

Case 1

Phonthep Angsuwatcharakon, MD.

Rungsun Rerknimitr, MD.

A 46-year-old man presented with chronic heartburn. EGD was done.



EGD showed salmon-pink patches extending beyond EGJ and some squamous islands were detected in this patch.

The diagnosis is Barrett's esophagus

Differential diagnosis is an inlet patch (heterotopic gastric mucosa)

Discussion:

Barrett's esophagus (BE) is defined as an intestinal metaplasia of the esophagus. The endoscopic finding of BE is salmon-color mucosa located above esophago-gastric junction¹. This can be associated with adenocarcinoma of the esophagus, the rate of cancer development is range between 0.5-3% per year². The duration for endoscopic surveillance in BE depends on the grade of dysplasia as in table³.

Table : Surveillance schedule for BE

Dysplasia	Follow-up endoscopy or management
None	3 year
Low grade	1 year until no dysplasia
High grade	Focal: every 3 months
	Multifocal: interventional therapy
	Mucosal irregularity: endoscopic mucosal resection

References

1. Badreddine RJ, Wang KK. Barrett's esophagus: pathogenesis, treatment and prevention. Gastrointest Endosc Clin N Am 2008;18:495-512.
2. von Rahden BH, Stein HJ, Weber A, Vieth M, Stolte M, Rosch T, et al. Critical reappraisal of current surveillance strategies for Barrett's esophagus: analysis of a large German Barrett's database. Dis Esophagus 2008;21:685-9.
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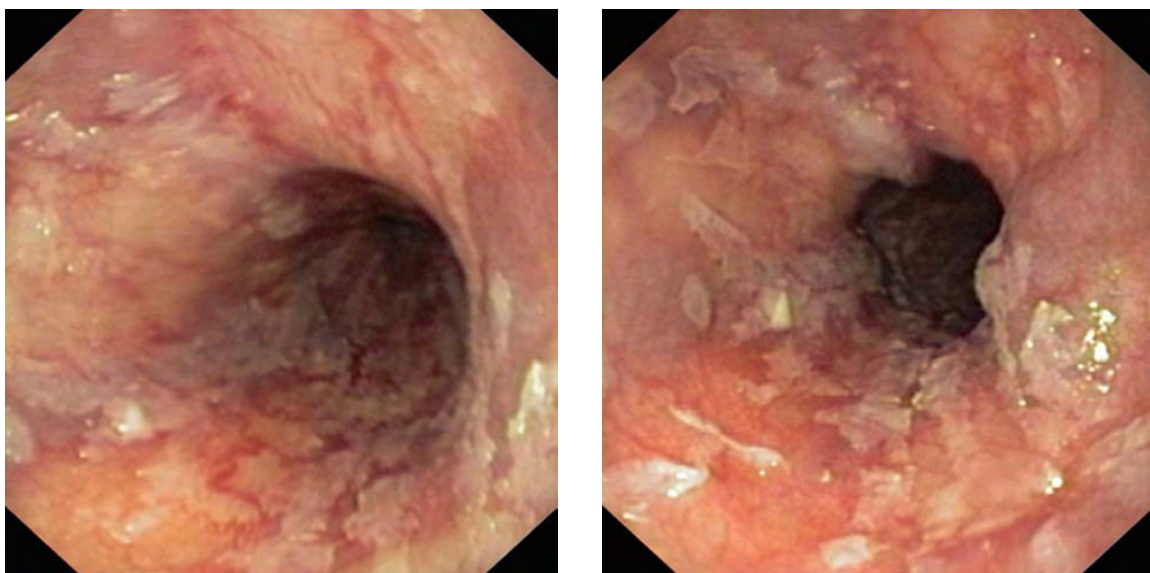


Case 2

Boonlert Imraporn, MD.

Rungsun Rerknimitr, MD.

A 60-year-old man with a history of old ischemic stroke presented with coffee ground contents from nasogastric tube for one day. His status was bed ridden and nasogastric feeding was required. Esophagogastroduodenoscopy was done as shown.



His EGD findings revealed nearly circumferential esophageal erosion with whitish mucosal slough extending from esophago-gastric junction up to 2 cm. above. His diagnosis was **reflux esophagitis grade C** according to the Los Angeles classification.

Discussion:

Most patients with gastro-esophageal reflux disease (GERD) are mainly classified into two categories: Non-erosive reflux disease (NERD) and erosive esophagitis. NERD accounts for approximately 70 percent of patients with GERD symptoms. When compared to NERD, patients with erosive esophagitis have more body mass index¹, more common esophageal dysmotility and more acid exposure from 24 hours

pH monitoring². The Los Angeles (LA) classification describes four grades of esophagitis severity from A to D, based on the extent of esophageal lesions known as mucosal breaks. The detail of LA classification is shown in the table below. The treatment of GERD includes lifestyle modification, medications or surgery. In case of severe esophagitis, long term proton pump inhibitors (PPI) are required to prevent the recurrence of esophagitis and to control the symptoms.

Los Angeles Classification of Reflux Esophagitis

Grade A	Mucosal break < 5 mm in length
Grade B	Mucosal break > 5 mm
Grade C	Mucosal break continuous between > 2 mucosal folds
Grade D	Mucosal break > 75% of esophageal circumference

References

1. Hampel H, Abraham NS, El-Serag HB. Meta-analysis: obesity and the risk for gastroesophageal reflux disease and its complications. *Annals of internal medicine* 2005;143:199-211.
2. Fass R. Epidemiology and pathophysiology of symptomatic gastroesophageal reflux disease. *The American journal of gastroenterology* 2003;98(Suppl):S2-7.



Case 3

Chatporn Kittitrakul, MD.

Rungsun Rerknimitr, MD.

A 66-year-old male patient, presented with 7 kgs weight loss within 2 months. He also had a history of bowel habit change for 2 months.

Esophagogastroduodenoscopy and colonoscopy were done as shown.

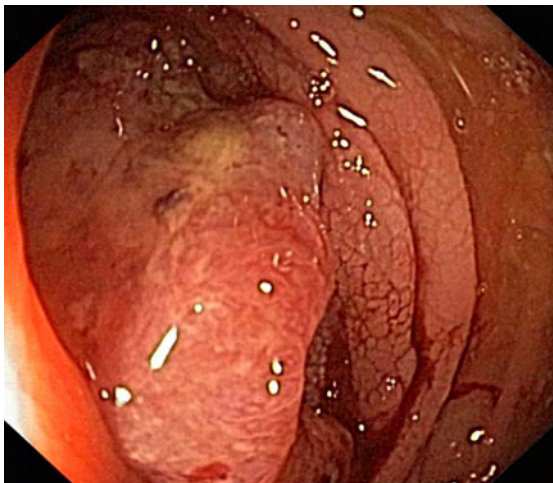


Endoscopic findings:

- A 3 cm. shallow irregular-surface ulcer in the distal esophagus (30-33 cm. from incisor)
- Circumferential fungating mass with easy contact bleeding, size 4x5 cm., was also found in the rectum. Scope was not able to pass through this mass.

Diagnosis:

- 1) Malignant esophageal ulcer.
- 2) Rectal mass with possible colorectal cancer.



