodenal ulcers with portal hypertensive duodenopathy. These lesions are not uncommon findings in cirrhotic patients. Portal

hypertensive duodenopathy can be found in 51.4% of portal hypertensive patients.[1] This condition has variable endoscopic findings. Patchy erythema is the most common lesions found in 46.3%. Others are erosions, diffuse erythema, ulcers, telangiectasia, and exaggerated villous pattern. Mixed lesions are found in 16.7% of the cases.

Previous endoscopic band ligation and coexistence of severe gastropathy were significantly more frequent in patients with portal hypertensive duodenopathy than in patients without this condition.[2]

Portal hypertensive duodenopathy was a source of overt bleeding in 6.7% and occult bleeding in 2.9% of cirrhotic patients. Histopathologically, vascular changes include either capillary congestion or capillary angiogenesis.

References

1. Barakat M, Mostafa M, Mahran Z, Soliman AG. Portal hypertensive duodenopathy: clinical, endoscopic, and histopathologic profiles. Am J Gastroenterol 2007;102:2793-802.
2. Menchen L, Ripoll C, Marin-Jimenez I, Colon A, Gomez-Camarero J, Gonzalez-Asanza C, et al. Prevalence of portal hypertensive duodenopathy in cirrhosis: clinical and haemodynamic features. Eur J Gastroenterol Hepatol 2006;18:649-53.

Case 5

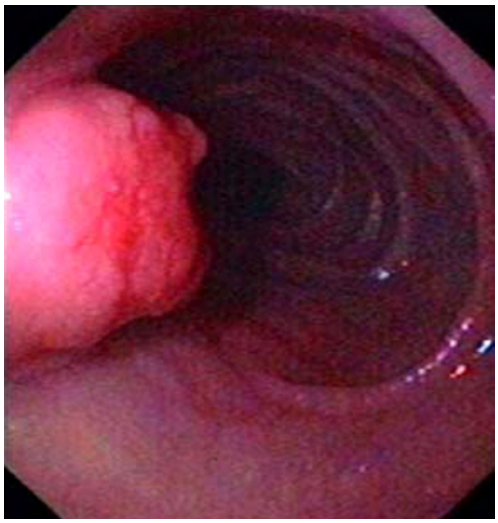
Sunthorn Treesaranuwattana, MD.

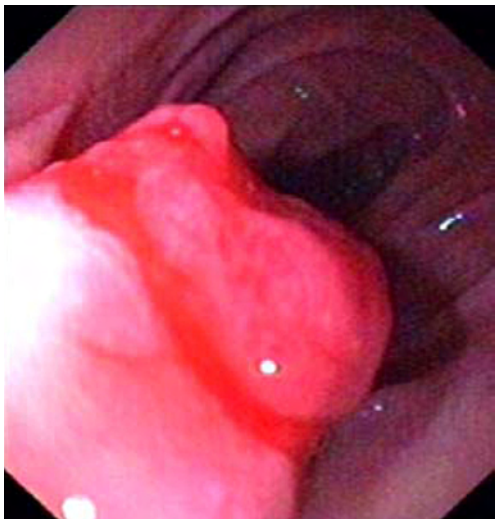
Chatporn Kittitrakul, MD.

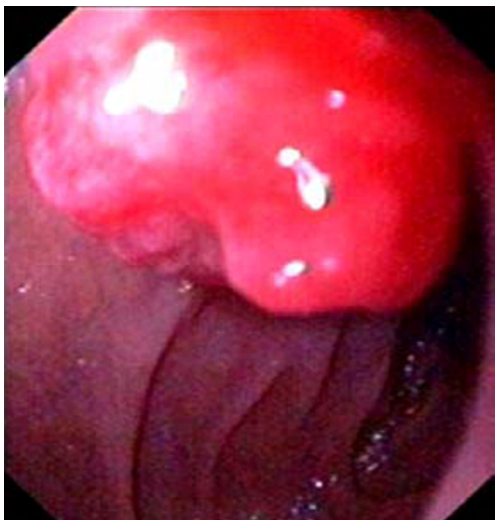
Rungsun Rerknimitr, MD.

A 53-year-old female patient, presented with dyspepsia for 2 weeks. She reported no associated symptoms.

Esophagogastroduodenoscopy was done and shown as figures.







Endoscopic findings:

- An enlarged ampulla of Vater with contact bleeding mucosa.

Diagnosis:

Ampullary tumor

Pathology report:

- Ampullary tumor biopsy: well differentiated adenocarcinoma

Discussion:

This patient presented with iron deficiency anemia without other alarming symptoms. Esophagoduodenoscopy was done to evaluate the cause of anemia despite no history of overt gastrointestinal bleeding. The ampullary mass with contact bleeding was identified as the site for occult bleeding. Since this ampullary mass is quite large with friable tissue then ampullary carcinoma was suspected. Biopsy was done and confirmed as ampullary adenocarcinoma. Typically, patients with ampullary mass present with jaundice, abdominal pain and fever. Jaundice is reported to be observed in 72-90% of these patients, but it is characterized by occasional fluctuation. Fever is present in 44% and abdominal pain occurs in 45% of patients.[1]

Standard treatment of ampullary adenocarcinoma is Whipple's resection but it contains high morbidity. Alternative treatment with endoscopic papillectomy is probably preferred in a selected case without local invasion. Hence, an accurate preoperative staging for ampullary neoplasm is mandatory before making therapeutic decisions. The use of endoscopic ultrasonography (EUS) and/or transpapillary intraductal ultrasonography (IDUS) as preoperative staging tools has been reported. Accuracy by EUS and IDUS in T staging was 63% and 78% respectively (P = .14)[2].

References

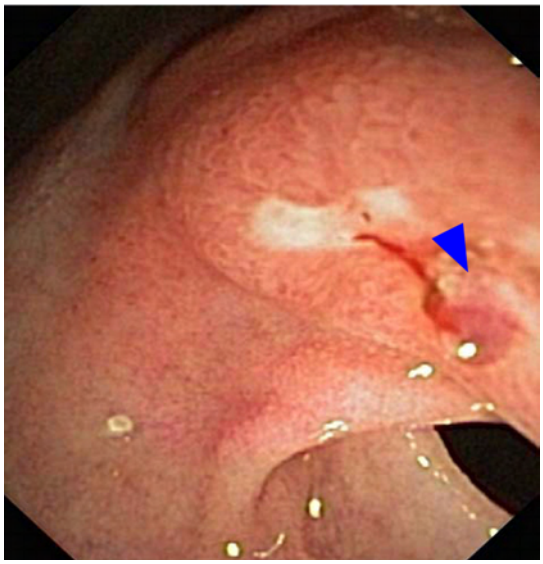
1. Kamisawa T, Tu Y, Egawa N, Nakajima H, Horiguchi S, Tsuruta K, et al. Clinicopathologic features of ampullary carcinoma without jaundice. J Clin Gastroenterol 2006;40:162-6.
2. Ito K, Fujita N, Noda Y, Kobayashi G, Horaguchi J, Takasawa O, et al. Preoperative evaluation of ampullary neoplasm with EUS and transpapillary intraductal US: a prospective and histopathologically controlled study. Gastrointest Endosc 2007;66:740-7.

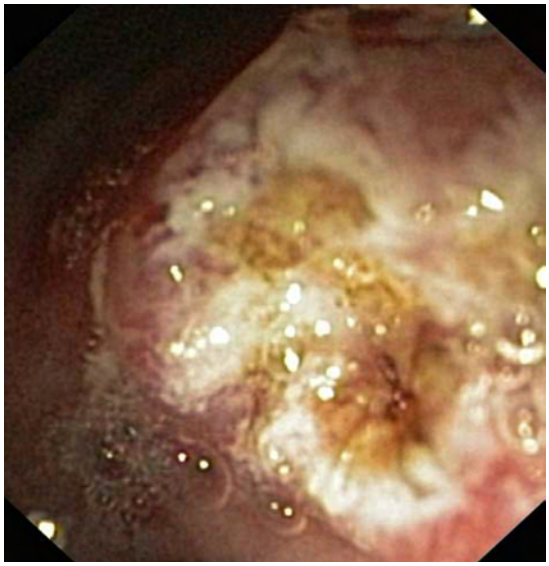
Case 6

Chatporn Kittitrakul, MD.

Rungsun Rerknimitr, MD.

A 90-year-old female patient, presented with shock from passing melena for 1 day. Esophagogastroduodenoscopy was done and shown as figures.





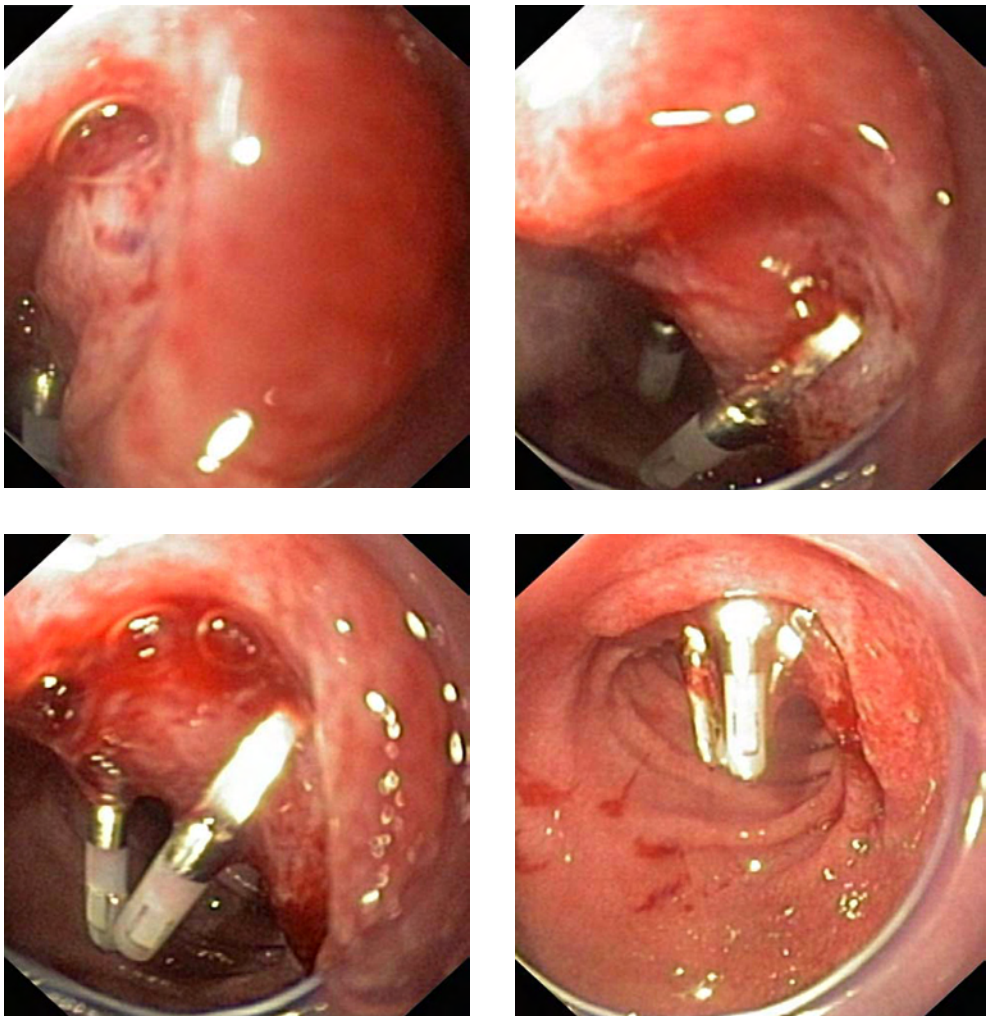
Endoscopic findings:

- Duodenal ulcer with non-bleeding visible vessel (blue arrow head) at posterior aspect of junction between duodenal bulb and second part, size 1 cm. in diameter. An argon plasma coagulator (APC) was applied at that non-bleeding visible vessel without immediate complication.

Diagnosis:

Duodenal ulcer with non-bleeding visible vessel

Later, she received intravenous proton pump inhibitor drip continuously and was kept NPO for 1 day. After then melena developed again and her hemoglobin dropped 1 g/dl from the baseline. This time, the limitation of the scope view occurred, this precluded further endoscopic therapy. Then the 2nd esophagogastroduodenoscopy with a transparent cap was done for a better view. Hemoclips were deployed over the bleeding site without complication.



After endoscopy with hemoclipped was done, she reported no recurrent bleeding and she could be discharged from the hospital within 4 days after the last endoscopy.

Discussion:

Endoscopic hemoclip hemostasis is a mechanical hemostatic method with initial hemostasis rates of 84.3-100%, rebleeding rates of 4.2-10%, and permanent hemostasis rates of 92.3-100%. The success rate is higher in cases in which endoscopic manipulation allows the clip to be placed at a more nearly perpendicular angle to the lesion. When obtaining a full view of a lesion is difficult, for example, the lesion on the posterior wall of the body or duodenal bulb, there is significant technical difficulty.[1] A nonrandomized prospective study compared bleeding control with the hemoclip with and without the aid of a transparent cap was done.[2] In this study the cap was only used in cases in which conventional clipping had failed.

There was no statistically significant differences between the patients treated with the cap and those treated without the cap with regard to the initial hemostasis rate (94.4% vs. 91.1%), the rebleeding rate (11.7% vs. 11.8%), or the permanent hemostasis rate (94.4% vs. 96.4%). Despite the non-significance finding, in authors' opinion, hemoclipping with the aid of the cap made it possible to clip a lesion too tangential to be applied.

References

1. Fuke H, Sato H, Saito K, et al. Benefits and limitations of endoscopic hemostasis for upper gastrointestinal bleeding with clipping and gel foam. Gastroenterol Endosc 1996;38:1993-8.
2. Kim JI, Kim SS, Park S, Han J, Kim JK, Han SW, et al. Endoscopic hemoclipping using a transparent cap in technically difficult cases. Endoscopy 2003;35:659-62.

