

*Treesaranuwattana S*  
*Amornsawadwattana S*  
*Rerknimitr R*  
*Sirimontaporn N*  
*Kittirakul C*

### CASE 1

A 56-year-old man presented with abdominal pain for two weeks. Physical examination was unremarkable. Ultrasonography of the upper abdomen was un-

remarkable 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.

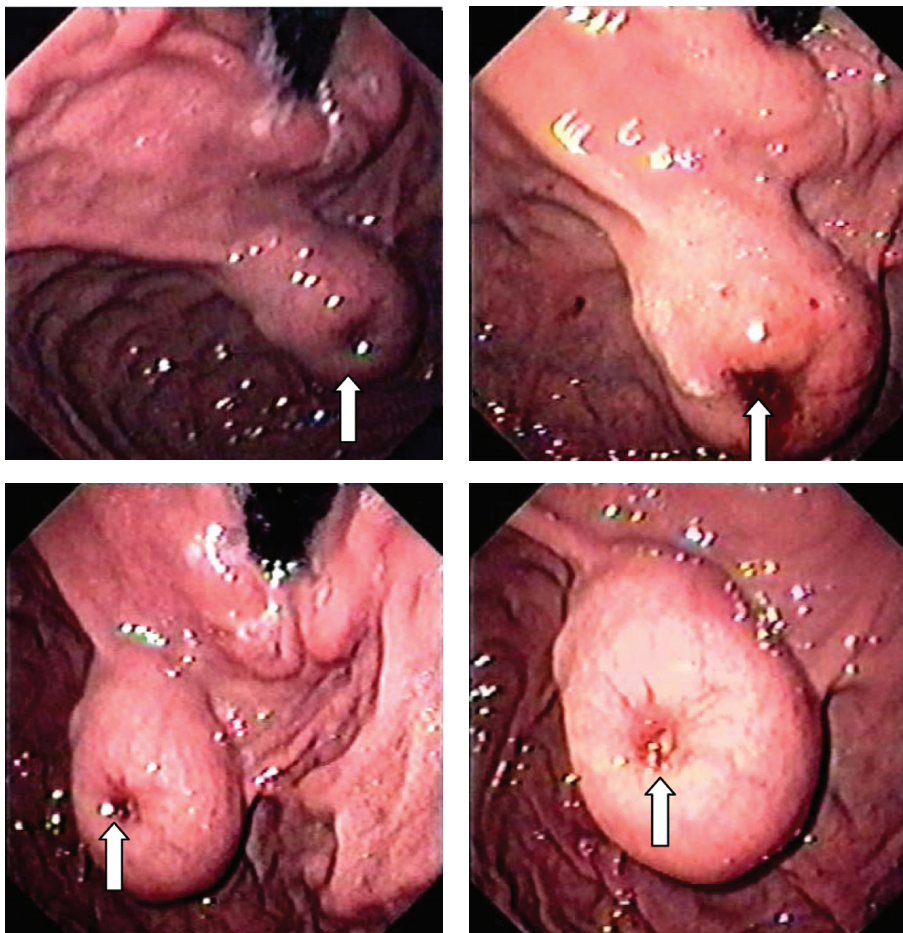


Figure 1.

### Pathological diagnosis

Gastrointestinal tumor (GIST) of the stomach. Degree of mitosis <5/50 HPF, immunohistochemical study: positive CD117 and CD34, negative S-100 and SMA.

### Discussion

GISTs are rare, accounting for only 0.1-3% of all GI malignancies<sup>(1)</sup> but they represent 80% of gastrointestinal mesenchymal tumor<sup>(2)</sup>. 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%)<sup>(3)</sup>. 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<sup>(4)</sup> 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 GISTs. 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<sup>(5)</sup>.