Pathology report:

- Esophageal ulcer: **moderately differentiated squamous cell carcinoma of the esophagus.**
- Rectal mass: **well-differentiated adenocarcinoma with mucin producing component.**

Discussion:

In this patient the indication for endoscopy is to work up for the cause of weight loss and the most likely cause was gastrointestinal malignancy. The characteristics findings of the esophageal ulcer and rectal mass were suggestive of malignant in origin. Although it is rare, synchronous multiple primary malignant neoplasm was the most likely diagnosis by the initial endoscopic findings which later confirmed by pathology. A prospective study from Hong Kong¹ showed that the incidence of multiple primary cancers beginning with squamous cell carcinoma of the esophagus was 10.8%. The overall synchronous tumors diagnosed simultaneously with the diagnosis of esophageal carcinoma were found in 28.1% of all multiple primary cancers. Hypopharynx was the most common site of other synchronous primary cancers (37.8%). Larynx and tongue were the second and the third common site, respectively. Colon cancer was found in 4.4% of reported synchronous primary cancers.

The decision for management in patients with multiple primary cancers is more difficult than esophageal carcinoma alone. From the previous studies^{1,2}, the treatment was largely determined by the stage of esophageal carcinoma. Patients with potentially curable esophageal lesions usually underwent curative resection, whereas patients with incurable lesions underwent palliative resection if possible. In the first study¹, there is no statistically significant difference between patients with synchronous cancers and without multiple primary cancers. Median survival was 8.5 months and 8.8 months respectively. The result of the second study was in the same manner². Another report from Japan demonstrated that the median survivals for multiple primary cancer and pure esophageal cancer were 19.6 months and 25.0 months respectively².

References

1. Poon R, Law S, Chu KM, Branicki FJ, Wong J. Multiple primary cancers in esophageal squamous cell carcinoma: incidence and implications. *Ann Thorac Surg* 1998;65:1529-34.
2. Nagasawa S, Onda M, Sasajima K, Takubo K, Miyashita M. Multiple primary malignant neoplasms in patients with esophageal cancer. *Dis Esophagus* 2000;13:226-30.

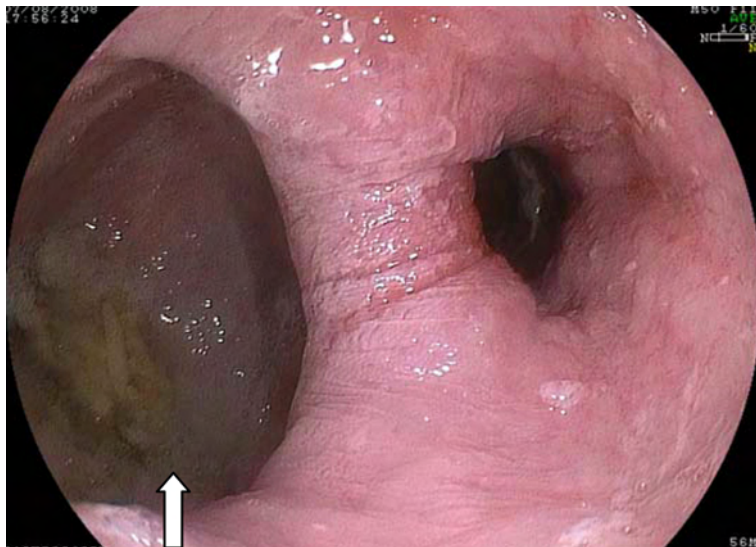


Case 4

Chatporn Kittitrakul, MD.

Rungsun Rerknimitr, MD.

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



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 classification¹, 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 Killian's triangle where the pharyngo-oesophageal diverticula locate, or due to separation of the esophageal wall above a zone of altered motility, as in the case of epiphrenic diverticula.

Epiphrenic diverticulum is usually single and asymptomatic.² 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 anti-reflux procedure. The surgery is usually performed through a thoracotomy. Currently there are minimally invasive operations³ developed for epiphrenic diverticula and resulting in good outcomes that comparable to traditional surgery.

References

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2. Costantini M, Zaninotto G, Rizzetto C, Narne S, Ancona E. Oesophageal diverticula. Best Pract Res Clin Gastroenterol 2004;18:3-17.
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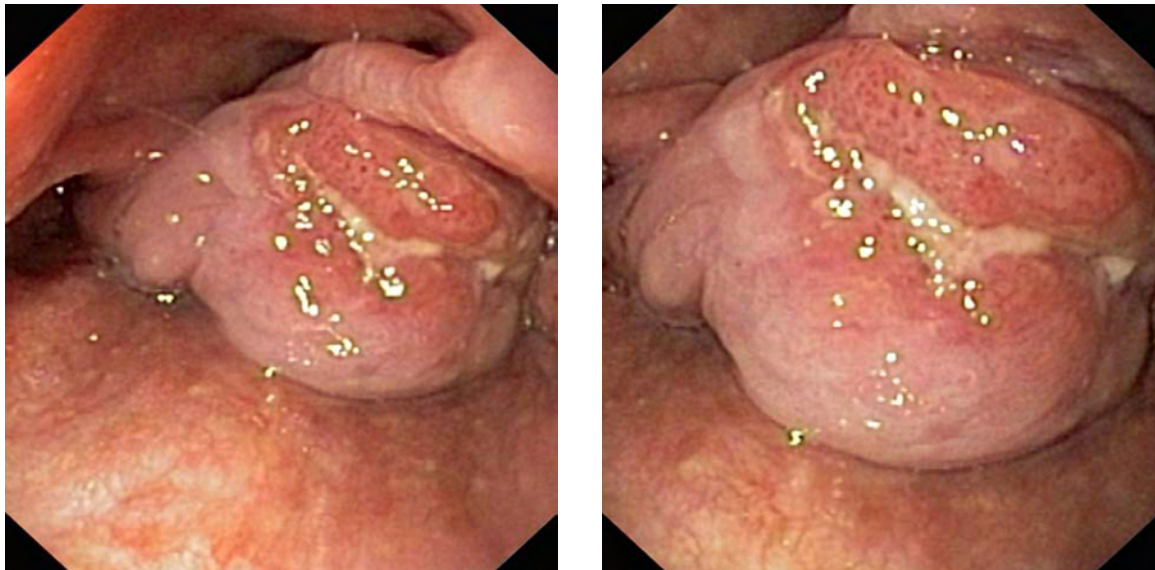
Case 5

Chatporn Kittitrakul, MD.

Rungsun Rerknimitr, MD.

A 69-year-old male patient, presented with iron deficiency anemia without history of gastrointestinal bleeding.

Esophagogastroduodenoscopy was done and shown as figures.



Endoscopic findings:

- A large irregular surface mass with ulceration at the posterior wall of cricoarythenoid fold, size 2 cm. in diameter. It compressed the larynx. Scope was able to pass through into the esophagus with some difficulty.
- Normal esophagus.

Diagnosis:

Hypopharyngeal cancer.

Pathology report:

- Hypopharyngeal mass: moderately differentiated squamous cell carcinoma

Discussion:

This patient presented with iron deficiency anemia without a history of gastrointestinal bleeding or ENT related complains. An esophagogastroduodenoscopy was performed to identify the cause of anemia. Then this hypopharyngeal mass was found.

Hypopharyngeal cancer is not common.¹ It accounts for 5% of all head and neck cancers. The most common presenting symptoms are dysphagia (53%), hoarseness (39%), neck mass (37%), weight loss (36%), sore throat (34%), otalgia (30%), and hemoptysis (5.5%). The tumors are advanced (> 50% stage 4) and 20% of cases deem unresectable at the initial presentation.

Surgical resection for hypopharyngeal carcinoma is very invasive and associated with increased morbidity including cosmetic deformities, swallowing disorder, aspiration pneumonia and speech defect. Radiotherapy or chemoradiotherapy are indicated as palliative treatments. However, these still can cause various complications such as oral pain, disorder of taste sensation and swallowing disorder. Recent developments in endoscopic diagnosis have enabled to detect an early hypopharyngeal carcinoma. At the early stage these lesions are just only the reddish or discolored lesions. Endoscopic mucosal resection with minimal invasiveness has been reported with good outcome.^{2,3} Endoscopic submucosal dissection has also been used in selected cases.⁴ This technique can obtained en bloc resection without local recurrence in one case series⁴.

