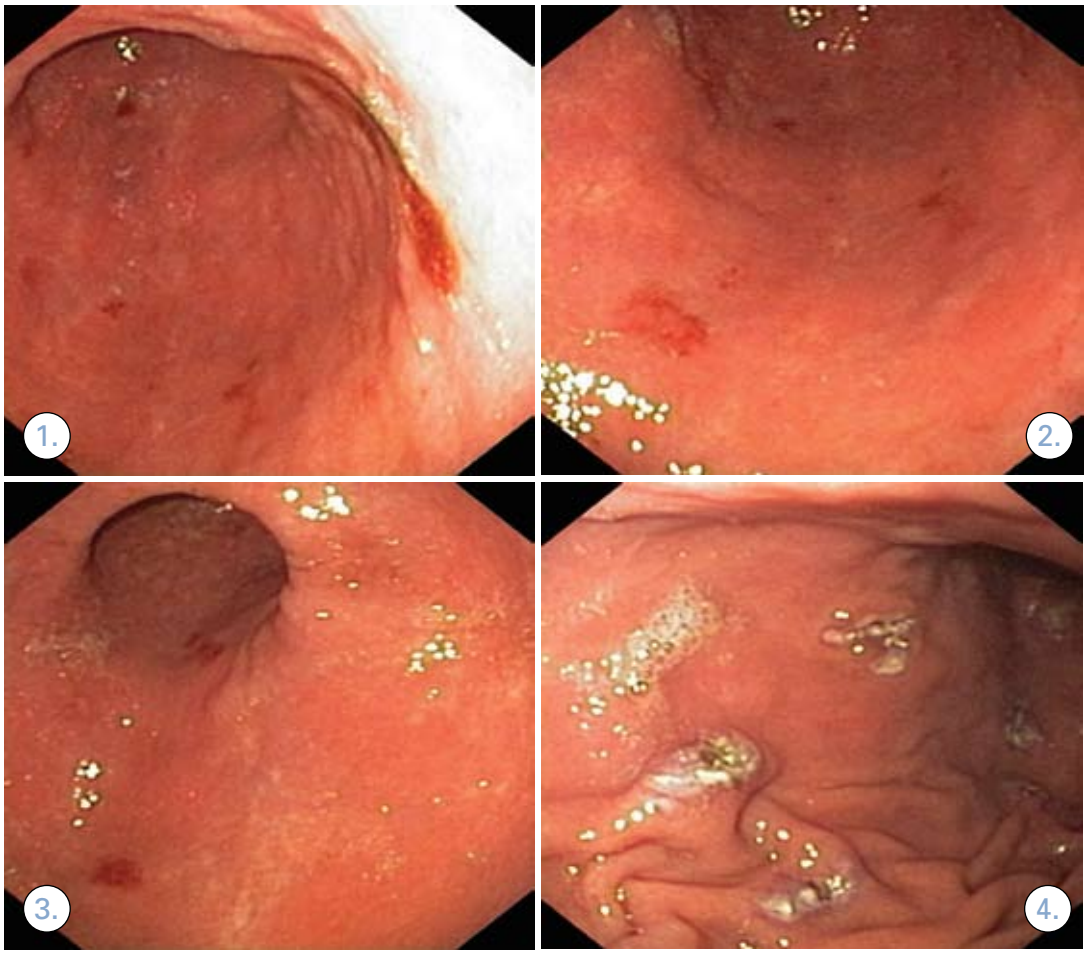
# Part 2
# Stomach

## Case 1

Sukprasert Jutaghokiat, MD.
Rungsun Rerknimitr, MD.

A 57 years old female, known case of diabetes mellitus and hypertension, presented with recurrent hematemesis.

EGD was done and showed as figure 1-4



The EGD showed discrete, flat, or slightly raised bright red lesions in body and antrum of stomach, with one in the figure 1 showed sign of recent bleeding. The diagnosis of gastric angiodysplasia was made.

The differential diagnosis are iatrogenic bleeding caused by instrument contact at the apex and Gastric antral vascular ectasia (GAVE)

The patient was treated by argon plasma coagulation (APC) at all angiodysplasia as in figure 4. There are no immediate complication.

## Discussion

Gastric angiodysplasia and gastric antral vascular ectasia are common forms of upper-GI vascular ectasia. These lesions are increasingly important sources of GI bleeding and most commonly observed in elderly patients.[1] Their presentation is either chronic, minimal bleeding, often present with iron deficiency anemia, or acute bleeding.

Gastric antral vascular ectasia has endoscopic finding that differ from angiodysplasia. Their typical findings are linear pattern of vascular ectasia that begin from gastric antrum to gastric body that resembled the stripes of a watermelon.[2]

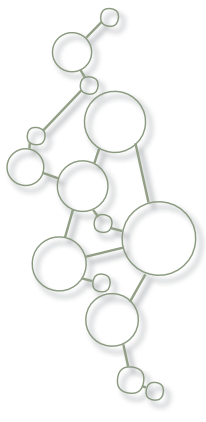
Endoscopic thermal ablation, including laser photo ablation and APC effectively controls bleeding and reduce transfusion need. Patients with gastric antral vascular ectasia require more treatment sessions that patients with gastric angiodysplasia.[3]

## References

1. Balasubramanian M, Kini S, Naik L, Kirtikumar, Deshpande J. Angiodysplasia of cardia of stomach-a case report. Indian J Pathol Microbiol 2004;47:18-20.

2. Barnett JL. Vascular ectasia of the gastric antrum. N Engl J Med 1998;339:166.

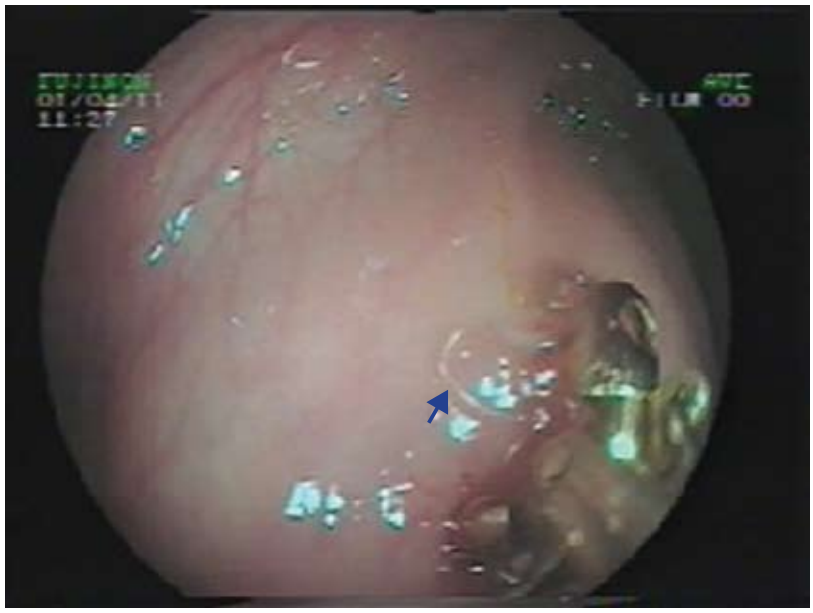
3. Pavey DA, Craig PI. Endoscopic therapy for upper-GI vascular ectasias. Gastrointest Endosc 2004;59:233-8.



# Case 2

Nopavut Geratikornsupuk, MD.
Rungsun Rerknimitr, MD.

A 64 year-old-male, bed ridden from old CVA and on NG tube feeding. He had history of coffee ground content and aspiration pneumonia for 2 times in last admission. EGD was done. Finding showed as figured.



The arrow showed the parasite which the posterior part of the worm is wider and looks like the whip handle, and the anterior part is long and thin. Trichuriasis (whipworm) was diagnosed.

