17TH GI LIVE

ENDOSCOPY DEMONSTRATION 2020

" ENDOMO : ENDOSCOPY AND MOTILITY INTERFACE "

24-26 February 2020

King Chulalongkorn Memorial Hospital Bangkok, Thailand

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Welcome Message

Dear colleagues,

In 2020, many things happened within the first and second months of the year including spread of the Coronavirus disease 2019 (COVID-19). Nevertheless, the 17th annual meeting of the Thai Association for Gastrointestinal Endoscopy (TAGE) is still going on. Theme of the conference in this year is a combination of endoscopy and motility. As part of activities of the conference, we have created this book which collected interesting cases and from our academic activities during 2019 and our colleagues. All cases have been selected and edited by several invited reviewers of the TAGE. I wish readers will learn some points from it. I would like to take this opportunity to thank the editor, all reviewers and faculties of TAGE for all years that we have worked hard together. Also, on behalf of TAGE, I would like to express our warm welcome to all participants of the 2020 TAGE annual meeting. I wish all readers and participants enjoy the book and meeting.

Warm Regards,

Pradermchai Kongkam, MD

President, the Thai Association for Gastrointestinal Endoscopy (TAGE)

February 2020

A Thousand and A Hundred Forty Peroral Endoscopic Myotomy for Esophageal Achalasia: 10 Years' Experience From A Single Tertiary Center

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Objective: Esophageal achalasia is most commonly treated with endoscopic dilation or laparoscopic Heller myotomy. Peroral endoscopic myotomy (POEM) has recently been described as a novel treatment for achalasia in humans. The aim of this study was to assess the clinical effectiveness and safety of POEM for treating esophageal achalasia performed in a single tertiary center within 10 years.

Methods: Between June 2010 to May 2019, POEM was performed in 1140 consecutive patients with achalasia. POEM procedure consisted of the following step: firstly, submucosal tunnel was created and extended below the lower esophageal sphincter (LES) onto the gastric cardia after a mucosal incision was made; then endoscopic myotomy of circular muscle bundles was done; finally, the mucosal entry was closed by hemostatic clips. The Eckardt score and manometry were used to evaluate the outcomes.

Results: POEM was successfully performed in 1115 of 1140 cases (97.8%). Mean procedure time was 42.1 min (range 33-86) and mean myotomy length was 9.5 cm (range 7-16). Mucosal perforations occurred in 23 (2%) patients during submucosal tunnel creation, major bleeding occurred in 15 (1.3%) patients, and 29 (2.5%) patients suffered pneumothorax immediate after procedure. All the complications were managed conservatively. During a mean follow-up period of 49.3 months (range 5-89 months), treatment success was achieved in 1005/1140 patients (88.2%). Mean LES pressure was 57.5 mmHg (29.2-83.1) and 15.6 mmHg (5.2-23.1) before and after the procedure (P = 0.000), respectively. Mean Eckardt score was 6.1 (4-11, median 6) and 0.6 (0-3, median 1) before and after POEM, respectively (P = 0.000). In a multivariate analysis, no independent predictors of treatment success was found. 215 patient (18.9%) developed mild reflux symptoms and required intermittent medication with proton pump inhibitors during the follow-up.

Conclusion: Our study demonstrated that POEM is a safe, and effective treatment for achalasia during a long-term follow-up. Further studies are warranted to compare the clinical outcomes of POEM with other treatment modalities.

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Rapid Drinking Challenge Evaluated by High-Resolution Esophageal Manometry in Upright Position Provides High Sensitivity and Specificity for Predicting Clinical Resolution After Treatment of Achalasia

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Objective: Rapid drinking challenge test (RDC) during high resolution esophageal manometry (HRM) in upright position is a simple but informative test to evaluate residual relaxation pressure at lower esophageal sphincter (LES) during the actual physiologic swallow. It has been shown to improve diagnostic yield of achalasia but its role in post-treatment evaluation is not clearly defined. We aimed to assess correlation between RDC and its role in predicting clinical response after treatment.

Methods: Consecutive patients with treatment-naïve achalasia completed GI symptoms questionnaires and underwent HRM and 100 ml RDC in upright position (Manoview device) at baseline and at one month after pneumatic dilation (PD) or peroral endoscopic myotomy (POEM). Mean residual LES relaxation pressure by using smart mouse tool during RDC and standard HRM metrics during 10 of 5 mL water swallows were analyzed. Upon 1-month follow-up, Eckardt score < 2 was considered an excellent response while the score of 2-3 was considered good response. ROC analysis was performed to compare performances of manometric parameters for predicting clinical response after treatment.

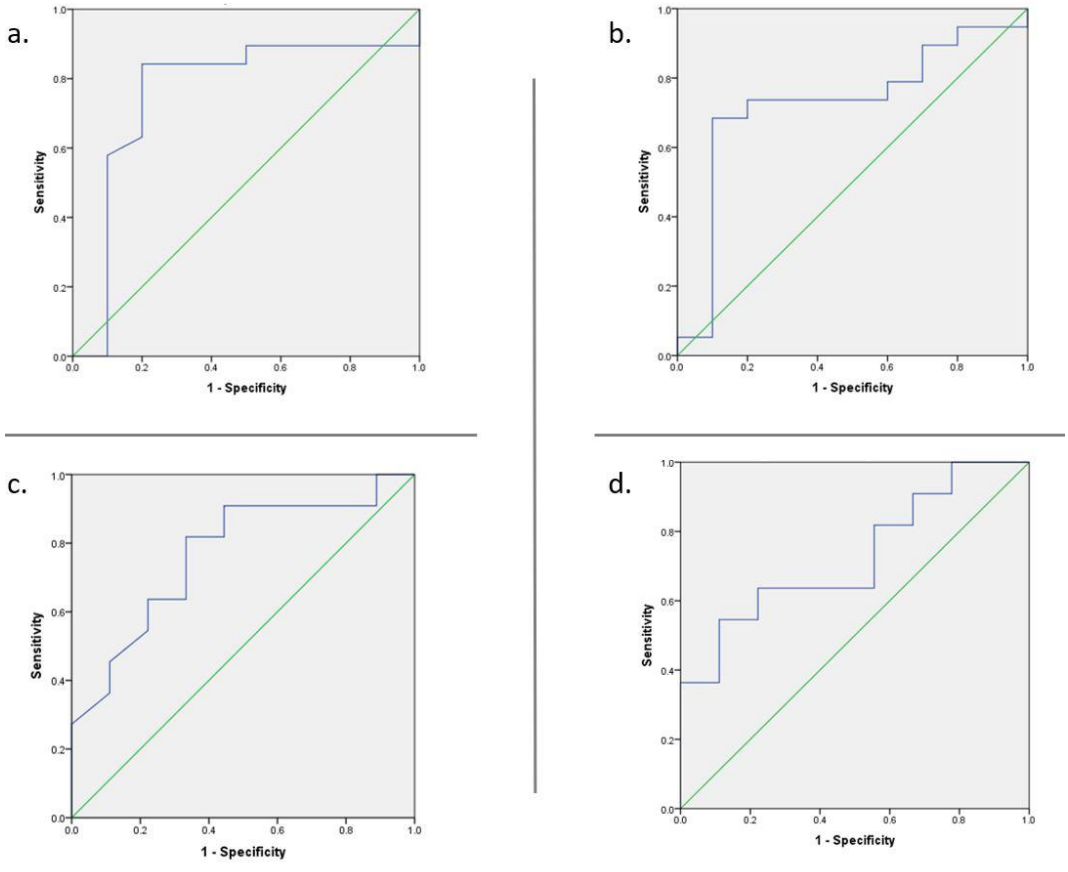
Results: 29 patients (21 females, age 48±15 years, median duration of symptoms 10 (4-12) months) were enrolled. Achalasia type II were the most common types [n=17 (58.6%), type I n=7 (24.1%), type III n=2 (6.9%), mixed II and III n=2 (6.9%), inconclusive n=1 (3.4%)] Pneumatic dilatation was performed in 23 (79.3%) patients [type I 5 (21.7%), type II 13 (56.5%)] and peroral endoscopic myotomy (POEM) in 6 (24.1%) patients [type I 2(28.6%), type II 4 (57.1%)]. At follow-up, % global symptom scores improved for 90% (80-100), Eckardt score significantly decreased [6 (5-7) to 1 (0-2.5), p<0.05] with dysphagia score (0-2) of 0 (0-0.5). 19 (65.5%) patients had excellent clinical symptoms improvement, while 5 (17.2%) had good clinical response. Patients with excellent clinical response had significantly lower post-procedural median integrated relaxation pressure (IRP) and residual LES relaxation pressure during RDC than those without excellent clinical response [7.9 (5-11.3) vs. 18.4 (13.7-23.9), and 15 (12.6-18.9) vs. 22 (16.2-26.3) mmHg, respectively, all p<0.05. LES relaxation pressure during RDC had similar AUROC for excellent response prediction [0.77 (95%CI 0.55-0.98) p=0.04] compared to median IRP during 10 wet swallows [0.76 (95% CI 0.55-0.97), p = 0.02] (Table1, Fig1). A cut-point residual LES relaxation pressure during RDC at 18.9 mmHg yielded highest sensitivity of 81.5% and specificity of 77.7%, positive likelihood ratio of 3.50 and negative likelihood ratio 0.24 for excellent response to PD/POEM.

Conclusion: Using HRM, LES relaxation pressure measurement during RDC provided high sensitivity and specificity in predicting excellent response after PD or POEM in patients with achalasia.

TABLE 1. Post- treatment high resolution esophageal manometry parameters comparing between achalasia patients with and without excellent clinical response.

	Excellent clinical response (Eckardt score <2) (n=19)	Non-excellent clinical response (Eckardt score >2) (10)	p-value
Manometric parameters			
Median IRP, mmHg	7.9 (5-11.3)	18.4 (13.7-23.9)	0.02
Percentage change of IRP, %	73.4 (33.6-81.3)	39.8 (-2.6-48.8)	0.05
Mean LES, mmHg	14.9 (11-18.2)	24.6 (15.8-27.8)	0.09
Percentage Change of LES, %	53.6 (15.0-78.9)	20.7 (-14.9-51.5)	0.11
Rapid Drinking Challenge phase			
LES relaxation pressure, mmHg	15 (12.6-18.8)	22 (16.2-26.3)	0.04
Integrated intrabolus pressure, mmHg	410 (87-1464)	1163 (710-4865)	0.09

IRP= integrated relaxation pressure; LES= lower esophageal sphincter
Data expressed as median (interquartile range)



1-month post procedure manometric parameters	AUROC (95% CI)	p-value
a. Median IRP	0.76 (95% CI 0.55-0.97)	0.02
b. Median IRP percentage change	0.73 (95% CI 0.52-0.93)	0.05
c. IES relaxation pressure in RDC	0.77 (95% CI 0.55-0.98)	0.04
d. Integrated intrabolus pressure in RDC	0.73 (95% CI 0.50-0.95)	0.09

Fig 1. AUROC of 1-month post-treatment manometric parameters for predicting excellent clinical response

The Efficacy of Cap-assisted Colonoscopy as Compared to Conventional in a Pediatric Population: A Randomized Controlled Trial

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Background: The use of cap-assisted colonoscopy (CAC) in adults reportedly shortens cecal and ileal intubation times (CIT and IIT, respectively) and improves cecal and ileal intubation rates (CIR and IIR, respectively) than standard colonoscopy (SC). However, no study to date has assessed the efficacy of CAC in children.

Materials and Methods: In total 44 children were randomized to CAC (n = 22) or SC (n = 22) and quality indicators were evaluated.

Results: The median ages of the CAC and SC groups were 9.7 years (range, 4.3–16.0) and 9.7 years (range, 3.9–13.5), respectively. The most common indication was suspected hematochezia (38.6%). The median CIT in the CAC and SC groups were 15 (range, 8–19) and 13.7 (range, 10–18) minutes, respectively ($p = 0.77$). The IIT in the CAC and SC group were 60 (range, 55–95) and 59 (range, 35–95) seconds, respectively ($p = 0.42$). The overall CIR was 100% and did not differ between groups. The IIR of the CAC and SC groups were 100% and 95.5%, respectively ($p > 0.99$). Good CIR and IIR were achieved and no complications occurred in either group.

Conclusion: There were no intergroup differences in quality indicators.

Acute Mesenteric Ischemia (AMI) as Initial Presentation of Noncirrhotic Portal Hypertension (NCPH): A Case Series

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INTRODUCTION

Worldwide, cirrhosis is the most common underlying reason for increased portal pressure and only less than 10% have noncirrhotic liver.[1] The underlying mechanism and its natural history have not been fully established. One of the causes of NCPH is chronic portal vein thrombosis (PVT) and complications include intestinal ischemia if the superior mesenteric vein is also involved.[3] Management is directed towards the underlying reason for the hypercoagulable state and will therefore need diagnostic work up. Thrombophilia may be secondary to various acquired and hereditary conditions. Acquired thrombophilia may be secondary to malignancy, pregnancy, surgery, or medications such as use of contraceptive and hormonal replacement therapy. Deficiencies in Protein C, Protein S, and antithrombin (AT) can also lead to thrombophilia.[4] Prevalence of hereditary thrombophilia in the general population varies from 0.2-0.4% for Protein C & Protein S deficiency, and 0.02% for AT deficiency.[5] Unfortunately, findings of mild splenomegaly associated with a normal liver may not trigger investigative efforts on its underlying cause.

CLINICAL PRESENTATION

Case 1 of 2

A year prior to present admission, a 30-year old female experienced progressive abdominal pain. She was diagnosed with acute abdomen and splenomegaly. She underwent exploratory laparotomy with intraoperative findings of necrotic segment of small intestines for which a segmental resection was done. Postoperatively, she took rivaroxaban but discontinued after 6 months because she felt well. No work up done on the underlying cause of mesenteric thrombosis and splenomegaly. She was asymptomatic in the interim until ten days prior to admission, she had diarrhea, fever, and abdominal cramps. These were later associated with bloatedness & early satiety hence admitted in a local hospital where piperacillin-tazobactam and metronidazole. While admitted, there was note of abdominal enlargement due to ascites. Paracentesis was done and drained two liters of ascitic fluid. No ascitic fluid studies done. Despite four days of antibiotics, fever and diarrhea persisted, prompting transfer to our institution. She has no other pertinent conditions in her medical history. She was nulligravid and previously on oral cyproterone acetate + ethinylestradiol & etonogestrel subdermal implant. Family history was negative for disorders of hypercoagulability. Upon admission, she was awake, oriented, tachycardic & febrile (38.1°C). Abdomen was distended, with normoactive bowel sounds, soft, nontender, with shifting dullness & obliterated Traube's space. No stigmata of liver disease.

Diagnostic Work-Up and Management

Portal Hypertension: Gastroscopy showed esophageal & gastric varices without signs of recent bleed and portal gastropathy (Fig 1). Serum albumin-ascitic fluid gradient was 20 g/L. Patient was maintained on propranolol & spironolactone.

Portal Vein Thrombosis (PVT): Whole abdomen CT scan with IV contrast showed PVT (Fig 2). The liver was normal. Work up for hereditary thrombophilia showed low Protein C, Protein S & AT blood levels. Rivaroxaban was started.

Pylephlebitis: Diagnostics showed leukocytosis with neutrophilic predominance. No active infiltrates on chest xray. Cultures of blood, urine, & stool were negative. Ascitic fluid had negative culture & MTB-PCR. There were no signs of malignancy on CT scan. Until the 12th hospital day, patient was still febrile but diarrhea & bloatedness

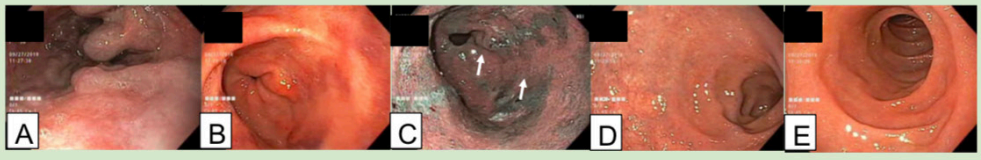

Figure 1. Gastroscopy pictures of Case 1
(A) Esophageal varices Grade II without signs of recent bleeding **(B)** The stomach showed portal gastropathy and gastric varices in the antrum were more evident under narrow band imaging (*white arrows*) **(C).** The duodenal bulb mucosa **(D)** was pale-looking with a violaceous hue in contrast to the normal pinkish mucosa of the second part of the duodenum **(E)**

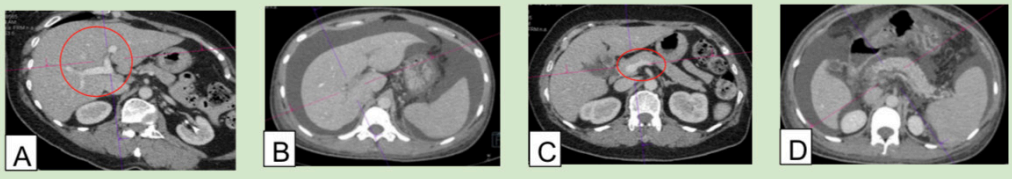

Figure 2. Axial view of the liver on CT scan with IV contrast of Case 1
(A) Image showing a normal opacification of the intrahepatic PV (within red circle) indicative of patency
(B) non-opacification of the intrahepatic PV in our patient.
(C) Image showing normal opacification of the splenoportal confluence (within red circle)
(D) non-opacification of the splenoportal confluence in our patient.

had resolved. Pylephlebitis was entertained after negative extensive search for focus of infection. Piperacillin-tazobactam completed 14 days & metronidazole for 10 days. Patient was afebrile for 2 days upon discharge.

Case 2 of 2

Four years prior to admission, an obese 24-year old male presented with severe abdominal pain and underwent exploratory laparotomy and resection of 143-cm long jejunum-ileum segment due to acute mesenteric ischemia secondary to mesenteric vein thrombosis. Work up showed Protein C and Protein S deficiency. Warfarin was started overlapped with low molecular weight heparin on the first five days. He was on warfarin for one year and shifted to rivaroxaban due to patient's preference. However, patient discontinued rivaroxaban intake because he was asymptomatic & was lost to follow up. Two years after discontinuation of anticoagulants, he was readmitted for bloatedness, vomiting and melena. Family history was pertinent only to an uninvestigated non-healing leg ulcer for both father and paternal grandfather. On admission, he was awake, alert, and oriented. Vital signs were normal. The abdomen was flat, with note of midline post-operative scar, with normoactive bowel sounds, soft, and nontender. The liver was not enlarged and the Traube's space was obliterated. No stigmata of liver disease. There were tarry stools on digital rectal exam.

Diagnostic Work-Up and Management

Initial hemoglobin level was 90 g/L, and platelet count was 77,000/mm^3. Liver functions tests were normal. Gastroscopy findings (Fig 3) include four large esophageal varices which do not collapse on insufflation and with red wales and cherry red spots from proximal to distal esophagus. There was portal gastropathy and a gastric varix in the cardia. Rubber band ligation of the esophageal varices was done. Whole abdomen CT scan with IV contrast (Fig 4) showed normal liver, splenomegaly, portovenous collaterals, thrombosis of the intrahepatic portal vein, splenoportal confluence, and superior mesenteric vein & minimal free fluid collections. Liver elastography revealed a noncirrhotic liver (F=0). Repeat Protein C and Protein S were still low four years after the episode of acute mesenteric ischemia. Patient was maintained on rivaroxaban and propranolol.

DISCUSSION

Both patients were apparently well until they presented with acute abdomen without any warning signs. At the time they underwent surgery for AMI, the only clue for having portal hypertension was splenomegaly. Both of them were doing well after surgery while on anticoagulant, but both also self-discontinued its intake and were lost to follow up because they were asymptomatic. Months after, both returned to the emergency room presenting with complications of NCPH.