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2. Nagai K, Kawada K, Nishikage T, et al. Endoscopic treatment for superficial hypopharyngeal carcinoma. *Stomach Intestine* 2003;38:331-8.
3. Muto M, Nakane M, Katada C, Sano Y, Ohtsu A, Esumi H, et al. Squamous cell carcinoma in situ at oropharyngeal and hypopharyngeal mucosal sites. *Cancer* 2004;101:1375-81.
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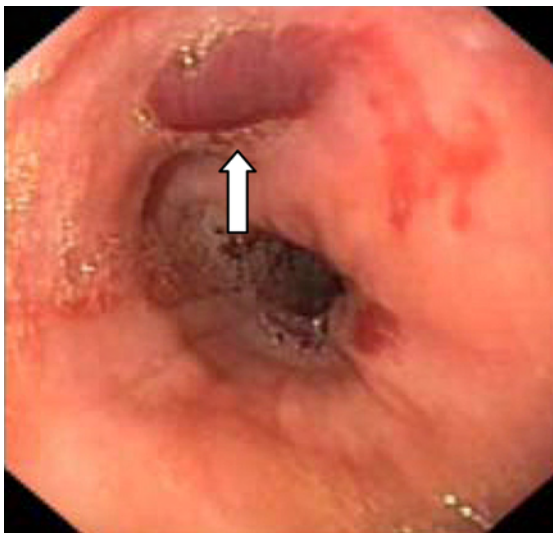
Case 6

Phonthep Angsuwatcharakon, MD.

Rungsun Rerknimitr, MD.

An 84-year-old man, previously diagnosed with pemphigus vulgaris by skin biopsy, presented with upper GI hemorrhage.

An EGD was done as shown.



The EGD showed discrete submucosal hemorrhage at mid-esophagus with easily peeled away of the superficial layer, resulting in erosions as shown (white arrow).

The diagnosis is pemphigus vulgaris involving the esophagus.

Discussion:

Pemphigus vulgaris is an autoimmune disease which has autoantibody to intercellular adhesion molecule of epidermal cells, causes easily separation of stratified columnar epitheliums. This involvement can be found at any stratified squamous epithelium¹. In patients with pemphigus vulgaris, esophageal involvement is ranged from 46-87.5%^{2,3}.

References

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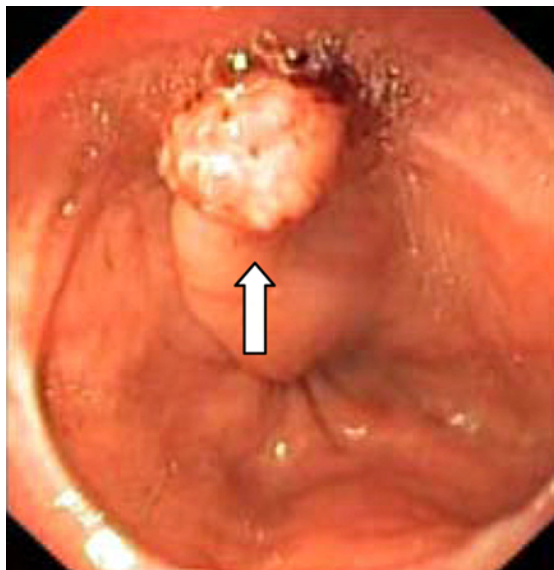
Case 7

Sunthorn Treesaranuwattana, MD.

Phonthep Angsuwatcharakon, MD.

Rungsun Rerknimitr, MD.

A 71-year-old woman, presented with dyspepsia. An EGD was done as shown.



The EGD showed a 0.8 cm. polypoid lesion with verrucous surface at distal esophagus (white arrow), just above the esophagogastric junction.

The diagnosis is squamous papilloma of the esophagus.

Differential diagnoses are: inflammatory polyp, foreign body impaction, cardiac polyp, and metastatic nodule.

Discussion:

Squamous papilloma of the esophagus is a rare condition, the reported incidence ranges from 0.01-0.45% from autopsy and endoscopic studies¹. Endoscopy findings are wart-like and polypoid mass, with average size of 0.5 cm². Locations of these lesions are different between Western and Eastern

countries especially in Japan. In Western countries, majority of lesions are found in the lower third of the esophagus due to chronic irritation such as reflux esophagitis^{2,3}. In Japan, majority of lesions are located in the middle esophagus because of high prevalence of chronic gastritis in this population¹. Human papilloma virus is isolated in 10-50% of cases^{1,2}.

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3. Carr NJ, Monihan JM, Sobin LH. Squamous cell papilloma of the esophagus: a clinicopathologic and follow-up study of 25 cases. *Am J Gastroenterol* 1994;89:245-8.



Case 8

Sukprasert Jutaghokiat, MD.

Rungsun Rerknimitr, MD.

A 49-year-old woman with a history of gastroesophageal reflux symptoms for 6 months presented for an endoscopy. Esophagogastrosocopy found a 3 mm. nodule in mid esophagus (Figure A, B). Computed virtual chromoendoscopy with Fujinon Color Enhancement (FICE) and magnification 100 hundred times was applied and showed abnormal vascular pattern as Figure C, D. Biopsy for pathology was compatible with esophageal papilloma.

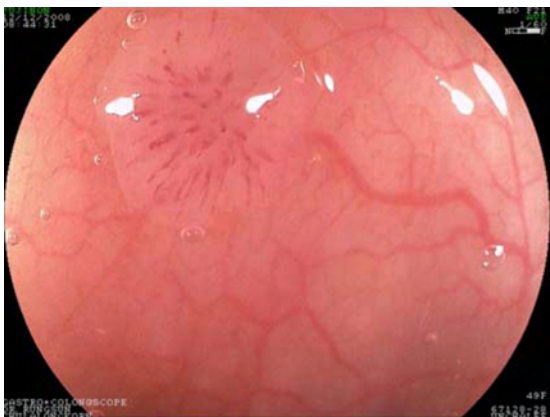


Figure A

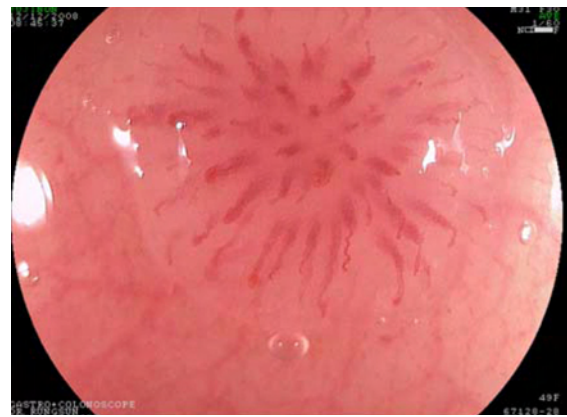


Figure B

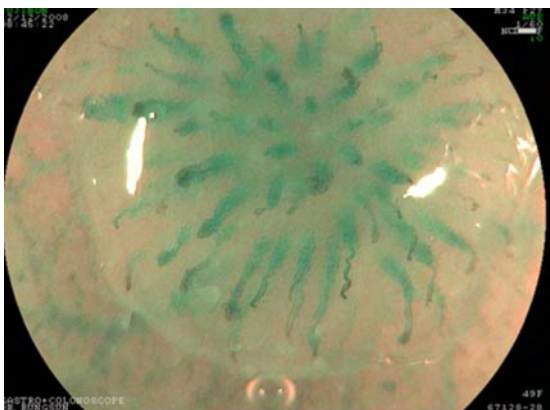


Figure C

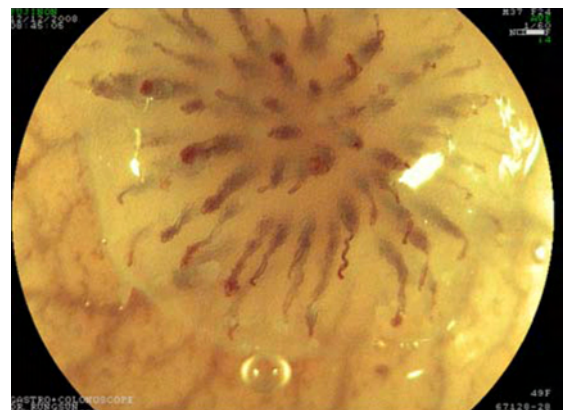


Figure D

Discussion:

Esophageal papillomas are benign epithelial lesions characterized histologically by fingerlike projections of tissue lined by an increased number of squamous cells and a core of connective tissue that contains small blood vessels. They are rare; their incidence in patients undergoing upper endoscopy has ranged from 0.01 to 0.45 percent¹. The etiology of esophageal squamous papilloma remains unclear. It has been suggested that chemical² (reflux), viral³ (human papilloma virus) or mechanical factors² (direct trauma) may contribute to the pathogenesis.

Most lesions do not cause symptoms but large lesions can cause dysphagia. Papillomas are generally amenable to endoscopic resection. A smaller than 1 cm. lesion can usually be removed with a forceps biopsy while a larger lesion requires an endoscopic mucosal resection. Recurrence after resection is infrequent².

References

1. Mosca S, Manes G, Monaco R, et al. Squamous papilloma of the esophagus: long-term follow up. J Gastroenterol Hepatol 2001;16:857.
2. Carr NJ, Monihan JM, Sobin LH. Squamous cell papilloma of the esophagus: a clinicopathologic and follow-up study of 25 cases. Am J Gastroenterol 1994;89:245.
3. Syrjanen, KJ. HPV infections and oesophageal cancer. J Clin Pathol 2002;55:721.



Case 9

Sukprasert Jutaghokiat, MD.

Rungsun Rerknimitr, MD.

A 35-year-old man experienced symptoms of heartburn for one year. His symptoms were usually relieved by anti-secretory agents until one month ago that he had more severe symptoms which partly controlled with medication. He had no weight loss. Esophagogastroduodenoscopy was done and shown as figure.



Figure A

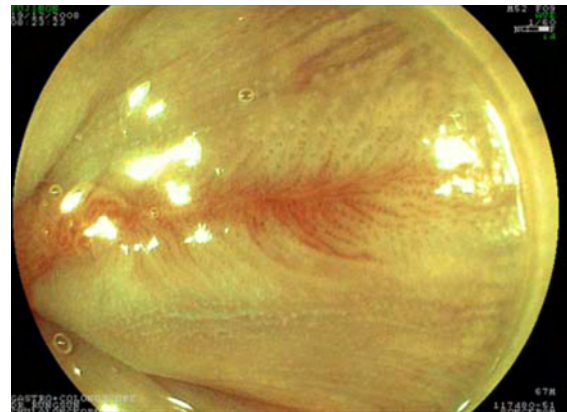


Figure B

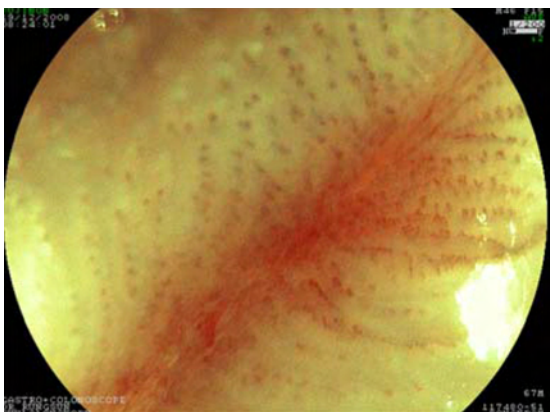


Figure C

EGD showed a linear mucosal break about 5 mm. in length extending from esophago-gastric junction. His diagnosis was **reflux esophagitis grade A according to the LA classification**. Computed virtual chromoendoscopy with Fuji Intelligent Color Enhancement (FICE) was applied to the lesion and shown as

figure B. After magnification about 100 times, the lesion revealed multiple tortuous, elongated and dilated intra-papillary capillary loops (IPCL) along the linear erosion.

Discussion:

Magnifying chromoendoscopy gives the ability to visualize the microstructure of gastrointestinal surface mucosa and mucosal vascularity in detail. Normal esophageal mucosa revealed intra-papillary capillary loops which showing dot-like structure with regular intervals¹. Dilation and elongation of IPCL were reported to be suggestive of inflammatory change in the esophagus including reflux esophagitis².

References

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Case 10

Sukprasert Jutaghokiat, MD.

Rungsun Rerknimitr, MD.

A 40-year-old man presented with GERD symptoms for six months. He had no alarm features. Esophagogastroduodenoscopy with narrow banded imaging (NBI) was done and shown as figures.

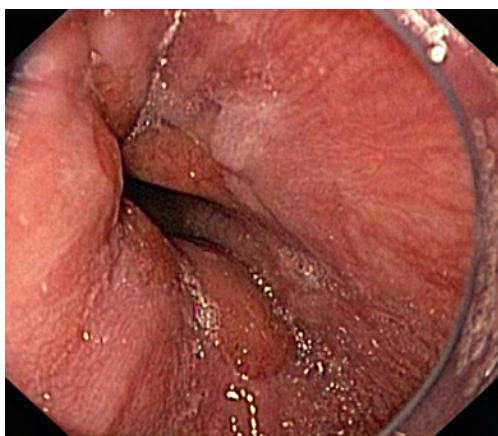


Figure A

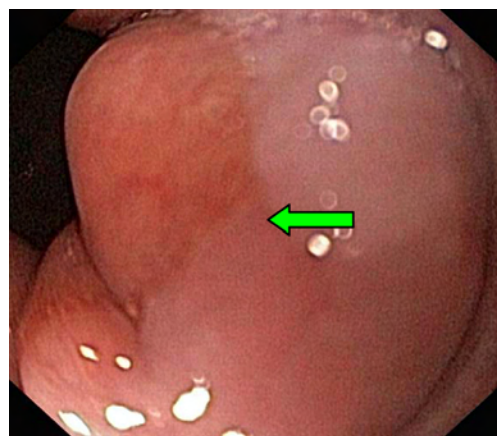


Figure B

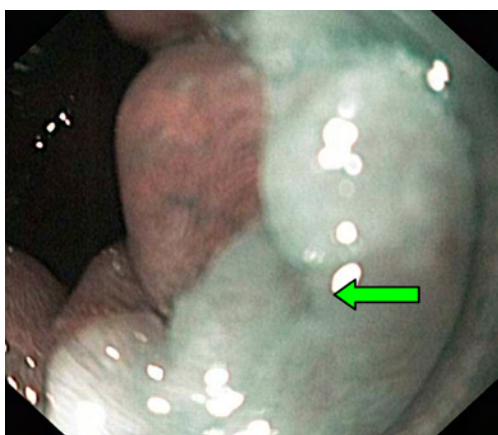


Figure C

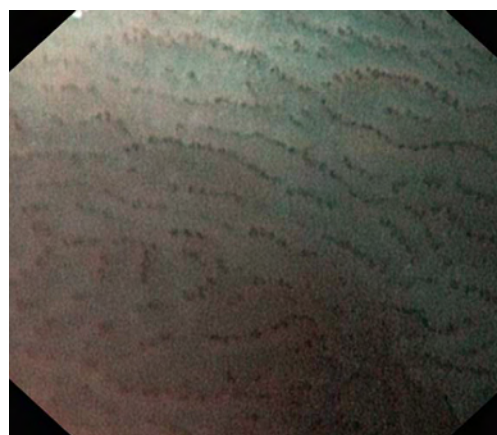


Figure D

White light EGD showed no evidence of erosive esophagitis at Z line. However when NBI system was applied, there was an apparent of a triangular fold (green arrow). Dilated and elongated intra-papillary

capillary loops were identified after using magnification system. Targeted biopsy from these lesions revealed **pathological reflux esophagitis**.