## Discussion

Trichuriasis is caused by infection with the nematode, *T. trichiura.* Trichuriasis is a common intestinal helminthic infection worldwide, and it is estimated that approximately one-quarter of the world population carries this parasite[1]. A high prevalence of infection is seen particularly in warm, moist climates, so it is most common in tropical regions.

Transmission of trichuriasis occurs via fecal-oral spread. Eggs are ingested, reach the intestine, and hatch to release larvae. Larvae develop into adult worms over a period of approximately two to three months. The male and female both live in the human intestine, with the thin end embedded in the bowel mucosa, and the thick end visible within the bowel lumen. The adults measure approximately 4 cm. in length. The male has a curved tail, which distinguishes it from the female. Adults live for one to three years.

Most infections with *T. trichiura* are asymptomatic. Clinical symptoms are more frequent with moderate to heavy infections. The most characteristic clinical finding in trichuriasis is rectal prolapsed. This occurs mainly in heavily infected individuals, and worms may be directly visible embedded in the mucosa of the prolapsed, inflamed rectum. Pica and finger clubbing are other potential clues to the diagnosis. The diagnosis of trichuriasis is made by stool examination for eggs. The eggs have a characteristic barrel shape. They are 50 x 20 μm, have a smooth thick wall, and have a hyaline plug at each end. Infected individuals may have a peripheral eosinophilia of up to 15 percent.

Treatment with either agent can be used. The guidelines in the United States recommend mebendazole (500 mg. as a single dose) or albendazole (400 mg. as a single dose)[2]. However, in heavy infections, a three-day regimen of mebendazole (100 mg BID) or albendazole (400 mg. QD) should be considered instead.

## References

1. Cooper, E. Trichuriasis. In: Tropical Infectious Diseases: Principles, Pathogens and Practice, vol 2, Guerrant, R, Walker, DH, Weller, PF, (Eds), Churchill Livingstone, Philadelphia 1999. p 955.

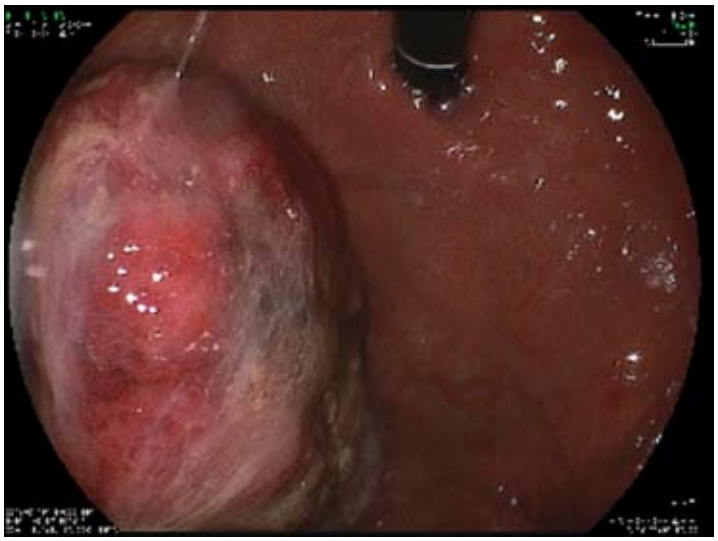
2. Drugs for Parasitic Infections. Medical Lett Drugs Ther; August 2004. online: www.medletter.com/freedocs/parasitic.pdf.

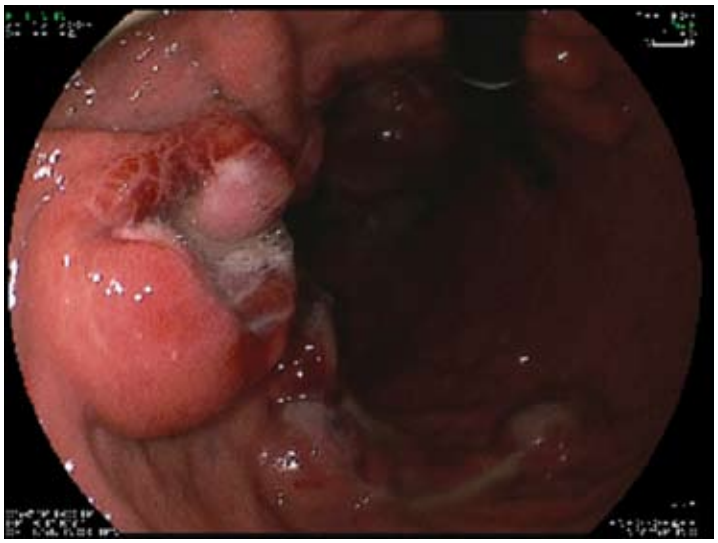
# Case 3

Wiriyaporn Ridtitid, MD.
Rungsun Rerknimitr, MD.

A 63 years old male presented with weight loss and melena for 6 months.

Esophagogastroduodenoscopy was done and showed as picture.