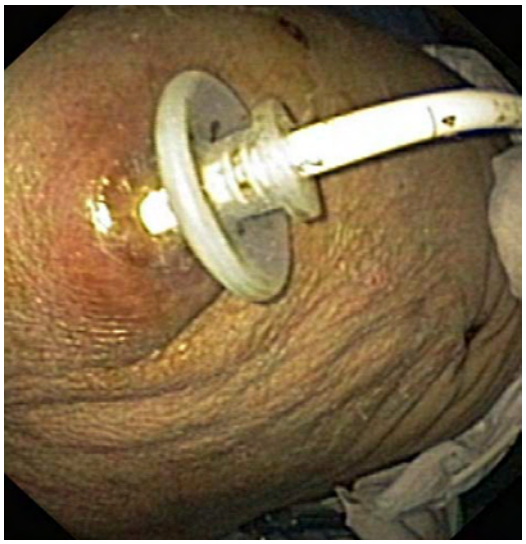
Case 7

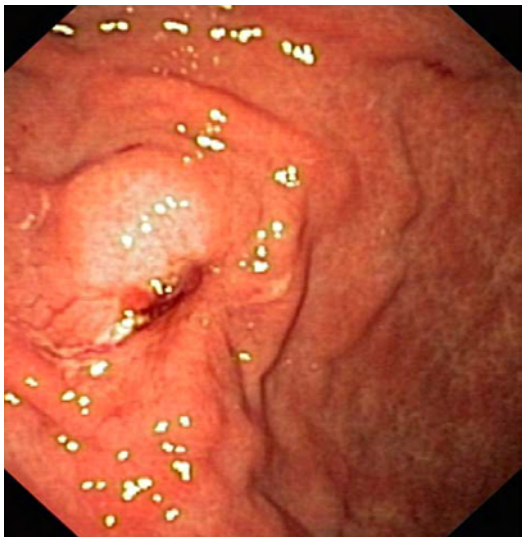
Chatporn Kittitrakul, MD.

Rungsun Rerknimitr, MD.

A 70 year-old-male patient, presented with leakage around percutaneous endoscopic gastrostomy (PEG) site after feeding. Skin under the external bumper of PEG appeared erythematous and swelling without discharge.

Esophagogastroduodenoscopy was done and shown as figure.





Endoscopic findings:

Submucosal swelling caused by the internal bumper of PEG at gastric body. The internal opening of PEG was obscured.

Diagnosis:

Buried bumper syndrome

Discussion:

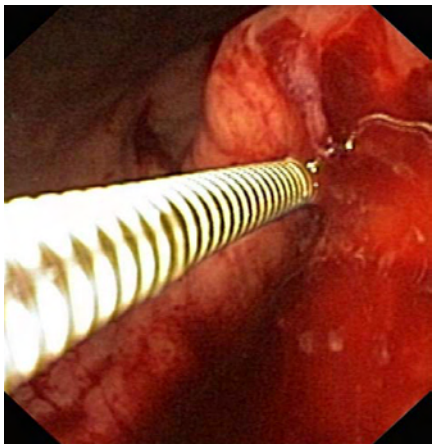
Buried bumper syndrome is defined as the migration of the internal bumper into the gastric or abdominal wall.[1] It is the well known as a long term complication post PEG that occurs in 2-6.1%.[2] Excessive tension to the skin between external and internal bumpers leads to ischemia and ulceration at the bumper site.[3] Subsequent healing causes mucosal overgrowth and burying the bumper so feeding cannot be possible.

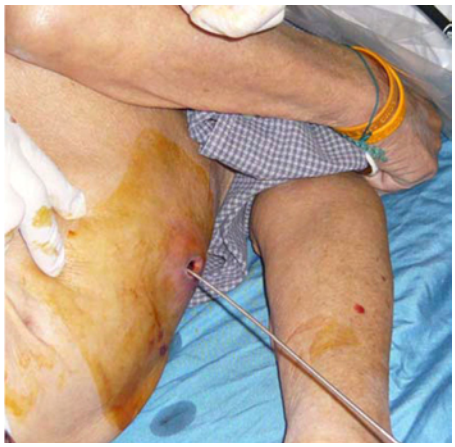
Many endoscopic methods were used to treat this syndrome. Needle knife technique and the push-pull T-technique are the methods that required advanced endoscopic skills. Both techniques had high failure rate. Other endoscopic method which used balloon dilator

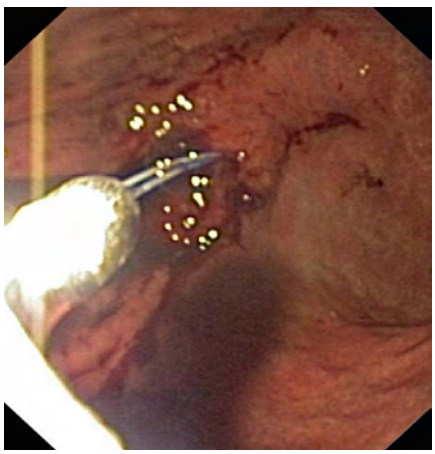
was described by Strock P and Weber J.[4] This technique can used if the internal opening of PEG was seen. Balloon dilator was inserted into the internal opening and impacted in the PEG. Traction was done to dislodge the internal bumper of PEG.

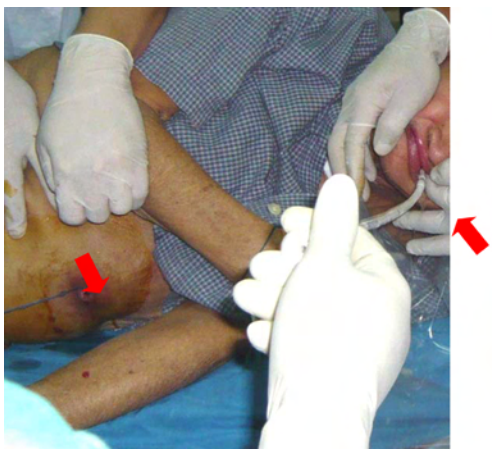
New endoscopic method which the use of ureteric catheter has been described.[3] External tube of PEG was shortened and ureteric catheter was inserted from the external opening of PEG into gastric cavity. Both ureteric catheter and external tube were tied together. Snare was used to trap the internal tip of the ureteric catheter and traction was done to dislodge the internal bumper of PEG.

In this case, we could not see the internal opening of the PEG. We used the biopsy forceps to make the hole from inside of the stomach through external opening of the old gastrostomy tube. The old gastrostomy tube was removed by pull technique. The tip of biopsy forceps was protruded from abdominal wall and tied with plastic thread. Then the biopsy forceps with plastic tread was pulled back inside gastric cavity and the standard procedure for PEG insertion was continued.









References

1. Schapiro GD, Edmundowicz SA. Complications of percutaneous endoscopic gastrostomy. Gastrointest Endosc Clin N Am 1996;6:409-22.
2. Ma MM, Semlacher EA, Fedorak RN, Lalor EA, Duerksen DR, Sherbaniuk RW, et al. The buried gastrostomy bumper syndrome: prevention and endoscopic approaches to removal. Gastrointest Endosc 1995;41:505-8.
3. Leung E, Chung L, Hamouda A, Nassar AH. A new endoscopic technique for the buried bumper syndrome. Surg Endosc 2007;21:1671-3.
4. Strock P, Weber J. Buried bumper syndrome: endoscopic management using a balloon dilator. Endoscopy 2005;37:279.

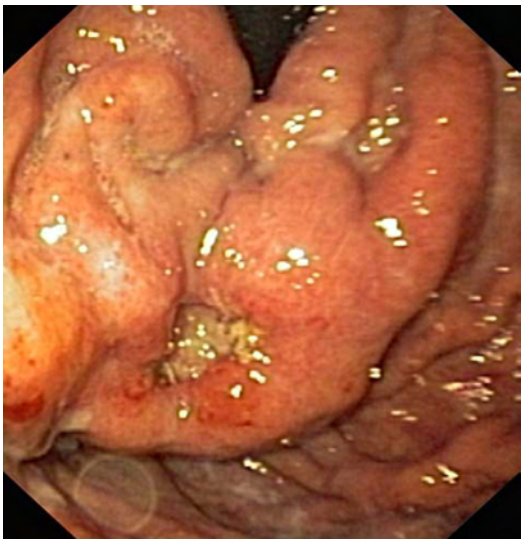
Case 8

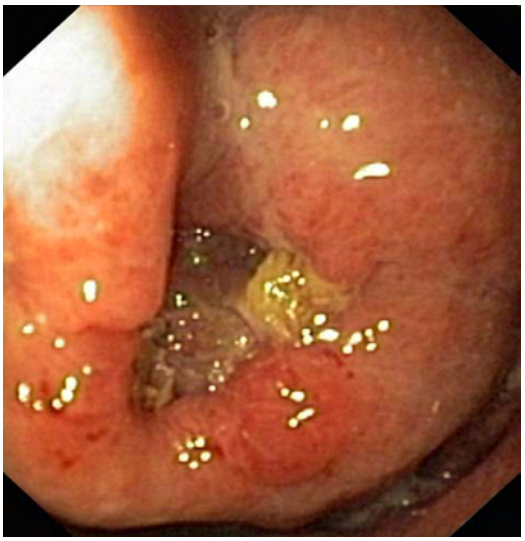
Chatporn Kittitrakul, MD.

Rungsun Rerknimitr, MD.

A 51-year-old male patient, presented with hematemesis for 2 hours. His underlying disease was alcoholic cirrhosis and he had previous hematemesis from gastric variceal bleeding 1 month ago. Upper endoscopy with glue injection for gastric varices was successfully done at that time.

Esophagogastroduodenoscopy was done and shown as figures.





Endoscopic findings:

Gastric varices at fundus, length 2 cm. with ulcer on top from the previous glue injection, no evidence of recent bleeding

Diagnosis:

Gastric varices with post glue injection ulcer

Intervention:

Glue injection was done to further obliterate gastric varices. No immediate complication occurred.

Discussion:

This patient presented with recurrent hematemesis resulted from an incomplete gastric variceal obliteration. From the endoscopic findings, gastric varices were still detected after 1 time of glue injection. Another finding was post glue injection ulcer which unlikely to be the cause of bleeding for this event. From the previous report from the King Chulalongkorn Memorial Hospital, primary success rate of glue injection for gastric variceal

bleeding was 58%.[1] Secondary success after repeat endoscopic glue injection was 12% (70% in total). Complications from glue injection were found in 54.2%, including epigastric discomfort (46%), post injection ulcer (38%), pulmonary emboli (8%) and mesenteric emboli (8%). The other recent trial from China reported that emergency hemostasis was achieved in 95.2%.[2] Complications occurred in 5.2%, including hemorrhage due to early expulsion of tissue glue (3.1%), septicemia (1%) and ectopic thrombosis (0.5%), such as splenic infarction.

References

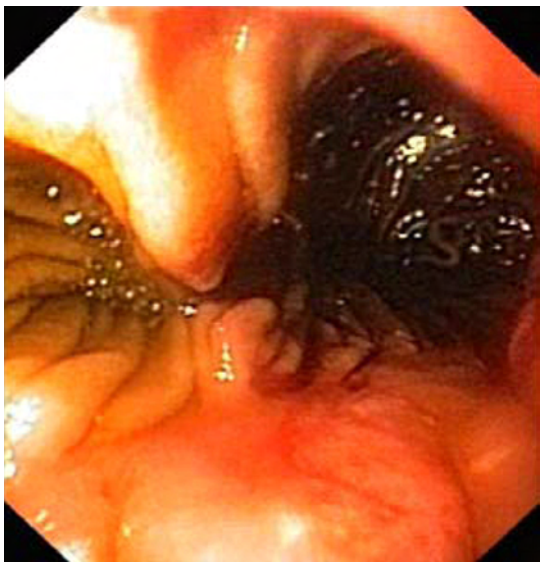
1. Noophun P, Kongkam P, Gonlachanvit S and Rerknimitr R. Bleeding gastric varices: results of endoscopic injection with cyanoacrylate at King Chulalongkorn Memorial Hospital. World J Gastroenterol 2005;11:7531-5.
2. Liu-fang C, Zhi-qiang W, Chang-zheng Li, Feng-chun C, Qi-yang H, En-qiang L, et al. Treatment of gastric varices by endoscopic sclerotherapy using butyl cyanoacrylate: 10 years' experience of 635 cases. Chin Med J 2007;120:2081-5.

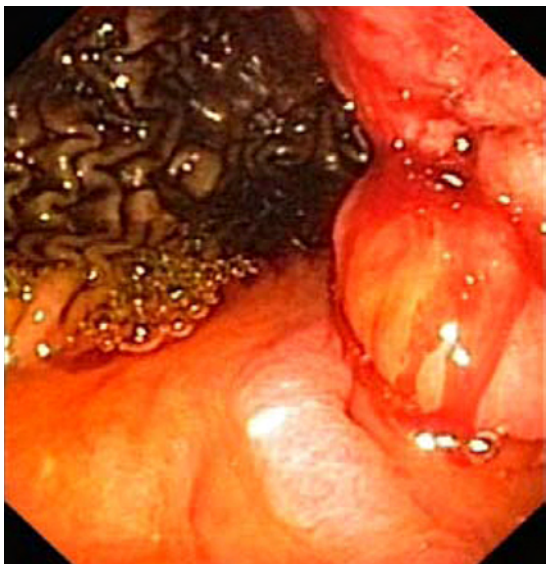
Case 9

Surachai Amornsawadwattana, MD.

Rungsun Rerknimitr, MD.

A 67- year-old male presented with melena and weight loss. He had a previous history of gastric surgery 50 years ago with unidentified indication and technique. Further investigation revealed multiple liver masses. A gastroscopy was performed as shown below.





Gastroscopic findings demonstrated a partial gastrectomy with Billroth II anastomosis anatomy. The remaining stomach showed thickening of rugae and friable gastric mucosa. No obvious mass and ulcer were detected. Random biopsy was done and histologic finding demonstrated only gastritis. He continued to have melena, then repeated gastroscopy with biopsy was done and finally disclosed moderately differentiated denocarcinoma of the stomach. Additional immunohistochemical stain showed positive stained for CK7+/CK20+. Liver biopsy was also performed and revealed metastatic gastric carcinoma. The final diagnosis was **gastric stump carcinoma with liver metastasis.** He later underwent a palliative gastrectomy to control GI bleeding. This patient died shortly after operation due to infection.

Discussion:

Gastric stump carcinoma (GSC) defined as a carcinoma developed from gastric remnant at least 5 years after gastric surgery for benign disease[1,2]. Risk of developing GSC is increased 4-7 fold after a time interval at least 20 years[2]. The presumed etiology was chronic duodenogastric reflux[1,2]. Clinical and experimental studies emphasized the important role of bile, but the precise mechanism is unknown[1]. Possible explanations were carcinogenic effect of bile acid, alteration of gastric pH and presence of bacterial flora plus unconjugated bile acid in gastric juice[1]. Type of reconstruction also influences risk of GSC, Billroth II procedure increases risk more than Billroth I procedure[2]. Treatment of choice is surgery if possible[1,2]. GSC has a poor prognosis, 5 years survival ranges from 7-20% with low respectability rate (38-40%) due to lymph nodes metastasis and local invasion[2].

References

1. Kondo K. Duodenogastric reflux and gastric stump carcinoma. Gastric Cancer 2002;5:16-22.
2. Sinning C, Schaefer N, Standop J, Hirner A, Wolff M. Gastric stump carcinoma-Epodemiology and current concepts in pathogenesis and treatment. Eur J Surg Oncol 2007;33:133-9.