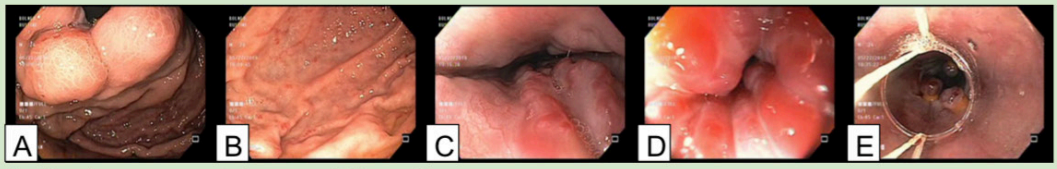

Figure 3. Gastroscopy pictures of Case 2
(A) Gastric varix in the cardia **(B)** Portal gastropathy **(C)** and **(D)** esophageal varices with red wales and cherry red spots **(E)** after rubber band ligation

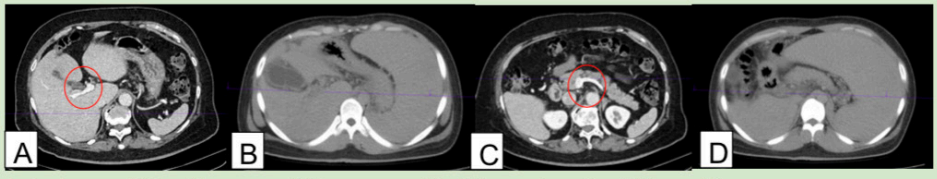

Figure 4. Axial view of the liver on CT scan with IV contrast of Case 2
(A) Image showing a normal opacification of the intrahepatic PV (within red circle) indicative of patency
(B) non-opacification of the intrahepatic PV in our patient
(C) Image showing normal opacification of the splenoportal confluence (within red circle)
(D) non-opacification of the splenoportal confluence in our patient

The most common initial presentation of NCPH is variceal bleeding. AMI is a known possible and morbid complication of NCPH secondary to chronic PVT, but rarely reported as initial presentation. Splenomegaly has been reported to be present in 95% of NCPH patients[2] and can be a clue that chronic PVT has been present. Although variceal bleeding in these noncirrhotic patients are reportedly more manageable compared to cirrhotic patients[7] with deranged liver function, variceal bleeding should still not be taken lightly. Unlike among cirrhotic patients where there has been extensive study on its management, there are no controlled studies on the prevention and management of gastrointestinal bleeding in noncirrhotic patients with chronic PVT. Hence, they are managed similarly with cirrhotic patients.[6] Anticoagulation has proved to be beneficial in patients with AMI from mesenteric vein thrombosis. If the associated risks of discontinuing anticoagulation, & need for regular follow up were emphasized, complications of chronic PVT might have been prevented. Chronic PVT may also be complicated by pylephlebitis and sepsis requiring about 4 weeks of antibiotic treatment8. In addition, the more worrisome event in these patients is recurrent thrombosis which may be fatal. Hypervigilance in patients with acquired risk factors for thrombophilia (eg, use of contraceptive drugs, obesity) on top of rare hereditary prothrombotic conditions is mandatory. Screening for hereditary thrombophilia is prudent before starting a patient on oral contraception.

CONCLUSION

AMI is an uncommon initial presentation of NCPH. It brings high morbidity and can be fatal without warning signs. Incidental finding of splenomegaly especially if associated with thrombosis should prompt thorough investigation on the underlying cause and see if complications have developed at the outset. In patients contemplated for contraception, its risk for thrombosis should be discussed especially among those who may have hereditary thrombophilia. NCPH secondary to chronic PVT can potentially have a morbid course and may require long-term anticoagulation. Due to the poorly defined natural history of NCPH, regular follow up on the status of portal hypertension and liver function are recommended. Risk for thrombus formation should be part of discussion and patient's understanding of his condition should be ensured especially among young patients who may take treatment and regular follow up lightly.

Keywords: case series, acute mesenteric ischemia, noncirrhotic portal hypertension, portal vein thrombosis, protein c and protein s deficiency

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The Effects of Continuous Positive Airway Pressure (CPAP) Treatment on Gastrointestinal Reflux (GER) Symptoms in Patients with Obstructive Sleep Apnea (OSA)

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Objective: OSA has been reported to be associated with nocturnal gastroesophageal reflux and CPAP treatment could decrease reflux events and esophageal acid contact time. We aimed to examine the effect of CPAP treatment on GER symptoms in patients with OSA and GERD.

Methods: We prospectively recruited 26 patients diagnosed OSA by polysomnography and had bothersome typical reflux symptoms (acid/food regurgitation or heartburn) for at least 3 months before the enrollment. 11 patients (42.3%) were prescribed CPAP and the other did not receive CPAP due to financial problems or denial of usage. The patients were initially interviewed regarding GI symptoms using a validated modified Rome III questionnaire. Gastrointestinal symptoms during the last 7 days were obtained by using questionnaires at baseline and follow up to compare the effects of CPAP and non-CPAP usage on GERD and other GI symptoms for a median of 7 (3-11.75) months in CPAP group and 14(13-18) months in non-CPAP group. Patients were allowed to receive GERD treatment as usual.

Results: Baseline characteristics were similar between the patients with and without CPAP use (Table 1). Most patients had severe OSA with similar baseline apnea-hypopnea index (AHI) (CPAP vs. no-CPAP: 50.9 ± 22.6 vs. 53.7 ± 17.4, p=0.75). Patients in CPAP arm had adequate usage (315±69 minutes/day and 85.4±11% used days) and they had normalized AHI after treatment (4.5±3.2). Only 1/15(6.7%) and 3/11(27.3%) patients with no-CPAP and CPAP usage had nocturnal reflux symptoms at baseline. Patients with CPAP usage significantly had higher rate of heartburn improvement than those without CPAP usage (7/7(100) vs. 3/6(50%), p = 0.03 (Fig1)). Rate of acid/food regurgitation improvement was not significantly different between patients with and without CPAP usage [4/7(57.1%) vs. 10/12((83.3%), p=0.31). (Fig 2) At the end of follow-up, global symptom severity score, rate of worsened bloating and belching and rate of proton-pump inhibitors (PPI) usage were not significantly different between groups (p>0.05). (Table 1) Multivariate analysis showed that adequate CPAP usage was independently associated with heartburn symptoms improvement after adjusted for gender, PPI usage and BMI with odds ratio= 14.4, 95%CI 1.0-205.5, p=0.05.

Conclusion: Adequate CPAP treatment significantly reduced heartburn but not regurgitation symptoms in OSA patients with predominant daytime typical GER symptoms. This study suggests that CPAP treatment might improve daytime heartburn symptoms by modulation of visceral hypersensitivity but not number of refluxes in GERD patients.

TABLE 1. Baseline and follow-up clinical profiles comparing between OSA patients with and without CPAP.

	OSA without CPAP usage (n=15)	OSA with CPAP usage (n=11)	p-value
Baseline data			
Age (year)	55.3 ± 13.5	55.5 ± 15.9	0.80
Gender (Male)	8 (53.3%)	5 (45.5%)	0.69
BMI (Kg/m^2)	27.4 ± 4.0	29.48 ± 5.3	0.28
Global GI symptoms score (0-10)	2 (1-4)	3.5 (2-5.5)	0.28
Typical reflux symptoms			
− Acid/food regurgitation, n (%)	12 (80%)	7 (63.6%)	0.41
− Heartburn, n (%)	6 (40.0%)	7 (63.6%)	0.23
Sleep parameters			
− AHI	50.9 ± 22.6	53.7 ± 17.4	0.75
− RDI	516 ± 24.6	54.2 ± 17.2	0.77
PPI usage, n (%)	3 (20%)	2 (18.2%)	1
Follow-up data			
Follow-up duration (months)	14 (13-18)	7(3-11.75)	
Global GI Symptoms score (0-10)	1 (1-7)	5(2-7)	0.10
Typical reflux symptoms			
− Acid/Food Regurgitation, n (%)	2 (16.7%)	3 (42.9%)	0.31
− Heartburn, n (%)	3 (50%)	1 (14.3%)	0.27
Acid/Food regurgitation Improvement, n (%)			0.31
− Improved	10 (83.3%)	4 (57.1%)	
− Not improved	2 (16.7%)	3 (42.9%)	
Heartburn Improvement, n (%)			**0.03**
− Improved	**3 (50%)**	**7 (100%)**	
− Not improved	3 (50%)	0 (0%)	
Worsened Bloating, n (%)	2 (13.3%)	1 (9.1%)	1
Worsened Belching, n (%)	1 (6.7%)	3 (27.3%)	0.28
PPI usage, n (%)	3 (20%)	2 (18.2%)	0.9
BMI (Kg/m^2)	28.0 ± 4.0	28.8 ± 4.9	0.72

AHI= Apnea–Hypopnea Index, RDI= Respiratory disturbance index
Data expressed as mean ± SD or median (interquartile range).

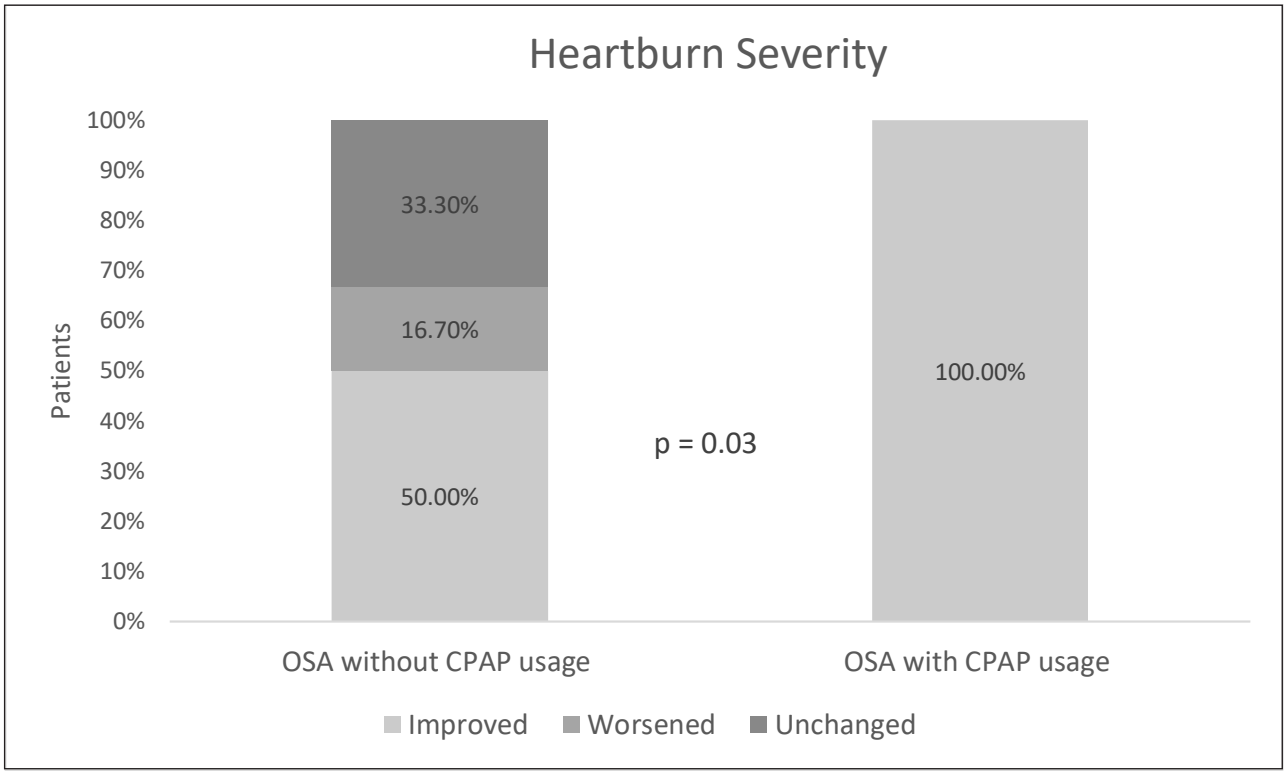

Fig 1. Follow-up heartburn severity changes in patients with OSA with and without CPAP treatment

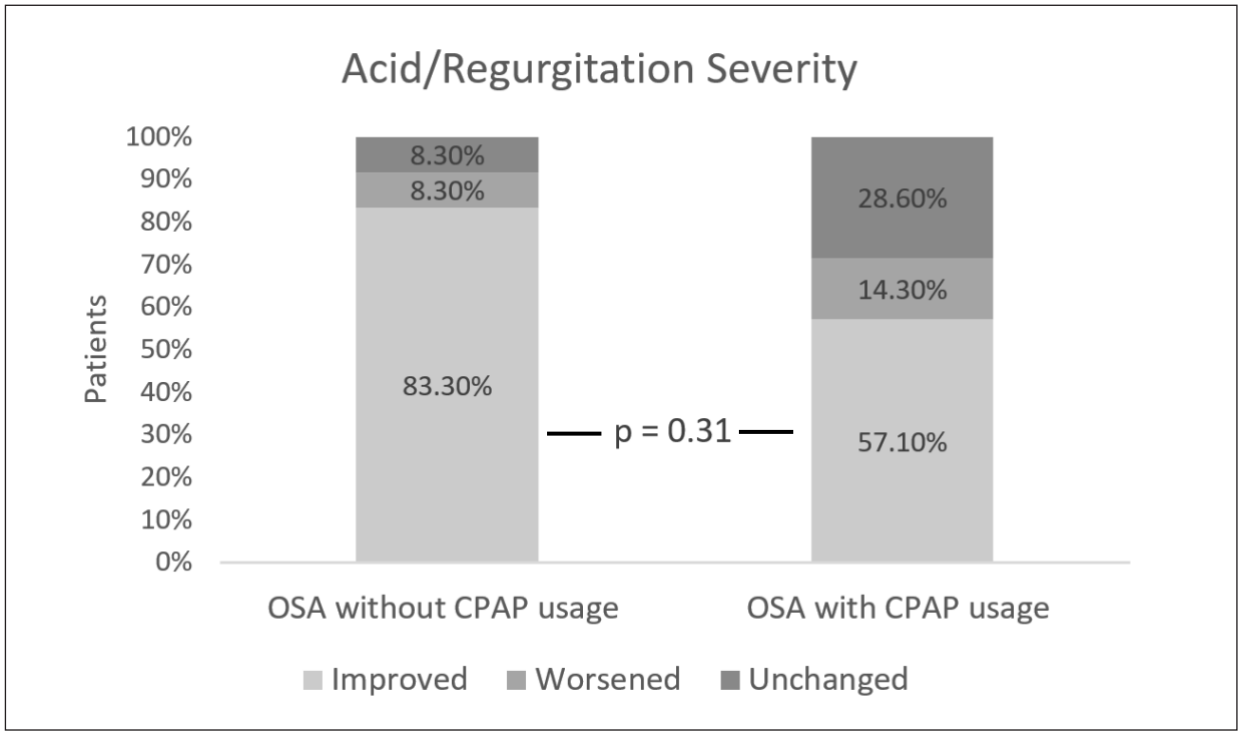

Fig 2. Follow-up acid/regurgitation severity changes in patients with OSA with and without CPAP treatment

Endoscopic Treatment for Colonic Obstruction Caused by Calcium Polystyrene Sulfonate (Kalimate®) Bezoar

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A case of 77 years old lady presented with severe pneumonia with acute respiratory distress syndrome was admitted to the ICU and received mechanical ventilation. Acute kidney injury with oliguria and hyperkalemia, with serum potassium varied from 4.95 – 5.39 mg/dL was noted. For the treatment of hyperkalemia, 14 repeated doses of oral Calcium Polystyrene Sulfonate (CPS) 30 gram (totally 420 gram) mixed with water was administered through the nasogastric tube in 7 days. During that period, she did not have any bowel movement despite receiving lactulose and milk of magnesia. Later, she developed generalized bowel dilation, feeding intolerance, and obstipation.

Plain abdominal radiography and CT scan were performed which showed long segment of CPS impaction from terminal ileum to descending colon causing small bowel obstruction was seen (Fig1A, 1B). Preserved intestinal mucosal enhancement was noted. Multiple doses of oral polyethylene glycol and rectal saline irrigation as well as intravenous metoclopramide were administered. Surgical consultation was done but conservative treatment was suggested. After two days of failed conservative treatment, colonoscopy was decided.

We performed colonoscopy using PCF 160AL (Olympus Medical, Tokyo, Japan) with CO_2 insufflation up to the hepatic flexure. The colonic mucosa was normal and large amount of impacted CPS bezoar was seen from descending colon. Five liters of saline irrigation showed dissolving surface of CPS bezoar and Pentapod grasping forceps (Olympus, Tokyo, Japan) was used for Fragmentation of the CPS bezoar (Fig 1C, 1D). After the procedure, the patient could able to pass large amount of CPS admixed with fecal content. Her abdominal distension improved and follow up abdominal radiography and CT scan without contrast showed improvement of CPS impaction (Fig 2).

Calcium Polystyrene Sulfonate is a Cation exchange resin that have been use for the treatment of hyperkalemia. When compared to Sodium Polystyrene Sulfonate, less incidence of side effect related to CPS such as ulceration, intestinal necrosis or obstruction have been reported (Table 1). Cautions should be made in patient with poor intestinal motility such as in critical care setting.

In summary, we report a case of CPS impaction, which is not a common condition. In case of failed conservative treatment, careful colonoscopy with irrigation might be a feasible treatment option for patient in absence of intestinal necrosis or perforation.

TABLE 1. Summary of case reports of Calcium Polystyrene Sulfonate induced gastrointestinal complication.

Author	Journal	Patient setting	Route of administration/ solvent	Dose of CPS	Type of complication	Management
Shioya	J Nippon Med Sch. 2007;74(5): 359-63.	77 year old woman with Diabetic ketoacidosis	Oral/ Sorbitol	15 mg, duration not mentioned	Ulcer with fistula	Surgery
Joo	J Korean Med Sci. 2009;24(6): 1207-11.	34 year old male with renal failure and intracerebral hemorrhage	Oral and rectal/ 20% dextrose water	60 gm enema over 2 days. 45 gm oral for 3 days	Colonic ischemia	Conservative
Gouturbe	Ann Pharmacother. 2011;45(2):e13.	73 years old man with colonic pseudoobstruction	Oral	60 gm over 4 days	Intestinal ischemia	Exploratory laparotomy
Tongyoo	J Med Assoc Thai. 013;96(12): 1617-20.	52 year old man admitted to ICU after surgical debridement of soft tissue infection	Oral/ water	360 gm in 5 days	Intestinal obstruction	Exploratory laparotomy
Castillo-Cejas	Rev Esp Enferm Dig. 2013;105(4): 232-4.	73 year old male with end-stage renal disease receiving hemodialysis	Oral/ water	Not mentioned	Colonic ischemia	Conservative
Lai	Endoscopy. 2013;45 Suppl 2 UCTN:E378-9.	86 years old male with renal failure after receiving angiotherapy for hepatocellular carcinoma	Oral/ Not mentioned		Intestinal obstruction	Enteroscopy with fragmentation

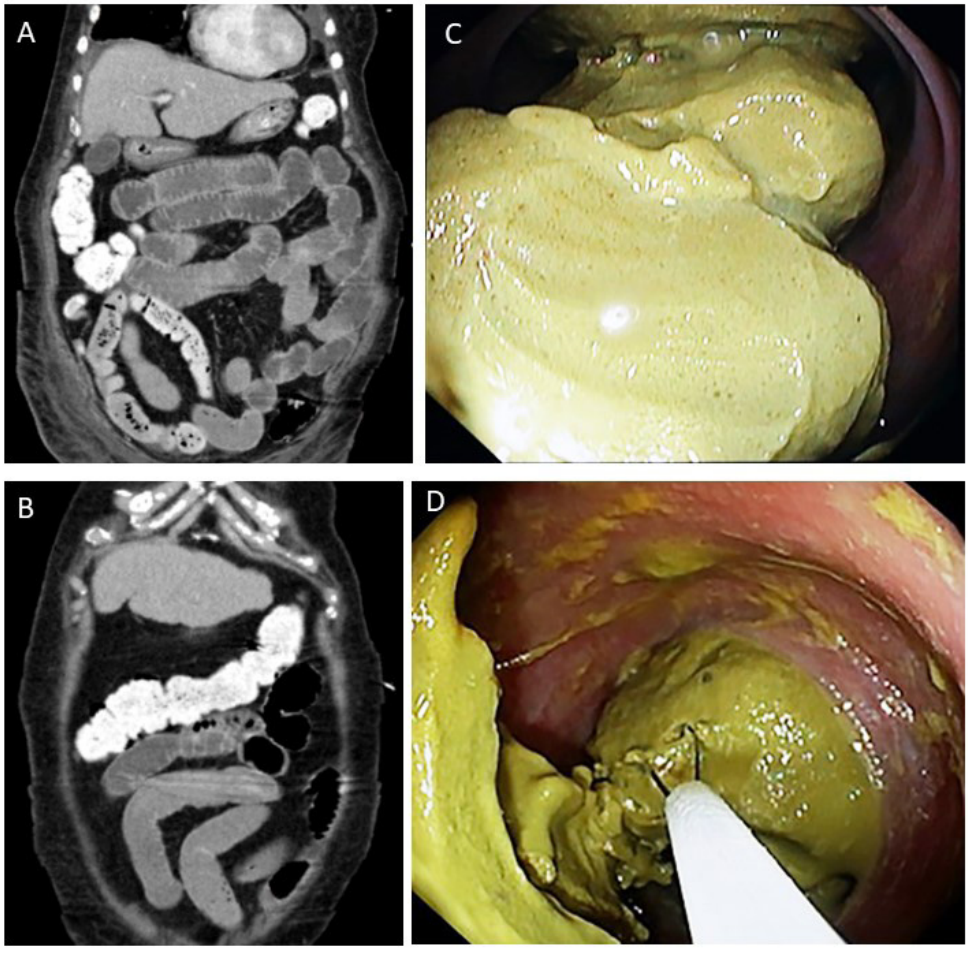

Fig 1. Computed tomography showed impacted Calcium Polystyrene Sulfonate in the colon causing small bowel dilation (A), (B). Colonoscopy was done and the bezoar was fragmented using the Pentapod grasping forceps and saline irrigation (C), (D).