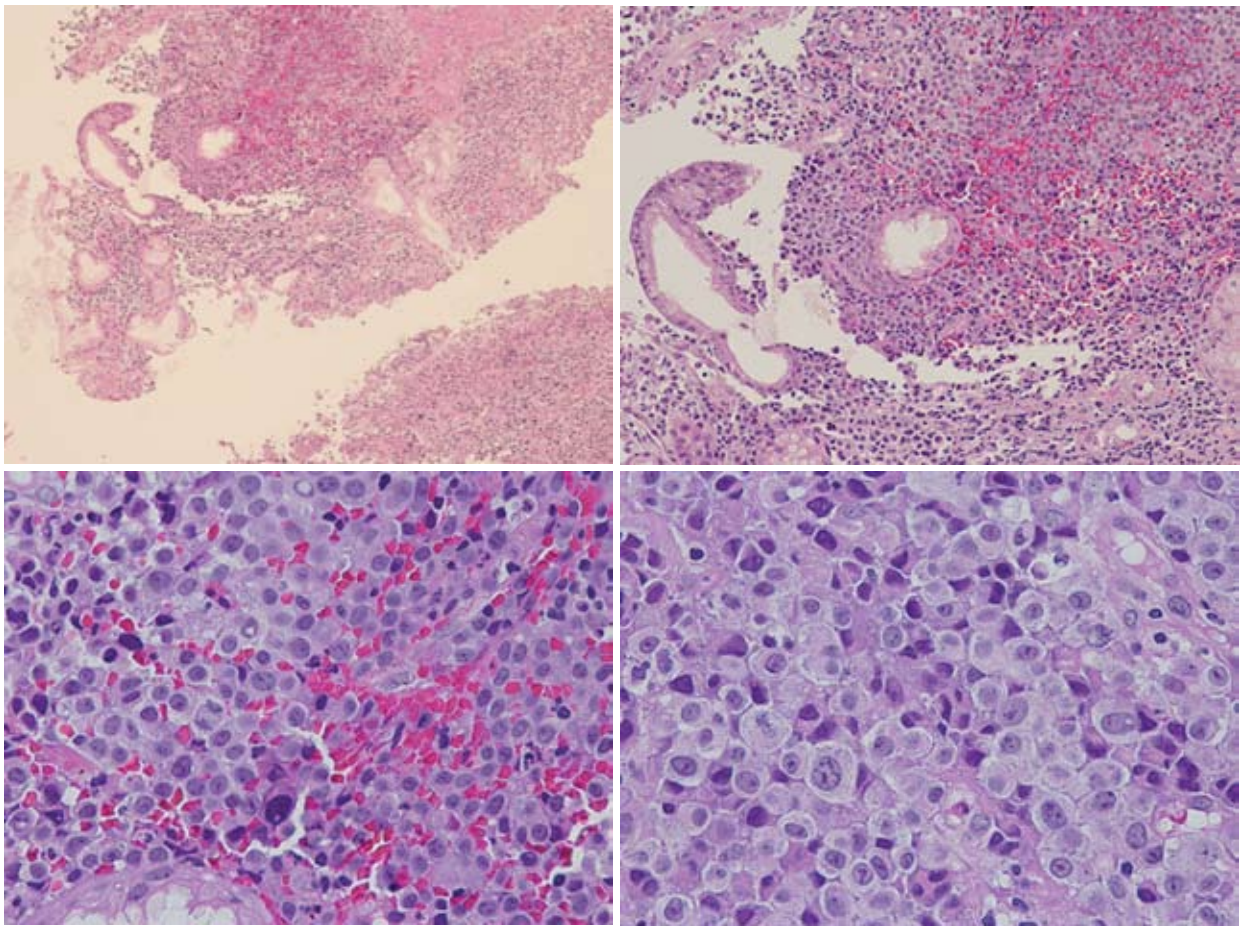




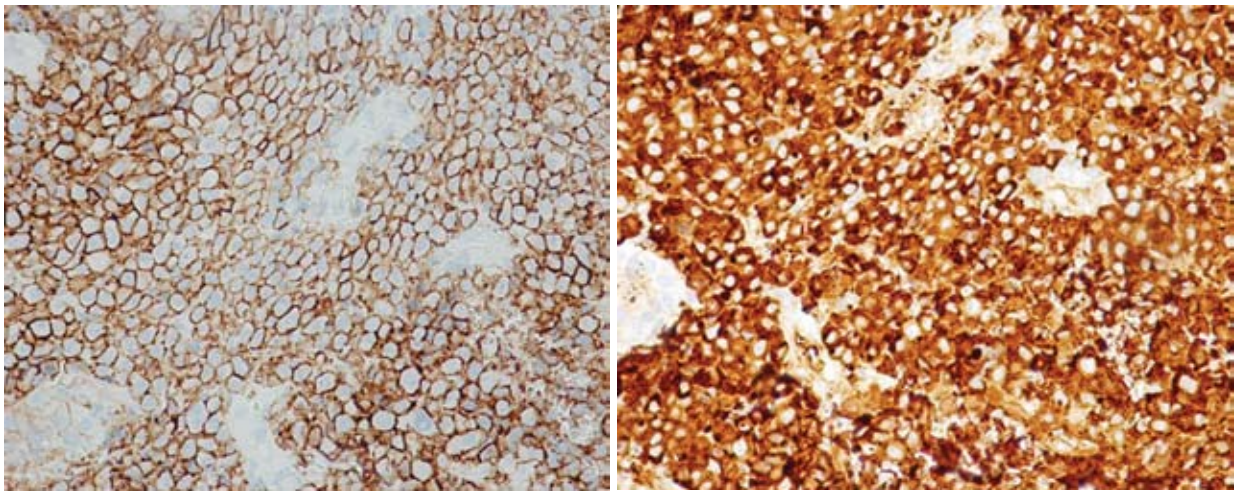
Esophagogastroduodenoscopy showed multiple round masses vary in size 1-4 cm. in diameter with necrotic ulcers on top at fundus and body of stomach.

The differential diagnosis are adenocarcinoma and hematologic malignancy.

Biopsy was done and pathological finding revealed plasmacytoid appearance tumor cells with large pleomorphic nuclei. The diagnosis is gastric plasmacytoma.



Immunohistochemical study was confirmed as figure; CD138 and Kappa were positive.



## Discussion

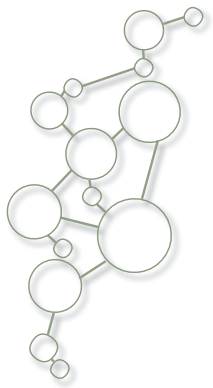
Gastrointestinal plasmacytomas are a relatively rare entity comprising less than 5% of all extramedullary disease[1]. It usually present with non-specific symptoms of anorexia, weight loss and abdominal discomfort rather than frank bleeding. The small bowel is the most common site of GI involvement by plasmacytoma (multiple myeloma), reportedly followed by the stomach, colon, and esophagus[2,3].

## References


1. Robinson H, Damasiewiczi M, Nemann DH, et al. Gastric involvement of plasmacytoma associated with t(4:14) and chromosome 13 deletion. Leukemia & Lymphoma 2006;47:1973-5.

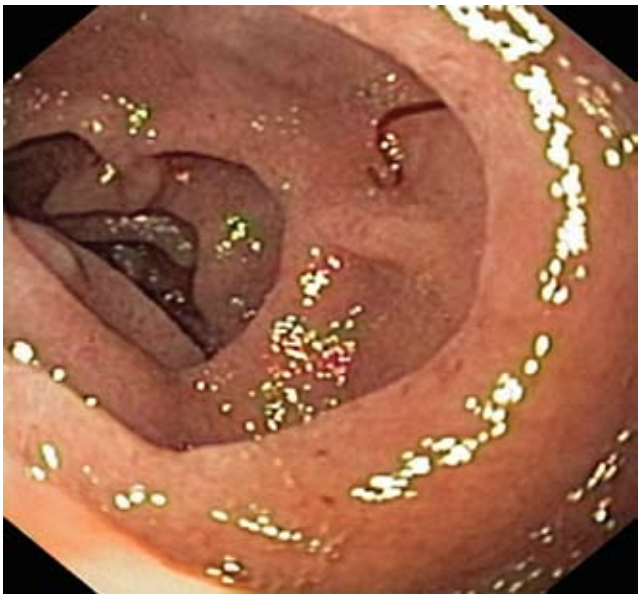
2. Chim CS, Wong WM, Nicholls J, et al. Extramedullary sites of involvement in hematologic malignancies-Case 3. Hemorrhagic gastric plasmacytoma as the primary presentation in multiple myeloma. J Clin Oncol 2002;20:344-7.

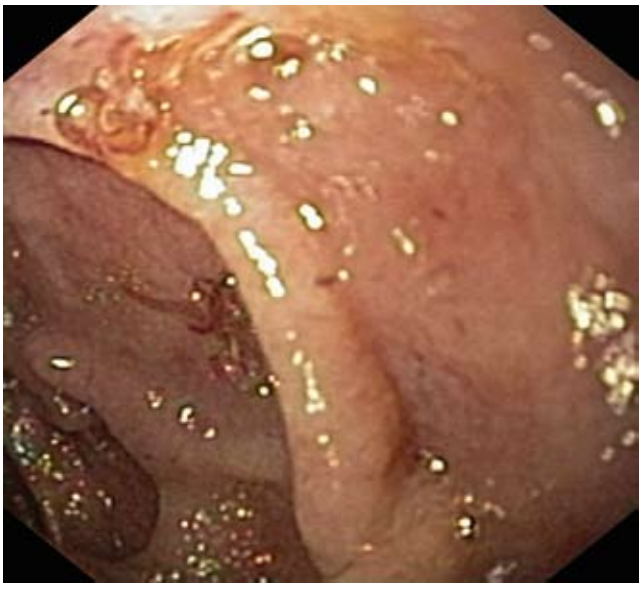
3. Spedini P, Marchetti G, Morandi S. Gastric localisation of multiple myeloma. Haematologica 2001;86:223.

# Case 4

Nathavut Sirimontaporn, MD.
Rungsun Rerknimitr, MD.





A 32 year-old man presented with anemia and fatigue for a month.

Esophagogastroduodenoscopy was showed as figure.

The esophagogastroduodenoscopy revealed hookworms along first and second part of duodenum.

The diagnosis is intestinal hookworms.