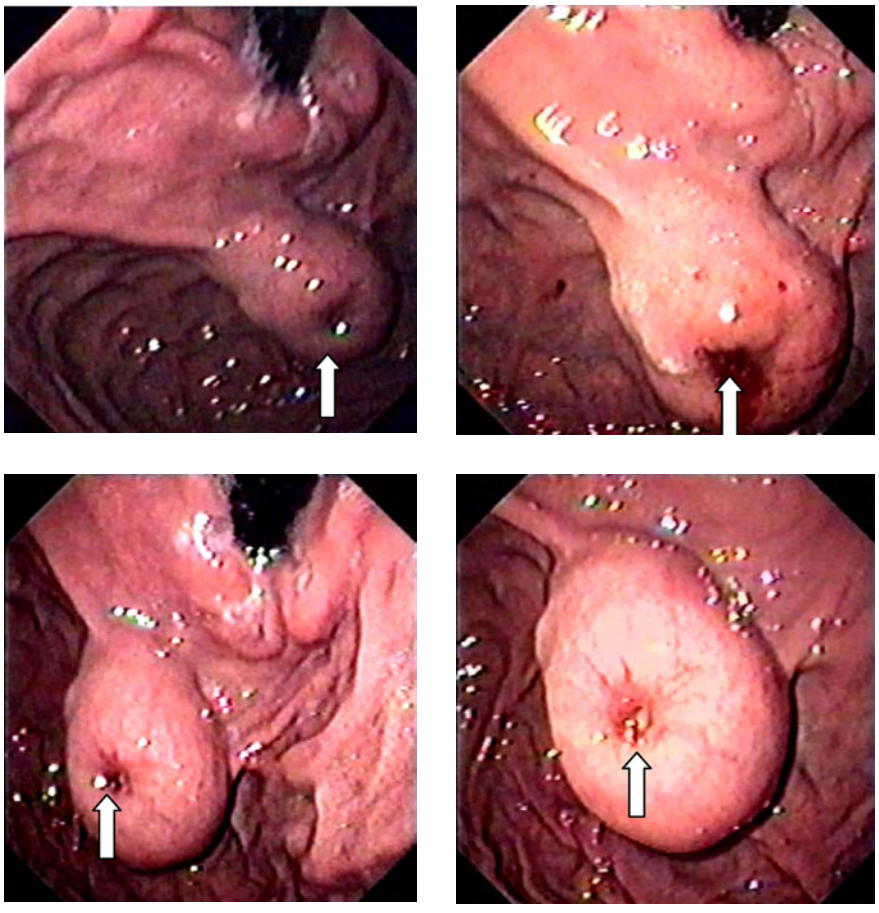
Case 10

Sunthorn Treesaranuwattana, MD.

Surachai Amornsawadwattana, MD.

Rungsun Rerknimitr, MD.

A 56-year-old man presented with abdominal pain for two weeks. Physical examination was unremarkable. Ultrasonography of the upper abdomen was unremarkable as well. A gastroscopy showed large submucosal mass at anterior greater curvature of the stomach. Mucosal ulceration at top of mass was depicted (white arrow). Wide wedge excision was later performed.



Pathological diagnosis:

Gastrointestinal tumor (GIST) of the stomach. Degree of mitosis ; <5/50HPF

Immunohistochemical study ; Positive CD117, CD34. Negative S-100, SMA

Discussion:

GISTs are rare, accounting for only 0.1-3% of all GI malignancies[1] but they represent 80% of gastrointestinal mesenchymal tumor[2]. Primary GISTs arise most commonly in the stomach (50-70%), followed by small intestine (25-35%), colon and rectum (5-10%), mesentery or omentum (7%), and esophagus (<5%)[3]. Patients generally present with non-specific symptoms including early satiety, bloating, gastrointestinal bleeding, fatigue from anemia, or obstruction. Small, clinically insignificant lesions may be found incidentally at endoscopy or at the time of surgery for other cancers.

The diagnostic studies are mainly radiographic studies which include CT scan that used for initial evaluation and surveillance for recurrence[4] or PET scan ([18F]fluoro-2-deoxy-D-glucose positron emission tomography. FDG-PET is a functional imaging technique that complements CT scan for detecting GISTs. PET scan can characterize ambiguous masses and monitoring response to therapy. Histologically, GISTs tend to fall into three categories of morphology, epithelioid, spindle cell, or mixed. Immunohistochemical analysis for KIT (CD117), expresses in 95% of GISTs. It should be performed to confirm a suspected diagnosis of GIST[5]. Five percent of GISTs, however, are KIT-negative. Such cases should be referred to an expert pathologist and may require KIT and PDGFRA mutation analysis. Prognostic factors are importantly depending on 2 characters that includes tumor sizes and mitotic index[5].

Risk	Tumor size (cm.)	Mitotic rate (per 50 HPF)
Very low	<2	<5
Low	2-5	<5
Intermediate	<5	6-10
	5-10	<5
High	>5	>5
	>10	Any mitotic rate
	Any size	>10

Treatments have been targeted by the three traditional cancer therapeutic modalities: surgery, chemotherapy, and radiotherapy. Surgery is effective for patients with resectable disease, but disease may recur in as many as 50% of individuals. Chemotherapy and radiotherapy have shown little efficacy[6].

Identification of KIT mutations led to the development of specific targeted therapies with tyrosine kinase inhibitors (TKIs). Therapy with the TKIs imatinib mesylate (STI571, Glevec; Novartis Pharmaceuticals, Basel, Switzerland) or sunitinib malate (SU11248, Sutent;Pfizer, Inc., New York, USA) is effective for unresectable, metastatic, and recurrent disease.

References

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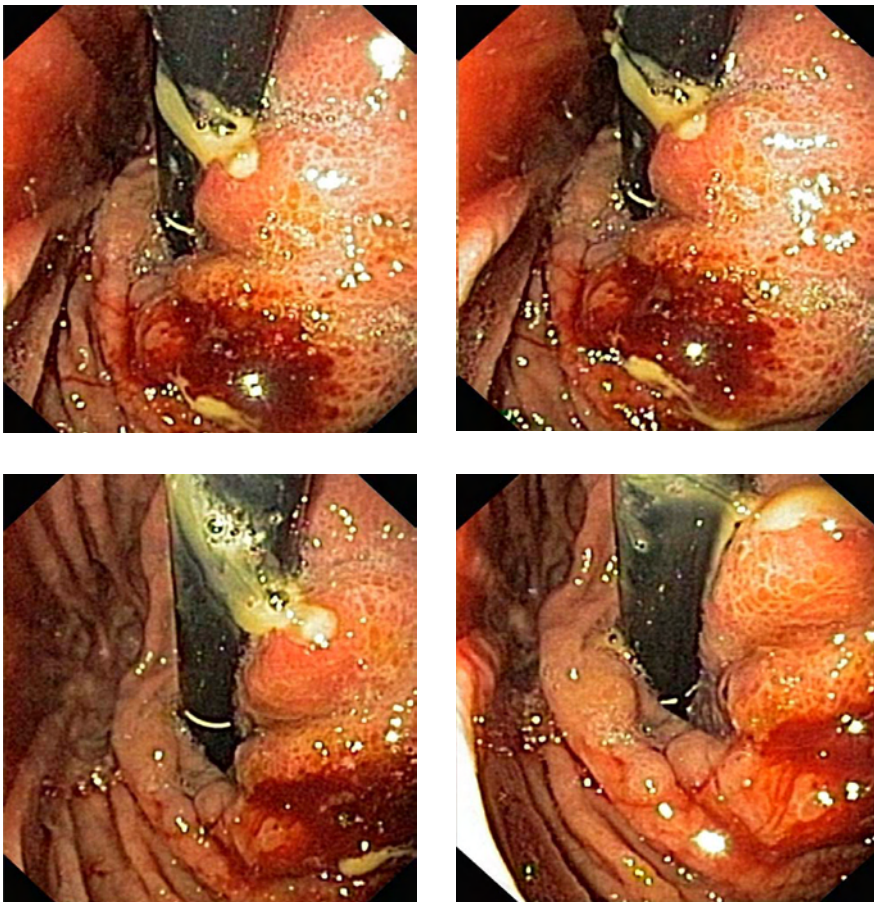
Case 11

Nathawut Sirimontaporn, MD.

Rungsun Rerknimitr, MD.

A 40-year-old male with a history of chronic hepatitis B and child C cirrhosis, presented with upper GI bleeding. An emergency upper gastroduodenoscopy showed bleeding gastric varices with severe portal hypertensive gastropathy. Cyanoacrylate injection was injected into the varices, bleeding stopped.

A follow-up upper GI endoscopy 3 days later was done as shown.



Diagnosis:

Pulurent material exploding from gastric varices.

Endoscopic procedures have become an essential tool for the diagnosis and treatment of gastrointestinal diseases, and every patient has the right to be examined and treated without risk of

transmission of infectious agents or complications that may result from inadequate reprocessing of endoscopes and endoscopic accessories. Bacterial infections have been acquired during endoscopy, caused for example by Salmonella sp., *Helicobacter pylori*[1] and Pseudomonas sp[2]. Viral diseases such as hepatitis B[3] and C[4] have also been transmitted during endoscopy. The majority of documented cases were caused by noncompliance with national and international reprocessing guideline. Gastric variceal abscess is one of the post procedure infection that might be one of the incidence.

References

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4. Bronowicki JP, Vernard V, Botte C, et al. Patient-to-patient transmission of hepatis C virus during colonoscopy. N Engl J Med 1997;337:237-40.

Case 12

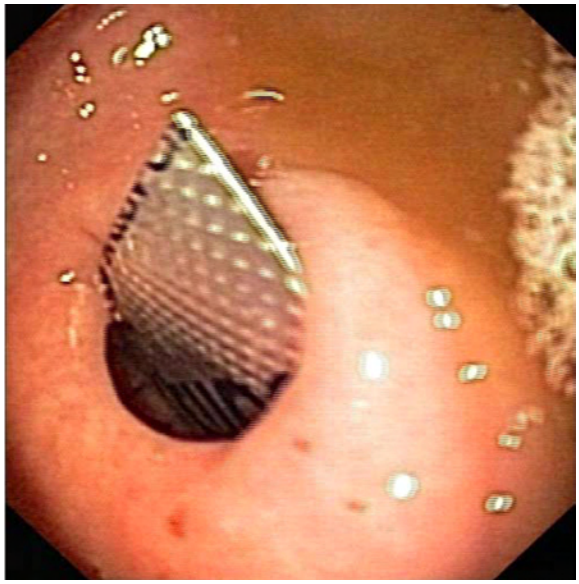
Pradermchai Kongkam, MD.

Nathawut Sirimontaporn, MD.

Rungsun Rerknimitr, MD.

A 71-year-old female, presented to the hospital with acute epigastric pain. Her past history is unremarkable except for her easily forgetfulness that was told by her family.

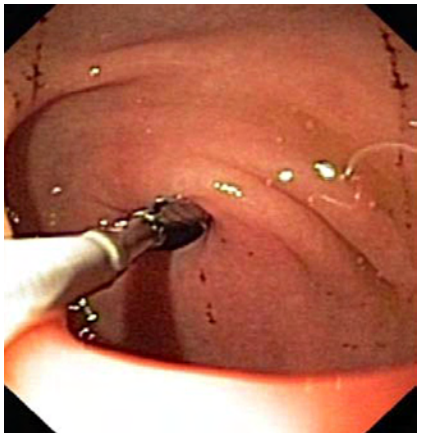
EGD was done as shown.

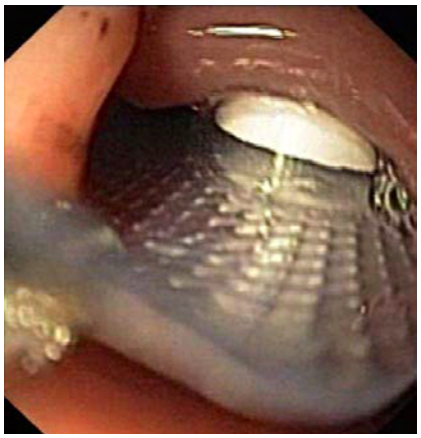


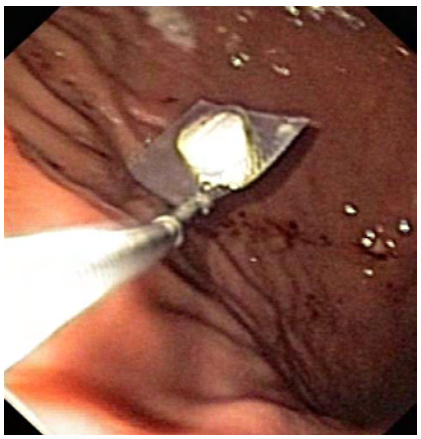
EGD showed a foreign body (sealed tablet) at the gastric outlet. The foreign body was successfully removed with a grasping forceps.

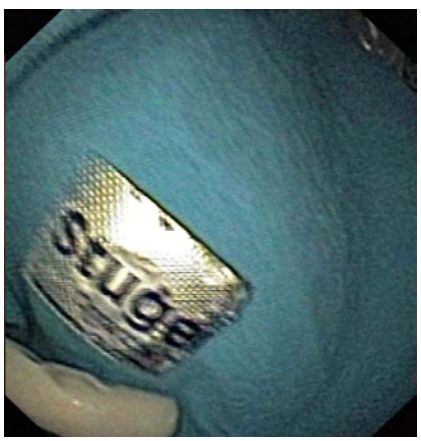
Diagnosis:

A foreign body (sealed tablet) in the stomach.









Discussion:

Most of the foreign body in stomach passes spontaneously, but 10-20% has to be removed endoscopically[1], and about 1% requires surgery[2]. Eighty percent of foreign body ingestions occur in pediatric age group[3], followed by toothless adults, prisoners and psychiatric patients. Children most often ingest coins, toys, crayons and ballpoints pen caps, whereas adults commonly tend to have problems with meat and bones. The most common locations of the obstruction are mid and distal esophagus followed by cervical esophagus and stomach respectively[4]. Once foreign body reaches the stomach, it has an 80-90% chance of spontaneous passage. The pylorus and duodenal lumen, however, may limit a passage of foreign body if it exceeds a certain size. Objects greater than 2 cm. in diameter may not pass spontaneously through the pylorus.

Although less than 1% of ingested foreign bodies cause perforation of the GI tract. However, a special shape object such as sharp or pointed objects may cause intestinal perforation; hence management of the gastric foreign body depends on the size and characters of the foreign bodies. Sharp foreign bodies such as pins, razor blades, toothpicks, and chicken bones should be endoscopically removed immediately[4]. Inert objects can be observed the progression with serial radiographs. If no movement is detected in 7 days after ingestion, removal by endoscopist is generally recommended[5].