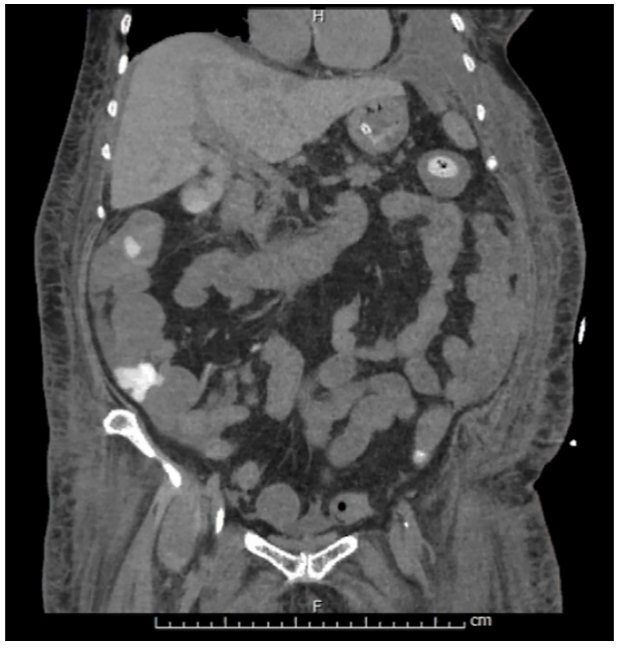

Fig 2. Computed tomography after the procedure showed improved small bowel dilation and only few amount of CPS remained in the colon.

Entero-Enteric, Entero-Colic, and Colo-Colic Fistulas in Moderately Active Crohn's Disease (CD) Treated with Vedolizumab: A Case Report

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Objective: We present a case of a moderately active Crohn's Disease (CD) with multiple internal fistulas treated with Vedolizumab, a gut-selective α4β7 integrin antagonist, a promising treatment option for fistulizing CD.

Clinical Presentation: The patient was a 32-year old male with CD for seventeen years. He presented with recurrent episodes of hematochezia, diarrhea, and abdominal pain. During the course of his disease, he was treated with mesalazine, corticosteroids, and azathioprine. Despite these, he was recurrently symptomatic and frequently hospitalized.

Diagnostic Work up and Management: While on mesalazine and azathioprine, patient had persistent right-sided abdominal pain without diarrhea. Colonoscopy showed skip areas of edematous, erythematous, and friable mucosa and pseudopolyps (Fig 1), with some difficulty advancing the scope. At 80cm level from the anal verge, there was a black, elongated and reflective material sliding as the scope was advanced or withdrawn (Fig 2). At this point, the endoscopist realized he had entered a colo-colic fistula and visualized the colonoscope shaft.

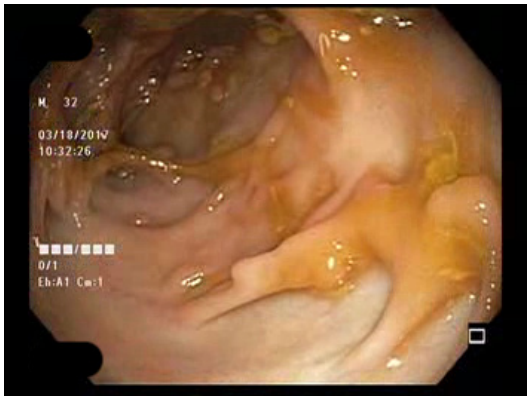

Fig 1. Colonic mucosa with edematous mucosa and multiple pseudopolyps

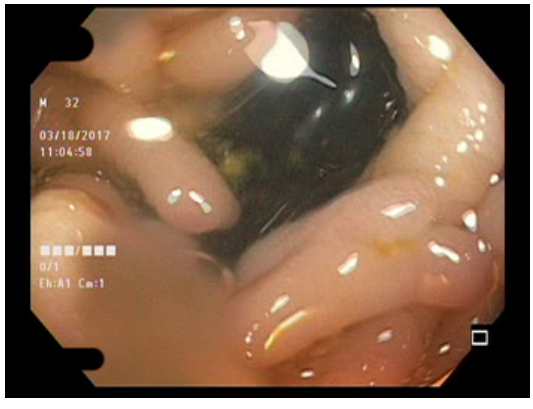

Fig 2. Colonoscope shaft through a colo-colic fistula

On withdrawal, two lumens were noted, one leading to the ileum and the other was a fistula leading to a colonic segment at 80 cm level. Histopathology revealed crypt distortion, neutrophilic infiltrates, and dilated and atrophic glands. Abdominal CT scan with triple contrast showed colo-colic, entero-enteric, and entero-colic fistulas involving the cecum, rectosigmoid, and ileum. Vedolizumab treatment given and completed 52 weeks course. Previous medications discontinued. His baseline Crohn's Disease Activity Index (CDAI) score of 390 (moderately active) improved to 146 (asymptomatic) after completion of vedolizumab, with improvement in quality of life and no hospitalizations. Repeat CT scan showed the same internal fistulas. Since completion of vedolizumab treatment, he remained in remission for 10 months as of this writing.

Conclusion: The treatment of fistulizing CD remains challenging. Vedolizumab is a promising treatment for such case. It can improve bothersome symptoms and patient's quality of life. However, its use in developing countries may be limited by its cost of treatment.

Esophageal Intramural Pseudo-diverticulosis as a Rare Cause of Hematemesis in a Patient with Alcoholic Cirrhosis

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A 52-year old man with history of alcoholic cirrhosis and alcohol dependence presented with hematemesis for six hours. His blood pressure was 82/44 mmHg. Laboratory showed anemia with hemoglobin of 5.0 g/dL (baselined hemoglobin 9.4 g/dL). EGD revealed large amount of blood clot in esophagus. After blood clot was removed, multiple small esophageal intramural pseudo-diverticulum was seen. One active bleeding from pseudo-diverticulum was seen at 22 cm from incisor. Three hemoclips were performed and hemostasis was achieved.

During a repeat endoscopy for reevaluation, biopsy of esophageal mucosa was done. The histopathological report showed benign stratified squamous epithelium with basal cell hyperplasia. Scattered neutrophils and lymphocytes in the epithelium were noted. No viral inclusion or fungus was observed.

DISCUSSION

Esophageal intramural pseudo-diverticulosis (EIP) is a rare benign esophageal disease with a bimodal peak pattern incidence during adolescence and the fifth to sixth decades of life. Co-morbidities associated with EIP include diabetes mellites, reflux esophagitis, chronic alcohol abuse, and corrosive esophageal injury. The most common presentation is intermittent or progressive dysphagia. Radiologic findings may reveal concentric thickening of esophageal wall and intramural air-filled vesicles on CT scan or variable-sized flask-shaped outpouching defects on barium esophagogram. The endoscopic findings of EIP include multiple small 2-4 mm flask-shaped esophageal diverticulum. The distributions of the outpouching are mainly diffuse in up to 60% but can be segmental with distribution in upper, middle, and lower esophagus in 14%,14% and 12%, respectively. Esophageal stricture can be commonly found with EIP in up to 70-96%. Dilated submucosal gland with inflammatory cells around the opening and submucosal fibrosis are observed on histological findings. The cornerstone of EIP management are treatment of its complications while controlling the underlying diseases.

EIP-related bleeding is a rare complication with only two previously published case reports. Our case demonstrated a severe spectrum of this rare entity where

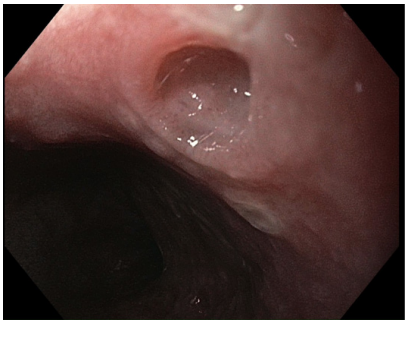
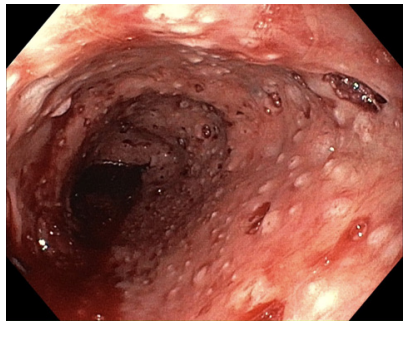


Fig 1. EGD performed 6 months ago revealed esophageal intramural pseudo-diverticulum



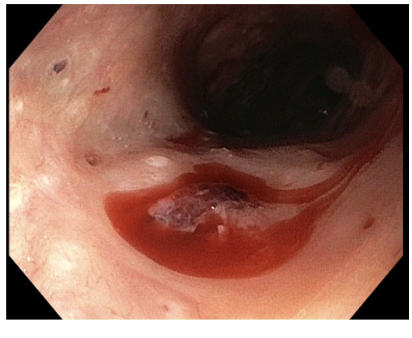


Fig 2. EGD showed multiple small esophageal intramural pseudo-diverticulum at mid to distal esophagus with one active bleeding from pseudo-diverticulum at 22 cm from incisor.

life-threatening hemorrhage can occur in the setting of alcoholic cirrhosis. Careful evaluation of each diverticular opening is paramount in identifying the culprit etiology, while hemostasis can be safely achieved using standard through-the-scope endoclips. Endoscopic dilatation can be safely performed when there is an associated esophageal stricture.

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Chewing Before Swallowing? A Commonly Overlooked Foreign Body Ingestion in Geriatric Population

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A 63-year old woman presented with odynophagia after swallowing her denture for six hours. A plain cervical x-ray showed a metal foreign body at mid-esophagus. An emergent upper endoscopy revealed a 4x3 cm denture in mid-esophagus (20 cm from incisor). It was successfully removed by rat-tooth forceps grasping the sharp metal edge of the denture.

DIAGNOSIS

Foreign body ingestion (denture in esophagus)

DISCUSSION

Denture ingestion is not uncommon with a rising incidence of 8.8%, especially in geriatric population. Dentures are typically made of polymethylmethacrylate which is radiolucent. As a result, they can be missed from plain film x-rays. Therefore, if clinical suspicion is high, a CT scan is warranted.

According to ESGE 2016 guideline, the decision for removing foreign body depends on the types and the locations of the objects. Denture is classified as sharp pointed irregular objects which needs to be emergently removed from esophagus within 2-6 hours to avoid debilitating morbidities such as esophageal necrosis. The recommended retrieval devices are grasping forceps, polypectomy snare, basket or retrieval net with transparent cap or latex rubber hood to protect mucosal injury. One of the key technical points is to grasp the foreign object by its sharp edge to avoid additional injury during the scope withdrawal. After removal, the patients can be discharged with a scheduled follow up. A dentistry evaluation for a better fit denture is essential to prevent recurrence of accidental ingestion.

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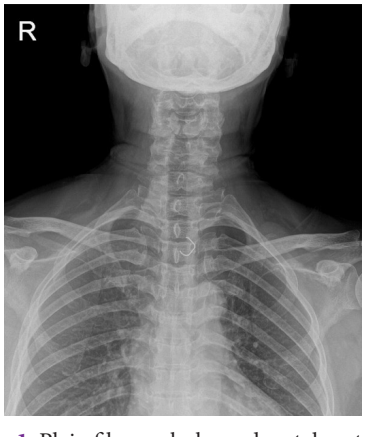

Fig 1. Plain film neck showed metal part of the denture at mid esophagus.

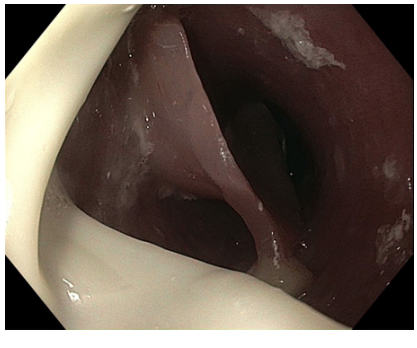

Fig 2. Denture size 4x3 cm was identified in mid-esophagus 20 cm from incisor.

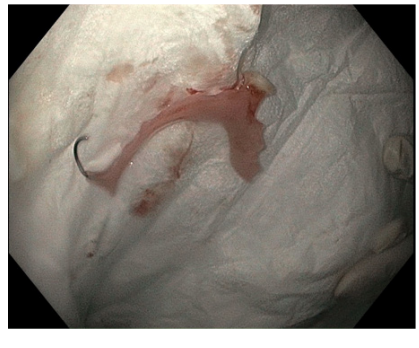

Fig 3. The denture was successfully by rat-tooth forceps.

Incidence and Risk Factors of Post ESD Coagulation Syndrome in Patients with Colorectal Neoplasm: A Systematic Review and Meta-Analysis

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Objective: Endoscopic submucosal dissection (ESD) has become more commonly used for large colorectal tumor to avoid the invasive surgery but it leads to several complications. Post ESD coagulation syndrome (PECS) is one of recognized complications describing as localized abdominal pain, fever, and leukocytosis after ESD. Several studies have tried to demonstrate the incidence and identify factors contributing to PECS. This meta-analysis was conducted to summarize and determine the incidence and predictors of PECS in patients with colorectal neoplasm.

Methods: MEDLINE and EMBASE databases were searched through November 2019 for studies on the features and risk factors of PECS in patients with colorectal neoplasm. The outcomes of interest were the incidence and patient and procedure related risk factors. Data from each study were combined using the random-effects, generic inverse variance method to calculate pooled incidence and risk ratio (RR) and 95% confidence intervals (CI). I^2 statistics was used to assess heterogeneity among studies, with I^2>50% indicates significant heterogeneity.

Results: Ten studies involving 2642 patients were included. The pooled incidence of PECS in patients with colorectal tumor is 10.1% (95%CI; 7.2%-13.0%, I^2=85.6%). A total of six factors were analyzed. Female sex (versus male), cecum as the location of tumor, tumor size ≥20 mm are associated with higher risk of PECS. Interestingly, rectum as the site of tumor is associated with lower risk of PECS compared to other parts of colon. However, old age (more than 60 years old), operation time ≥90 minutes, and muscularis propria involvement are not significantly associated with PECS risk.

Discussion: Our meta-analysis has demonstrated that female sex, the site of tumor: cecum, and tumor size were the predictors of PECS occurrence in patients with colorectal neoplasm. These findings raise the endoscopist awareness on high risk patients to develop PECS.

A Comparison on the Combination of Linked Color Imaging and Endocuff-Assisted Colonoscopy, Linked Color Imaging Alone, Endocuff-Assisted Colonoscopy Alone, and High-Definition Colonoscopy for Adenomas Detection: A Randomized Trial

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Objective: An advanced technology using mucosal image enhancement (Linked Color Imaging, LCI) or mucosal exposure device (Endocuff Vision assisted colonoscopy, EAC) showed improving adenoma detection rate (ADR). However, the effective of combination of both techniques has been unknown. Therefore, we investigated whether the combination of the LCI and EAC, LCI alone, and EAC alone could increase in ADR when compared with the white-light imaging (WLI).

Methods: We performed a prospective randomized study. Consecutive participants age 50 - 75 years who underwent screening colonoscopy were randomized (1:1:1:1) into colonoscopy by LCI+EAC, LCI alone, EAC alone and WLI using a computer-generated randomization. The sealed envelope was opened before the colonoscopy. All colonoscopies were performed with a high-definition colonoscope (EC 760ZP-V/L, Fujifilm Co, Japan) and done by endoscopists with the recorded ADR ≥ 35%. Data on participant characteristics, quality of bowel preparation, cecal intubation time, withdrawal time and polyp characteristics were collected. A primary outcome was ADR. A secondary outcome was the number of adenomas per colonoscopy (APC) and cecal intubation time.

Results: A total of 820 participants were randomized. Twelve participants were excluded; poor bowel preparation (n=7), incomplete colonoscopy (n=2), and needed to remove Endocuff for sigmoid passage (n=3). The remaining 808 participants (mean age 61 years, 65% female) completed study (Fig 1). The participants' characteristics and withdrawal time were not different among the 4 groups. The cecal intubation time in the LCI+EAC (5.9 ± 3.0 minutes) and EAC (6.4 ± 3.5 minutes) were significantly shorter than those in the WLI (8.3 ± 8.5 minutes) ($p<0.01$, respectively). The ADR for the LCI+EAC, LCI alone, EAC alone, and WLI was 59.5%, 54.4%, 53.0% and 48.5%, respectively. The ADR was significantly higher in the LCI+EAC than in the WLI ($p=0.03$). The APC for the LCI+EAC, LCI alone, EAC alone, and WLI was 1.32, 1.23, 1.19 and, 0.95 respectively. The APC was significantly higher in the LCI+EAC than in the WLI ($p=0.02$) (Fig 2). Although the ADR and APC in the LCI alone and EAC alone were higher than those in the WLI, there were no statistically significance. After adjusting for age, male sex, smoker, family history of colon cancer, body mass index and withdrawal time, the LCI+EAC was significantly associated with an increase in ADR when compared with the WLI (odds ratio, 1.58; 95% confidence interval, 1.05-2.37).

Conclusion: Our study shows that either LCI or EAC alone had a trend for better adenoma detection than WLI but their combination was significantly superior. Endocuff could speed up the cecal intubation time if there was no difficulty in passing the sigmoid colon.