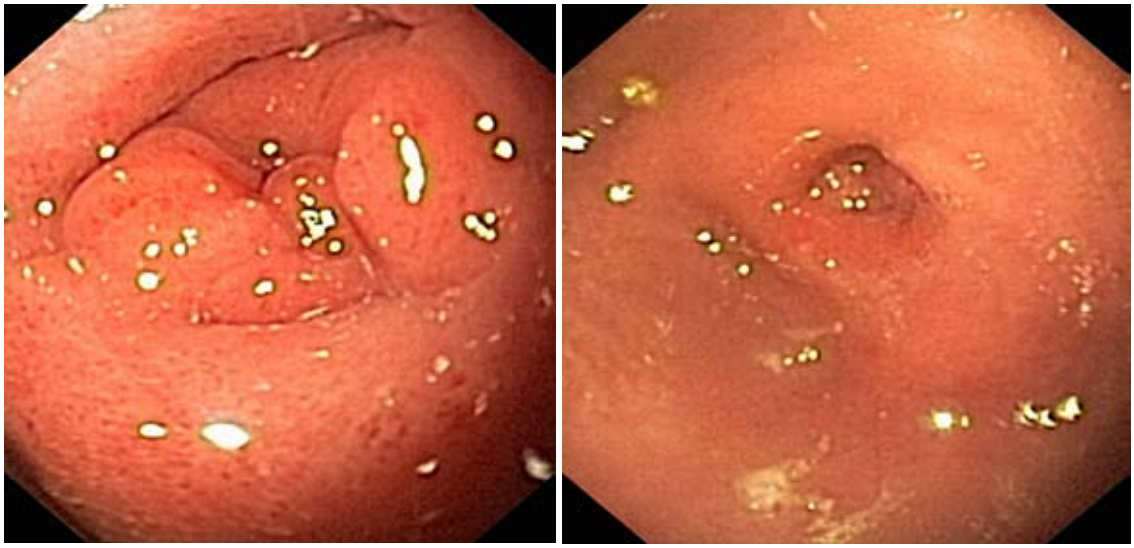
Treatment is 400 mg. of albendazole once.

# Case 5

Wiriyaporn Ridtitid, MD.
Rungsun Rerknimitr, MD.

A 44 year-old man presented with nausea and vomiting with weight loss for a month.

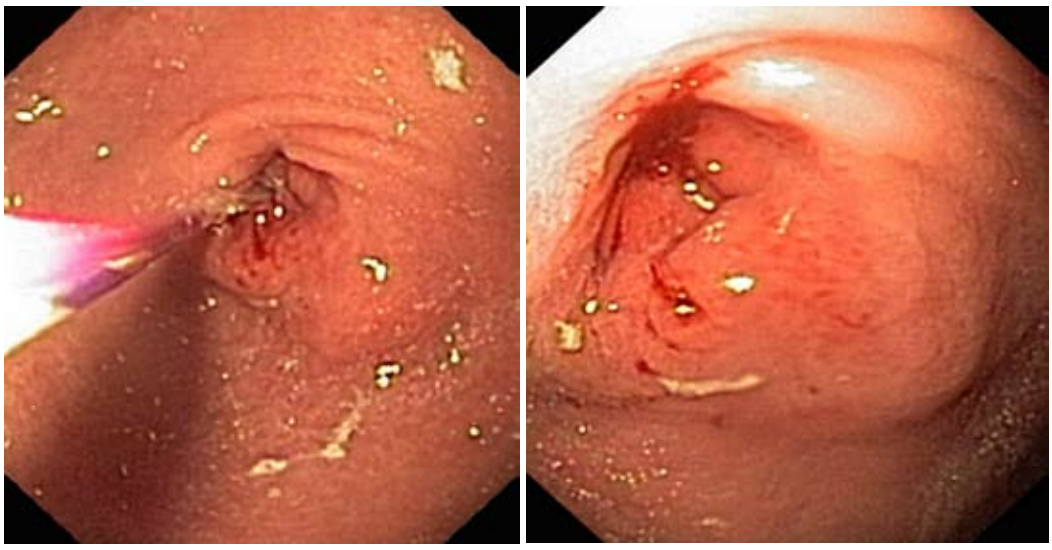
Esophagogastroduodenoscopy was showed as figure.



Esophagogastroduodenoscopy revealed swelling and deformity at pylorus with minimal opening of pylorus and can't pass scope through duodenum.

The diagnosis is benign pyloric stenosis.

Pneumatic balloon dilatation was performed as figure.



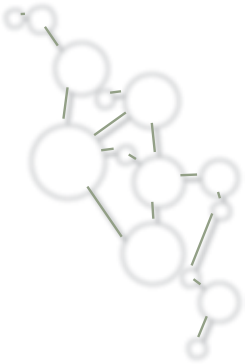
## Discussion

Causes of benign pyloric stenosis are peptic ulcer, anastomotic stricture, and NSAIDs[1]. Outcome of balloon dilatation can Symptomatic relief 70-80%. Median time to recurrence is 9 months. The overall symptomatic-free rates in 12, 24, 36, and 48 months were 85.3%, 78%, 68.8%, and 68.8%, respectively[2].

## References

1. Kim JH, Shin JH, Bae JI, et al. Gastric outlet obstruction caused by benign anastomotic stricture: treatment by fluoroscopically guided balloon dilation. J Vasc Interv Radiol 2005;16:699-70.

2. Yusuf, Tony E, Brugge, et al. Endoscopic therapy of benign pyloric stenosis and gastric outlet obstruction. Current Opinion in Gastroenterology 2006; 22:570-3.

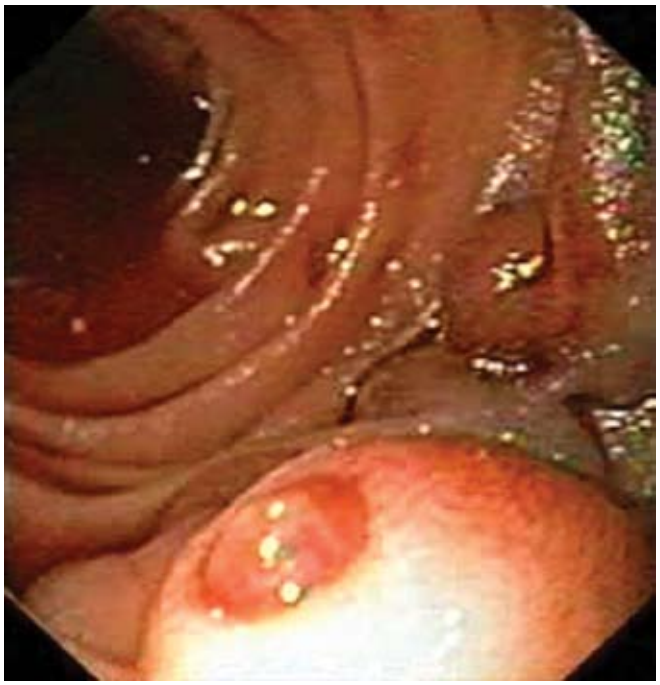


# Case 6

Chatporn Kittitrakul, MD.
Rungsun Rerknimitr, MD.

A 45 years old male patient, presented with hematemesis for 1 hour. He felt dizziness when he was standing. He had pale and orthostatic hypotension.

Esophagogastroduodenoscopy was done and shown as picture.



**Endoscopic finding:** duodenal varices with nipple sign.

## Discussion

Duodenal varices are rare as compared with esophageal or gastric varices. Most common causes are due to hepatic causes (2/3 of cases) and the others are extrahepatic causes (1/3 of cases)[1]. Cirrhosis is the most common hepatic cause (30% of all cases)[2,3]. The extrahepatic causes are result from occlusion of one of the major vessels in the area: the splenic, portal, or superior mesenteric vein. Thrombotic disorders are one of the first causes that should be investigated[1,2]. Previous surgical trauma with adhesions, obstructive tumors and severe pancreatitis have also been noted to cause duodenal varices[1-4].

Diagnosis of duodenal varices is almost always made during endoscopy for investigation of GI bleeding. The diagnosis has also been made on barium studies, splenoportography, angiography and laparotomy. When duodenal varices are diagnosed by endoscopy 60% have varices elsewhere in the GI tract[1]. 50% of these are gastroesophageal varices. 40% of patients with portal hypertension have duodenal varices at angiography, but they are rarely clinically significant because they often do not penetrate the submucosa.