Discussion:

With magnifying chromoendoscopy, the terminology of minimal erosive reflux esophagitis (MERD) is accepted as a part of the spectrum of GERD especially in Japan. By using conventional endoscopy, these lesions cannot be identified and are usually diagnosed as non-erosive reflux esophagitis (NERD). Narrow band imaging (NBI) facilitates mucosal surface evaluation and may improve the endoscopic diagnosis of gastroesophageal reflux disease. NBI improves visualization of Z line to evaluate micro-erosions and identification of intra-papillary capillary loops (IPCL). In addition, NBI is able to detect the presence of increased vascularity or dilated IPCL at Z line and micro-erosions which are not seen on conventional endoscopy¹. However this new diagnostic modality requires validation for clinical practice after more research studies².

References

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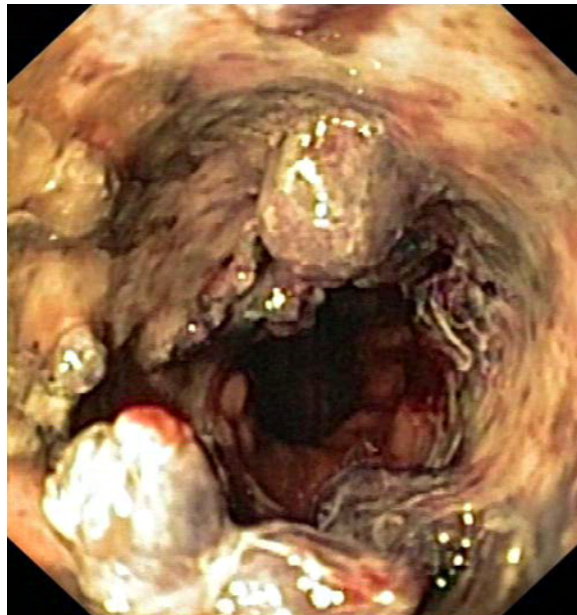


Case 11

Surachai Amornsawadwattana, MD.

Rungsun Rerknimitr, MD.

A 38-year-old male with active alcohol consumption presented with hematemesis for 1 day. He denied use of any medication, caustic ingestion. EGD was performed and picture of distal esophagus was displayed.



Endoscopic findings revealed black necrotic esophageal mucosa at the distal esophagus that stop abruptly at the esophagogastric junction. Stomach and duodenum appeared unremarkable. The diagnosis was **acute necrotizing esophagitis**.

Discussion:

Acute necrotizing esophagitis (ANE) is a rare and severe form of acute esophagitis. It was first described by Goldenberg, et al in 1990². Esophageal mucosa appears circumferentially black in color (black esophagus) due to mucosal necrosis¹. Lower third of esophagus is mostly affected and there is an abrupt

interruption at the esophagogastric junction, while gastric mucosa appeared unaffected¹. Etiologies remain unknown, but ischemic, toxic, traumatic and infectious mechanisms have been proposed as causes¹. Most patients are symptomatic and some present with upper GI bleeding². Complication including perforation, mediastinitis, and stricture were reported². There is no specific treatment¹, mortality depends on the underlying condition, but the overall mortality rate has been reported as 31.8%².

References

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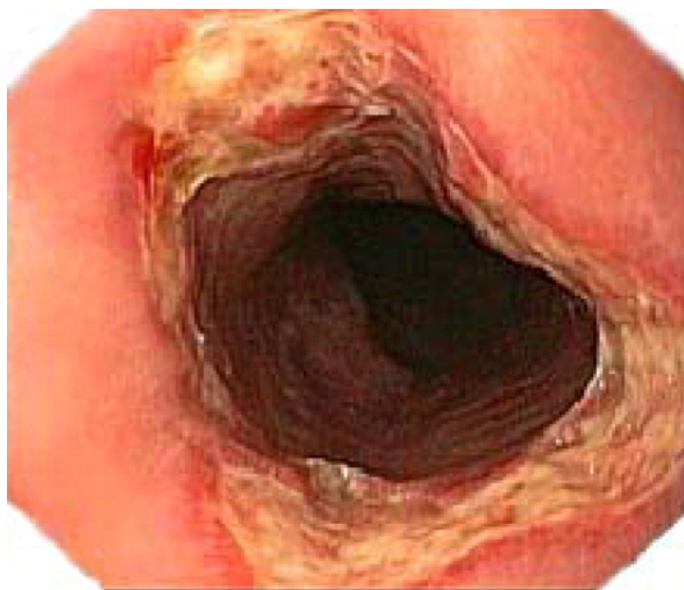
Case 12

Roongruedee Chaiteerakij, MD.

Panusit Petcharapirut, MD.

Rungsun Rerknimitr, MD.

A 20-year-old healthy male patient presented with acute odynophagia for 2 days. EGD showed the following image.



Upper endoscopy showed a large circumferential deep ulcer in mid-esophagus with relatively normal surrounding mucosa. The rest of esophageal examination was completely normal.

Histological examination revealed esophageal ulcer with diffuse infiltration of neutrophils and eosinophils in the mucosa. Neither organism nor malignant cells was found. In retrospect, we found that he had taken doxycycline for acne for 1 week. The diagnosis was **pill-induced esophagitis**. After being treated with local anesthetic agent and discontinuation of the drug, his symptom improved within a few days.

Differential diagnoses:

CMV esophagitis, HIV related esophagitis, herpes esophagitis, and deep fungi infection of the esophagus.

Discussion:

Drug-induced esophagitis is not uncommon in clinical practice, however, most cases are under-diagnosed or underreported resulting in lower than the exact incidence of this condition¹. More than 70 drugs causing esophageal injury have been reported including antibiotics, particularly tetracycline, doxycycline, and clindamycin. These account for over 60% of the cases². Other medications causing esophageal injury are non-steroidal anti-inflammatory drugs (NSAIDs), potassium chloride, ferrous sulphate, ascorbic acid, quinidine, aspirin, captopril, and bisphosphonates, etc².

Mechanisms for esophageal injury include concentration and pH of the chemicals, sustained or slow release formulation, method of delivery (tablet or gelatin capsule which becomes sticky during dissolution when taken with inadequate water or taken under recumbent position), size, and shape of the pills². Predisposing patient factors include habit of drug ingestion; decreased production of saliva due to old age, use of anticholinergic agents, connective tissue diseases; pre-existing esophageal dysmotility or abnormal anatomy and condition that prolonged esophageal transit time, particularly supine position^{2,3}.

Several mechanisms of injury are varied among different drugs², for example, doxycycline, tetracycline, ascorbic acid, and ferrous sulphate produce acidity when dissolved; clindamycin, potassium chloride, and quinidine have direct caustic effect to esophageal mucosa. Potassium chloride can also cause esophageal damage by producing local hyperosmolarity.