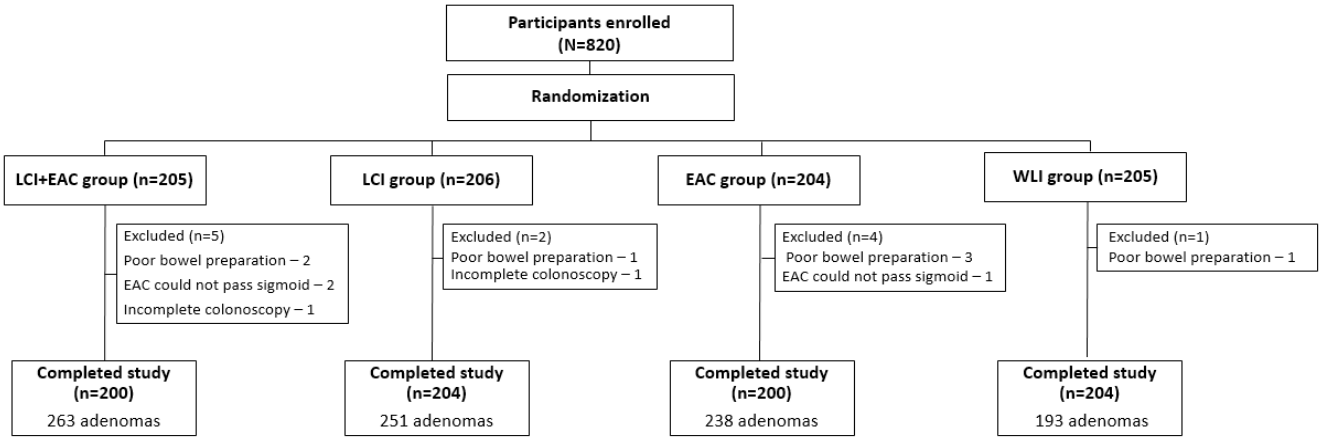

Fig 1. Study flow and randomization.
(LCI, Linked-color imaging; EAC, Endocuff Vision assisted colonoscopy; WLI, white light imaging)

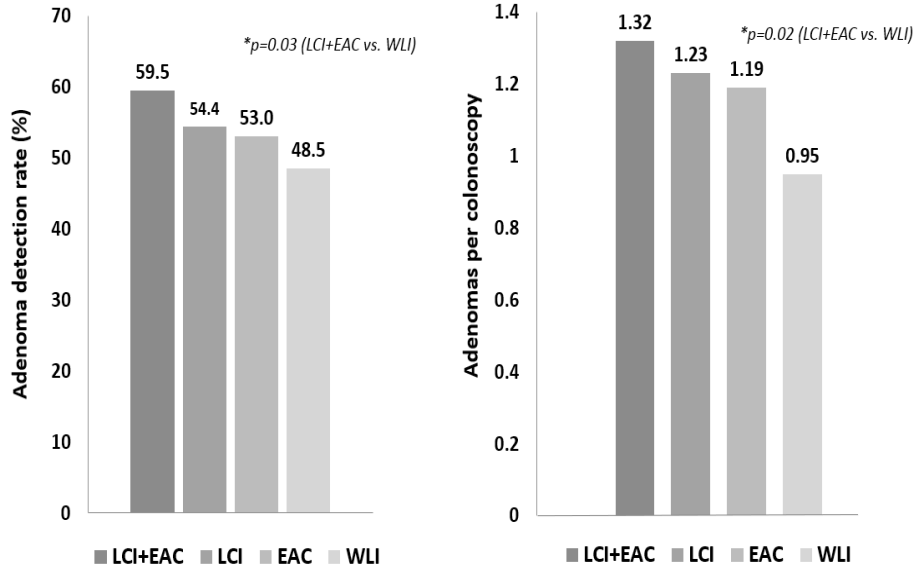

Fig 2. Adenoma detection rate (ADR) and adenomas per colonoscopy (APC) among 4 grops.
(LCI, Linked-color imaging; EAC, Endocuff Vision assisted colonoscopy; WLI, white light imaging)

Esophageal Manometry Yields Better Predictive Value in Assessing Clinical Outcomes than Esophageal Transit Scintigraphy After Treatment for Achalasia

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Objective: High resolution esophageal manometry (HRM) and esophageal transit scintigraphy can be used as tools to evaluate clinical response after treatment in patients with achalasia. Their performances in predicting clinical outcomes and correlation with symptoms improvement have not been well-studied. We aimed to evaluate the performance of both tests in clinical response prediction after treatment in patients with achalasia.

Methods: Consecutive patients with treatment-naïve achalasia completed GI symptoms questionnaires and underwent HRM in upright position (Manoview device, 10 of 5 ml water swallows) and esophageal transit scintigraphy at baseline and at one month after pneumatic dilation (PD) or peroral endoscopic myotomy (POEM). A reduction of Eckardt score to less than 2 was defined as an excellent response, score of 2-3 was a good response, while the score of > 3 was considered as non-responders. ROC analysis was conducted to compare performances of manometric and esophageal transit scintigraphy parameters for predicting clinical response.

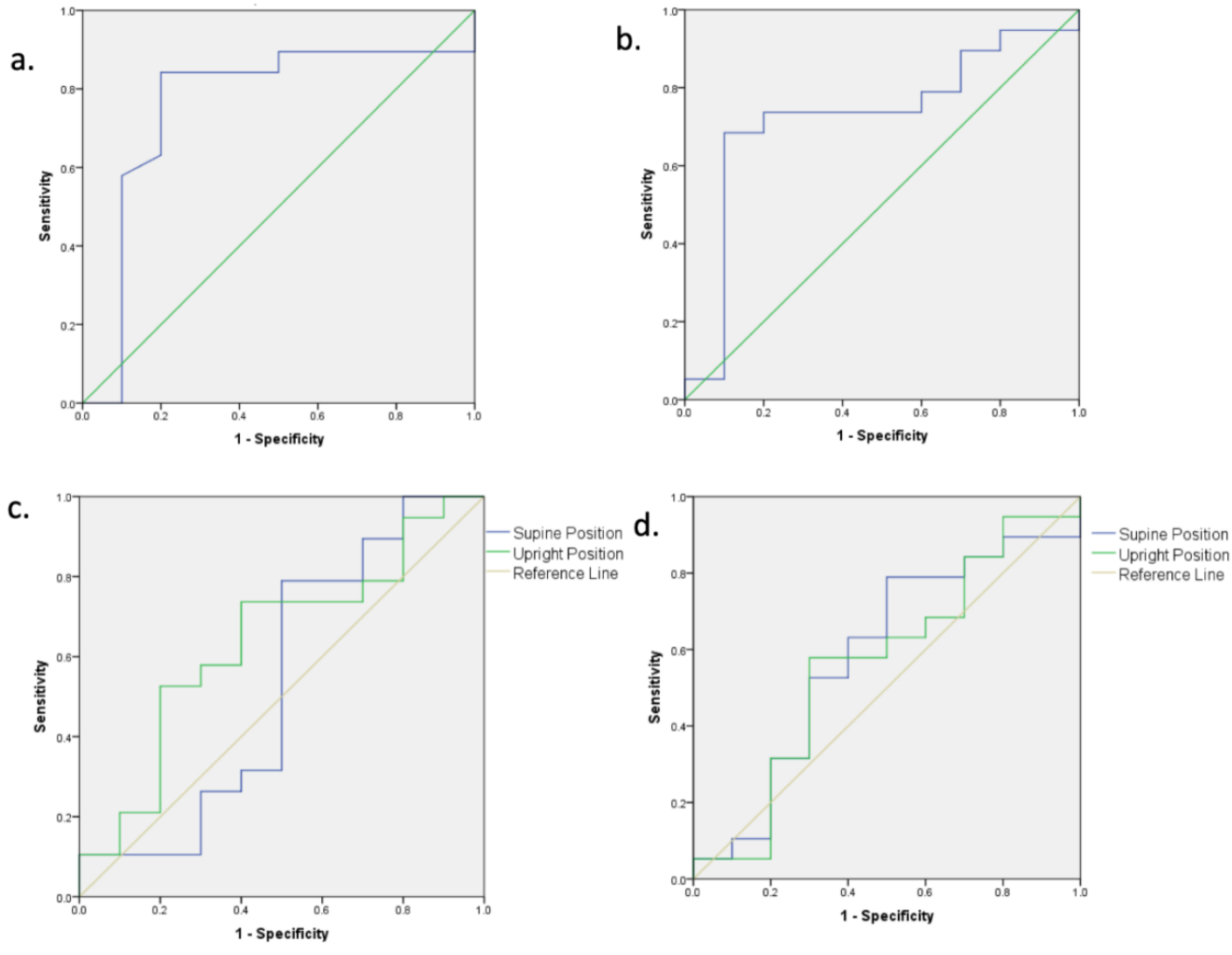
Results: 29 patients (21 females, age 48±15 years, median duration of symptoms 10 (4-12) months) were enrolled. Achalasia type II and type I were the most common types (58.6% and 24.1%, respectively). PD was performed in 22 (75.9%) patients and POEM in 7 (24.1%) patients. Of these, 65.5% achieved excellent response and 17.2% had good response at 1-month at treatment (Table 1). Patients with excellent clinical response had significantly higher percentage of median integrated relaxation pressure (IRP) reduction and significantly lower post-procedural IRP compared to those with non-excellent clinical response (% IRP reduction 73% (33.6-81.3) vs. 39.8% (-2.6-48.8), p= 0.05; IRP 7.9 (5-11.3) vs. 18.4 (13.7-23.9) mmHg, p=0.02, respectively. ROC analysis using a median IRP threshold of 14.4 mmHg and IRP reduction percentage of 48.3% demonstrated sensitivity values of 84% and 80% and specificity values of 80% and 73.7%, respectively in predicting excellent clinical response to treatment. Esophageal transit scintigraphy parameters including esophageal retention activities at 2 and 10 minutes in both supine and upright and their percentage changes did not show statistically significant correlation with clinical outcomes. (Table 1, Fig 1)

Conclusion: Postprocedural median IRP and IRP reduction percentage correlate well with the clinical response after PD/POEM. In contrast, esophageal retention activity at 2 and 10 minutes in both supine and upright position were not associated with symptomatic improvement. We propose that high resolution esophageal manometry should be the principle modality for assessing clinical response after treatment of achalasia.

TABLE 1. High resolution esophageal manometry and esophageal transit scintigraphy parameters comparing between achalasia patients with and without excellent response at 1-month after pneumatic balloon dilatation or peroral endoscopic myotomy.

	Excellent clinical response, Eckardt score <2 (n=19)	Non-excellent clinical response, Eckardt score ≥2 (n=10)	p-value
Manometric Parameters			
– **Median IRP, mmHg**	**7.9 (5-11.3)**	**18.4 (13.7-23.9)**	**0.02**
– **Percentage change of IRP, %**	**73.4 (33.6-81.3)**	**39.8 (-2.6 – 48.8)**	**0.05**
– Mean resting LES pressure, mmHg	14.9 (11-18.2)	24.6 (15.8-27.8)	0.09
– Percentage change of LES, %	53.6 (15.0-78.9)	20.7 (-14.9-51.5)	0.11
Esophageal Transit Scintigraphy parameters			
– Esophageal retention at 2 minutes, %			
• Supine	38 (21-54.6)	46.9 (17.5-78.2)	0.58
• Upright	17.5(9.1-59.9)	45.9 (11.1-61.1)	0.65
– Esophageal retention at 10 minutes, %			
• Supine	35(13.3-45.3)	33.2 (9.5-65.2)	0.75
• Upright	11.7(7.4-45.2)	33 (11.2-54.7)	0.23
– Esophageal retention percentage change at 2 minutes, %			
• Supine	33.4 (2.6-54.4)	32.6 (-14.7-54.7)	0.72
• Upright	49.9 (4.7-81.2)	23.1 (8.7-73.2)	0.85
– Esophageal retention percentage change at 10 minutes, %			
• Supine	44.1 (10.6-67.6)	16.9 (-13.3-67.6)	0.46
• Upright	69.7 (20.1-83.1)	39.7 (10.5-82.4)	0.58

IRP= Integrated Relaxation Pressure, LES= Lower Esophageal Sphincter
Data expressed as median (interquartile range)



1-month post treatment manometric & esophageal scintigraphy parameters	AUROC (95% CI)	p-value
a. Median IRP	0.76 (95% CI 0.55-0.97)	0.02
b. Median IRP percentage change	0.73 (95% CI 0.52-0.93)	0.05
c. Esophageal retention at 10 minutes		
• Supine	0.54 (95% CI 0.29-0.79)	0.75
• Upright	0.64 (95% CI 0.42-0.86)	0.23
d. Esophageal retention percentage change at 10 minutes		
• Supine	0.58 (95% CI 0.52-0.93)	0.34
• Upright	0.56 (95% CI 0.33-3.80)	0.58

Fig 1. Area under ROC (AUROC) of high resolution esophageal manometry and esophageal transit scintigraphy parameters for predicting excellent clinical response at 1 month after treatment.

Is Prophylactic Endoscopic Clipping Closure Useful for Preventing Post ESD Coagulation Syndrome (PECS) in Patients with Large Colorectal Neoplasm? The Answer from a Meta- analysis

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Objective: Endoscopic submucosal dissection (ESD) is an approved minimally invasive treatment for large colorectal tumor. Post ESD coagulation syndrome (PECS) is a complication describing as the inflammatory response condition (fever, abdominal pain, and leukocytosis) after ESD. Endoscopic clipping closure has been introduced to prevent this complication. The data on efficacy of closure is inconclusive among published literature. We conducted the meta-analysis aimed to determine the efficacy of prophylactic role of endoscopic clipping closure for PECS prevention in patients with large colorectal neoplasm.

Methods: We searched MEDLINE and EMBASE databases through November 2019 for published studies that compare the prophylactic efficacy of endoscopic clipping closure for preventing PECS in patients with colorectal neoplasm. The outcome of interest was the rate of PECS occurrence. Data from each study were combined using the random-effects, generic inverse variance method of DerSimonian and Laird to calculate pooled risk ratio (RR) and 95% confidence intervals (CI). I^2 statistics was used to assess heterogeneity among studies, with I^2>50% indicates significant heterogeneity.

Results: Four studies were included in the meta-analysis and reported a total of 412 and 863 patients receiving and not receiving endoscopic closure, respectively. The occurrence of PECS in patients receiving prophylactic closure is comparable to control group with pooled RR of 0.55 (95% CI;
0.17-1.75, I^2= 68.3%).

Conclusion: This meta-analysis could not demonstrate the prophylactic efficacy of endoscopic clipping closure for PECS in patients with large colorectal tumor. However, the analysis was restricted by the small sample size. Further randomized controlled trials are still needed to clarify this effectiveness.

The "Predict, Resect and Discard" Strategy for a Surveillance Recommendation Based on Number and Size of Polyps Without Pathology Assessment

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Objective: The US Multi-Society Task Force (US-MSTF) on Colorectal Cancer for post-polypectomy surveillance recommends the interval of colonoscopy based on polyp histology and number. In practice, majority of colonic polyps are small polyps (<10 mm). The "predict, resect and discard" strategy for these polyps is a cost-saving approach because no pathology required. In order to gain a wide acceptance in clinical practice, this strategy should have a high negative predictive value >90% for high-risk adenoma (HRA) which is the main determination for surveillance recommendations. Therefore, we aimed 1) to determine the negative predictive value (NPV) for a diagnosis of HRA based on polyp size and 2) to compare the concordance of surveillance recommendation based on size and number of polyp with the US-MSTF recommendations.

Methods: The electronic database of screening colonoscopy between January 2007 and December 2018 was retrospectively reviewed. In our practice, all identified polyps were resected and sent to a pathologist except diminutive polyps (size <6 mm) at the rectosigmoid colon. HRA was defined as adenoma with high-grade dysplasia, or villous adenoma. Our surveillance interval based on number and size of polyp is shown in Table 1. The primary outcome was the NPV for a diagnosis HRA of polyp size <10 mm and number needed for pathological assessment (NNP) to detect one HRA. The secondary outcome was the agreement in surveillance interval recommendation between the two methods. The US-MSTF recommendations on surveillance interval were used as a reference standard.

Results: A total of 9339 subjects (mean age 61, 61% female, 7511 polyps) were enrolled. None of polyps size <10 mm (n=6644) had cancer. For polyp size <10 mm, the NPV for diagnosis of HRA were 97.9% (95% confidence interval [CI], 97.6%-98.2%) and the NNPs to detect one HRA were 48. When compared our surveillance interval based on no pathology assessment with the US-MSTF recommendations, the surveillance interval agreement was 97.4% (95% CI, 97.1% – 97.7%) (Fig 1). Our surveillance recommendation could reduce 89% of specimens to be sent for pathology and 93% of patients could be immediately assigned for the surveillance interval with 97.4% agreement to the US-MSTF recommendation.

Conclusion: Our study supports the "predict, resect and discard" strategy of polyp smaller than 10 mm. It could save the number of pathology examinations with agreeable surveillance interval to the US-MSTF recommendation.

TABLE 1. Agreement of the surveillance interval between our proposed recommendation based on number and size of polyp without pathology assessment and the US Multi-Society Task Force recommendation

	Our proposed recommendation based on size and number of polyps	US Multi-Society Task Force on colorectal cancer recommendation	Surveillance interval (years)	Agreement (%, 95%CI)
1.	No polyp	No polyp	10	100% (99.9 -100.0)
2.	1-3 small polyps <10 mm	1-2 tubular adenomas < 10 mm	5-10	95.2% (94.4 - 96.0)
3.	≥ 4 small polyps <10 mm	3-10 tubular adenomas <10 mm	3	71.2% (66.0 - 75.9)
4.	≥1 polyps ≥10 mm	≥1 adenomas ≥10 mm ≥1 adenomas with villous feature ≥1 adenomas with high-grade dysplasia ≥1 sessile serrate lesion with dysplasia	3	98.1% (96.7 - 98.9)
		Overall		97.4% (97.1 - 97.7)

Epidemiology and Financial Burden of Pancreatic Cancer-related Hospitalizations In the Philippines: An 11-Year Cross Sectional Study of The National Health Insurance Database

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INTRODUCTION

Problem Statement

Pancreatic cancer is a global problem. In 2018, there were 458,918 new cases of pancreatic cancer that were registered worldwide. In the same year, there were 432,242 cases who died from pancreatic cancer.[1] Although primary pancreatic cancer is not featured among the ten most common cancers in 2018, it still ranked 7th in mortality rates among the 36 cancers included in the Global Cancer Statistics (GLOBOCAN). The almost equal number of new cases to number of deaths may reflect the low chance of survival from primary pancreatic cancer. In fact, its Mortality-to-Incidence Ratio (MIR) is as high as 94.2%[1], making pancreatic cancer one of the most fatal malignancies with very poor prognosis.

However, incidence data provided in the GLOBOCAN 2018 are produced by population-based cancer registries (PBCRs). Although these PBCRs may cover nationwide populations, more often they only cover subnational areas, such as selected urban areas, particularly in countries undergoing economic development. In the Philippines, the GLOBOCAN 2018 had sources of data on incidence of pancreatic cancer from three local cancer registries in Manila, Rizal, and Cebu for the years 2008 to 2012 with rates applied to the 2018 population while data on mortality were from the World Health Organization (WHO) mortality database.[11] These data are outdated and likely to be only partial of the true values of data regarding primary pancreatic cancer.

Significance

Analysis of pancreatic cancer epidemiology may help the medical field to be updated of its current status and how it can be addressed in the community level. Given the high case fatality rate from pancreatic cancer, and the fact that a highly effective treatment is still to be discovered, the best chance of helping this population subgroup is to identify them early in the disease process when surgery can be curative. Evaluation by a gastroenterologist plays a critical role in this identification process.

Being able to present actual facts and numbers, including how much is being spent by both the government and patients in the diagnosis and management of primary pancreatic cancers, may help us see the reality of how big an impact it is when pancreatic cancer takes its toll on the patient's health and resources. Hopefully, information from epidemiologic studies will help in the analysis, formulation and implementation of policies that will help in the assurance of availability of medical expertise and hospitals with adequate diagnostic equipment in different parts of the Philippines.

BACKGROUND INFORMATION AND REVIEW OF LITERATURE

The latest version of the global cancer statistics, GLOBOCAN 2018[1], presented figures focusing on illustrating epidemiologic profile and worldwide impact of the different cancer diseases. In the Philippines, data came from three registries in Manila, Rizal, and Cebu as mentioned by the agency that collated data for GLOBOCAN.[11] Population and regional coverage of these three registries were unknown. Caution however was given in interpreting data especially those coming from low- to middle-income countries because of possible inadequate recording and data keeping. No information regarding this concern was specified for Philippine data.

Incidence and mortality rates

Based from GLOBOCAN 2018, there were 405,389 new cases of pancreatic cancer worldwide among 15 years old and above, with a crude incidence rate of 7.2 per 100,000 population. For the Philippines alone, the crude incidence rate was 4.3 per 100,000 population. For the year 2018, there were 3,123 new cases of pancreatic cancer among Filipinos aged 15 years old and above.[2]

Looking at the trend of incidence rates of pancreatic cancer in the Philippines among 15 years old and above since the earliest available data in 1983-1987, the incidence rate trend appears to be fluctuating between 4 and 8 per 100,000. If we plot the absolute number of new cases diagnosed with pancreatic cancer for each time period with available data, there is a notable and worrisome steep increase after the year 2007.[3-10] Improved data collection and computerized data retrieval can be a reason. Increased awareness of the serious threat of the disease might have also contributed to the observed trend but a real and actual surge in pancreatic cancer cases should be considered.