To date, there has been limited experience of treatment for bleeding duodenal varices. Treatments for duodenal variceal bleeding include endoscopic procedures[5] (band ligation, sclerotherapy, or clipping), surgery (variceal ligation, duodenal resection, and extrahepatic portosystemic shunts), and interventional radiologic procedures (transileocolic vein obliteration, and balloon-occluded retrograde transvenous obliteration).

## References

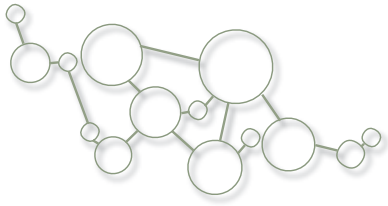
1. Linder S, Wiechel K. Duodenal varicose veins. Surg Endosc 1991;5:31-5.

2. Amin R, Alexis R, Karzis J. Fatal ruptured duodenal varix: A case report and review of the literature. Am J Gastroenterol 1985;80:13-8.

3. Hashizume M. Vascular anatomy of duodenal varices: Angiographic and histopathological assessments. Am J Gastroenterol 1993;88:1942-5.

4. Cottam DR, Clark R, Hayn E, Shaftan G. Duodenal varices: A novel treatment and literature review. Am Surg 2002;68:407-9.

5. Machida T, Sato K, Kojima A, Takezawa J, Sohara N, Kakizaki S, et al. Ruptured duodenal varices after endoscopic ligation of esophageal varices: an autopsy case. Gastrointest Endosc 2006;63:352-4.

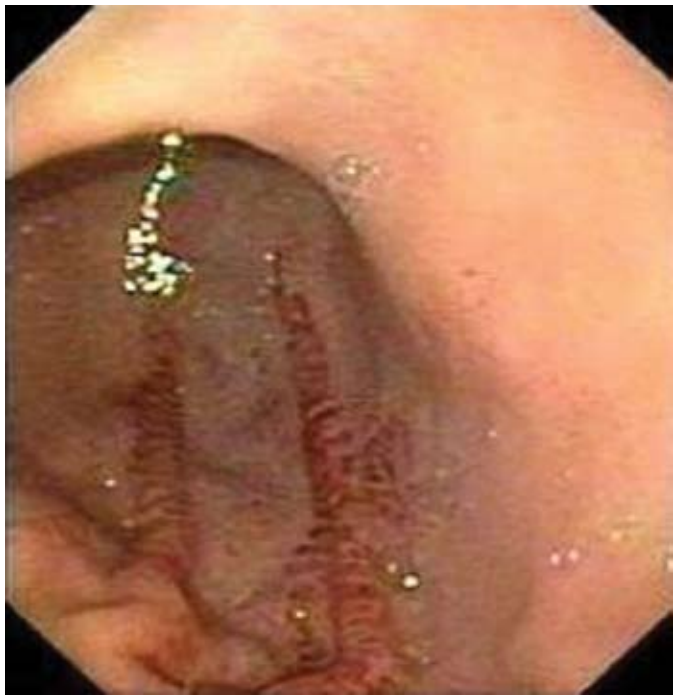


# Case 7

Roongruedee Chaiteerakij, MD.
Rungsun Rerknimitr, MD.

A 67 years old female patient, presented with anemic symptoms for 4 days. She also had melena for 3 weeks. Her underlying disease is end stage renal disease and she received hemodialysis 2 times/week.

Esophagogastroduodenoscopy was done and shown as figure



**Endoscopic findings:** Many red patches in linear array in the antrum of the stomach.

**Diagnosis:** Gastric antral venous ectasia (GAVE) syndrome

## Discussion

GAVE syndrome refers to the ecstatic dilatation of venous plexuses in the gastric antrum due to portal hypertension (synonym: **"**water melon stomach**"**). Oozing hemorrhages are not uncommon. Approximately 30% of patients with GAVE syndrome will have cirrhosis[1]. GAVE syndrome can be distinguished from portal hypertensive gastropathy (PHG) in that GAVE generally has more antral involvement and the classic features of GAVE syndrome including gastric ectasia, gastric dilation, thrombi, increased spindle cell proliferation, and fibrohyalinosis may be seen on biopsy[2].

The classic non-cirrhotic patient with GAVE is a middle aged female with autoimmune disease[3]. More specifically, in this study of 45 consecutive patients with GAVE, 71% were women, mean age was 73 years, most presented with occult blood loss, and 62% were transfusion dependent[3]. Autoimmune connective tissue disorders were present in 62%, with 31% having Raynaud's, 20% sclerodactyly, and 100% atrophic gastritis. Hypergastrinaemia was present in 76%[3]. GAVE syndrome has been associated with several disease states, including scleroderma,[3,4] various other autoimmune diseases,[3,5] bone marrow transplantation,[6] and chronic renal failure[3,7]. Non-cirrhotic patients are more likely to have a classic "watermelon" stomach with linear lesions within the antrum whereas in cirrhotics the disease is more often diffuse[2,3].

## References

1. Payen JL, Cales P. Gastric modifications in cirrhosis. Gastroenterol Clin Biol 1991;15:285-95.

2. Payen JL, Cales P, Voigt JJ, Barbe S, Pilette C, Dubuisson L, et al. Severe portal hypertensive gastropathy and antral vascular ectasia are distinct entities in patients with cirrhosis. Gastroenterol 1995;108:138-44.

3. Gostout CJ, Viggiano TR, Ahlquist DA, Wang KK, Larson MV, Balm R. The clinical and endoscopic spectrum of the watermelon stomach. J Clin Gastroenterol 1992;15:256-63.

4. Murphy FT, Enzenauer RJ, Cheney CP. Watermelon stomach. Arthritis Rheum 1999;42:573.

5. Schoonbroodt D, Horsmans Y, Hoang P, Poncelet-Maton E, Laka A, Geubel A. (Vascular gastric anomalies, CREST syndrome and primary biliary cirrhosis: efficacy of ethinyl estradiol-norethisterone combination). Gastroenterol Clin Biol 1994;18:649-51.

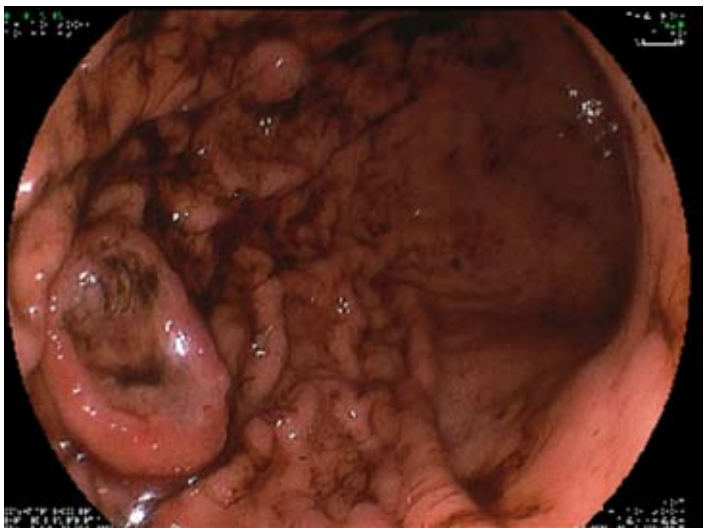
6. Tobin RW, Hackman RC, Kimmey MB, Durtschi MB, Hayashi A, Malik R, et al. Bleeding from gastric antral vascular ectasia in marrow transplant patients. Gastrointest Endosc 1996;44:223-9.