Typical scenario is young adults, with no prior history of esophageal symptoms, taking doxycycline for acne with little or no fluid before immediately going to bed followed by a sudden onset of odynophagia and retrosternal burning pain several hours later^{1,4,5}. However, the symptoms can manifest up to ten days after drug exposure⁶. Other presentations are hematemesis, melena, abdominal pain, and dysphagia or weight loss due to stricture formation^{2,7}.

The diagnostic test of choice is upper an upper endoscopy which can demonstrate a wide spectrum of findings; typical are discrete single or multiple ulcers with normal surrounding mucosa in the mid-esophagus, the most affected location (accounting for two-thirds of cases) resulting from anatomic narrowing due to external compression by aortic arch or left atrium^{1,5,7-9}. Other findings are mucosal edema, erythema, and superficial erosions. Differential diagnoses are herpes or cytomegalovirus-induced esophagitis that tend to have more wide-spread distribution and usually associated with immunocompromised status; candida esophagitis in which ulcers typically occur on a diffuse plaque background; reflux esophagitis in

which lesion is almost always occurs in the distal esophagus; and Crohn's disease that is usually accompanied with other evidence of Crohn's disease in the intestine.

Histological examination has no pathognomonic findings¹. The biopsy specimen usually reveals necroinflammation with nonspecific esophagitis^{1,8}.

This condition is self-limited. Therefore, no specific treatment is needed². Symptoms usually resolve within 2 to 6 days after discontinuation of the drugs⁵. Local anesthetic agent and antacids may help relieve the pain, however, the benefit of using these agents, including anti-secretory drugs and proton-pump inhibitors remain unclear^{1,6,8}.

The most important issue for critical management is making the correct diagnosis and cessation of the offending drug (s)^{1,5}. Patients should be emphasized to take the drugs with at least 200 cc of water in the upright position. Avoiding potentially injurious medication and changing to alternative agents if feasible are important measures to prevent drug-induced esophageal injury.

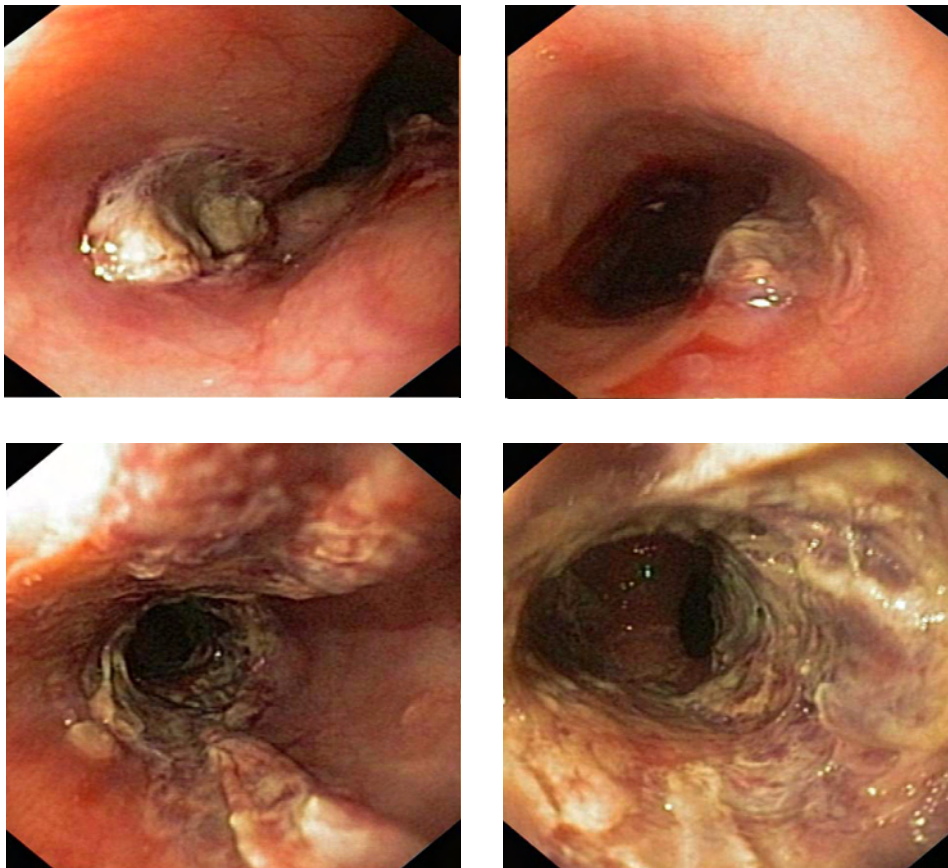
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Chatchai Kriengkirakul, MD.Rungsun Rerknimitr, MD.

A 35-year-old female with an underlying SLE who had been taking prednisolone 60 mg./day complained with headache for 1 month. She was admitted to receive her antifungal agent. During admission she developed passing melena per rectum and her hematocrit dropped from 27% to 21%. An EGD was done as shown.



Endoscopic findings:

Multiple esophageal ulcers extending from the upper esophagus downward to esophagogastric junction. The largest one at distal esophagus had circumferential thickening. All ulcers were deep, friable and

covered with thick yellow exudate without necrotic base. Biopsy from the esophageal ulcers was done. Pathology showed fibrinonecrotic tissue. Acute ulcer with the presence of atypical epithelial cell change, compatible with herpes esophagitis was detected.

Diagnosis:

Herpes esophagitis

Discussion:

Herpes esophagitis can occur as primary infection or reactivation of the latent virus (most common) of laryngeal, superior cervical and vagus nerves. It is associated with immunocompromised hosts more often than immunocompetent hosts. It can be found in all ages. Clinical presentations are severe odynophagia, heartburn, fever, nausea, vomiting, chest pain, bleeding, tracheoesophageal fistula. Differential diagnoses of infectious esophagitis in immunocompromised hosts are candida, viruses (HSV and CMV), mycobacteria, nocardia, other fungal infections (cryptococcosis, histoplasmosis, blastomycosis, and aspergillosis). Lesions are mostly found in the distal esophagus.^{1,2} Endoscopic findings of herpes esophagitis can be described in 3 stages.

- Early: vesicles, normal mucosa between the lesions
- Later: sharply demarcated ulcers with raised edges
- Late: necrotic foci, plaques, confluent ulcers

Histologic findings are multinucleated giant cells, with ground-glass nuclei and eosinophilic inclusions (Cowdry type A inclusion bodies) that occupied up to one-half of the nuclear volume.

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Chatchai Kriengkirakul, MD.

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Rungsun Rerknimitr, MD.

A 81-year-old male with an underlying non-small cell lung cancer stage IV has been treated with chemo therapy and steroid complained with progressive dysphagia for 1 month. An EGD was done as shown.



Endoscopic findings:

A large (3x2 cm.) oval shape ulcer with some necrotic base and exudate at the distal esophagus. There were multiple small shallow ulcers in the midesophagus.

Pathology demonstrated sparse distorted atypical cells with enlargement and intranuclear inclusion (white arrow). Patient's CMV-viral load by PCR was 2,010 copies/ml.

Diagnosis:

CMV esophagitis

Differential diagnoses:

Herpes esophageal ulcer, Pill induced ulcer, and HIV esophageal ulcer.