Data on mortality rates of pancreatic cancer in the Philippines are also lacking. Data taken from the World Health Organization (WHO) of pancreatic cancer mortality rates among Filipinos 15 years old and above, an increasing trend can also be noted like that of the incidence rates but true value is difficult to validate, especially when several time points are lacking.

Costs of hospitalization

Treatment and hospitalization costs are mainly contributed by surgical and endoscopic procedures received by these patients. Understandably, those who have non-metastatic disease and therefore surgical candidates, tend to have higher hospitalization costs compared to those diagnosed with advanced disease and only given palliative care. In a Surveillance, Epidemiology, and End Results (SEER) Medicare database study that included 15,037 pancreatic cancer patients diagnosed between 2000 and 2007, total direct medical costs were estimated from Medicare payments. Results showed that the mean total costs of hospitalization was highest among the surgical candidates amounting to $134,000 (approximately Php 7M) while those with metastatic disease had mean total costs of $49,000 (approximately Php 2.5M), with an average cost of $61,700 (approximately Php 3.2M). Majority of the expenses were of cancer-related procedures, surgical and endoscopic.[12]

OBJECTIVES

Primary Objective

To describe the annual pancreatic cancer-related hospitalizations in the Philippines based on the national health insurance (PhilHealth) database from 2008-2018

Secondary Objectives

1. To describe the region-specific distribution of pancreatic-cancer related hospitalizations
2. To describe the annual number and disposition of pancreatic cancer-related hospitalizations
3. To describe the age and sex distribution of all pancreatic cancer-related hospitalizations
4. To describe the annual gross death rate of patients with pancreatic cancer-related hospitalization
5. To determine annual total cost of hospitalization and annual total reimbursements of pancreatic cancer-related hospitalizations
6. To determine the annual total Out-of-Pocket (OOP) expenses by patients

METHODOLOGY

Type of Study, Time Period, and Target Population

This is a cross sectional descriptive study including all patients with a diagnosis of pancreatic cancer who filed for insurance claims (PhilHealth) from January 1, 2008 to December 31, 2018. No exclusion criteria employed.

Description of study procedure, data Collection, and data analysis

All patients with a discharge diagnosis of primary pancreatic cancer with the following International Classification of Diseases version 10 (ICD-10) codes were included: C25.0 malignant neoplasm of head of pancreas, C25.1 malignant neoplasm of body of pancreas, C25.2 malignant neoplasm of tail of pancreas, C25.3 malignant neoplasm of pancreatic duct, C25.4 malignant neoplasm of endocrine pancreas; malignant neoplasm of islets of Langerhans, C25.7 malignant neoplasm of other parts of pancreas; malignant neoplasm of neck of pancreas, C25.8 malignant neoplasm of overlying lesion of pancreas, C25.9 malignant neoplasm of pancreas unspecified. All PhilHealth claims with the above ICD codes, between January 1, 2008 and December 31, 2018 were extracted electronically from the database.

Data on demographics of patients of included hospitalizations and their respective institutions of

confinement were obtained. Standard age brackets by the WHO were used to allow comparison with GLOBOCAN and other worldwide data. *Total Actual Charges* and *Total Claims Amount* were extracted from each included patient.

All data were encoded and analyzed using *Microsoft Excel*. Results were presented as frequencies with percentage or means whenever applicable. *Annual Gross Death Rate, Annual Total Cost of Hospitalizations, Annual Total Reimbursements,* and *Total Out-of-Pocket Expenses* were computed accordingly.

RESULTS

There were a total of 6,624 pancreatic cancer-related hospitalizations based on submitted insurance claims from 2008 to 2018 (Table 1).

Distribution of included hospitalizations from 2008-2017 showed that majority were primarily from NCR (27.6%) and Region III (11.4%). Only a small number of hospitalizations were recorded from the Mindanao regions of CARAGA (0.8%) and ARMM (0.3%). The following were the number of hospitalizations and percentage distribution for the other regions in the Philippines: Region I (n=345, 6.3%), Region II (n=207, 3.7%), Cordillera Administrative Region (n=137, 2.5%), Region IV (n=525, 9.5%), Region V (n=188, 3.4%), Region VI (n=512, 9.3%), Region VII (n=449, 8.2%), Region VIII (n=86, 1.6%), Region IX (n=102, 1.9%), Region X (n=247, 4.5%), Region XI (n=279, 5%), and Region XII (n=223, 4%).

When the number of pancreatic cancer-related hospitalizations in 2018 were included, there were a total of 6,624 hospitalizations with submitted insurance claims. Table 2 below shows data for patients aged 15 years old and above. More than half were of male patients (61.5%).

TABLE 1. Agreement of the surveillance interval between our proposed recommendation based on number and size of polyp without pathology assessment and the US Multi-Society Task Force recommendation

	Pancreatic Cancer-Related Hospitalizations ***(N=6,624)***
Age, n (%, mean)	
>15 yrs old	6,611 (99.8%, 62.8 years)
<15 yrs old	13 (0.2 %, 5.8 years)
Sex, n (%)	
Male	4,075 (61.5%)
Female	2,549 (38.5%)
*Patient Type, (%)	
Member	3867 (70%)
Dependent	1641 (30%)
*Social Sector, (%)	
Government	1,738 (31.5%)
Private	3,770 (68.5%)
*Institution Class, n (%)	
Level 1 Hospital	949 (18%)
Level 2 Hospital	1919 (35%)
Level 3 Hospital	2586 (47%)

*Data from 1,117 hospitalizations (including 1 patient <15 yrs old) for the year 2018 were not available and not included in the count, Hence percentages were computed using N=5,508.

TABLE 2. Annual Number of Pancreatic Cancer-Related Hospitalizations and Disposition In the Philippines of patients 15 years old and above from 2008-2018

Year	Male	Female	Total	DISPOSITION			
				Expired	DAMA**	Improved/ Recovered	Others***
2008*	2	0	2	-	-	-	2
2009*	6	6	12	-	-	-	12
2010*	23	15	38	-	-	-	38
2011*	270	177	447	-	-	-	447
2012*	302	151	453	-	-	-	453
2013*	300	167	467	-	-	-	467
2014	600	321	921	87 (9.5%)	52 (5.7%)	782 (85.7%)	-
2015	639	343	982	108 (11.1%)	64 (6.6%)	810 (83.3%)	-
2016	701	380	1,081	121 (11.2%)	61 (5.7%)	894 (83%)	5
2017	711	394	1,105	120 (11%)	63 (5.8%)	920 (84%)	2
2018	517	599	1,116	106 (9.5%)	98 (8.8%)	908 (81.3%)	1 (absconded) 3 (transferred)
Total	**4,071** (61.5%)	**2,553** (38.5%)	**6,624** (100%)	**542**	**338**	**4,314**	**1,430**

*No data available on final disposition of patients
**Discharged Against Medical Advice
***Disposition was designated N/A, transferred, or absconded

From 2011 onwards, the number of pancreatic cancer-related hospitalizations is steadily increasing (Fig 1), with the male population consistently greater in number than the females, except for the year 2018 when hospitalizations of female patients (n=599) slightly exceeded those of the males (n=517).

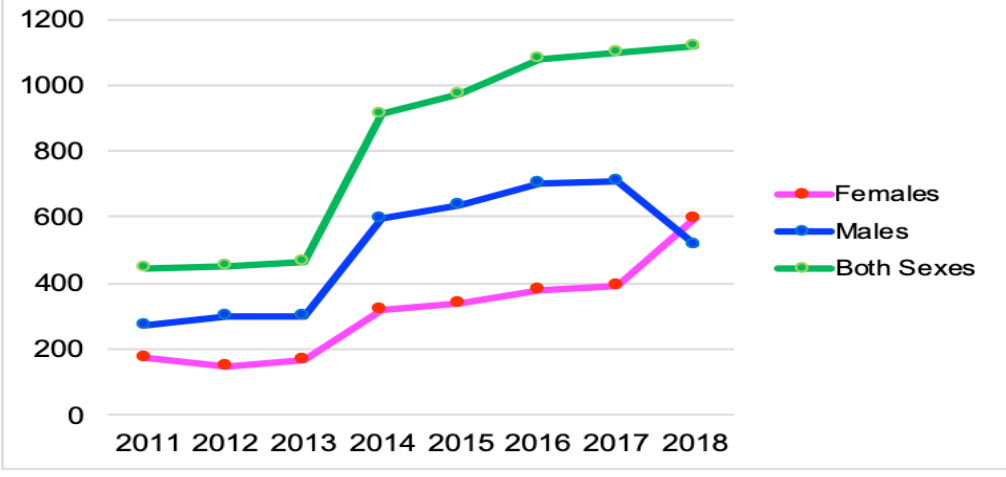

Fig 1. Number of Pancreatic Cancer-Related Hospitalizations of patients 15 years old and above from 2011 to 2018

Of the 6,624 pancreatic cancer-related hospitalizations with submitted insurance claims from 2008 to 2018, the age and sex distribution showed that majority were of elderly patients aged 60-69 years old (n=2161, 32.6 %) and were predominantly of the male sex (n=4,075, 61.5%). The *annual Gross Death Rate* for the years 2014-2018 were steady at 9.5-11.2% for patients 15 years old and above.

Data on *Total Actual Charges* and *Total Claims Amount* from each included hospitalization were taken from years 2011 to 2018 and were summed up as *Total Cost of Hospitalization* and *Total Reimbursements* for each year, respectively. The *Annual Total Cost of Hospitalizations* from government and private institutions combined ranged from Php 5M to 182M and the *Annual Total Reimbursements* ranged from Php 6M to 16M, comprising of 8.4-38.2% of the *Annual Total Cost of Hospitalization*.

Fig 2 below shows the steadily increasing A*nnual Total Cost of Hospitalization* and *Annual Out-of-pocket expenses* for the year 2011 to 2018. Noticeable in the line graph is that the Annual Total Reimbursements have remained stable below Php 20M.

DISCUSSION

Epidemiology of primary pancreatic cancer in the Philippines is still not well defined. Basing on observations in the clinics and hospital admissions, data were limited to the patients you only see in your own institution. So far, the identifiable source of greater data acquisition is from the health insurance institution that receives all insurance claims from various healthcare facilities nationwide.

The annual number of pancreatic cancer-related hospitalizations in this study was demonstrated to be steadily increasing from 2011 to 2018. This is congruent with the data from the *International Agency for Research on Cancer (IARC)* from 1983 to 2018, showing a steep increase in the number of new cases of pancreatic cancer after the year 2007. There was however a difference in actual number of cases for the year 2018. In this present study, there were 1,116 cases of pancreatic cancer patients in 2018 while GLOBOCAN registered a total of 3,123 patients. The patients unaccounted for in the present study may likely be those without insurance membership, or inadequate data keeping from the hospital level up to provincial and regional levels.

It is also commonly observed that primary pancreatic cancer is the disease of the elderly. Almost all of the patients in this study were 15 years old and above (99.8%) mostly in the 6th and 7th decades of life. It is predominantly seen in the male population with a male-to-female ratio almost steady at 2:1. Results of the present study is parallel to the *GLOBOCAN 2018* results (Table 1).

Most of the recorded hospitalizations were from

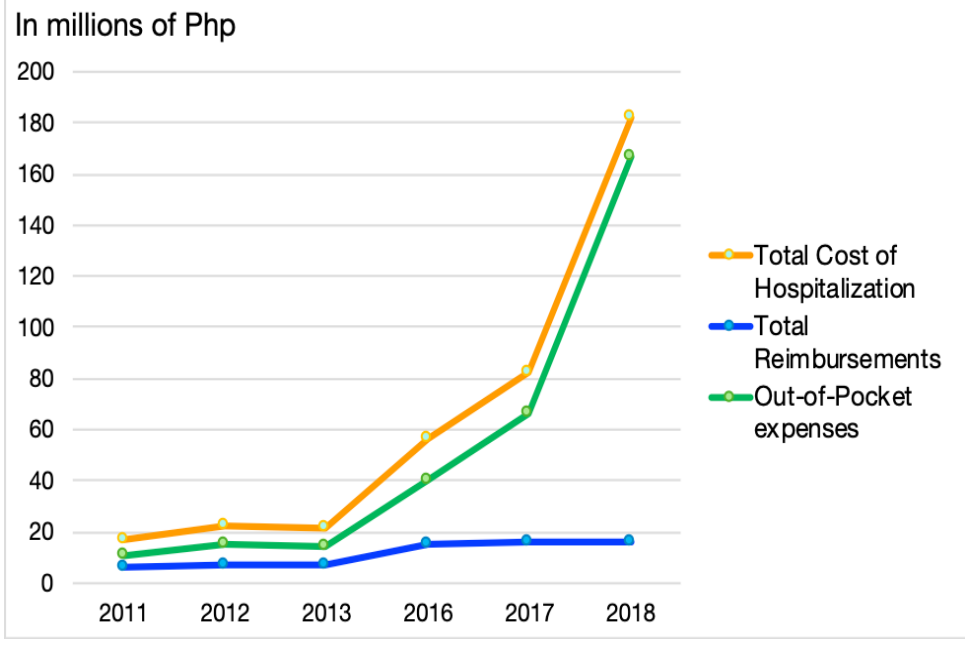

Fig 2. Annual total cost of hospitalization, total reimbursements, and total Out-of-Pocket expenses for pancreatic cancer-related hospitalizations from 2011-2018, Government and Private Sectors Combined

*Data on *Total Actual Charges* for the years 2014 and 2015 were lacking in a majority of hospitalizations, and were not included in the graph.

private institutions (68.4%) since there are more hospitals of this type that are accessible and are actually branching out into different parts of the country as they are being managed quite well by corporations. Moreover, government hospitals are likely to be of no vacancy with a long line up of patients who want to be admitted or transferred to avail of cheaper hospital charges and possibly free professional fee. Moreover, Hospital costs may influence a patient's disposition of hospitalization. Though desire to be treated continuously is every family's perspective, some may be forced to even be *Discharged Against Medical Advice (DAMA)*. Around 5% to 8% of hospitalizations lead to DAMA, not far from the computed *Annual Gross Death Rate* of 9% to 11%. The *Annual Gross Death Rate* has remained steady in these values for the past 5 years. As more new cases of pancreatic cancer are expected to be diagnosed in the coming years, more patients are expected to die from the disease even with a steady annual gross death rate.

Primary pancreatic cancer has a high mortality-to-incidence ratio, and a diagnosis made at an early stage is considered favorable. Part of the evaluation of disease status are CT scan or MRI scan machines that are usually not available in the provinces. Moreover, endoscopic ultrasound and trained endosonographers, are available only in 8 institutions in the Philippines and all of these are situated in the National Capital Region (NCR). Endoscopic Retrograde Cholangiopancreatography (ERCP) is also one of the common procedures that pancreatic cancer patients undergo. Facilities and qualified endoscopists may not be readily available in the provinces and Level 1 and Level 2 Hospitals. Pancreaticobiliary surgeons may also be not available in distant provinces. Hence, there is a tendency for patients to not seek consult for recurrent or persistent symptoms and develop advanced disease. Some still need to travel to nearby cities or provinces, even to NCR just to have the appropriate diagnostic evaluation and treatment. Logistics of finances and availability of accompanying relative may influence the delay in consult. These things may explain the highest number of pancreatic cancer-related hospitalizations coming from the NCR and the neighboring Regions III and IV. While CARAGA and ARMM regions in Mindanao as well as Region VIII in the Visayas, recorded the least number of these hospitalizations.

Hospitalizations of patients both with early and advanced stage pancreatic cancer potentially costly. The *Annual Total Cost of Hospitalization* for government and private sectors combined, showed the increasing trend from 2011 up to 2018 reaching a peak of Php 182M. In the Philippines, any diagnosis of pancreatic cancer, whether early or advanced, is designated to a fixed medical case rate. This case rate amount shall be deducted by the healthcare institution from the member's total hospital bill, including the professional fees, prior to discharge. During review of the included hospitalizations in this study, the *Total Claims Amount* was highly variable in 2008-2013 until in 2014 that the *Total Claims Amount* for each hospitalization was fixed at Php 14,200. In the background of a steadily increasing *Annual Total Cost of Hospitalization* and a plateau of *Annual Total Reimbursements* (based on fixed medical case rate), the *Annual Out-of-Pocket expenses* is also increasing, highest in 2018 amounting to Php 167M, which is 91.6% of the *Annual Total Cost of Hospitalization*.

CONCLUSION AND RECOMMENDATIONS

New cases and hospitalizations from pancreatic cancer have been demonstrated in this study to be steadily increasing for the past 10 years. Even the cost of hospitalization and out-of-pocket expenses are increasing as well. Since pancreatic cancer has a dismal prognosis and a potentially devastating effect, physically and financially, development of strategies for screening individuals high risk for pancreatic cancer would be beneficial. The goal of discovering an effective screening strategy that is truly preventive of pancreatic cancer, as the case of timely colonoscopy for colon cancer, remains a dream. While research on targeted therapies and cost-effective screening strategies are still underway, a thrust to ensure distribution in the Visayas and Mindanao provinces of available facilities such as CT scans, MRI, and endoscopic ultrasound (EUS) and doctors who are trained in performing EUS, ERCP, and pancreaticobiliary surgery may have to be addressed hand-in-hand by the private and government bodies. This will enable arrival at a diagnosis of pancreatic cancer early in the disease and a potentially curative surgery is still possible.

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Downhill Esophageal Varices

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CASE HISTORY

A 68-year-old male with history of chronic hepatitis C cirrhosis underwent upper endoscopy for surveillance of varices. An upper endoscopy showed purplish longitudinal tortuous submucosal vessels, located at the proximal one third of the esophagus (Fig 1). The varices were not observed at the distal part of the esophagus and gastric cardia. This patient was diagnosed with downhill esophageal varices and further investigation revealed an obstruction of superior vena cava (SVC) from lung cancer.