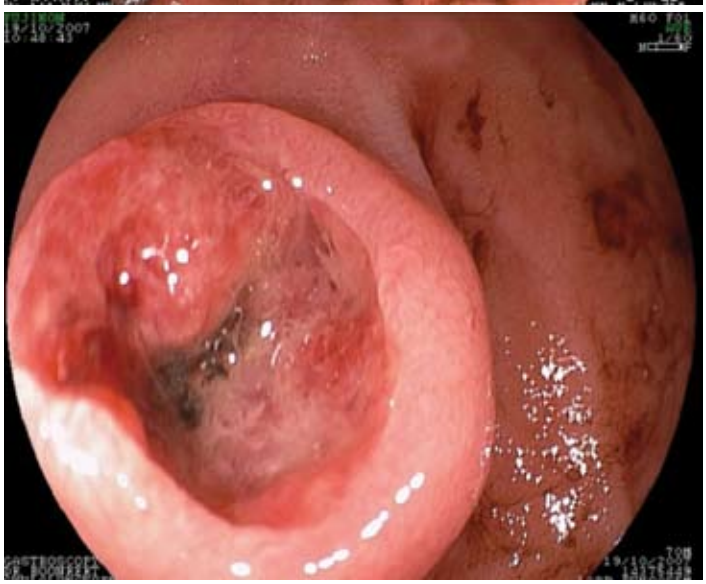
7. Herman BE, Vargo JJ, Baum S, Silverman ED, Eisold J. Gastric antral vascular ectasia: a case report and review of the literature. J Nucl Med 1996;37:854-6.

# Case 8

Boonlert Imraporn, MD.
Rungsun Rerknimitr, MD.

A 60 years old male, developed upper GI bleeding with coffee ground contents. He has no abdominal pain. His underlying disease is squamous cell lung cancer with brain metastasis.





In this case, pathology revealed metastatic squamous cell cancer. He was treated with palliative chemotherapy. No recurrent bleeding occurred.

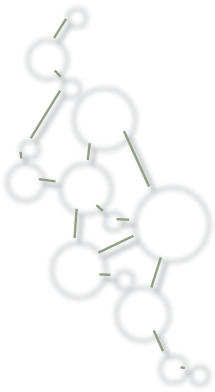
## Discussion

Hematogenous metastases to the stomach are a rare event. The most frequent tumors involved in secondary gastric sites are melanoma, breast, and lung cancer. Most patients with gastrointestinal metastases are asymptomatic[1]. Abdominal pain is the most frequent (80% of the cases) symptom in the symptomatic patient. Differential diagnosis are lymphoma, ectopic pancreas and carcinoid tumor. These lesions may present with three different appearances: (1) multiple nodules of variable size with a central ulcer; (2) submucosal, raised, and ulcerated at the tip and defined as **"**volcano-like**"**; and (3) raised areas without a central ulcer. In these patients, the prognosis is very poor.

## References

1. Kadakia SC, Parker A, Canales L. Metastatic tumors to the upper gastro-intestinal tract: endoscopic experience. Am J Gastroenterol 1992;87:1418-23.

2. Green LK. Hematogenous metastases to the stomach. A review of 67 cases. Cancer 1990;65:1596-600.

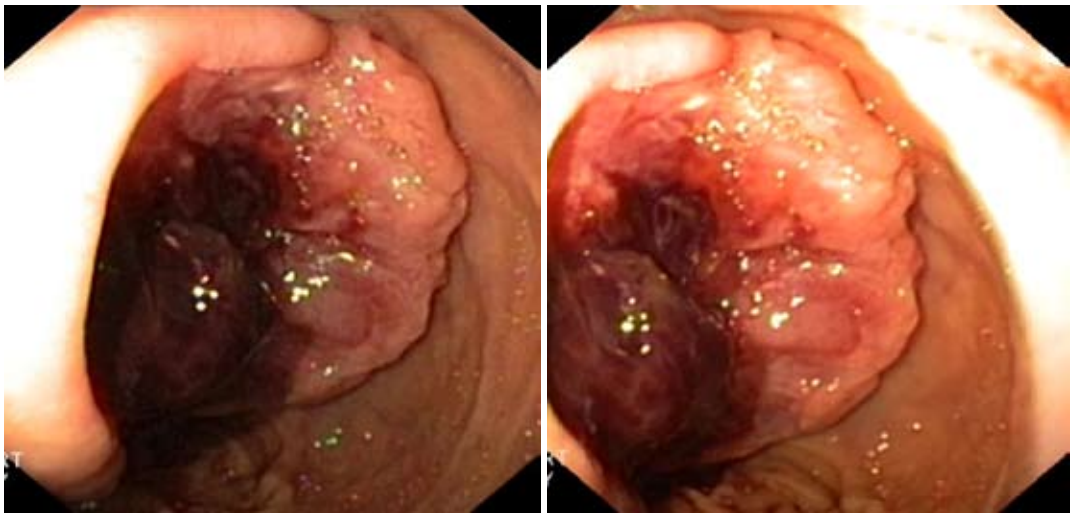


# Case 9

Sukprasert Jutaghokiat, MD.
Rungsun Rerknimitr, MD.

A 44 years old female presented with early satiety and palpable abdominal mass at epigastrium for 6 months.

EGD was done and showed as pictures.



In this case, pathology revealed spindle shaped cells with positive staining for CD 117. His diagnosis is duodenal gastrointestinal tumors (GISTs). CT scan showed large 10 cm. mass with central necrosis involving second part of duodenum without distant metastasis.

## Discussion

Gastrointestinal stromal tumors are the most common mesenchymal neoplasm of the gastrointestinal tract and are highly resistant to conventional chemotherapy and radiotherapy. Such tumors usually have activating mutations in either KIT (75–80%) or PDGFRA (5–10%), two closely related receptor tyrosine kinase. Most of GISTs occurred in stomach and small intestines. Duodenal GISTs are rare conditions but possess poorer prognosis. Aggressive gastrointestinal stromal tumors have a defined pattern of metastasis to the liver or throughout the abdomen Tumor size and mitotic activity were two principal factors in the risk stratification. Imatinib mesylate, a small-molecule kinase inhibitor, has proven useful in the treatment of recurrent or metastatic gastrointestinal stromal tumors.