References

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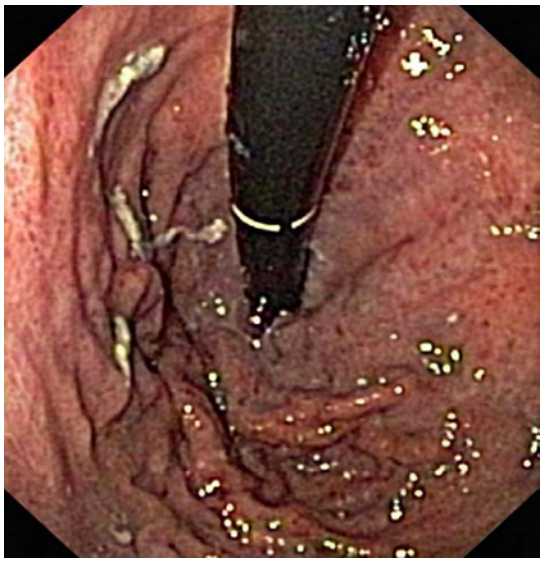
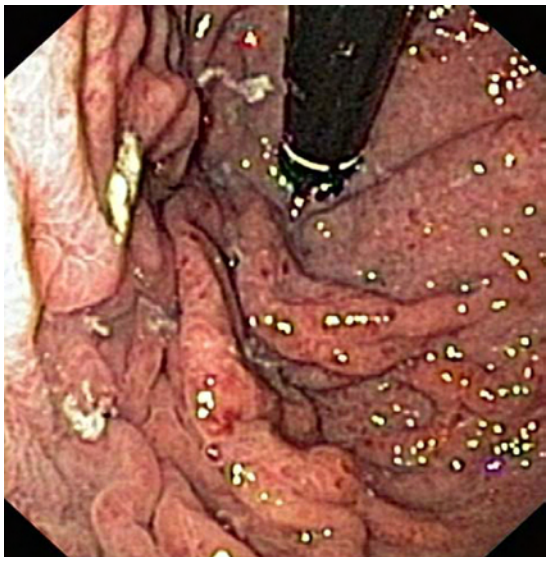
Case 13

Nathawut Sirimontaporn, MD.

Rungsun Rerknimitr, MD.

A 53-year-old female, known case of alcoholic liver cirrhosis, came for an elective EGD for surveillance of esophageal varices. The finding is shown as pictures.





Fundus:

Erythematous gastric mucosa with mosaic pattern and diffuse petichial hemorrhage was deteted.

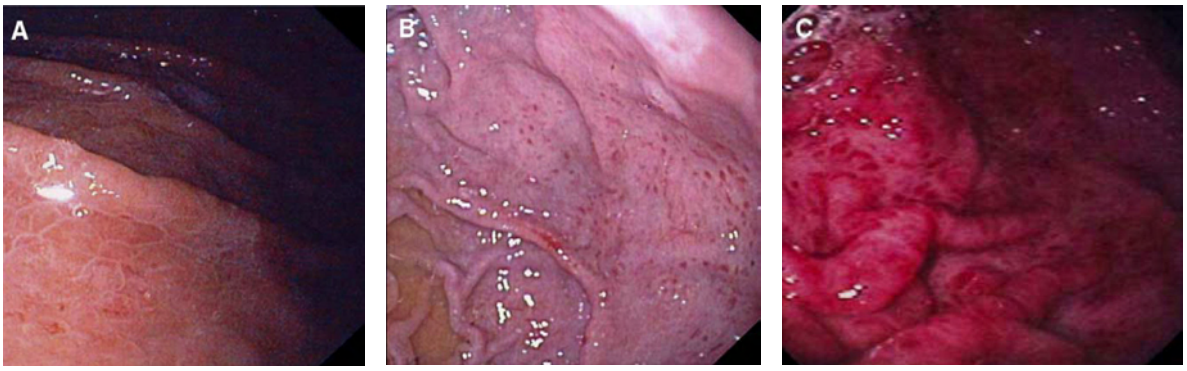
Diagnosis:

Portal hypertensive gastropathy (PHG).

Discussion:

Portal hypertensive gastropathy is a common complication of portal hypertension characterized by ectasia of the gastric mucosal capillaries and submucosal veins. The diagnostic of PHG is made endoscopically not histologically. Currently the best-described and best-validated PHG classification has been developed by the Northern Italian Endoscopic Club[1]. The key endoscopic feature is a mosaic-like pattern (snake skin appearance) that seen mainly in the fundus and cardia with less frequently seen in the antrum.

The mosaic-like pattern is described as mild when the areola is uniformly pink, moderate if the center is red, and severe if the areola is uniformly red.



A[2]: Mild PHG B: Moderate PHG C: Severe PHG

Beta-blocker seems to be an important treatment for PHG, the combination of nadolol and isosorbide mononitrate appeared similar to nadolol alone in preventing the first episode of PHG bleeding[3]. In addition, propanolol has been shown to reduce the risk of recurrent bleeding from PHG[4]. Furthermore, somatostatin can also arrest acute bleeding from PHG. An uncontrolled study with octreotide for a treatment of acute PHG related bleeding showed that the result was better than vasopressin or omeprazole[5]. The use of transjugular intrahepatic portosystemic shunts (TIPS) has been studied in some small series which show a good result in the resolution of PHG. Nevertheless, liver transplantation reverses portal hypertension most effectively.

References

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Case 14

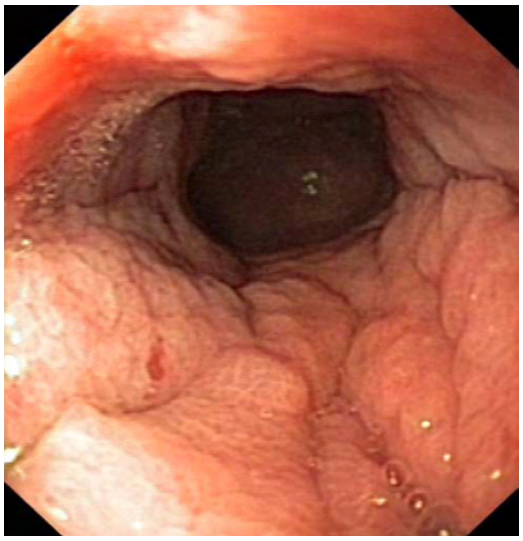
Nathawut Sirimontaporn, MD.

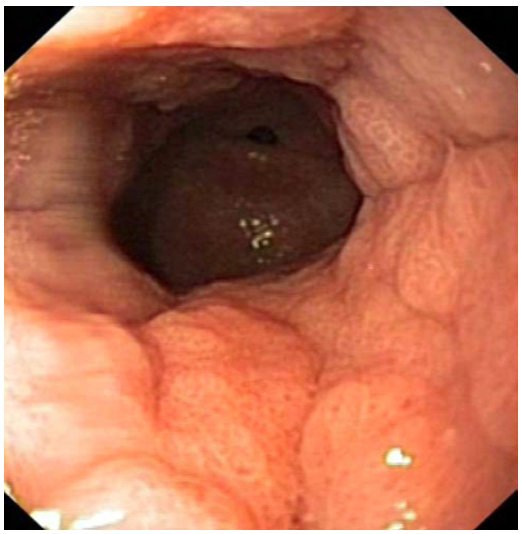
Naruemon Klaikaew, MD.

Rungsun Rerknimitr, MD.

A 47- year-old female presented to the hospital with dyspepsia, nausea and vomiting that increasing in severity. She also complained of decreasing in her appetite which made her body weight drop down 7 kgs during the past 3 months.

An EGD was done as pictures.





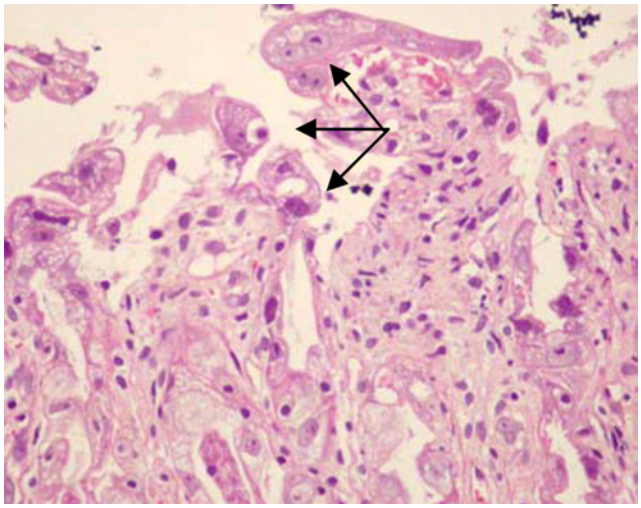
Endoscopic findings showed, diffuse mucosal thickening, contact bleeding and poor distensibility of the gastric body. This process caused narrowing of the gastric lumen as leather bag (linitis plastica).

Biopsies from the body of stomach was done

Diffuse infiltration of atypical glands lining with cells (dark arrow) possess of pleomorphic nuclei with vacuolated cytoplasm, ulceration, necrosis and mitosis.

Diagnosis:

Poorly differentiated adenocarcinoma of the stomach causing linitis plastica. Differential diagnoses are; lymphoma, amyloidosis, post corrosive restriction of the stomach, Menetrier's disease, and radiation induced gastric retraction.



Discussion:

Linitis plastica is a form of diffuse infiltrative gastric adenocarcinoma. This condition is marked by thickening and fibrosis of gastric wall like a leather bottle by having malignant cells being scarcely distributed in the fibrous stroma. Frequently, gastric mucosa is spared of malignant invasion, making an endoscopic diagnosis very difficult. The most common site of gastric linitis is the antrum and pyloric regions (with some variable spreading proximally towards gastric body). The fundus is least often involved[1].

Since macroscopic features do not often permit the distinction between benign and malignant lesions, multiple endoscopic biopsies are required. The CT scan and the endoscopic ultrasonography may be useful for a diagnosis of gastric linitis and also for the evaluation of the local extension[2]. Since Linitis plastica contains a very poor prognosis. Distant node metastasis and peritoneal seeding (carcinomatosis) make surgical excision rarely beneficial[3].

References

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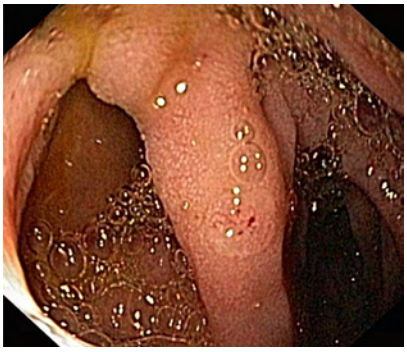
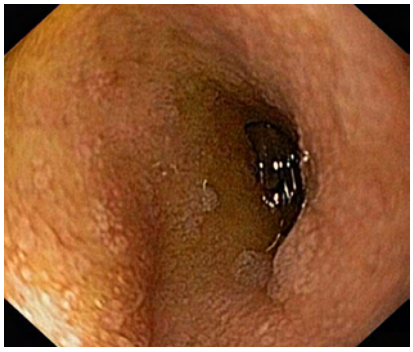
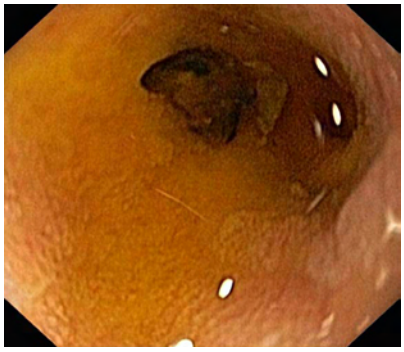
Case 15

Phonthep Angsuwatcharakon, MD.

Boonlert Imraporn, MD.

Rungsun Rerknimitr, MD.

A 71-year-old man presented with chronic dyspeptic symptoms. Esophagogastroduodenoscopy was done and shown as figures.

An EGD revealed an out pouching of the duodenal bulb. Mucosa covering the out pouching area was not typical small villi. There was a stone fragment resided in the pouch. There was a tunnel containing bile at the tip of the pouch as well.

Diagnosis:

Cholecystoduodenal fistula containing a stone.

Discussion:

Cholecystoduodenal fistula in adults often presents as a complication of duodenal ulcers or gallstones eroding into the duodenum.[1] However it can be rarely associated with congenital disease such as choledochal cyst[2]. The gallstones can entry into the bowel through a biliary enteric fistula. Sixty percent of

the fistulas are cholecystoduodenal fistulas. However, cholecystocolonic and cholecystogastric fistulas can occur[3]. Pressure necrosis by the gallstone against the biliary wall causes erosion and fistula formation. The complications of this fistula include gallstone ileus, gallbladder carcinoma, weight loss and malabsorption.

References:

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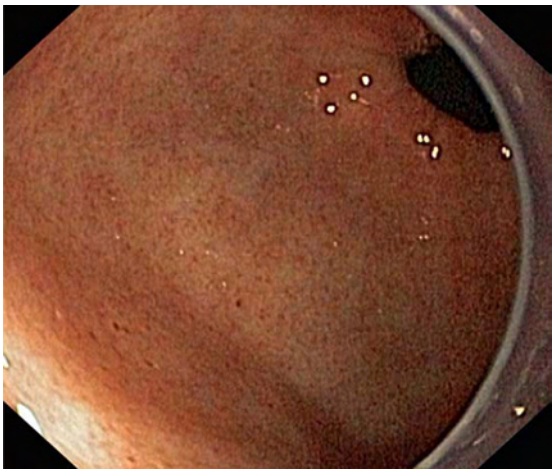
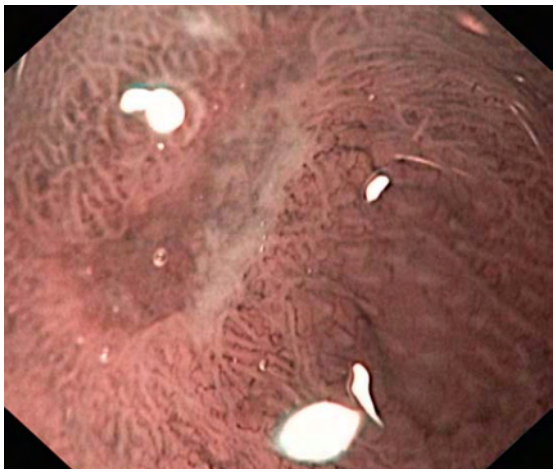
Case 16

Boonlert Imraporn, MD.

Rungsun Rerknimitr, MD.

A 65-year-old man complained dyspeptic symptom for many years. He had no alarm features. He had no underlying disease. An esophagogastroduodenoscopy was done and shown as figures.





In this case, EGD showed only very small focal depressed area in the antrum. After NBI with magnification was applied, this area revealed a loss of normal pit pattern and microvascular structure. Targeted biopsy was done and the pathological diagnosis was consistent with a small focus of signet ring cell type adenocarcinoma of stomach. His diagnosis was early depressed type gastric cancer. A repeat targeted biopsy also revealed a similar result. Elective subtotal gastrectomy was done later after complete cancer staging.