DIAGNOSIS

Downhill esophageal varices

DISCUSSION

Downhill esophageal varices, also known as proximal esophageal varices, are uncommon condition, representing 0.4-10% of esophageal varices.[1] "Downhill" is named based on the cephalo-caudal direction of variceal blood flow.[2] The most common reported cause of downhill esophagus is SVC compression especially from mediastinal malignancy such as thymoma, lymphoma or lung cancer.[3] Apart from malignant SVC obstruction, other various benign etiologies were also reported in the literature; e.g. complication of central catheterization, thyroid goiter, Behçet disease and mediastinal fibrosis.[4]

Proximal and middle third of esophagus are predominantly supplied by venous drainage of azygos

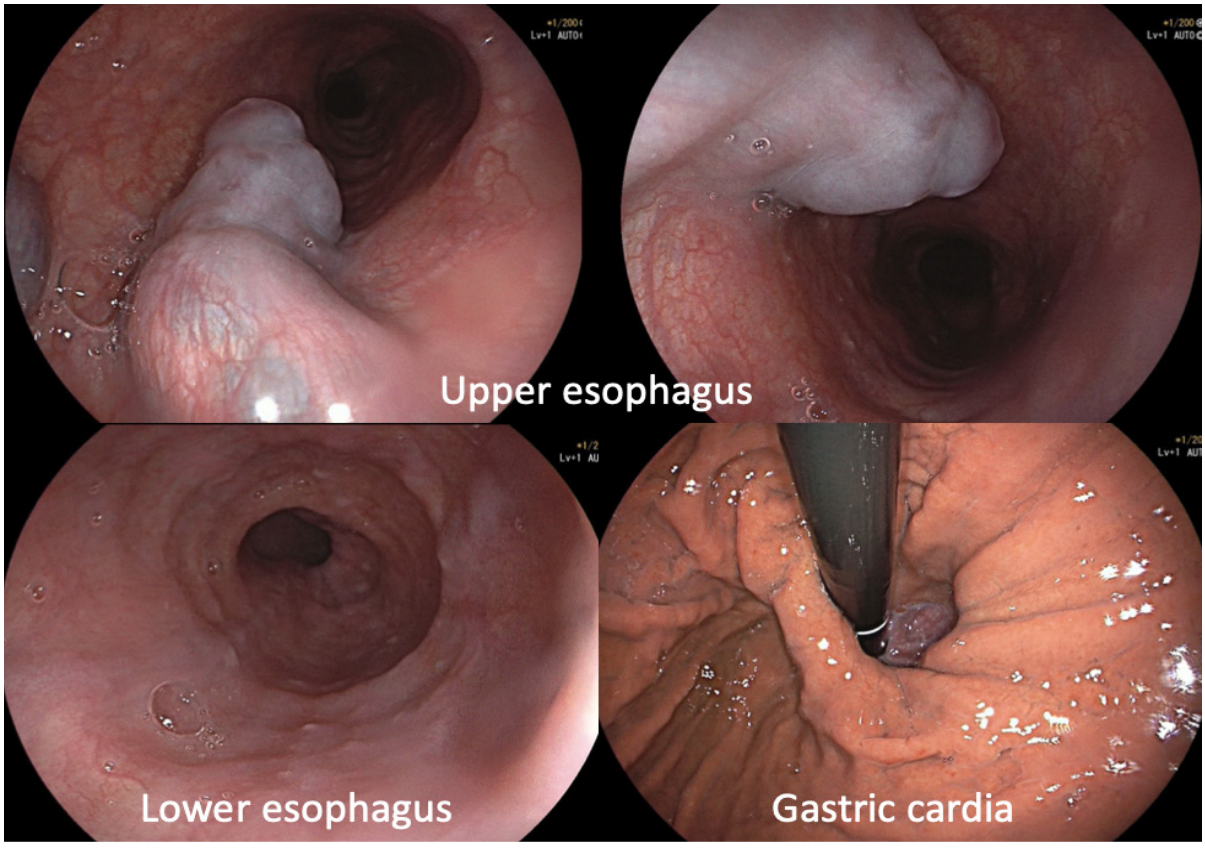

Fig 1. Upper endoscopy revealed dilated and tortuous submucosal vessels at proximal esophagus which did not present in lower esophagus or gastric cardia.

systems. When the obstruction of SVC occurs, the retrograde flow of blood through the collateral vessels results in dilatation of vein and varices formation.[2] If the obstruction is above the level of azygos vein, the varices will be confined to the upper and middle part of the esophagus. While the obstruction below azygos vein leads to development of varices along the entire esophagus.[5] Unlike distal or uphill esophageal varices, downhill varices are not related to portal hypertension.[6] Gastrointestinal bleeding is a very rare presenting symptom. The deeper location in submucosa of proximal esophageal varices, compared to more superficially located distal esophageal varices, contributes to lower risk of bleeding complication.[2]

There is no standard treatment guideline for downhill varices currently. The cornerstone of the treatment is the correction of SVC obstruction.[7] Traditional endoscopic intervention such as band ligation, sclerotherapy or balloon tamponade were reported in several bleeding case series. Although rare, downhill esophageal varices are like a warning sign which could lead to a fatal condition. Correction of the underlying etiology should be promptly done to prevent further serious complications.

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Achalasia Cardia

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CASE HISTORY

A previously healthy 54 year-old-woman presented with regurgitation of undigested food. She had dysphagia which involves both solid and fluid and progressively worsened for 6 months. She lost her weight for 9 kg. Esophagogastroduodenoscopy (EGD) was performed. Diffuse dilatation of esophageal lumen with retained food was seen (Fig 1, 2). No peristalsis of the esophagus was observed. The upper endoscope could pass through esophagogastric junction without difficulty. The retroflex view of EGD showed normal gastric cardia (Fig 3). The rest of the EGD findings were normal. High-resolution esophageal manometry found impaired lower esophageal sphincter relaxation (integrated relaxation pressure >15 mmHg), loss of esophageal peristalsis and pan-esophageal pressurization (Fig 4), which were compatible with achalasia cardia type 2. Her dysphagia symptom was much improved after treatment with pneumatic balloon dilation.

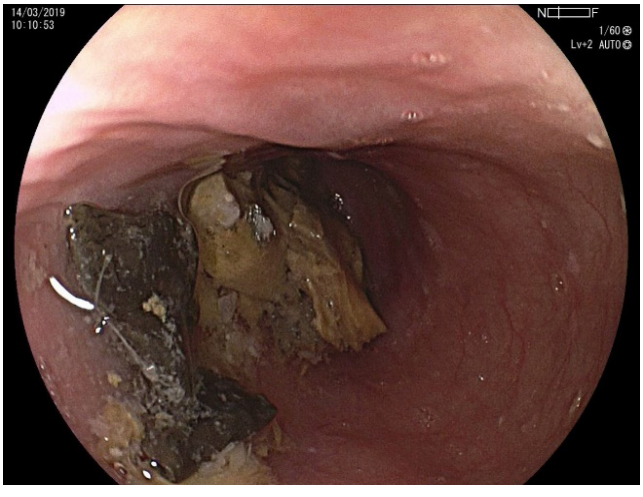

Fig 1. EGD revealed retained food in the dilated esophagus

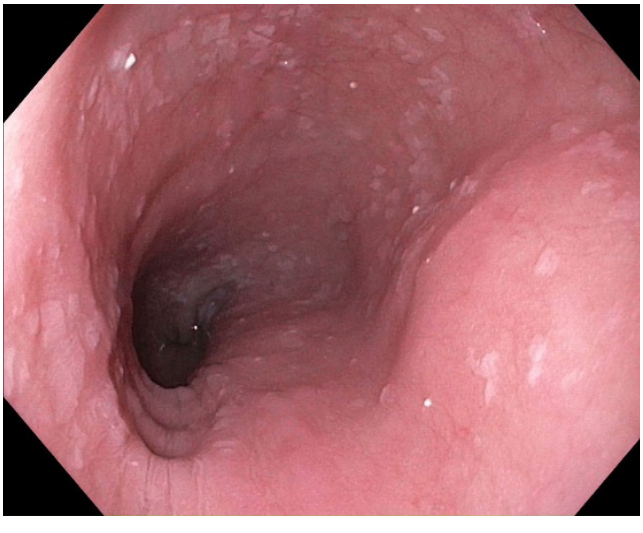

Fig 2. After complete removal of the retained foods, EGD revealed dilated and tortous esophagus with unremarkable mucosa.

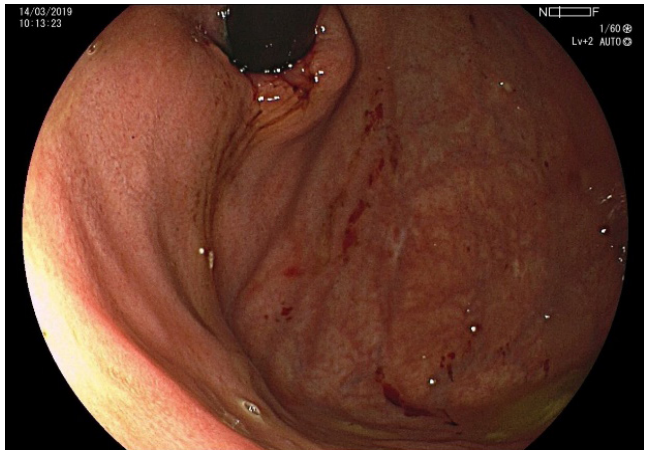

Fig 3. Retroflex view demonstrated no mass at gastric cardia and fundus

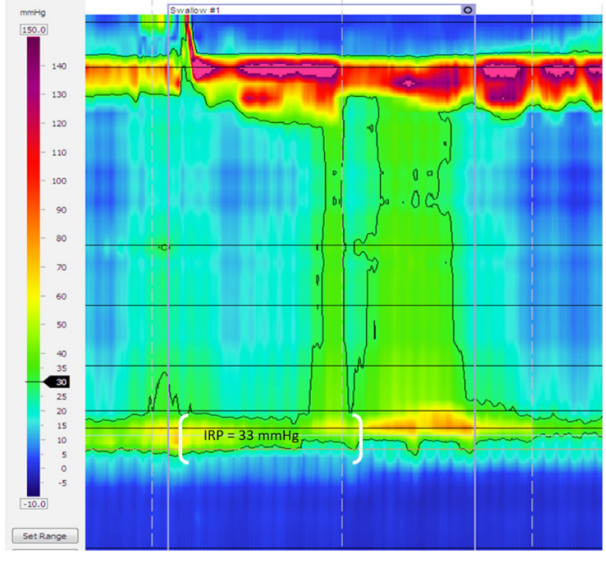

Fig 4. High-resolution manometry showed impairment of lower esophageal sphincter relaxation (high integrated relaxation pressure, IRP), absence of peristalsis, and pan-esophageal pressurization

DIAGNOSIS

Achalasia cardia

DISCUSSION

Achalasia cardia is an esophageal motility disorder from ganglion cell defect in myenteric plexus of the lower esophageal sphincter and body of esophagus resulted in impairment of lower esophageal sphincter relaxation and absent esophageal peristalsis. Dysphagia of both solid and liquid, food regurgitation and retrosternal chest pain are typical presentations. EGD is necessary in suspicious patients before making diagnosis of achalasia to exclude mechanical obstruction such as peptic stricture, and esophageal mass, and to exclude pseudoachalasia, such as adenocarcinoma of gastric cardia, especially individuals with advanced age (> 50 years old) at symptom onset, short duration of symptom (< 1 year) and marked weight loss.[1,2]

The spectrum of endoscopic findings in achalasia ranges from normal-looking esophagus to a dilated and tortuous sigmoid-like esophagus. Although there is no pathognomonic endoscopic finding of achalasia, some features are highly suggestive for achalasia, including dilatation of the esophageal lumen, abnormal liquid and/or retention of food in esophagus, thickening and whitish change of the esophageal mucosal surface, feeling of resistance at the esophagogastric junction that suddenly gives away when the endoscope popping into stomach and abnormal contraction of esophageal body. The stasis of fluid and food contents in the esophagus may cause esophagitis, mucosal erosions, and Candida esophagitis. Recent study presented a "Pinstripe pattern" of distal esophagus, the presence of longitudinal superficial wrinkles by conventional white light, staining observation using indigo carmine spraying, and narrow-band imaging, as a new predictor to detect early achalasia.[3,4]

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Endoscopic Submucosal Dissection in Esophageal Adenocarcinoma

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CASE HISTORY

A 33-year-old Thai female underwent an EGD due to a 2-month history of both solid and liquid food dysphagia. She had previously been diagnosed with gastroesophageal reflux. However, she did not respond to the treatment with PPI. EGD, using high-definition white light and NBI mode, discovered a solitary 0.7 cm firm, slightly elevated, smooth nodular lesion covered by an intact epithelium with central shallow ulceration at mid esophagus. (Figs 1&2) Esophageal biopsy was obtained. The pathological report was compatible with esophageal adenocarcinoma.

EUS revealed a 3x6 mm hypoechoic lesion in mid esophagus involving 1st, 2nd and superficial 3rd layer of esophageal wall without lymphadenopathy. Endoscopic submucosal dissection (ESD) was subsequently performed (Figs 3 - 8). The final pathology showed mucosal nodule composed of atypical round cells arranging in cribriform and polygonal pattern; confirmed adenocarcinoma with squamous differentiation with free margin. A superficial invasion of 200 μm into submucosal layer was noted.

DIAGNOSIS

Endoscopic submucosal dissection in esophageal adenocarcinoma

DISCUSSION

Esophageal adenocarcinoma is rare, accounting for only 1–4% of esophageal cancer in Asian population.[1] Adenocarcinoma of the esophagus is associated with the reflux of gastric acid into the esophagus, usually in the setting of Barrett's esophagus.[2] Endoscopic submucosal dissection (ESD) is currently considered as a curative standard treatment for esophageal carcinoma with M1 (intraepithelial) or <200 μm M2 (invasion into the lamina propria) involvement with no lymphovascular invasion, while tumors with lymphovascular invasion or deep submucosal invasion greater than 200 μm should be treated as advanced carcinomas due to the increased risk of lymph node metastasis.[3] Endoscopic ultrasound is an essential adjunct modality to delineate the depth of invasion prior to resection.[4] Overall, ESD has led to excellent *en bloc* resection rates as high as 95%-97%

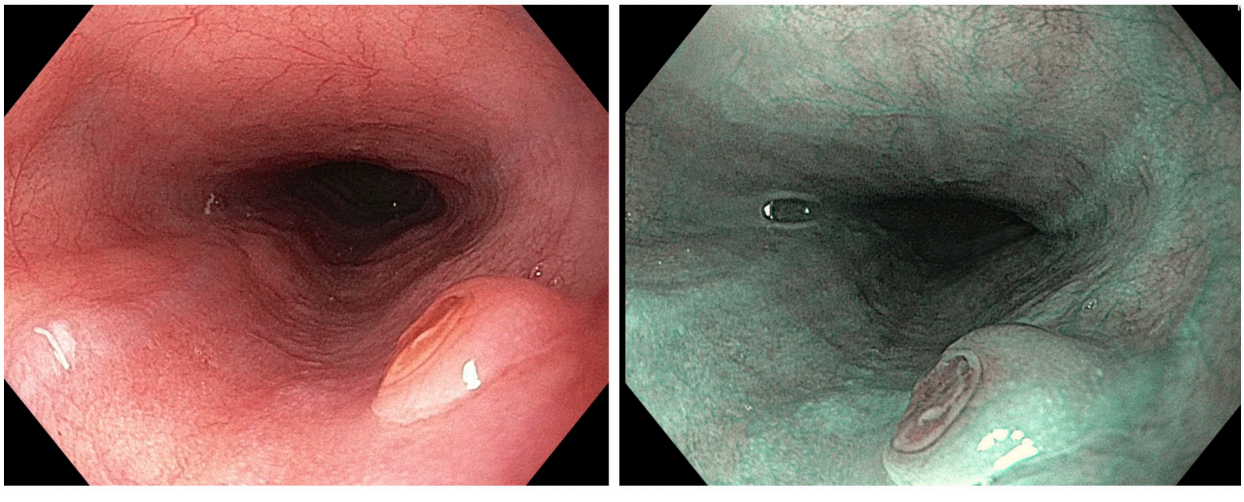
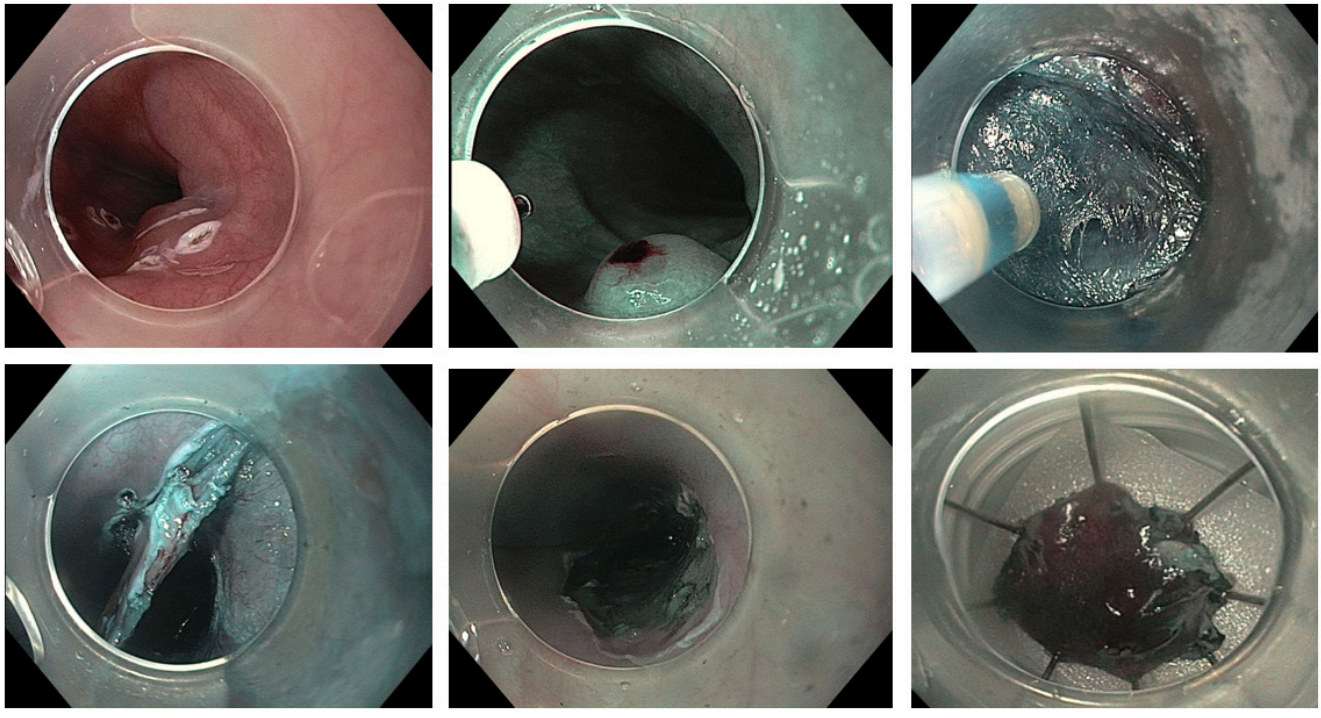


Fig 1&2. A 0.7 cm. firm, slightly elevated, smooth nodular lesion covered by an intact epithelium with central shallow ulceration at mid esophagus.



Figs 3-8. Demonstrated ESD technique starting by marking the lesional border followed by circumferential incision and dissection of the lesion.

in esophageal adenocarcinoma with an acceptable risk of bleeding, stricture, and perforation.[1] This case underscores the importance of a thorough endoscopic and endosonographic evaluation of a subepithelial lesion in the esophagus. An early diagnosis and prompt resection are the cornerstone of esophageal cancer management.[1]

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Gastric Gastrointestinal Stromal Tumor

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CASE HISTORY

A 70-year-old Thai man was referred from community hospital with the complaint of intermittent right upper quadrant abdominal pain, fatigue and anorexia for 2 months. He denied history of overt gastrointestinal blood loss, bowel habit change or family history of GI malignancy. His body weight was not change during the past 2 months. The physical examination revealed moderate pallor without palpable abdominal mass or lymphadenopathy. Complete blood count showed hematocrit of 17.4 %, white blood count of 5,600 cells/mm^3 and platelet of 420,000/mm^3. He was initially transfused with 3 units of red cell which increased his hematocrit to 36%. He was scheduled for upper endoscopy later and was discharged with twice-daily oral PPI, ferrous sulfate and folic acid. Two weeks later, EGD was performed and revealed a 4-cm round subepithelial mass with clean-based ulcer on top at upper gastric body (Fig 1). Multiple biopsies were done at the ulcer edge. The pathological result showed spindle cell neoplasm with positive immunohistochemical staining of CD117 (diffuse), CD34, SMA (focal), and desmin (focal) with negative of S100.