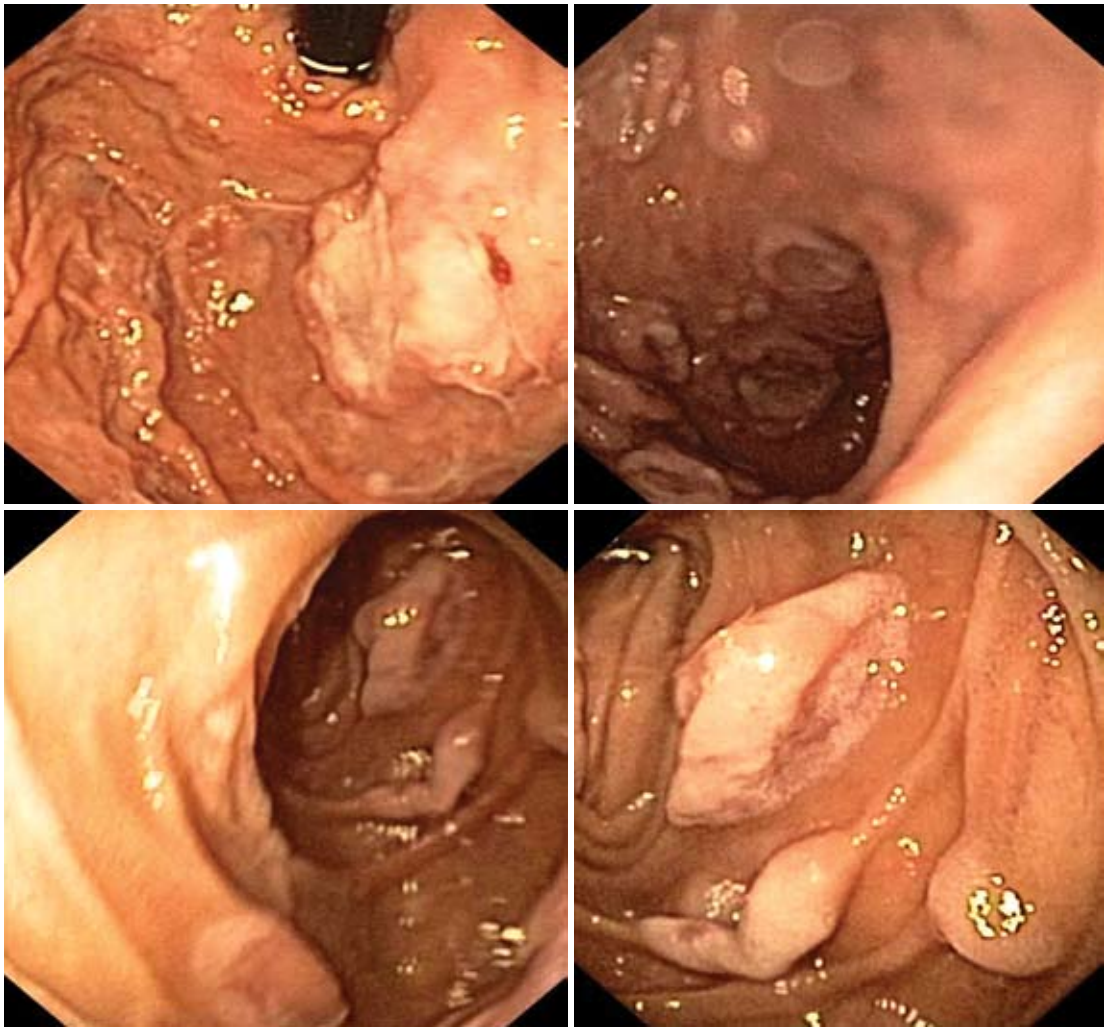
## Reference

1. Rubin BP, Heinrich MC, Corless CL. Gastrointestinal stromal tumour. Lancet 2007;369:1731-41.

# Case 10

Boonlert Imraporn, MD.
Rungsun Rerknimitr, MD.

A 40 years old male developed upper GI bleeding with coffee ground contents. His underlying disease is symptomatic HIV infection with CD4 count = 170 cell/ml.



In this case, pathology showed non Hodgkin lymphoma (NHL). He was referred to hematologist for chemotherapy.

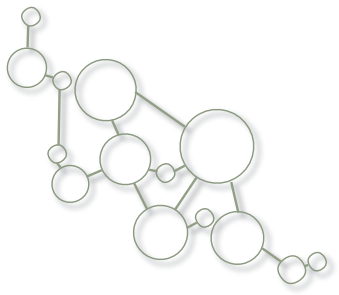
## Discussion

Development of lymphoma is considered an AIDS-defining condition[1]. HIV associated NHL typically has an aggressive presentation with rapidly growing disease and prominent B symptoms[2]. The gastrointestinal tract is a common site including unusual sites such as anus and rectum. Prognosis is generally poor, with 2-year survival rates of 10-20%. However, survival is improved with HAART regimen.

## References

1. 1993 revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. MMWR Morb Mortal Wkly Rep 41:1,1992.

2. Kaplan LD: Clinical management of human immunodeficiency virus-associated non-Hodgkin's lymphoma. J Natl Cancer Inst Monogr 1998;23:101.

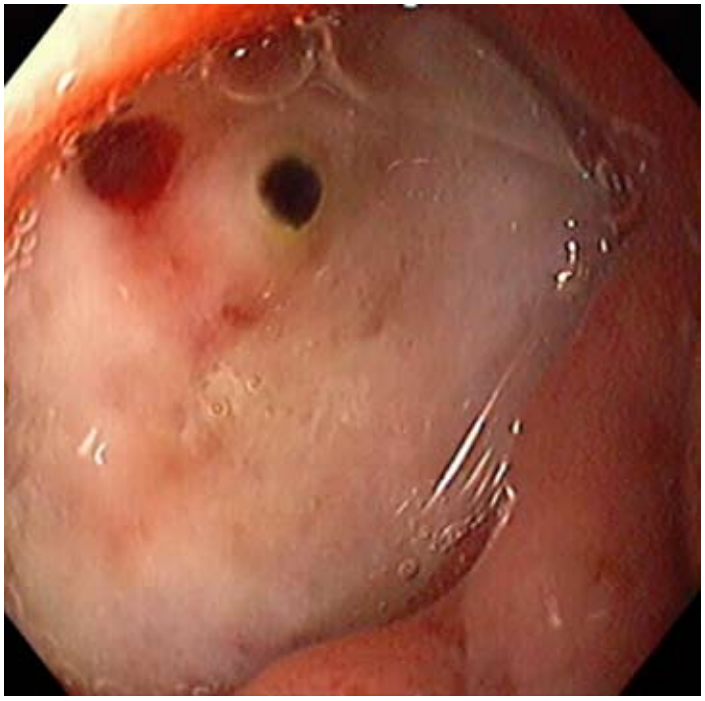


# Case 11

Sukprasert Jutaghokiat, MD.
Rungsun Rerknimitr, MD.

A 45 year-old man presented with acute hematemesis for 8 hours. He had no underlying disease but he regularly took NSAIDs. His vital sign was stable.

EGD was done and showed as figure.



EGD finding showed large duodenal ulcer at duodenal bulb with non bleeding visible vessel and nearby one flat black pigmented spot.

Combined adrenaline injection and bipolar coaptation were done.

## Discussion

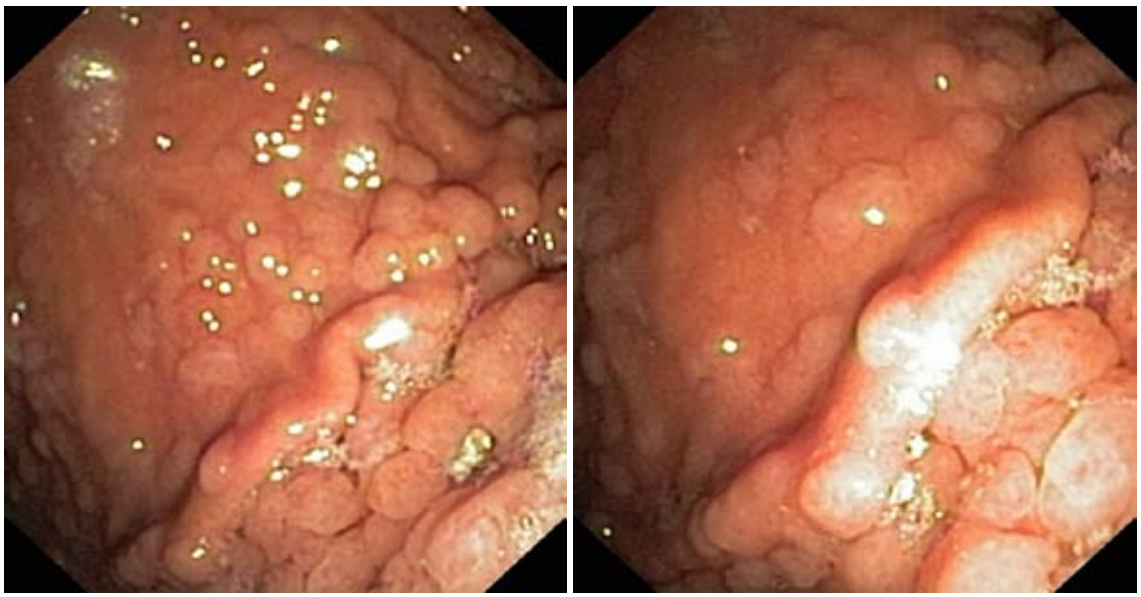
Peptic ulcer disease is the most common specifically identified cause of acute upper GI bleeding. The incidence rate of bleeding from duodenal ulcer is approximately twice that of gastric ulcer. The ulcer on posteroinferior wall of duodenal bulb are most likely to bleed or rebleed. The rebleeding rate depends on endoscopic stigmata. The ulcer with non-bleeding visible vessel and flat pigmented spot carry about 43% and 10% rebleeding rate respectively. Eventually this patient requires endoscopic interventions to prevent recurrent bleeding.