Discussion:

Narrow band imaging (NBI) with magnification possesses the role to detect small gastric lesions and is useful for targeted biopsy. Depressed-type early gastric cancer is difficult to detect due to subtle changes in color and shape. NBI can enhance the visualization of such small and ill defined lesions. In addition it can identify the demarcation line of depressed lesions[1]. The abnormal pit pattern from magnifying

NBI including irregular arrangement and size together with destructive pattern strongly correlates with the presence of gastric cancer[2,3]. However further studies are needed to show the benefit of a routine use of NBI in the clinical practice especially in the countries with low incidence of gastric cancer.

References

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3. Tanaka K, Toyoda H, Kadowaki S, et al. Features of early gastric cancer and gastric adenoma by enhanced-magnification endoscopy. J Gastroenterol 2006;41:332-8.

Case 17

Sukprasert Jutaghokiat, MD.

Rungsun Rerknimitr, MD.

A 69-year-old man presented with congestive heart failure and marked anemia. He denied a history of melena. He had past medical history of spontaneous and recurrent epistaxis since childhood. Physical examination revealed diffuse small telangiectasia at both palms and soles (Figures A, B). An esophagogastroduodenoscopy was performed and showed diffuse small telangiectasia in the gastric antrum (Figures C, D) and duodenum (Figure E). CT of the upper abdomen showed an aneurysm of right hepatic vein (Figure F).

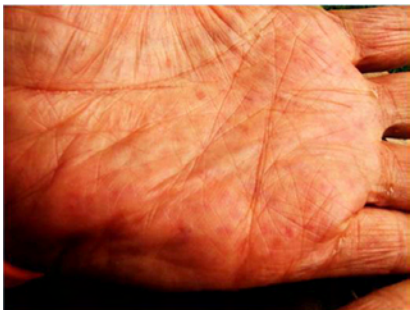

Figure A

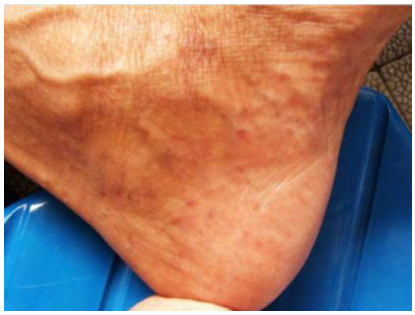

Figure B

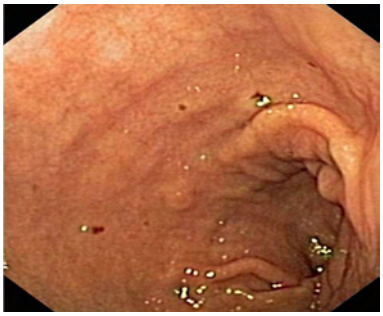

Figure C

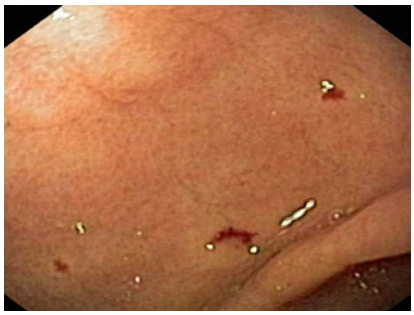

Figure D

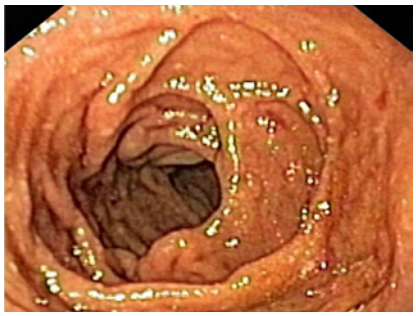

Figure E

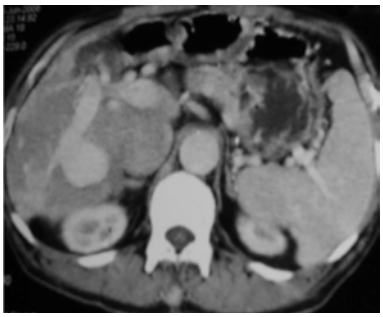

Figure F

Diagnosis:

Hereditary hemorrhagic telangiectasia (Osler-Weber Rendu syndrome)

Discussion:

This patient was diagnosed as hereditary hemorrhagic telangiectasia (Osler-Weber Rendu syndrome) according to Curacao diagnostic criteria[1]. Clinical manifestations of this syndrome are epistaxis, gastrointestinal bleeding, iron deficiency anemia, mucocutaneous telangiectasia and arteriovenous malformation of internal organ such as pulmonary, hepatic and cerebral circulation. HHT is inherited as an autosomal dominant with the two major genes mutation on chromosome 9 and 12[2,3].

Gastrointestinal bleeding often has an onset in the 5th to 6th decade of life and often presents as iron deficiency anemia which is more common than acute gastrointestinal bleeding. Abnormal telangiectasia can occurred throughout GI tract but stomach and duodenum are the most common sites. Most of patients can have a conservative management by iron supplement and blood transfusion. While other modalities are estrogen-progesterone hormone[4], anti-fibrinolytic drug[5] and endoscopic ablasion.

References

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Case 18

Sukprasert Jutaghokiat, MD.

Rungsun Rerknimitr, MD.

A 61-year-old man presented to the hospital with severe acute epigastric pain for 15 hours followed by fresh blood hematemesis. He denied any underlying disease. On arrival, the patient had high blood pressure, tachypnea and tachycardia. Physical examination showed mild pallor. Bowel sound was absent with mild abdominal distention and no peritoneal sign. Nasogastric lavage revealed fresh blood. His chest X-ray demonstrated a wide mediastinum (Figure 1) and a plain film of the abdomen showed small bowel ileus. He was sent for an emergent EGD. The findings are shown as figures 2 A, B, C. Gastroduodenal infarction was suspected and he was transferred for an emergent CT of the whole abdomen including angiography. **Acute aortic dissection (Stanford type B) with hepatic and intestinal infarction was diagnosed** (Figure 3A - B). After patient had been stabilized, an explore laparotomy was done. Gangrene of gallbladder, spleen, descending and sigmoid colon were detected. Cholecystectomy, splenectomy and left half colectomy were performed. Within the same day, fenestration of infrarenal abdominal aorta to provide blood flow to the gut was also successfully done. After surgery, the patient did well without further abdominal pain and no bleeding recurred.

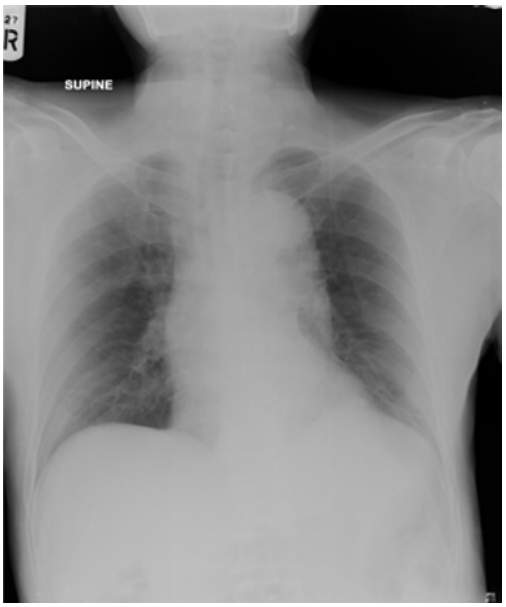
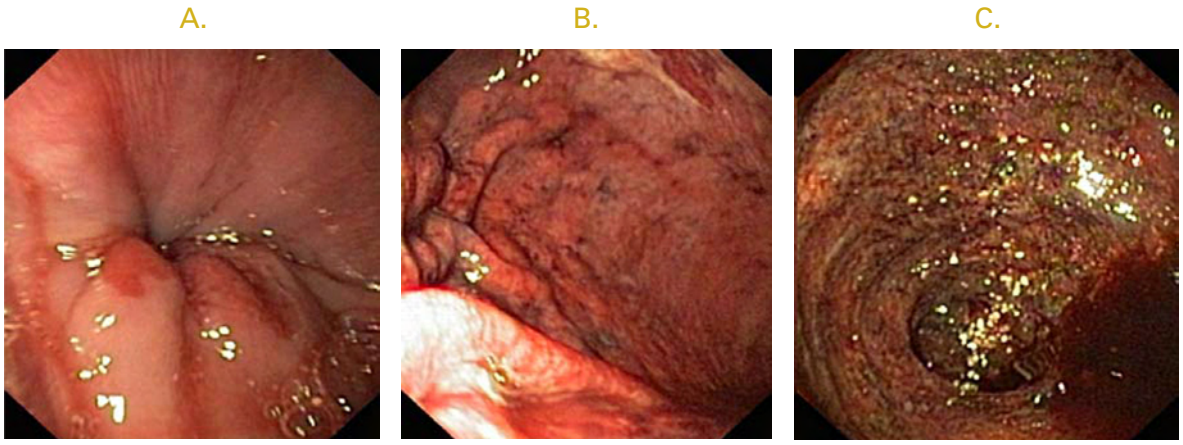
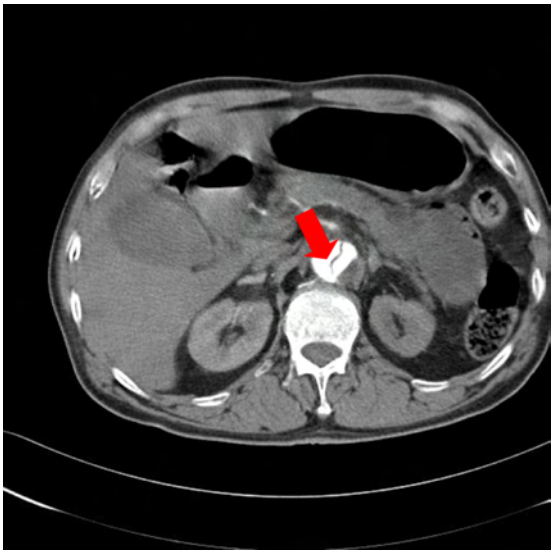


Figure 1 : A. Chest X-ray showing widening of the mediastinum.



Figures 2 : A. Demonstrating normal esophagus, B. & C. showing diffuse mucosal infarction containing necrotic slough of stomach and duodenum.



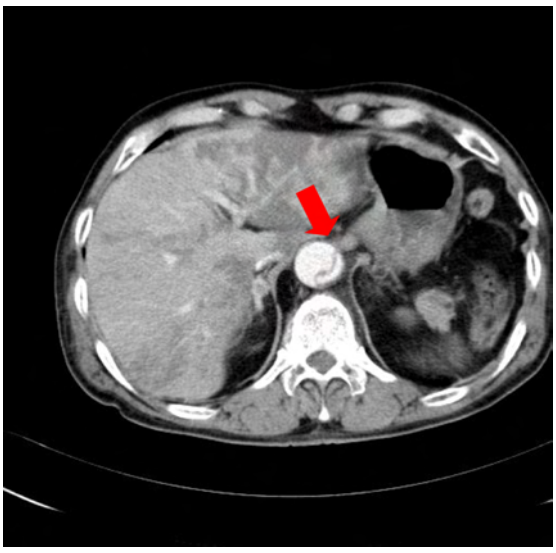


Figure 3 : A. An abdominal CT scan showed aortic dissection extending from descending thoracic aorta to entire intra-abdominal aorta, occlusion of celiac axis is noted.

B. Showing hepatic infarction (red arrows indicating a false lumen).

Discussion:

This patient presented with acute upper gastrointestinal bleeding from gastroduodenal ischemia was suffered from acute aortic dissection. In general, patients with uncomplicated dissecting aortic aneurysm type B are best treated with conservative therapy[1]. However, this patient had a complicated course of acute aortic dissection type B with disruption of blood flow to all major intra-abdominal aortic branches. Then emergent aortic fenestration in acute abdominal dissection was indicated to effectively relieve organ from gangrene. However, this procedure still carries a high mortality rate[2]. In contrast, endovascular stent-grafting is a less invasive alternative and has a lower morbidity

rate especially in subacute and chronic dissection[3]. However, the outcome is not as good as surgery for acute dissection[4,5].

References

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2. Panneton JM, Teh SH, Cherry KJ, Jr., et al. Aortic fenestration for acute or chronic aortic dissection: an uncommon but effective procedure. J Vasc Surg 2000;32:711-21.
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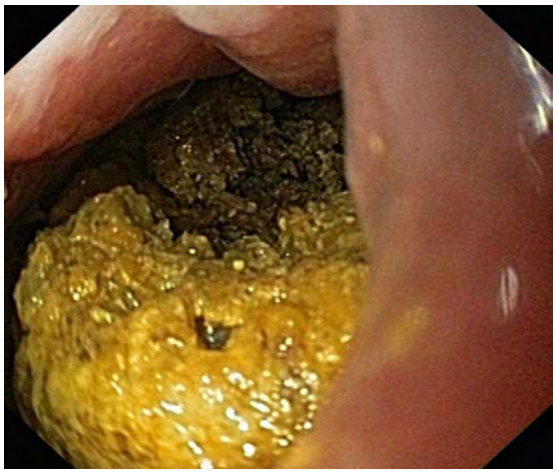
Case 19

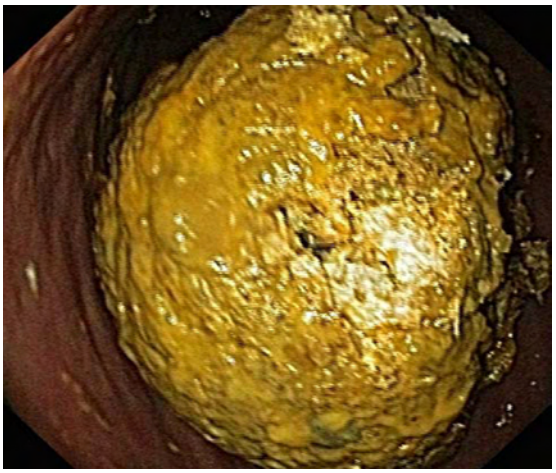
Sunthorn Treesaranuwattana, MD.

Sukprasert Jutaghokiat, MD.

Rungsun Rerknimitr, MD.

67-year-old man underwent Bilroth II gastrectomy from duodenal perforation twenty-five years ago. He experienced a severe upper abdominal pain which relieved with vomiting of undigested food for seven days. An esophagogastroduodenoscopy was done and shown as figure.