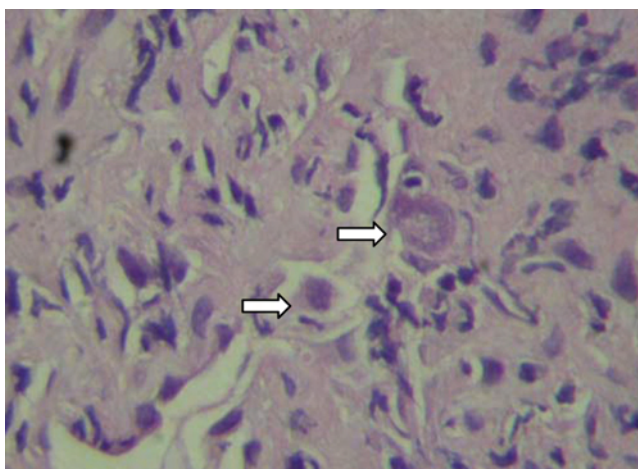
Discussion:

Risk factors of CMV infection in patients without AIDS are recipients of solid organ and bone marrow transplants and those who receive immunosuppressive medications. CMV infection can involve GI tract from the oropharynx to the anus. Majority of lesions occur in the large intestine and duodenum. It can present many symptoms including pain, ulceration, bleeding, diarrhea, and perforation.¹ Gastrointestinal ulceration is the most common presentation (70%) and gastrointestinal bleeding is the 2nd common presentation (58 in patients without AIDS). Gastrointestinal ulceration occurs from ischemic process, which can be described by narrowing of capillary lumens by swollen endothelial cells after CMV infection.²

Endoscopic features for alimentary tract are varies; inflamed mucosa alone (15%), ulceration alone (35%), inflammatory mucosa associated with ulcer (45%), submucosal tumor with ulcer (5%)³. The most common feature is multiple ulcers with at least one large ulcer³. The typical pathology of GI tract involvement by CMV in patients without AIDS is diffuse ulcerations and necrosis with scattered CMV inclusions.

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Phonthep Angsuwatcharakon, MD.

Rungsun Rerknimitr, MD.

A 53-year-old man with suicidal idea recently ingested a glass of household-bleaches containing hydrochloric acid. An EGD was done.

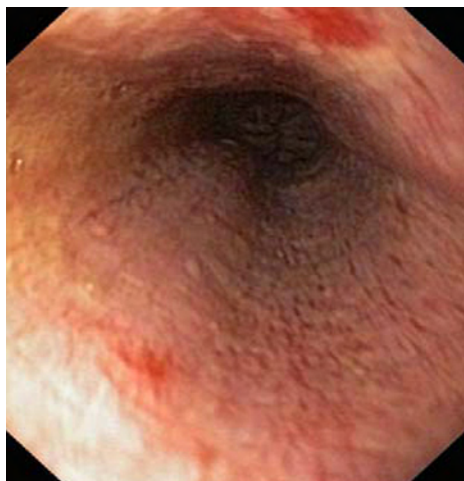


Figure A

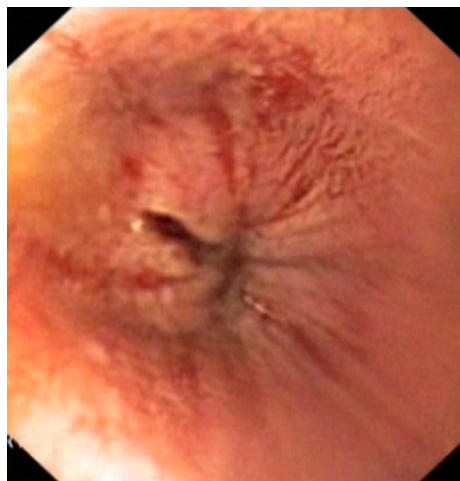


Figure B

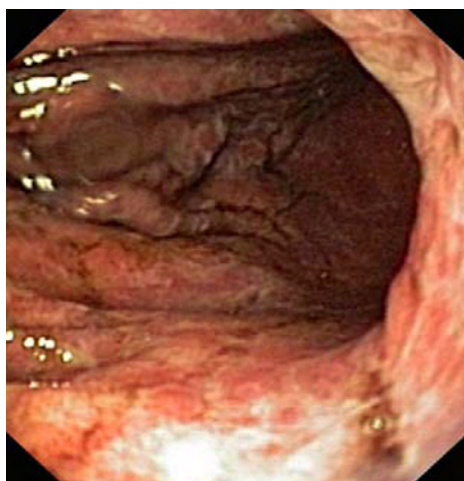


Figure C



Figure D

Endoscopy showed;

Figure A-B, endoscopic view of the esophagus: Circumferential whitish exudates along the esophagus, few small shallow ulcers were seen.

Figure C-D, endoscopic view of the stomach: Diffuse shallow ulcers with exudates in the gastric body. Erythematous and swollen mucosa were observed at the gastric antrum.

Diagnosis:

Corrosive esophagitis grade 2a

Discussion:

Ingestion of caustic agents can cause esophageal injury. Flexible gastroscopy with gentle insufflations is a safe procedure for grading the injuries. The preferred time for endoscopy is 12-24 hours after ingestion¹ and can be safe up to 96 hours². Contraindications for gastroscopy are third degree burn of hypopharynx¹ and clinical or radiographic suspicion of esophageal perforation². Endoscopy is usually avoided between 5-15 days of ingestion² since the tissue is soft, liquidified and easy to be perforated. Grading of esophageal injuries according to the degree of burns provides prognostic and therapeutic implications. With superficial injuries (grade 0-2a) patients can be discharged safely whereas with deeper injuries (grade 2b-3) patients requires an intensive care and good monitoring^{1,2}.

Table Grading of caustic esophageal injury²

Grade	Endoscopic findings	Stricture development (%)
0	Normal esophagus	0
1	Mucosal edema and hyperemia	0
2a	Friability, hemorrhage, erosions, blisters, whitish membranes, exudates and superficial ulcerations	0
2b	Deep or circumferential ulceration, in addition to 2a lesions	71.4
3a	Small and scattered area of necrosis	100
3b	Extensive necrosis	100

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Surachai Amornsawadwattana, MD.

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A 58-year-old male with dyspepsia underwent an EGD. Endoscopic findings are shown as figures.