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

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

1. DeMatteo RP, Lewis JJ, Leung D, *et al.* Two hundred Gastrointestinal stromal tumors: recurrence patterns and prognostic factors for survival. *Ann Surg* 2000;231:51-8.
2. Miettinen M, Lasota J. Gastrointestinal stromal tumors: definition, clinical, histological, immunohistochemical and molecular genetic features and differential diagnosis. *Virchows Arch* 2001;438:1-12.
3. Miettinen M, Majidi M, Lasota J. Pathology and diagnostic criteria of gastrointestinal stromal tumors (GISTs): a review. *Eur J Cancer* 2002;38(Suppl 5):S39-S51.
4. Demetri GD, Delaney T. NCCN;sarcoma. *Cancer Control* 2001; 8:94-101.
5. Fletcher CD, Berman JJ, Corless C, *et al.* Diagnosis of gastrointestinal stromal tumors: a consensus approach. *Hum Pathol* 2002;33:459-65.
6. Joensuu H, Flecher C, Dimitrievic S, *et al.* Management of malignant gastrointestinal stromal tumors. *Lancet Oncol* 2002; 3:655-64.

Table 1.

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

## CASE 2

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.

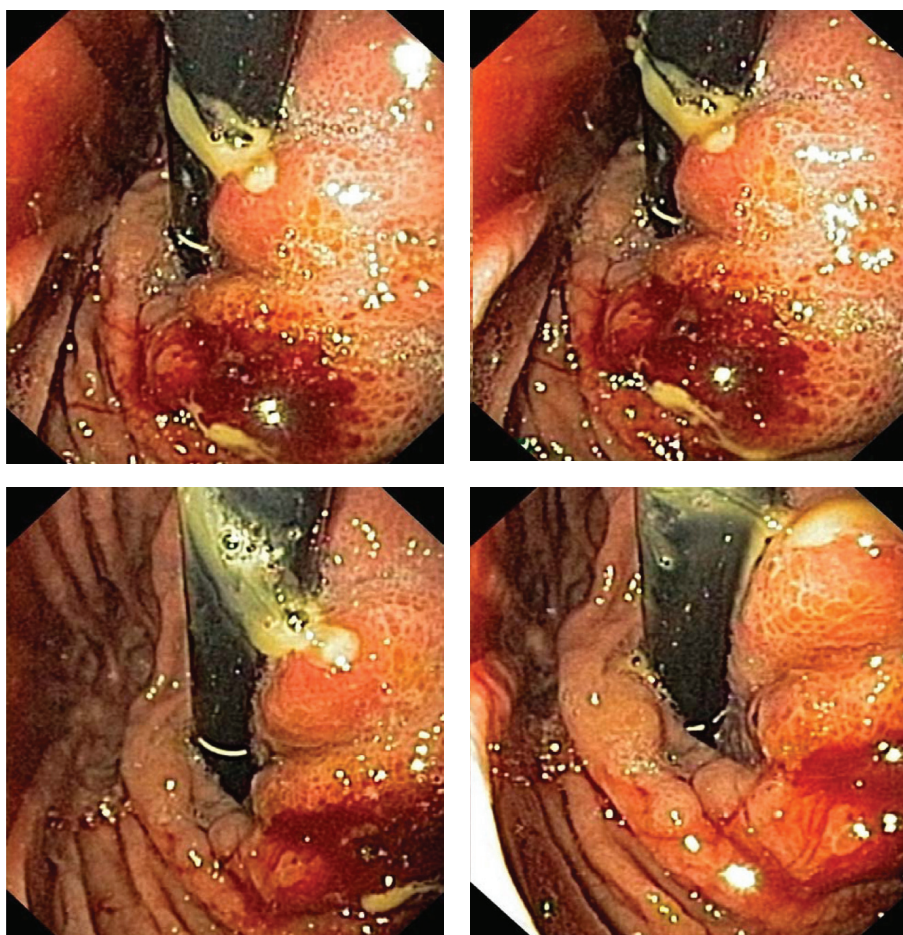


Figure 2.

### Diagnosis:

Pululent material exploring 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*<sup>(1)</sup> and *Pseudomonas* sp.<sup>(2)</sup> Viral diseases such as hepatitis B<sup>(3)</sup> and C<sup>(4)</sup> 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

1. Langenberg W, Rauws EA, Oudbier JH, *et al.* Patient to patient transmission of *Campylobacter pylori* by fiberoptic gastroduodenoscopy and biopsy. *J Infect Dis* 1990; 161:507-11.
2. Moayyedi P, Lynch D, Axon A. *Pseudomonas* and endoscopy. *Endoscopy* 1994; 26:554-8.
3. Birnie GG, Quigley Em, Clements GB, *et al.* Endoscopic transmission of hepatitis B virus. *Gut* 1983; 24:171-4.
4. Bronowicki JP, Vernard V, Botte C, *et al.* Patient-to-patient transmission of hepatic C virus during colonoscopy. *N Engl J Med* 1997; 37:237-40.