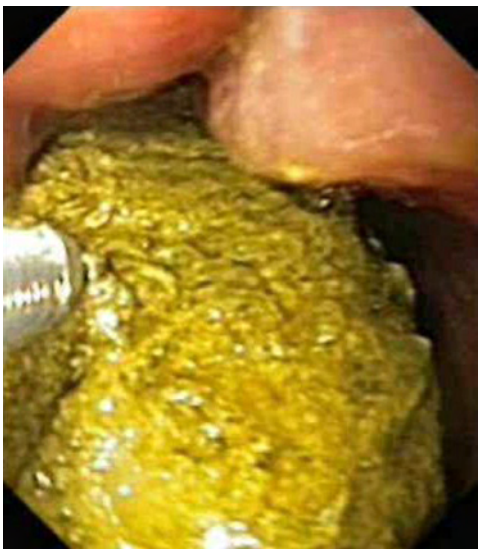


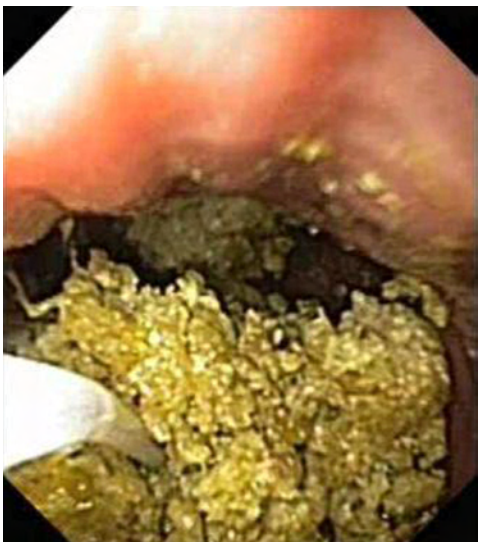


EGD showed the upper part of large bezoars seen just distal to esophago-gastric junction and occupying the entire stomach. His diagnosis was gastric bezoars. Disruption of bezoars by tip catheter and mechanical lithotripter (shown as figures below) were done. Water irrigation and snare capture were performed to facilitate the removal process.

Discussion:

Gastric bezoars characterize by the accumulation of foreign ingested material in the form of masses or concretions and are classified by their composition into three major types including phytobezoars, trichobezoars and phamacobezoars. The most common form is phytobezoars which compose of vegetable. This usually occurs in patients with gastric dysfunction such as delayed gastric emptying time.





The common manifestations include abdominal pain, vomiting, early satiety, anorexia and weight loss. The current gold standard is upper gastrointestinal endoscopy. In case of phytobezoars, mechanical fragmentation and chemical dissolution with enzyme therapy can be performed. Some agents including cellulose, acetylcystiene, coca-cola[1,2] and papain[3] have been reported to be useful.

References

1. Ladas SD, Triantafyllou K, Tzathas C, Tassios P, Rokkas T, Raptis SA. Gastric phytobezoars may be treated by nasogastric Coca-Cola lavage. Eur J Gastroenterol Hepatol 2002;14:801-3.
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3. Walker-Renard P. Update on the medicinal management of phytobezoars. Am J Gastroenterol 1993;88:1663-6.

Case 20

Sukprasert Jutaghokiat, MD.

Rungsun Rerknimitr, MD.

A 45-year-old woman presented with chronic dyspepsia for 6 months. She had no alarm features. She had no underlying disease. An EGD with Fujinon intelligent color enhancement (FICE) system was done and shown as figures.

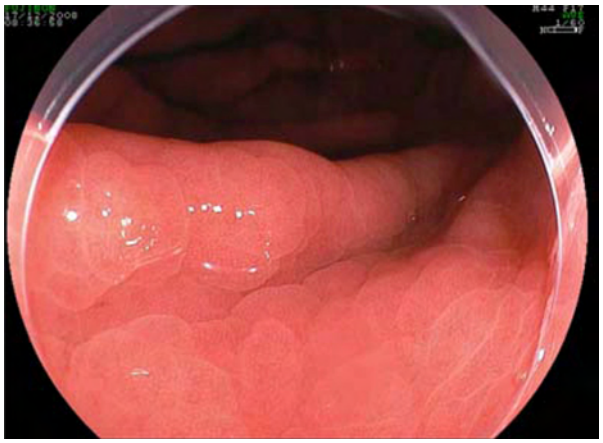


Figure A

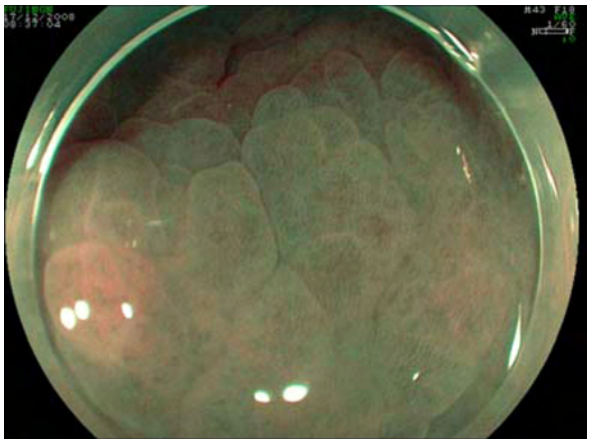


Figure B

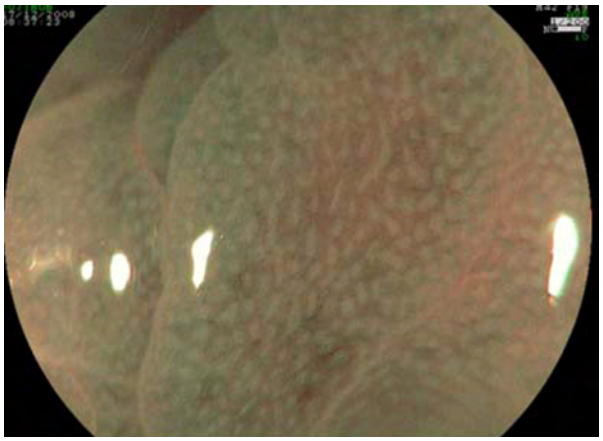


Figure C

Her diagnosis was **nodular gastritis from *Helicobacter pylori* infection.** The nodularity of gastric mucosa was seen under white light endoscopy. After switching the mode to FICE system nodularity of mucosa with regular pit pattern was described. Her result of rapid urease test was positive.

Discussion:

Nodular gastritis (NG) is a form of chronic gastritis with moderate inflammation. There are significant eosinophilic infiltrations in superficial lamina propria and lymphoid hyperplasia of gastric mucosa. It is associated with the presence of *Helicobacter pylori* infection[1,2]. This finding is more frequent in children than in adults and usually regresses after *H. pylori* eradication. Recent report in adults also represents that nodular gastritis regresses significantly after *H. pylori* therapy with a proton pump inhibitor and two antibiotics[3].

References

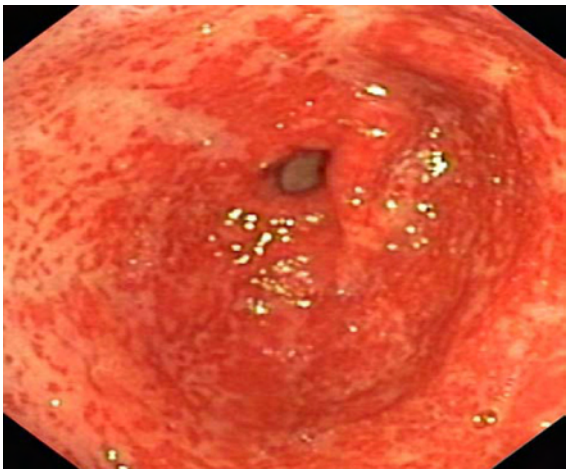
1. Maghidman S, Cok J, Bussalleu A. Histopatholigal findings in nodular gastritis. Experience at the Cayetano Heredia National Hospital. Rev Gastroenterol Peru 2001;21:261-70.
2. Rudelli A, Vialette G, Brazier F, Seurat PL, Capron D, Dupas JL. Helicobacter pylori and gastroduodenal lesions in 547 symptomatic young adults. Gastrointerol Clin Biol 1996;20:376-3.
3. Manisha Dwivedi, Sri Prakash Misra and Vatsala Misra. Nodular gastritis in adults: Clinical features, endoscopic appearance, histopathological features, and response to therapy. J of Gastroenterol and Hepatol 2008;23:943-7.

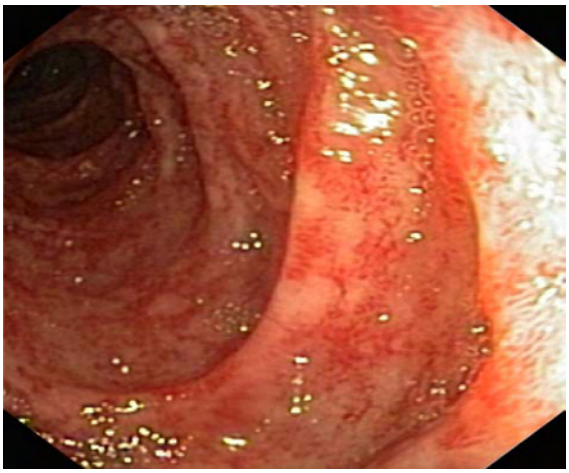
Case 21

Chatchai Kriengkirakul, MD.

Rungsun Rerknimitr, MD.

A 67-year-old woman, presented with passing black stool for 4 days prior to admission. Her underlying disease was transitional cell carcinoma of the kidney. She underwent right nephrectomy and chemoradio-adjuvant therapy lately. She looked markedly pale without jaundice. Rectal examination was done and found melena. NG lavage found coffee ground. Hematocrit dropped from 33% to 17% within 1 week. Patient received 4 unit of packed red cell transfusion. EGD was done as shown.





Endoscopic findings:

Diffuse vascular ectasia along gastric fold and upper part of duodenum with contact bleeding.

She was diagnosed as **vascular ectasia (watermelon stomach).** An argon plasma coagulation (APC) was applied for therapy.

Discussion:

Gastric Antral Vascular Ectasia (GAVE) accounts as 4% of non-variceal upper GI bleeding. It sometimes is associated with cirrhosis, connective tissue disorders, bone marrow transplantation, and chronic renal failure. Clinical presentations are varies from chronic iron-deficiency anemia to heavy acute gastrointestinal bleeding. The first-line treatments are laser or argon plasma coagulation and treatment for

the underlying disease. Other treatments are estrogen and/or progesterone, tranexamic acid, thalidomide and surgical antrectomy in unresponsive cases.[1]

Vascular ectasia of the whole intestine as a cause of recurrent gastrointestinal bleeding after high-dose chemotherapy has been reported in a 17 year-old patient who was diagnosed with early relapse Hodgkin's disease and treated with salvage chemotherapy followed by high-dose chemotherapy and autologous stem cell transplantation. He presented with gastrointestinal bleeding 5 months later. Vascular ectasis was diagnosed by endoscopy with histological confirmation of the gastric antrum, duodenum, ileum, and entire colon.[2]

References

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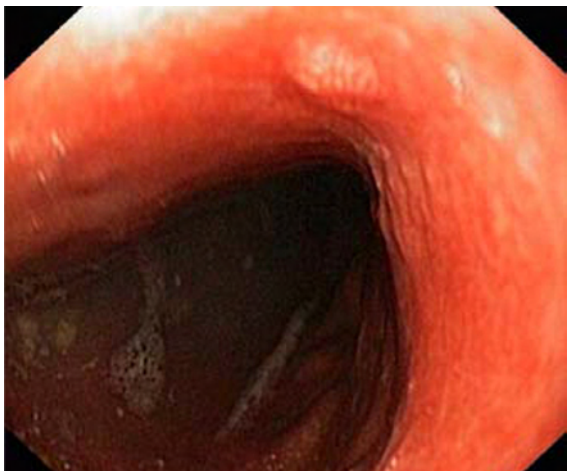
2. Schmidmaier R, Bittmann I, Götzberger M, Straka C, Meinhardt G, Eigler A. Vascular ectasia of the whole intestine as a cause of recurrent gastrointestinal bleeding after high-dose chemotherapy: Endoscopy 2006;38:940-2.

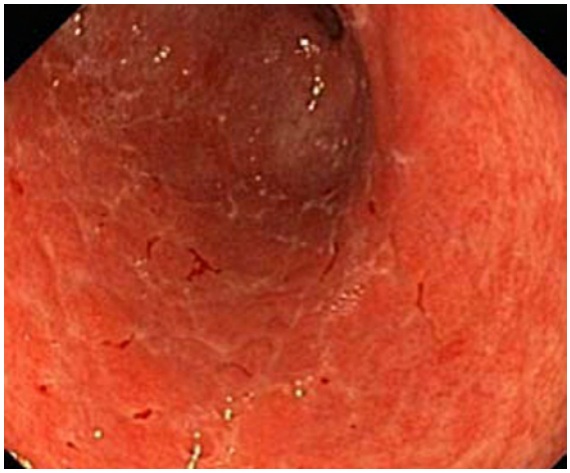
Case 22

Chatchai Kriengkirakul, MD.

Rungsun Rerknimitr, MD.

A 38-year-old female without previous medical history came to hospital because of dyspepsia and weight loss in 4 months. Her examination showed mild anemia, no jaundice, others were unremarkable. EGD was done as shown.





Endoscopic findings:

Diffuse erythrematous gastric mucosa without normal rugae pattern. There were some large mosaic cracks containing heme in the gastric body. Gastric biopsy revealed moderate chronic and acute erosive gastritis with atypical lymphoproliferative infiltration. No *H.pylori* was seen. An immuno-histochemical stain showed negative antibody study for both CD 20 and CD 3 (C- Kappa: C- Lambda ratio was 5:1).

Diagnosis:

Gastric MALToma without Helicobacter pylori infection.

Discussion:

Gastric MALT lymphoma, an extranodal marginal zone B cell lymphoma has an indolent behavior. It represents about 40% of gastric

lymphomas. The median age at diagnosis is approximately 60 years, with a wide age range. The male-to-female ratio is equal. Etiology and pathogenesis are associated with[1] *H. pylori* infection (90% of cases) and eradication of infection leads to regression of the lymphoma in most of early cases[2]. There was an evidence for antigen-driven B cell proliferation (*H. pylori* antigens non-H. pylori antigens)[3]. Genetic studies, found that t(11;18) (q21;q21) translocation is more frequent in cases regional lymph nodes spreading and also associated with cases that do not respond to *H. pylori* eradication, even when disease is localized. The most common presentations are epigastric or abdominal pain (53%), and dyspepsia (32%). Nausea, gastric bleeding and B symptoms (fever, night sweats, weight loss) are unusual. Serum levels of LDH and β2-micro globulin are usually normal. Low-grade gastric MALT lymphomas are most commonly located in the antrum (41%) but may be multifocal in 33% of the cases. Lesions may appear as ulcers in 47%, erosions in 23%, or erythema in 30%. Gastric MALT lymphoma cells usually express pan-B antigens (CD19, CD20, and CD79a). Staging and risk assessment should be evaluated with EGD plus multiple biopsies, complete blood counts, LDH, β2-microglobulin, bone marrow aspirate and biopsy and CT of the chest, abdomen and pelvis. For localized MALToma with positive for Helicobacter infection, treatment can be only with antibiotics. In H. pylori-negative cases or failed antibiotic therapy, choices for treatment are irradiation and systemic chemotherapies (systemic chemotherapy and/or immunotherapy with anti-CD20 monoclonal antibodies) depend on stage of the disease. However, surgery is not recommended. Evaluation and follow-up after treatments are EGD with multiple biopsies taken 2-3 months after *H. pylori* eradication and at least twice a year for 2 years to monitor histologic regression of the lymphoma, then endoscopic and systemic follow-up (blood counts and adequate radiological or ultrasound examinations) can be done once a year.[1,2]

References

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2. Morgner A, Schmelz R, Thiede C, Stolte M, Miehlke S. Therapy of gastric mucosa associated lymphoid tissue lymphoma. World J Gastroenterol 2007;13:3554-66.