DIAGNOSIS

Gastric gastrointestinal stromal tumor (GIST), spindle cell type

DISCUSSION

GIST is the most common malignant mesenchymal tumor of the gastrointestinal tract. However, it accounts for less than 1% of all gastrointestinal tumors.[1] The most common site is the stomach (60-70%) which mainly involves upper body and fundus. The clinical manifestation depends on the size and the location of the tumor. Patient presentation can be varied from asymptomatic, which is the most common, to occult or overt gastrointestinal bleeding, abdominal pain, and less commonly, gut obstruction. The endoscopic findings of gastric GIST include smooth- or ulcerated-surface, oval or round, bulging mass covered with normal-appearing mucosa. Pressuring by a biopsy probe shows a firm consistency.[2,3] Typical endoscopic ultrasound (EUS) findings of GIST comprise of homogenous hypoechoic lesion originating from 4^{th} or 2^{nd} layer of gastric wall (Fig 2).[4] Calcification inside the lesion can be found resulting in EUS finding

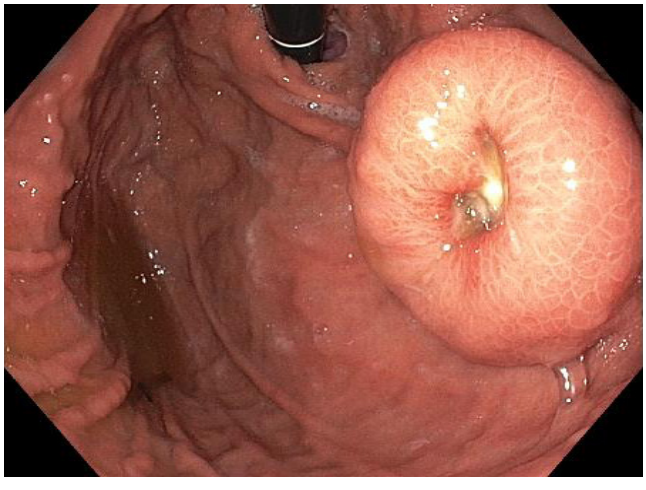

Fig 1. An upper endoscopy in retroflexion revealed a 4-cm round subepithelial mass with central ulcerated umbilication at lessor curve of upper gastric body

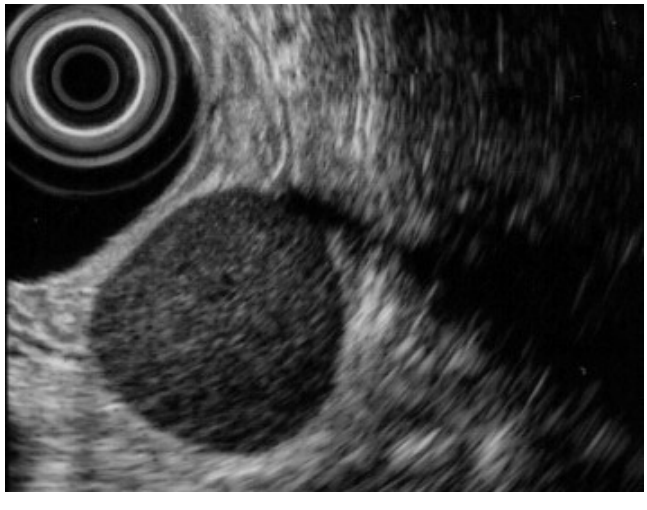

Fig 2. EUS findings revealed a round, homogeneous, hypoechoic tumor arise from the 4^{th} layer (muscularis propria) of the stomach

of heterogeneous echogenicity with cyst shadowing foci.[5] The alarming EUS features suggestive for malignant potential are tumor size larger than 5 cm, irregular extraluminal borders, local invasion, and heterogeneous appearance.[6] Histopathology of GIST is classified into 3 types as spindle cells (70%), epithelioid cells (20%), and mixed type (10%).[7] The National Comprehensive Cancer Network guidelines suggest GIST's size >2 cm or smaller with high-risk features (irregular borders, cystic spaces, ulceration, echogenic foci, heterogeneity) should undergo resection. Complete surgical resection is the treatment of choice for local and early stage. There are many modalities of endoscopic resection for gastric GIST such as endoscopic submucosal dissection (ESD), endoscopic submucosal excavation (ESE), submucosal tunneling endoscopic resection (STER) and endoscopic full-thickness resection (EFTR) depending on the size, level of muscularis propria involvement and growth pattern (intrinsic or extrinsic).[8] In case of locally-advanced, metastatic, or recurrent disease, Imatinib (a tyrosine kinase inhibitor) is the first-line treatment.[9]

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Gastric Hyperplastic Polyp

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CASE HISTORY

A 68-year-old Thai male with diabetic nephropathy and stage 4 chronic kidney disease was undergone gastroscopy for iron deficiency anemia evaluation. There was a large hyperemia, lobulated mass with some foci of erosions and ulcerations at greater curvature of stomach (Fig 1). With magnified narrow band imaging (M-NBI), there was papillary projection epithelium with whitish margin and no obviously abnormal microsurface and microvascular pattern. Biopsy was done and showed elongated, branching foveolar epithelium set in abundant stroma with cystic dilatation at the deeper part. The epithelial lining consists of single layer of the foveolar mucin-rich cells. The edematous lamina propria was infiltrated by lymphocytes and plasma cells (Fig 3).

DIAGNOSIS

Gastric hyperplastic polyp

DISCUSSION

Gastric hyperplastic polyp is the second most common polyp in the stomach following the fundic gland polyp.

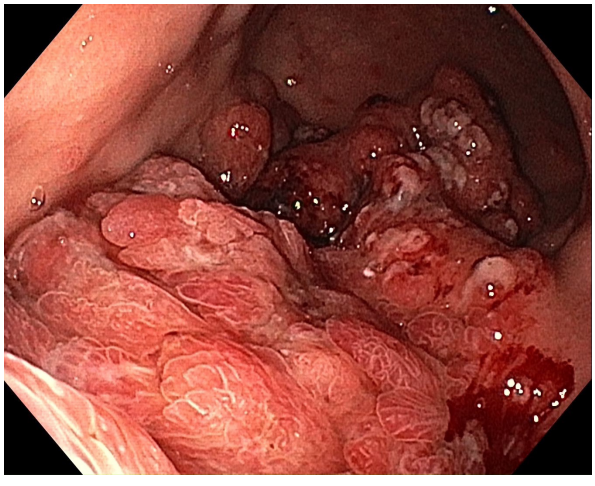

Fig 1. White light endoscopy showed a large hyperemia lobulated mass with some foci of erosion and ulceration with contact bleeding at the greater curvature of the stomach

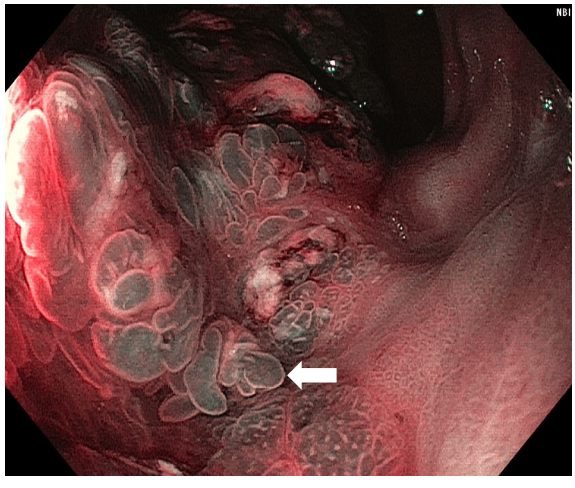

Fig 2. M-NBI showed papillary projection epithelium with whitish margin (arrows) and no obviously abnormal microsurface and mircrovascular pattern

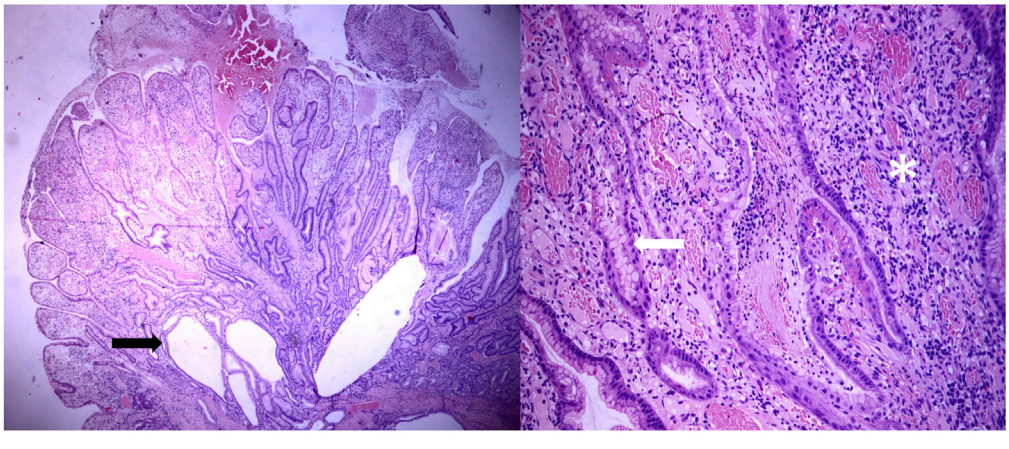

Fig 3. Histology showed elongated, branching foveolar epithelium set in abundant stroma with cystic dilatation at the deeper part (black arrow). The epithelial lining consists of single layer of the foveolar mucin-rich cells (white arrow). The edematous lamina propria was infiltrated by lymphocytes and plasma cells (white star).

The prevalence of gastric hyperplastic polyp was 13%.[1] The incidence increases with age that mostly in elderly patients at 65-75 years of age.[2] Gastric hyperplastic polyp associates with inflammation of gastric mucosa including *Helicobacter pylori (H. pylori)* infection, autoimmune gastritis, atrophic gastritis and cytomegalovirus infection.[3] Gastric hyperplastic polyp is low risk for malignancy; however, 5-37% of those has intestinal metaplasia (IM), 20% has focal dysplasia and 2-6% has adenocarcinoma.[3,4] The predictors of malignant transformation were polyp size larger than 1 cm, pedunculated feature, and post-gastrectomy.[5] The typical endoscopic finding of gastric hyperplastic polyp on M-NBI are the marginal crypt epithelium showed a white, curved morphology, forming a regular widened brownish intervening part (Fig 2), correlated with hyperemia and edematous lamina propria in histologic features. The edematous stroma causing separation of glands and containing with numerous inflammatory cell.[6,7]

We should remove the polyps causing symptoms (eg. anemia or obstruction) or the size of polyps larger than 1 cm. Biopsy at surrounding mucosa to evaluate *H. pylori* infection, atrophic gastritis and IM is recommended. If the pathology shows gastric hyperplastic polyp with dysplastic change, surveillance gastroscopy within 1 year is suggested.[8]

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Kaposi Sarcoma

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CASE HISTORY

A 40-year-old man with a history of untreated Human Immunodeficiency Virus (HIV) infection (CD4 = 47 /μL), presented with progressive dysphagia and odynophagia for 5 months. During the same period, he noticed multiple discrete violaceous papules/plaques at his trunk and extremities. Esophagogastroduodenoscopy revealed 25-mm diameter purplish lobulated mass at the pharynx (Fig 1) and various sizes of multiple dark reddish subepithelial lesions at the antrum of the stomach (Fig 2). Multiple biopsies were obtained from the antral lesions. Histopathology showed vascular and spindle cell proliferation in lamina propria, compatible with Kaposi sarcoma involving the gastrointestinal (GI) tract. Finally, this patient was treated with antiretroviral therapy combined with systemic chemotherapy.

DIAGNOSIS

Kaposi sarcoma with GI tract involvement in Acquired Immune Deficiency Syndrome (AIDS)

DISCUSSION

Kaposi sarcoma is an angio-proliferative tumor caused by human herpes virus 8 (HHV-8), first described by a Hungarian dermatologist named Dr. Moritz Kaposi in 1872.[1] Kaposi sarcoma is mostly found in nontreated HIV patients (CD4 <200/μL) but can also develop in patients with immunosuppressive therapy. Most patients with Kaposi sarcoma present with cutaneous lesions. GI tract involvement of Kaposi sarcoma is often asymptomatic and indicates poor prognosis.[2] Some clinical factors may predict the occurrence of GI tract involvement of Kaposi sarcoma including CD4 <100/μL, men who have sex with men and the presence of cutaneous Kaposi sarcoma. GI tract can be involved by Kaposi sarcoma in any part from the oropharynx to the rectum, but it occurs frequently in the stomach and small intestine. Clinical presentation includes abdominal pain, dysphagia/odynophagia, nausea/vomiting, iron deficiency anemia, GI bleeding, and rarely mechanical obstruction or bowel perforation.[3]

Endoscopically, GI tract involvement of Kaposi sarcoma ranges from a red maculopapular lesion to a darker purplish nodular or polypoid lesion. In more severe disease, patients may present with a volcano-like mass with a central umbilication or ulceration which can bleed on contact. Although the majority of GI tract involvement of Kaposi sarcoma can be identified easily on endoscopy, in some situations it may resemble common benign lesions (peptic ulcer, granulation tissue)

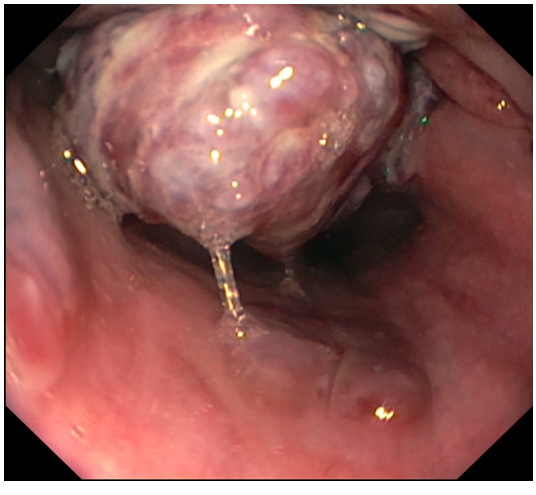

Fig 1. A 25-mm purplish lobulated mass at the pharynx

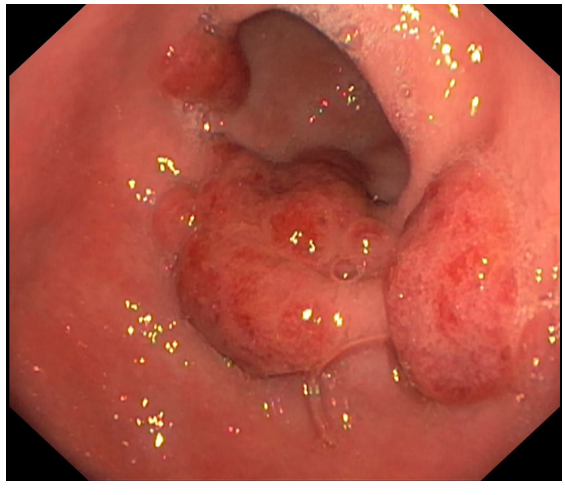

Fig 2. Various sizes of multiple reddish subepithelial lesions at the antrum of the stomach

as well as neoplasms (gastrointestinal tumor, spindle cell melanoma, angiosarcoma). Endoscopy with biopsy allows for pathological confirmation of spindle cell proliferation with HHV-8 latent nuclear antigen (HHV8 LNA) expression.[3] However, due to submucosal nature of tumor growth, endoscopically obtained biopsy may be too superficial in the absence of mucosal invasion cases. In such situations, an endoscopic ultrasound guided biopsy can increase the diagnostic yield.[4] Treatment with combination of antiretroviral therapy and systemic chemotherapy have been associated with improved morbidity and mortality in individuals with visceral Kaposi sarcoma.[3]

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Metastatic Gastrointestinal Malignant Melanoma

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CASE HISTORY

A 44-year-old man had undergone Billroth II gastrojejunostomy for 20 years due to complicated gastric ulcer, and history of black-pigment mass at right first toe and right groin node with significant weight loss for 4 months. He came to emergency department with hematemesis and syncope. EGD was performed and showed ulcerated black-pigment masses and nodules at cardia, fundus, body, afferent and efferent limb of jejunum (Figs 1-4). Biopsy was not performed due to previous confirm pathological diagnosis from right first toe and right groin node. Multiple metastases in lungs, liver and skin also suspected from the cross-sectional imaging study.

DIAGNOSIS

Metastatic malignant melanoma in GI tract

DISCUSSION

Malignant melanoma involving the GI tract may be either primary or metastatic lesion. Primary melanomas of the GI tract are lower incidence rate, more aggressive and worse prognosis.[1] The primary melanoma may arise from the neural crest cells[2] or neoplastic transformation of amine-precursor uptake and decarboxylation (APUD) cells within the gut.[3] GI Metastatic melanoma is found during endoscopic workup in 1-4% of primary cutaneous melanoma and may spreading via vascular and lymphatic system.[4] GI metastatic melanoma are often multiple and

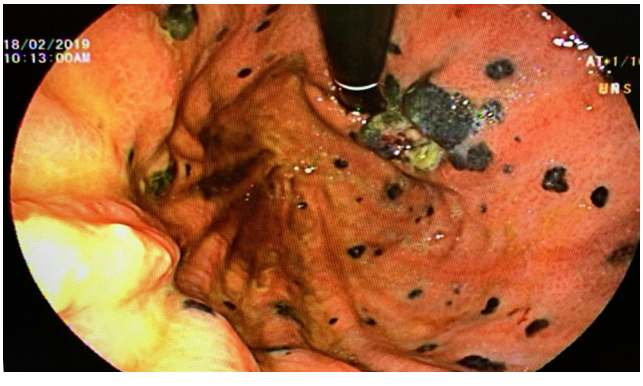

Fig 1. Multiple black-pigmented nodules at fundus and ulcerated black-pigmented masses at cardia

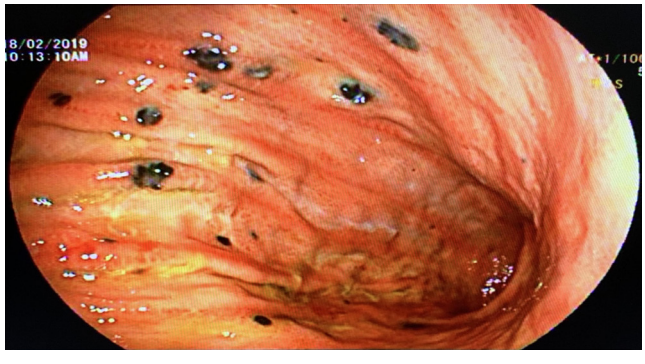

Fig 2. Multiple black-pigmented nodules at gastric body

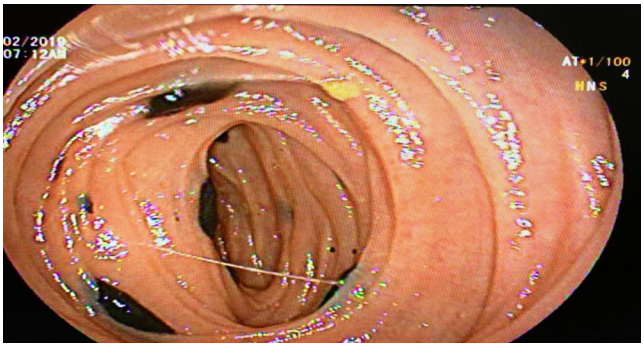

Fig 3. Multiple black-pigmented nodules at efferent limb

Fig 4. Multiple black-pigmented nodules and masses at afferent limb

can also be found extra-intestinal, different from primary GI melanoma. The common sites of GI luminal organ metastases were small bowel (35.6%), colon (28.2%), stomach (22.7%), and oral cavity and esophagus (9.3%).[5] Endoscopic finding may be presented in different form, such as large ulcerated mass, nodular lesion, submucosal mass or black pigmented lesion as in this patient. For ulcerated mass like lesion, metastasis neoplasm from breast and lung, adenocarcinoma or lymphoma should be concerned. In term of pathological diagnosis of primary mucosal melanoma is supported when precursor lesion or melanosis is shown[1], such as junctional melanocytic proliferation within the mucosa. In contrast, in case of metastatic gastrointestinal lesion that occur after spontaneous regression of primary cutaneous melanoma, the histology is associated with lymphocytic infiltration of the dermis with melanophages, vascular proliferation, and reparative fibrosis[6] Diagnosis of metastatic melanoma is generally made by radiographic contrast studies or endoscopic evaluation based on symptoms and location of the disease. Cross sectional imaging such as Computed tomography (CT) together with Positron Emission Tomography (PET) has shown to be useful, with higher accuracy of PET/CT in identifying small lesion[7] Gastrointestinal melanoma can appear as infiltrative lesion in 30%, polypoid lesion in 28%, cavitary lesion in 24%, and exo-enteric mass in 17%.[7] However, tissue biopsy is necessary to make the diagnosis and special immunohistochemical stains, including HMB-45 and S100, are useful to confirm the diagnosis of malignant melanoma[8] Treatment for metastatic melanoma includes surgical resection, immunotherapy, targeted therapy, and possibly radiation therapy to symptomatic sites. If a patient's functional status is acceptable, surgical resection should be firstly considered as it is effective in palliating symptoms and prolong survival.[9]

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Double Pylorus

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CASE HISTORY

A 50-year-old Thai male developed severe epigastric pain and heart burn for 3 months as well as an 18 kg of weight loss. He denied NSAID, tobacco and alcohol use. He had been taking only antihypertensive drugs. Physical examination was unremarkable except for only mildly pale conjunctivae. No abdominal tenderness. The blood tests showed only microcytic anemia with a hematocrit of 26%. He was treated with proton pump inhibitor (PPI) and prokinetic drugs over the past 2 months without symptom improvement.