### CASE 3

A 56-year-old male patient, presented with dyspepsia for 1 month. He denied any weight loss. Esophagogastroduodenoscopy was done as shown.

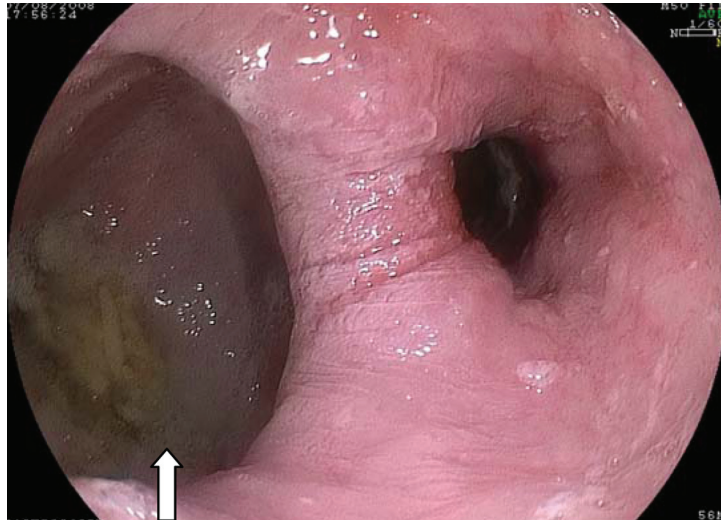


Figure 3.

#### Endoscopic findings

An outpouching of esophageal mucosa at lower esophagus (white arrow), 6 cm. in diameter, with minimal food content.

#### Diagnosis

Epiphrenic diverticulum

#### Discussion

Esophageal diverticula are rare. They can be divided into 3 categories according to locations: pharyngo-esophageal, parabronchial and epiphrenic diverticula. Another classification, Rokitansky classification<sup>(1)</sup>, provides more useful information of the etiopathogenesis of esophageal diverticula. This classification divided esophageal diverticula to 2 types: traction diverticula and pulsion diverticula. Traction diverticula are the result of a chronic inflammatory process starting from the mediastinal lymph nodes (usually from a granulomatous disease) which involves the esophageal wall. These diverticula are commonly seen in the vicinity of the carina (parabronchial diverticula). Pulsion diverticula are the result of an altered pressure gradient inside the esophageal lumen, which determines their formation through *loci minoris resistentiae* in the esophageal wall. These develop naturally, as the Killan's triangle where the pharyngo-oesophageal diverticula locate, or due to separation of the esoph-

ageal wall above a zone of altered motility, as in the case of epiphrenic diverticula.

Epiphrenic diverticulum is usually single and asymptomatic.<sup>(2)</sup> It occurs less frequently than Zenker's diverticulum. It is usually asymptomatic and found accidentally by imaging studies. When symptoms are present, the most frequent symptoms are dysphagia and regurgitation of indigested food. Surgery is the mainstay for treatment. The decision whether to operate must be balanced between patient's symptoms and the operative risk including the presence of surgical expertise. Traditional surgical treatment for an epiphrenic diverticulum consists of esophageal myotomy, diverticulectomy (or diverticulopexy), and an antireflux procedure. The surgery is usually performed through a thoracotomy. Currently there are minimally invasive operations<sup>(3)</sup> developed for epiphrenic diverticula and resulting in good outcomes that comparable to traditional surgery.

#### REFERENCES

1. Posthletwait RW. Diverticula of the esophagus. Surgery of the Esophagus. Norwalk: Appleton Century Crofts; 1986:129-60.
2. Costantini M, Zaninotto G, Rizzetto C, *et al.* Oesophageal diverticula. Best Pract Res Clin Gastroenterol 2004; 18:3-17.
3. Varghese TK, Marshall B, Chang AC, *et al.* Surgical treatment of epiphrenic diverticula: a 30-year experience. Ann Thorac Surg 2007; 84:1801-9.