Case 23

Chatchai Kriengkirakul, MD.

Rungsun Rerknimitr, MD.

An 91-year-old male complained with anorexia and weight loss 8 kgs within 8 month, and later developed RUQ abdominal pain with jaundice. Recently he passed melena and his hematocrit decreased from 31% to 27%. An EGD was done as shown.

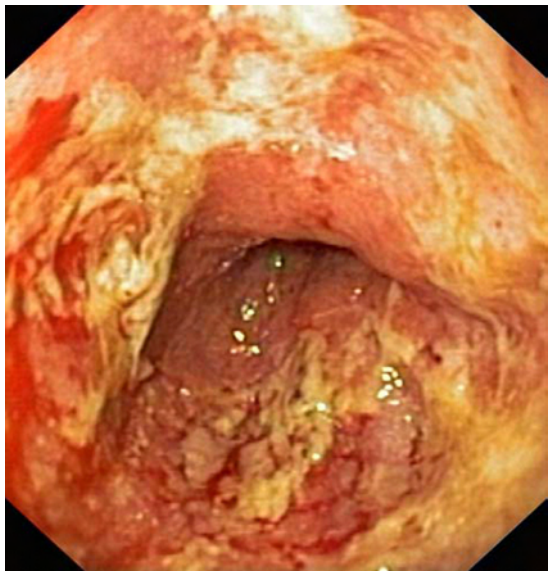

Figure A

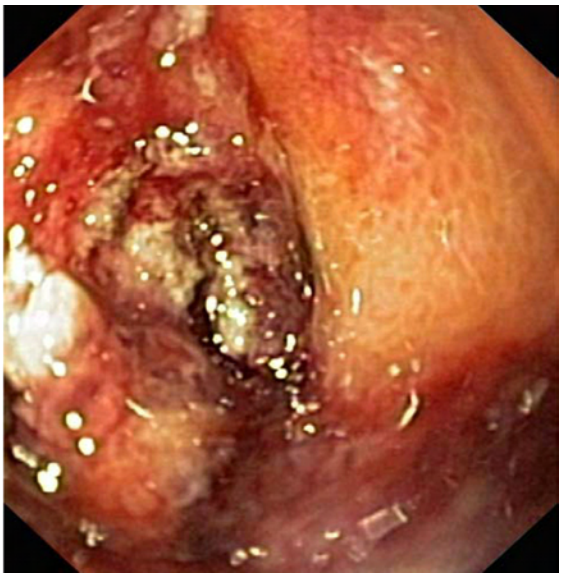

Figure B

Endoscopic findings:

Severe reflux esophagitis LA class D (Figure A). In the duodenal blub, the lumen was compromised by an invading tumor with contact bleeding (Figure B). This tumor later was confirmed as a gallbladder carcinoma.

Diagnosis:

Gallbladder cancer invading duodenal bulb with severe reflux esophagitis.

Discussion:

Malignant tumors of the gallbladder found 1-2% of resected gallbladder and 0.5% of autopsies. It is the 6 th most common cancer of GI tract and is found in 3% to 4% of all GI tumors. More than 80% of patients with gallbladder carcinoma have gallstones but gallbladder carcinoma develops in less than 0.5% of patients with gallstones[1] .The risk factors for gallbladder carcinoma are gallstones size more than 3 cm. (10-fold higher risk of cancer than smaller stones), calcified gallbladder, a long common channel formed by the union of the pancreatic and common bile ducts, chronic typhoid carrier state, chronic cholecystitis, age > 50 yr, female sex, gallbladder polyps (> 1 cm.), smoking, family history, and ethnicity (Mexican Americans and Native Americans)[2]. Symptoms and signs include right upper quadrant pain (>80%), nausea, fatty food intolerance, anorexia, weight loss, fever, and chills. In advance disease, patient may have continuous abdominal pain, obstruction of bile duct with jaundice (>30%), a palpable mass, hepatomegaly and ascites. Laboratory findings are often unremarkable until obstructive jaundice developed. In addition, there is no reliable tumor marker for early detection of this disease[3]. Preoperative diagnosis of gallbladder carcinoma is difficult because it is nonspecific in presentation. The sensitivity and specificity of CT abdomen are near 90%. CT of the abdomen typically shows intense irregular enhancement occur at the periphery of primary lesion. The locations for local tumor invasion can be the first part of duodenum or hepatic flexure of the colon[4].

References

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2. Lièvre A. 44th Congress of the ASCO (American Society of Clinical Oncology). J Chir (Paris) 2008; 145:504-8.
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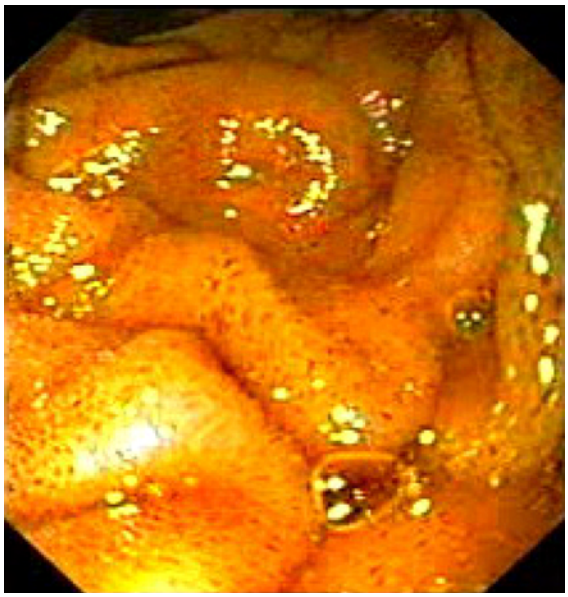
4. Saito A, Noguchi Y, Doi C, Mukai K, Fukuzawa K, Yoshikawa T, et al. A case of primary adenosquamous/squamous cell carcinoma of gallbladder directly invaded duodenum. Hepatogastroenterology 1999;46:204-7.

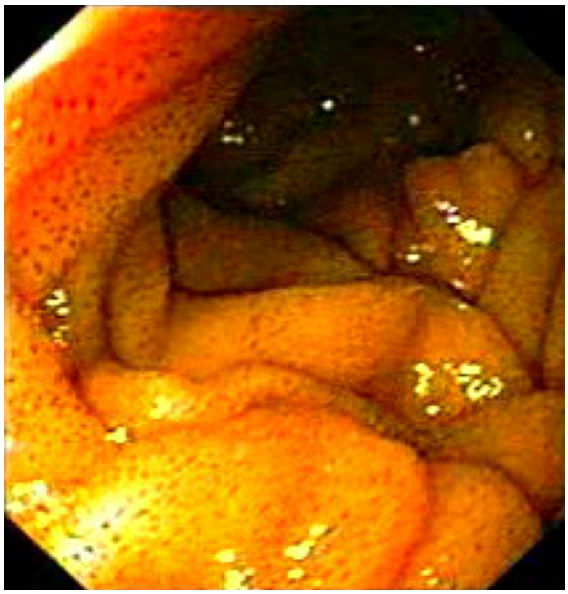
Case 24

Apichart Suramethakul, MD.

Rungsun Rerknimitr, MD.

A 50-year-old female, with a history of hypertension and dyspepsia underwent a gastroscopy.





Gastroscopy revealed a discrete, flat, small dark brown spots (a pepper sprinkled appearance) with in the duodenal mucosa.

Diagnosis:

Pseudomelanosis duodeni

Discussion:

Duodenal pseudomelanosis is a very rare and reversible condition. This pigment corresponds principally to the accumulation of ferrous sulfide and calcium inside the lysosomes of macrophages, located in the lamina propria[1]. This condition occurs predominantly in elderly female patients and is strongly linked to chronic illnesses, including systemic hypertension, chronic renal failure, upper gastrointestinal bleeding and diabetes mellitus[2,3]. Moreover, it is related to the use of common medications, including hydralazine, propranolol, thiazide diuretics, furosemide and ferrous sulfate.

From an etiological point of view, it is suggested that in cases of pseudomelanosis duodeni, iron metabolism may be impaired and iron may pool within the macrophages. However, this etiology and its detailed clinical significance remain obscure.

References

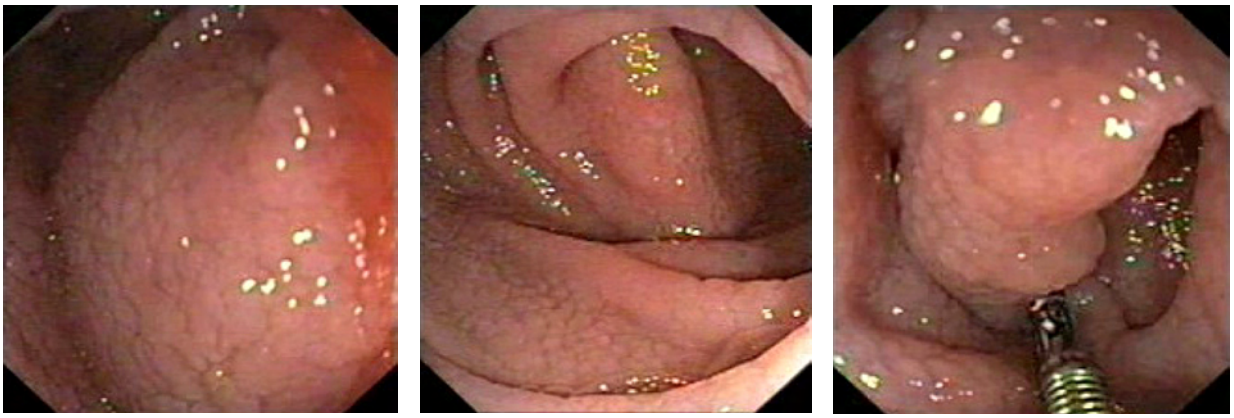
1. Treeprasertsuk S, Thong-Ngam D, Suwangool P, Kullavanijaya P. Pseudomelanosis duodeni: association with hypertension and chronic renal failure: case report. J Med Assoc Thai 2000;83:964-8.
2. Giusto D, Jakate S. Pseudomelanosis duodeni: associated with multiple clinical conditions and unpredictable iron stainability - a case series. Endoscopy 2008;40:165-7.
3. West B. Pseudomelanosis duodeni. J Clin Gastroenterol 1988;10127-9.

Case 25

Apichart Suramethakul, MD.

Rungsun Rerknimitr, MD.

A 14-year-old male patient with a history of chronic diarrhea for 6 months underwent a gastroduodenoscopy.



Gastrodudenoscopy revealed the **"**mosaic**"** appearance and loss of duodenal folds or scalloping fold which is significantly associated with duodenal villous atrophy. This is compatible with **celiac sprue.**

Discussion:

Celiac sprue is a disorder of the immune system whereby the normal lining of the small bowel is damaged by an allergic type of reaction from a protein in wheat, barley and rye called gluten. The normal frond-like tiny tubular projections within the jejunum, called villi, are badly damaged. With the result of gluten immune reaction, nutrients are no longer absorbed properly, thus malnutrition occurs. Comparing to the previous normal lining with its numerous pinpoint projections of light, this lining is smooth and reddened or inflamed[1,2]. This is a very abnormal appearance. The associated diseases are dermatitis herpetiformis, DM, selective Ig A deficiency and Down syndrome[3].

References

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3. Neuhausen SL, Steele L, Ryan S, Mousavi M, Pinto M, Osann KE, et al. Co-occurrence of celiac disease and other autoimmune diseases in celiacs and their first-degree relatives. J Autoimmun 2008 Sep;31(2):160-5.

Case 26

Phonthep Angsuwatcharakon, MD.

Rungsun Rerknimitr, MD.

A 52-year-old woman with systemic mucormycosis and septic shock had developed duodenal ulcer perforation and been treated with a simple closure. Eight days after a simple closure, she developed an active upper gastrointestinal bleeding.

EGD was done and showed as figure A-D

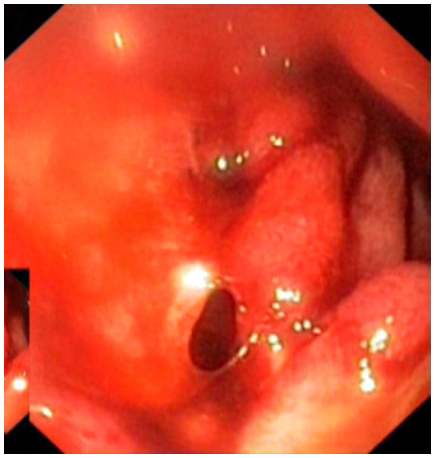

Figure A

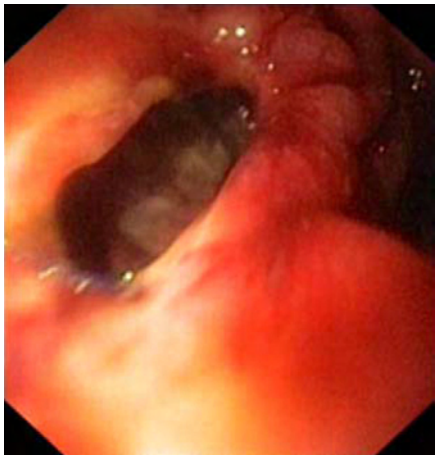

Figure B

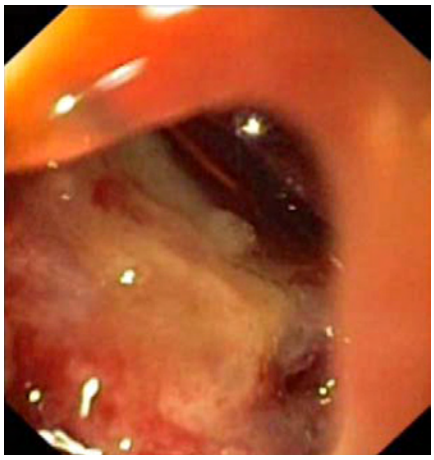

Figure C

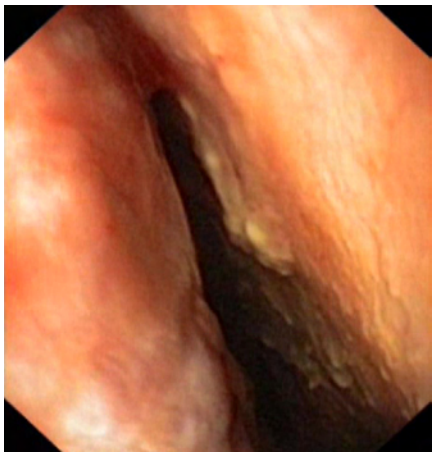

Figure D

Endoscopic showed:

Figure A-B: A 2 cm-long duodenal ulcer with perforation.