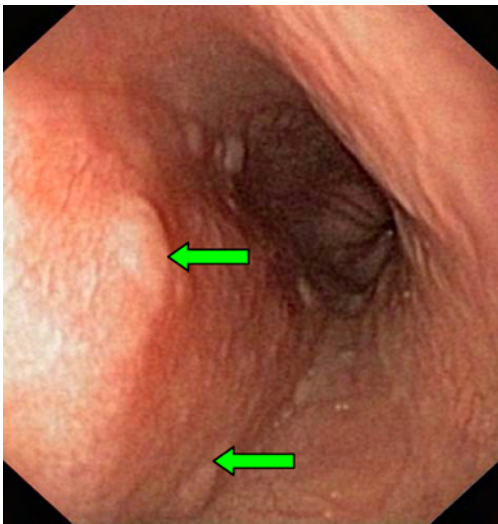


Figure A

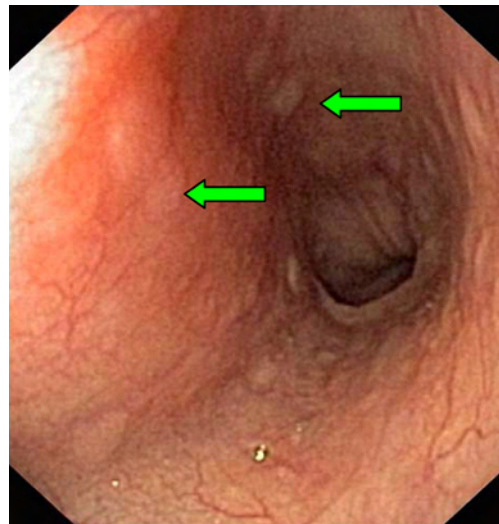


Figure B

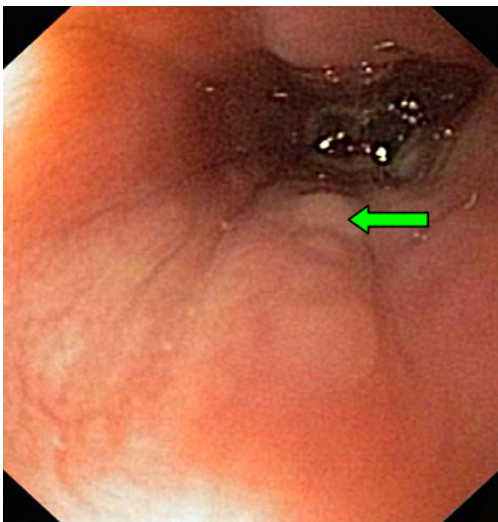


Figure C

Esophagoscopy revealed multiple, flat, grey white, oval shape lesions (green arrows) varying in size scattered in the distal esophagus. The diagnosis is **glycogenic acanthosis**.

Discussion:

Glycogenic acanthosis is a benign thickening of esophageal squamous epithelium of undefined etiology¹. It is commonly found in the fifth to sixth decades of life and becomes

larger and increases in number with advanced age^{1,2}. Endoscopic findings of glycogenic acanthosis are elevated grey white plaques that range in diameter from 1 to 15 mm. and located more prominent in the lower esophagus than in the upper esophagus². They can be found about 20-40% during routine endoscopic examinations². Thickening of large squamous epitheliums filled with glycogen is mainly found from histology². Extensive glycogenic acanthosis is associated with GERD, Cowden's syndrome and Celiac disease^{1,2}.

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A 28-year-old woman came to the hospital for the fourth time for an elective esophageal dilation after a diagnosis as corrosive agent induced stricture of mid-esophagus. A gradual stepwise esophageal dilation with increasing in size of Maloney dilator (42Fr, 44 Fr and 48 Fr) was done. The immediate esophagoscopy after dilation (Figure A) showed a linear deep mucosal tear with bleeding at 10 cm. from incisor. **iatrogenic intrathoracic esophageal perforation** was diagnosed.

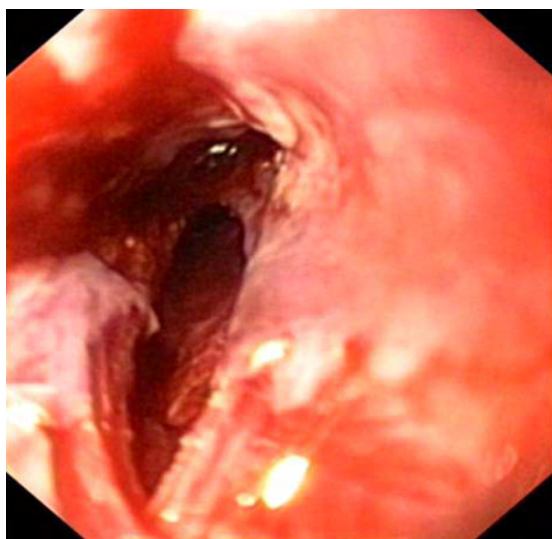


Figure A: Esophagoscopy showed linear deep mucosal tear with bleeding.

A Polyflex esophageal stent (Boston Scientific, Natick, MA) with 18-23 mm. in diameter and 150 mm. in length was inserted under fluoroscopy and conscious sedation. Her chest X-rays after procedure (Figure B) showed that the stent was placed at mid-esophagus and pneumomediastinum and subcutaneous emphysema were noted.

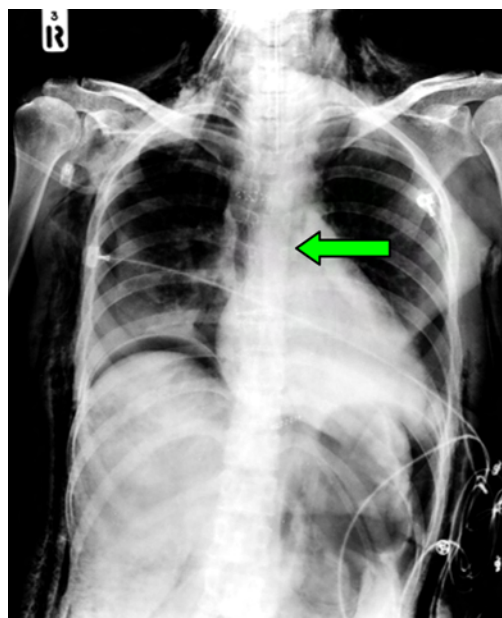


Figure B: Chest X-rays showed esophageal stent in place (green arrow), pneumomediastinum and subcutaneous emphysema.

At 3 months later, an esophagoscopy with removal of the stent revealed completely healed perforation (Figure C).

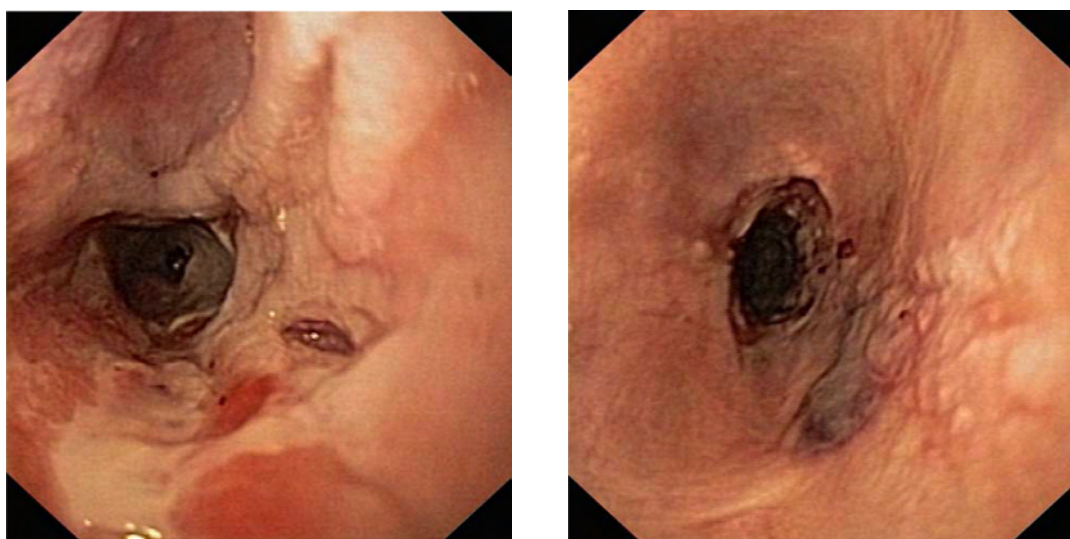


Figure C: Esophagoscopy after the stent removal showing healed ulcer with patent esophageal lumen

Discussion:

Esophageal perforation is a life threatening condition that requires rapid diagnosis and treatment. The mortality rate depends on the time of recognition and the cause of perforation. Although surgery plays a major role of treatment, morbidity rate is still significant. Anastomotic leakage can develop and additional

procedures are usually required¹. Esophageal stenting is another option that provides a better outcome and lower mortality rate in well selected conditions²⁻⁴. Failure to remove metal esophageal stent becomes a history when a plastic Polyflex stent arrives.

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