# Case 12

Boonlert Imraporn, MD.
Rungsun Rerknimitr, MD.

A 30 year-old man with known history of familial adenomatous polyposis came to the hospital for EGD.

EGD finding was shown as pictures.



EGD findings revealed multiple small gastric polyps located in fundus. His diagnosis is multiple fundic gland polyps.

## Discussion

Fundic gland polyps (FGPs) are the most common gastric polyps in both familial adenomatous polyposis (FAP) and sporadic patients. FGPs are reported to occur in 12.5 to 84% of patients with FAP whereas sporadic FGPs are identified in 0.8 to 1.9% of non-FAP patients undergoing upper gastrointestinal endoscopy. FAP associated FGPs tend to be more numerous, occur at a younger age, and have a more equal gender distribution. The pathogenesis of FGPs remains uncertain. FGPs have generally been regarded as non-neoplastic lesions. Neoplastic progression of FGPs in FAP patients has occasionally been reported, including the development of a large dysplastic gastric polyp or even infiltrating gastric cancer[1-4]. Despite the lack of more exact

estimates of the risk of tumor progression in patients with FAP and fundic gland polyposis, molecular evidence indicates that FAP associated FGPs are neoplastic polyps. Similar to the presence of other neoplastic polyps of the upper gastrointestinal tract in patients with FAP, the presence of fundic gland polyposis may warrant close endoscopic surveillance[5].

## References

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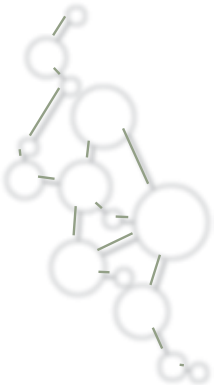
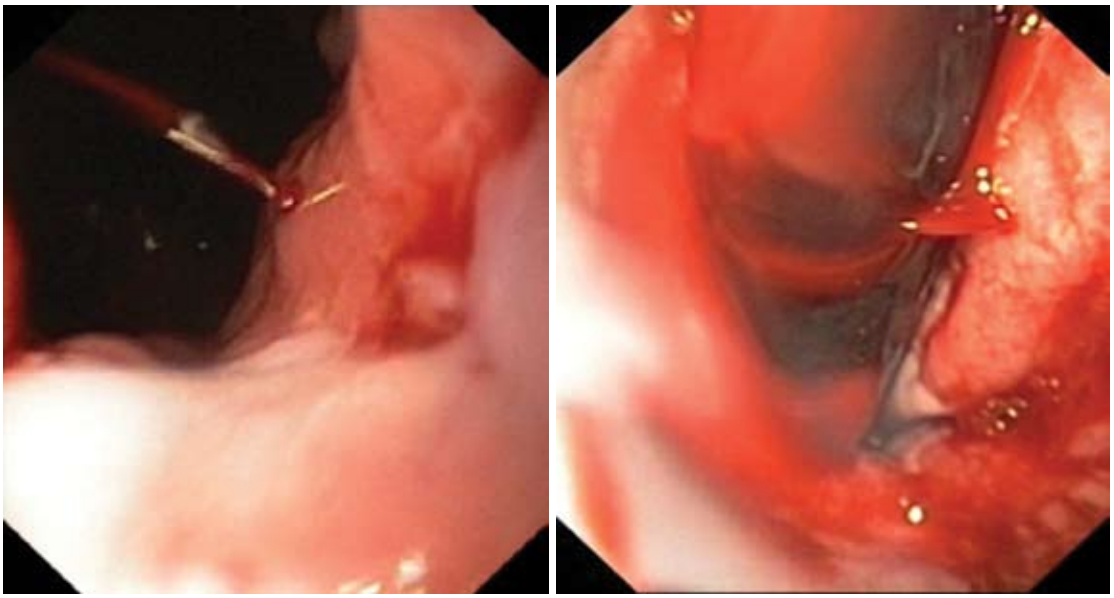


# Case 13

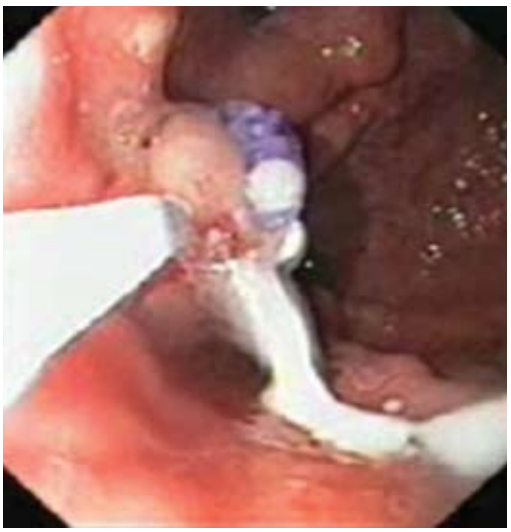
Nathavut Sirimontaporn, MD.
Rungsun Rerknimitr, MD.

A Thai 36 years old man, present with hematemesis 10 hrs prior to admission. It was the first episode of hematemesis. He had no previous melena. He was diagnosed as having HIV positive and on HAART since 7 years ago, and his HIV condition was well-controlled.

Esophagogastroduodenoscopy was shown as pictures



EGD Stomach: Gastric varices at fundus length 1.0 cm with active bleeding from varices, No GU



The cyanoacrylate glue was injected intravariceal via endoscope, and the results was shown

**Diagnosis:** Active gastric variceal bleeding with successfully stopped bleeding by intravariceal injection of cyanoacrylate glue.

## Discussion

Gastric varices are abnormally dilated submucosal veins in the stomach that can be a life-threatening cause of upper GI hemorrhage. Gastric varices are caused most commonly by cirrhosis with associated portal hypertension. Gastric varices are less prevalent than esophageal varices and are present in 5%-33% of patients who have portal hypertension, with an incidence of bleeding ranging from 3%-30%[1] Larger varices, those with associated with red spots, and those found in patients who have advanced liver disease are more likely to bleed[2], risk factors for bleeding gastric varices include variceal size, severity of cirrhosis based on Child-Pugh score, and red wheals or cherry red spots on the variceal wall[2].

Gastric varices are sometimes difficult to detect by endoscopy, small gastric varices often are mistaken for a mucosal fold, gastric varices are identified based on their shape (grapelike cluster) and color (bluish tinge), in difficult situation, endoscopic ultrasound has been demonstrated to be superior to endoscopy in the diagnosis of gastric varices but the clinical role of endoscopic ultrasound in the diagnosis of gastric varices remains to be established[3]. Angiography also can identify gastric varices, however, angiography usually is performed when severe upper GI bleeding precludes adequate diagnosis examination.

Management of gastric varices includes several modalities, preliminary trials have shown that both endoscopic obturation using cyanoacrylate (Histoacryl)[4] and gastric variceal ligation[5] are promising tools in the management of gastric variceal bleeding. The mechanism of arresting variceal bleeding is different in both modalities. Cyanoacrylate glue polymerized rapidly and plugs the lumen if injected into a varix[4]. In contrast, ligation of varices results in strangulation and necrosis of varices[6].

There are trial showed that cyanoacrylate injection was superior to gastric variceal ligation in terms of arresting acute bleeding and prevention of rebleeding[7] and found to be safe in terms of lower ulcer rates induced by glue injection than the gastric variceal ligation group.

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