Figure C-D: Retroperitonal organs visibly seen through the perforation.

Diagnosis:

Perforated duodenal ulcer.

Discussion:

EGD is an important procedure for post-gastrointestinal surgery patients when they developed bleeding[1]. An immediate EGD after gastrointestinal surgery is concerned for suture breakdown, bleeding, leakage, perforation, or abscess caused by endoscopic intubation, torque or insufflations[1]. Risk of postoperative EGD perforation depends upon general medical status, type of surgery and surgical repair site. EGD should be avoid during the first 2-4 days postoperatively because of the sutured site is weakest. In addition, EGD should be postponed until 1 week after surgery to permit postoperative pain and wound edema to be subsided. It has been shown that endoscopic benefits may exceed the risks when EGD is performed more than 7 days after surgery[1].

Reference

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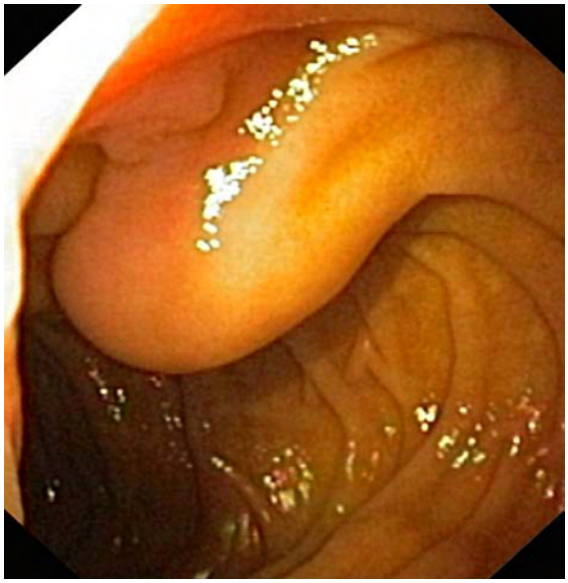
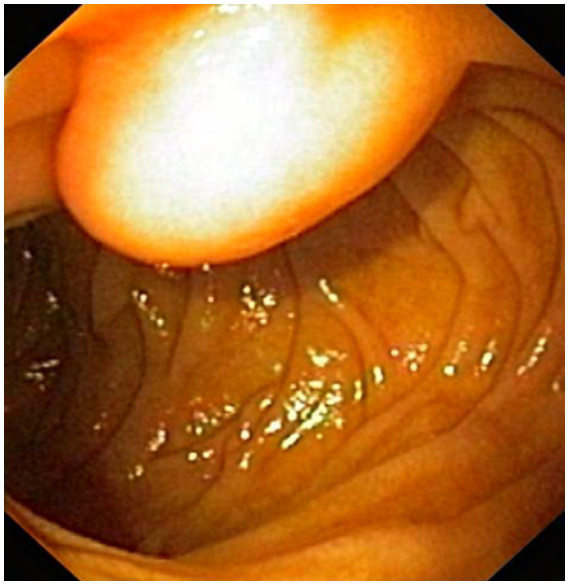
Case 27

Surachai Amornsawadwattana, MD.

Rungsun Rerknimitr, MD.

A 79-year-old female, underlying stroke presented with melena. An esophagogastroduodenoscopy (EGD) was performed and findings are shown s figures.





A yellowish submucosal mass at the second part of duodenum adjacent to the ampulla of Vater. Duodenal lipoma was diagnosed.

Discussion:

Lipoma is a benign tumor consists of mature fat tissue surrounded by fibrous capsule[1]. GI lipoma can be found throughout the GI tract, but the most common site is colon[2]. It is usually solitary, but multiple lesions were also reported[1]. Duodenal lipoma is considerably rare when compare to gastric lipoma[1]. It is often asymptomatic and found incidentally during endoscopic examination. However, a size of greater than 4 cm. can cause GI bleeding, abdominal pain, intussusception and pressure ulceration[1]. Endoscopic finding of lipoma is a well defined elevated yellowish submucosal mass covered with normal mucosa and infrequently has ulceration on top[1,2]. Unnecessary surgery or endoscopy can be withheld by the findings of CT scan. A well-circumscribed mass with uniform fat attenuation is suggestive the diagnosis of

lipoma[1]. Treatment of choice in symptomatic case is surgical removal, but in small lipoma, endoscopic snaring or enucleation can also be performed[3].

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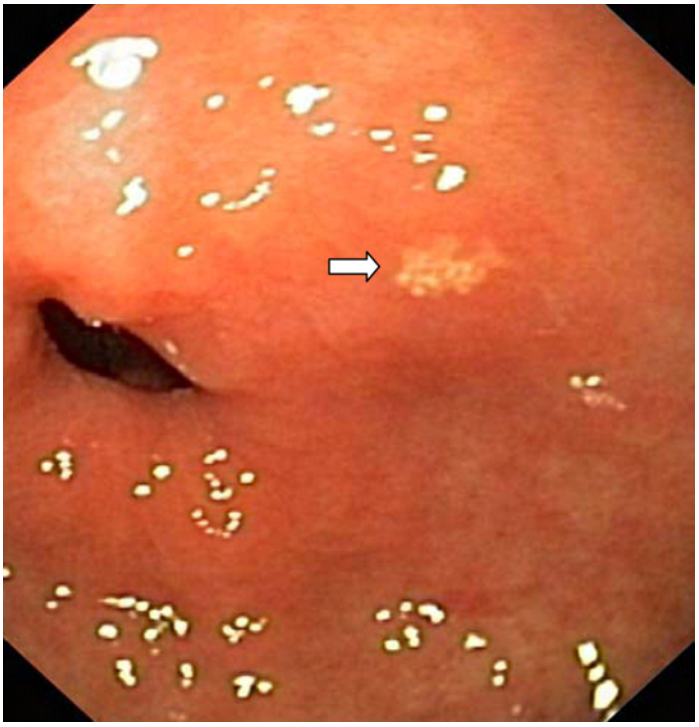
Case 28

Surachai Amornsawadwattana, MD.

Naruemon Klaikaew, MD.

Rungsun Rerknimitr, MD.

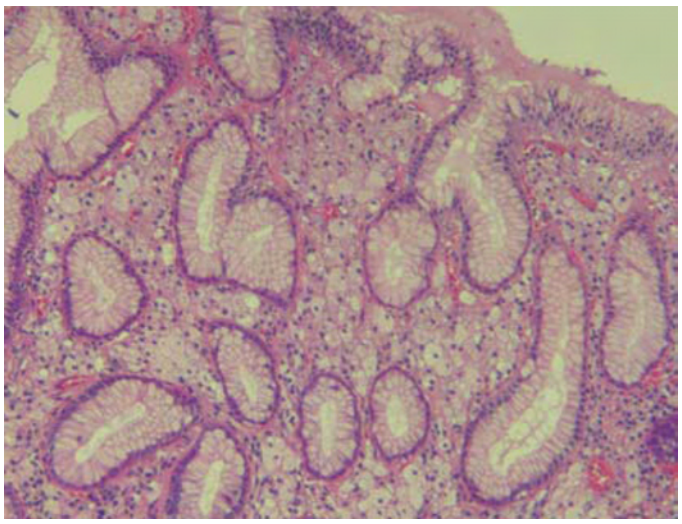
A 54-year-old woman presented with dyspepsia. The initial physical examination was unremarkable. Then EGD was performed.

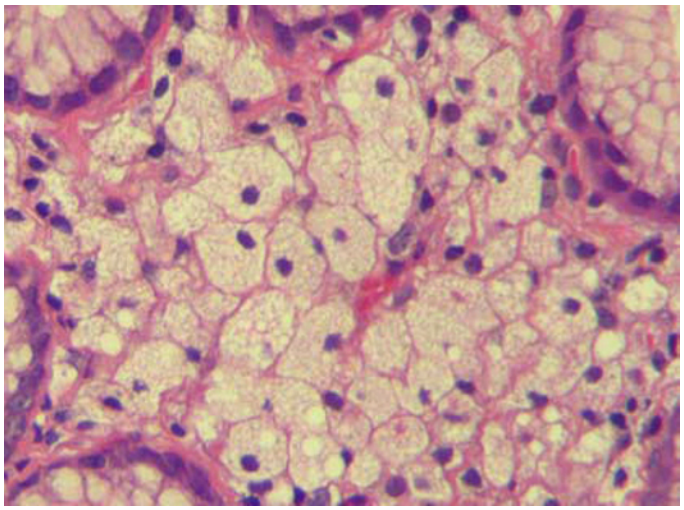


Endoscopic finding disclosed a 0.3-cm., yellowish, granular mucosal lesion was seen at the prepyloric area (white arrow). Biopsy was taken and histologic examination was done. It revealed numerous foamy histiocytes infiltrate within lamina propria without the presence of inflammatory cells. The diagnosis was **gastric xanthelasma.**

Discussion:

Gastric xanthelasma, also known as gastric xanthoma or gastric lipid island, is an uncommon benign condition in the stomach[1,2,3]. It was first reported by Orth in 1887 as lipid laden macrophage detected in gastric mucosa[2]. The incidences from endoscopic studies vary





from 0.018 to 0.8%, on the contrary to a higher percentage, 58% from an autopsy case series[2]. They usually locate in the antrum and pyloric region[1,3], but body and fundus are also seen[1]. The xanthelasmas are rarely found outside the stomach, but the lesions in esophagus, duodenum and colon were also reported[1,3]. Pathogenesis is unknown, but the most accepted hypothesis is the healing process after mucosal damage which brings about the accumulation of fat or cholesterol[1,2]. Endoscopic appearances of xanthelasma are yellow to white, well-demarcated plaque or nodule with size varying from 1 to 10 mm[1,2]. Multiple lesions are also observed[1-4]. Histopathology of gastric xanthelasma is the numerous foamy macrophages exclusively infiltrate in lamina propria layer[1]. Major differential diagnosis is signet ring carcinoma which stains positive for cytokeratin and mucicarmine. Gastric xanthelasma stains negative for cytokeratin and mucicarmine, but positive for Sudan black, oli red O and CD 68[1]. Other differential diagnoses are whipple disease and *Mycobacterium avium-intracellulare* infection[1].

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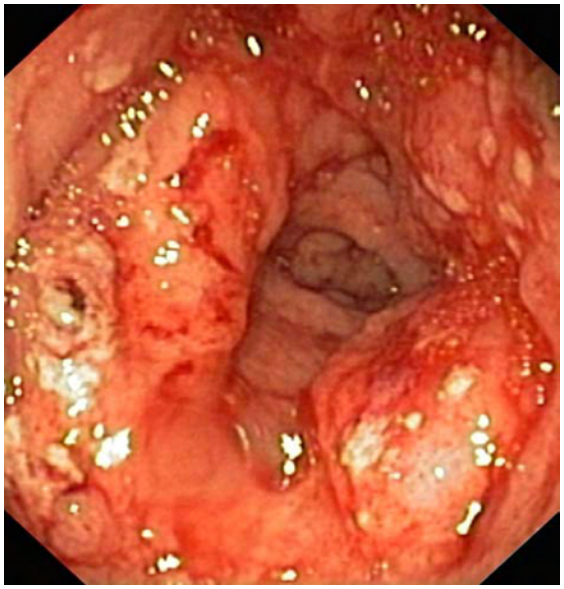
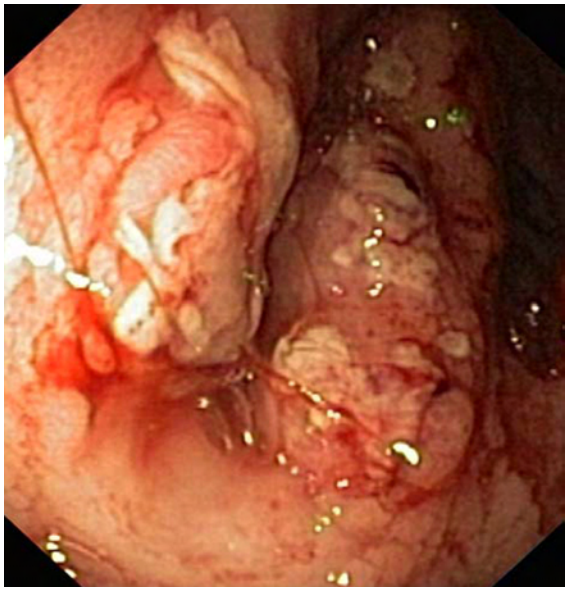
Case 29

Chatchai Kriengkirakul, MD.

Rungsun Rerknimitr, MD.

A Thai male 70 year-old man presented with hematemesis for 1 day. NG lavage was done and shown a lot of bloody content. An EGD was done as shown





EGD findings: diffuse submucosal mass with ulceration and on top exudate varies in size 1-3 cm, contact bleeding of the whole stomach especially antrum was observed.

Pathological report:

Diffuse large B-cell lymphoma (mixed positive and negative CD 20).

Discussion:

Primary diffuse large B-cell lymphoma of the stomach (PGDLCL) shows an incidence of 1/100,000 in Western countries. Majority of the cases are aggressive lymphoma. No risk factors have been clearly identified in PGDLCL. However, there are some evidences that atrophic gastritis, immunodeficiency

condition (especially solid-organ transplant treated with cyclosporine) may predispose patients for PGDLCL. The role of *H. pylori* infection is controversial. PGDLCL occurs more frequently in male with median age range of 50-60 years. Majority of patients report epigastric pain (70%), dyspepsia (30%), weight loss (40%), epigastrium mass (uncommon), bleeding and perforation (rare). The efficacy of endoscopic biopsy is very high, with diagnostic accuracy of 70-90%. Treatment of choice for PGDLCL irrespective of anatomic site of the lesion is rituximab plus antracycline-based combination chemotherapy.[1,2] Complications of chemotherapy include gastric outlet obstruction, bleeding and gastric perforation are rare. In one report[3], there were six patients developed gastric bleeding before and during chemotherapy treatment, all patient were treated conservatively with blood transfusions. Two patients did not response to the conservative therapy, one underwent angiography with embolization, and the other underwent a subtotal gastrectomy. The role of consolidation radiotherapy is debatable. The addition of radiotherapy was associated with a lower local relapse rate compared with chemotherapy alone. Surgical management is controversial. In addition to chemotherapy, *H.pylori* should always be eradicated in localized or extensive PG-DLBCL with concomitant low grade MALT component.

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