The EGD was performed and showed two communicating channels between the antrum and the duodenal bulb and a partially healed, clean-base ulcer was seen at the mucosal ridge between the two opening channels (Figs 1&2). The gastric antrum biopsy showed only chronic antral gastritis, no evidence of *Helicobacter pylori* infection. In retrospect, he had had EGD and colonoscopy done according to iron deficiency anemia and positive fecal occult blood for 2 years ago. At that time, the EGD revealed one focal area of thin gastric wall at lesser curvature of the antrum along with diffuse erythematous change of surrounding mucosa (Fig 3).

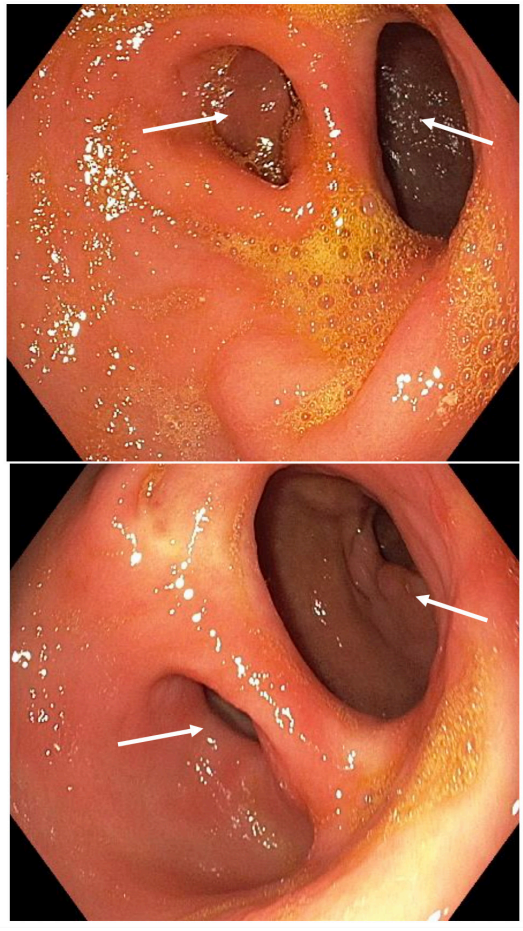
DIAGNOSIS

Acquired double pylorus

DISCUSSION

Double pylorus has many synonyms such as pyloric duodenal fistula, gastroduodenal fistula and double channel pylorus. It was classified into congenital or acquired double pylorus. There are no specific gastrointestinal symptoms for double pylorus. The patients may present with abdominal discomfort, chronic dyspepsia, vomiting, or gastrointestinal bleeding.[1-3]

Most cases of double pylorus were accidentally found from upper endoscopy with the reported prevalence between 0.001% to 0.4%.[4] Diabetes mellitus, cirrhosis, chronic renal failure, and systemic lupus erythematosus, and corticosteroid or NSIAD use had been reported to be associated with double pylorus.[5-8] The differential



Figs 1&2. Two communicating channels between pylorus and duodenal bulb, compatible with double pylorus (white arrow).

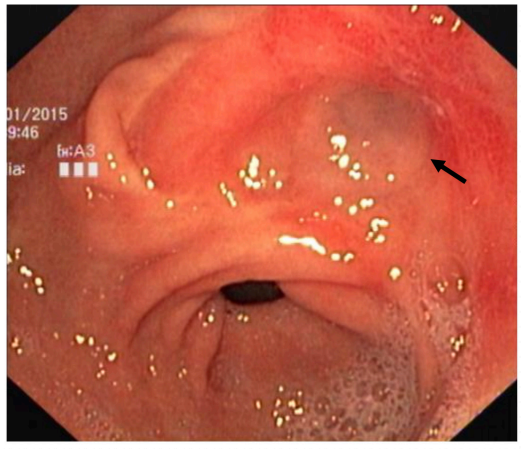


Fig 3. Image of prior EGD approximately 2 years from current presentation, showed generalized erythematous mucosa and one focal thin mucosa, almost being fistula (black arrow).

diagnoses were congenital double pylorus and gastric diverticulum. When performing side-viewing endoscopy such as ECRP and EUS, care must be considered while intubating through pylorus in patients with this rare entity to avoid serious complications especially perforation.

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Foreign Body Removal

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CASE HISTORY

A 43-year-old Thai female with bilateral temporomandibular joint ankylosis causing severe trismus and mandibular deformity. She underwent dental procedure under general anesthesia. During the endotracheal tube exchange procedure, the plastic tube exchanger was accidentally dropped into the right main bronchus (Fig 1). The pulmonologist has attemped to remove the plastic tube via bronchoscopy. Unfortunately, the tube was broken and parts of them were ingested by patient. Three hours after ingestion, gastroenterologist was consulted for removal of foreign body under general anesthesia. Abdominal radiography demonstrated several pieces of the plastic tube in her stomach and three pieces of the fragmented tube already passed to small intestine (Fig 2). The upper endoscopy revealed two fragments of plastic tube, size 0.5 x 3 cm, in the duodenal bulb (Fig 3). Multiple attempts of snare retrieval was done, however this caused more fragmentation, then a retriever net basket (Roth Net, US endoscopy, Ohio, USA) (Fig 4) was used and successfully removed all fragments. Follow up abdominal imaging demonstrated spontaneous migration of the residual fragments (Fig 5).

DIAGNOSIS

Foreign body removal

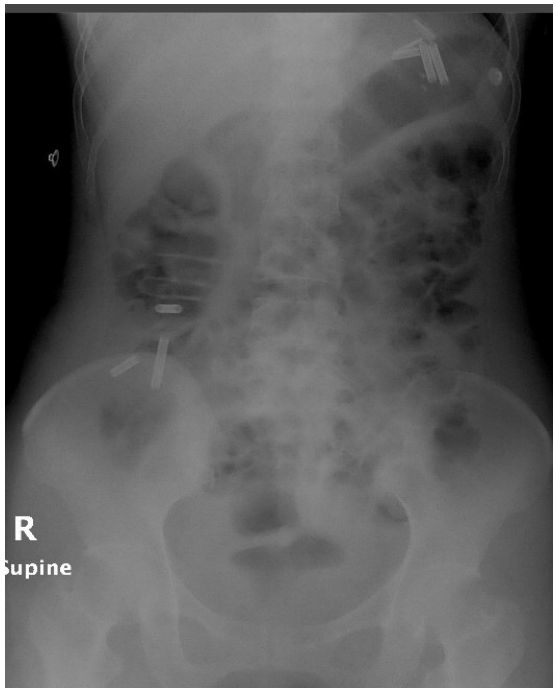

Fig 2. Abdominal radiograph showed several fragments of plastic tube in stomach and three pieces of plastic tube in small intestine

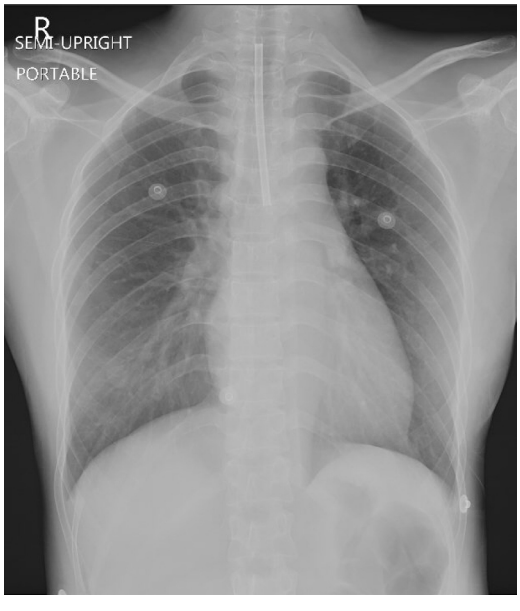

Fig 1. Chest x-ray demonstrated plastic tube in patient's bronchus

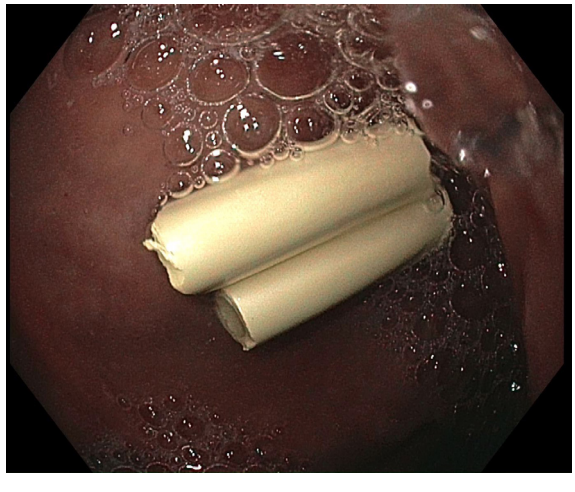

Fig 3. Two fragments of plastic tube found in duodenal bulb

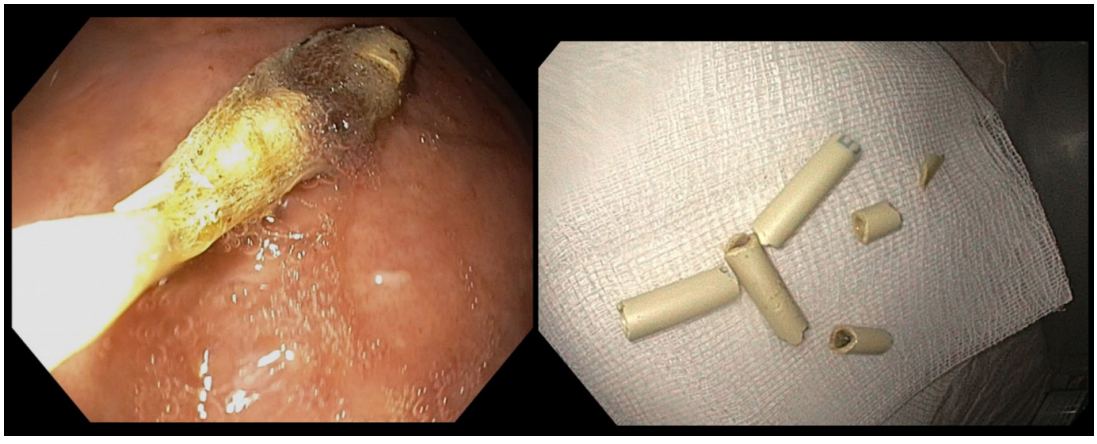

Fig 4. Successful removal with retriever net basket

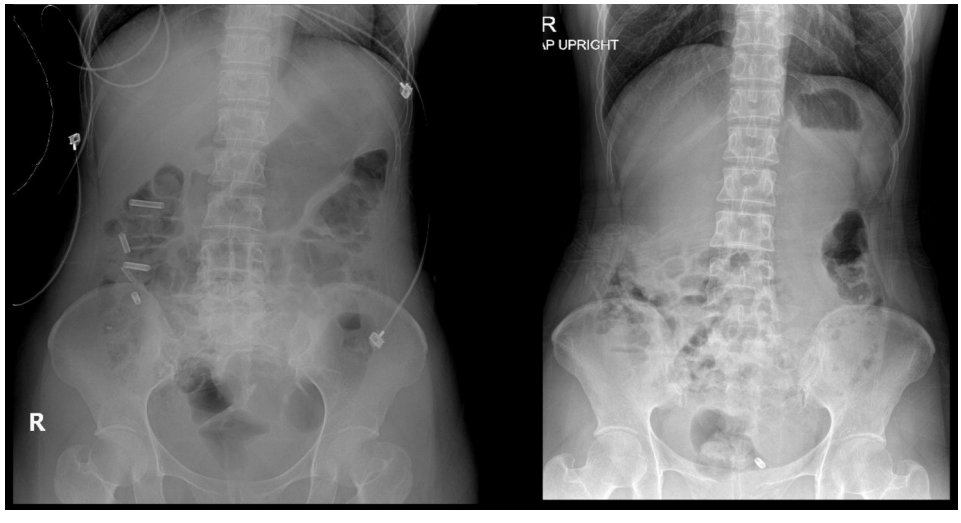

Fig 5. Follow up plain radiographs demonstrated propagation of the rest of foreign body pieces.

DISCUSSION

Although the majority of foreign bodies in upper gastrointestinal tract can pass spontaneously[1], up to one-third of them especially a sharp point object in stomach can lead to complication.[2] In this patient, endoscopic removal was attempted because of the sharp edge of the plastic tube fragments. Before endoscopic removal, the airway protection was crucial especially in this patient according to the underlying condition, therefore, endotracheal intubation was indicated. According to the ESGE guideline, the proper timing of endoscopy to remove the sharp point object in stomach should be within 24 hours.[3] Selection of suitable devices for objects removal depends on the type and the location of the foreign body (3). In this patient, net basket was used in order to grab multiple pieces object at a time and it could also prevent esophagogastric junction and pharyngeal damage.

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Gastric MALT Lymphoma

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CASE HISTORY

A 74 years old lady with underlying of type 2 diabetes, rheumatoid arthritis, and interstitial lung disease presented to the division of gastroenterology due to anemia. Esophagogastroduodenoscopy (EGD) was performed and a well-defined mucosal discoloration area located at the greater curvature of the stomach was identified (Fig 1). Narrow band imaging was applied and showed loss of mucosa surface with dilated superficial vasculature (Fig 2). Mucosal biopsy showed atypical lymphoproliferation. Immunohistochemistry showed CD20 (+), CD3(-), CD43 (+), Bcl6 (-), Bcl2 (+), CyclinD1 (-), CD5(-), CD10(-), CD23(-), Kappa(-), Lambda(-), and Ki67 (+, 5-10/HPF) compatible with extra nodal marginal zone lymphoma of mucosa-associated lymphoid tissue (MALT lymphoma). *Helicobacter pylori* testing both on histology and serology were negative and the patient was referred to the hematologist for chemotherapy after failed antibiotics triple therapy.

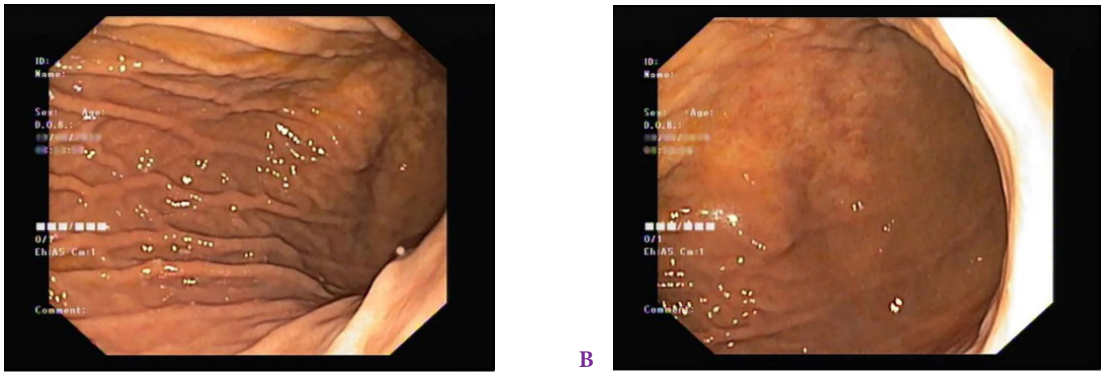
DIAGNOSIS

Gastric MALT lymphoma

DISCUSSION

Gastric MALT lymphoma is the most common type of MALT, which is a subtype of primary extranodal non-Hodgekin lymphoma.[1] It has a strong correlation with *H. pylori* infection, with 92- 98% *H. pylori* detection in surgical specimen.[2] On the other hand, the pathogenesis of *H. pylori* negative gastric MALT lymphoma is associated with genetic variations, other kinds of infection, or autoimmune disease. As a result, response to antibiotics is much less in *H. pylori* negative disease (28% versus 75% in *H. pylori* positive).[3] Since most of gastric MALT is indolent, the clinical presentation of gastric MALT is poorly specific and might range from pain, loss of appetite and weight loss to vomiting or bleeding.[4]

Most common location of gastric MALT is the antrum but may occur in any part of the stomach.[5] Endoscopic findings such as ulceration, exophytic, hypertrophic mucosa, petechial hemorrhage, mixed pattern or even normal appearing mucosa were described.[6] Using Magnify imaging, 75% of patients with gastric MALT showed disappearance of glandular structure, irregular branching abnormal vessels or alteration of connecting venule. These mucosal changes disappeared after successful treatment of MALT either by chemotherapy, antibiotics or radiation.[7,8] These microstructural changes sometimes resemble diffuse type gastric cancer and tissue biopsy is necessary to make the diagnosis.[8] Apart from luminal endoscopy or computed tomography, the depth of involvement and locoregional lymph node could be evaluated by endoscopic ultrasound.[9] When comparing between different endoscopic appearances, polypoid lesion was associated with longer time to achieve complete remission and higher recurrence when compared with infiltrative or ulcerative type.[10]



A B

Fig 1. White light imaging showed focal mucosal discoloration of the gastric body mucosa in far view (A) and close up view (B)

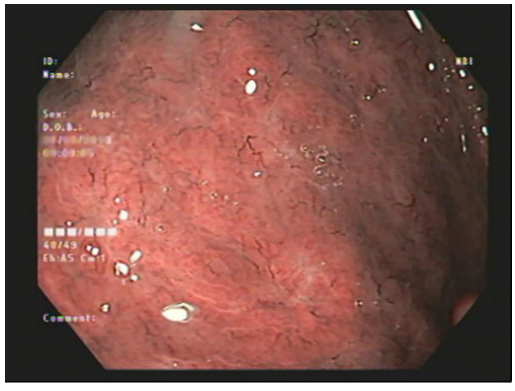


Fig 2. On NBI mode, the lesion showed loss of mucosal pattern with increased irregular vasculature

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Combined Biliary and Duodenal Obstruction from Pancreatic Adenocarcinoma

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CASE HISTORY

A 74-year-old male presented with vomiting suspecting for gastric outlet obstruction (GOO). He had a past history of metastatic pancreatic adenocarcinoma at head and uncinated process of the pancreas. He had previously been treated with a biliary self-expandable metal stent 6 months ago (SEMS) (Fig 1). Physical examination revealed abdominal distension and positive splashing sign without icteric sclera. Plain abdominal film showed plenty amount of gastric content suspected of GOO (Fig 2). Given advanced stage of the primary disease of pancreatic adenocarcinoma, a duodenal stenting as a palliative treatment procedure for duodenal obstruction was scheduled. Upper endoscopy demonstrated supra-ampullary malignant duodenal obstruction. A double channel therapeutic gastroscope (GIF2TH180; Olympus Medical Systems Corp, Tokyo, Japan) was used. Passage through stenotic lumen was successfully performed with a Bending cannula catheter (Swingtip, Olympus America Inc, Lehigh Valley, Pennsylvania,USA) and a 0.035 guide wire (Acrobat2; Wilson-Cook Medical Inc., Winston-Salem, NC, USA) was used to pass through lumen of the catheter across the obstruction. The guide-wire was then removed. Contrast media was injected for assessing position and extension of the obstruction, any further obstruction distally could then be excluded. A 0.035 guide-wire (Jagwire; Boston Scientific, Natick, MA, United States) was passed through the catheter to lie with its tip as distal as possible. The catheter was removed and delivery system of the self-expandable metal stent (SEMS) was placed over the wire, the Uncoverd SEMS (UCSEMS) 90 x 22 mm. (The WallFlex™, Boston Scientific Co., Na-tick, Mass., USA) was placed over the GW. Precise positioning of the SEMS before and during deployment was shown fluoroscopically in real time. Subsequent CT abdomen confirmed proper position of the SESMS (Fig 3).

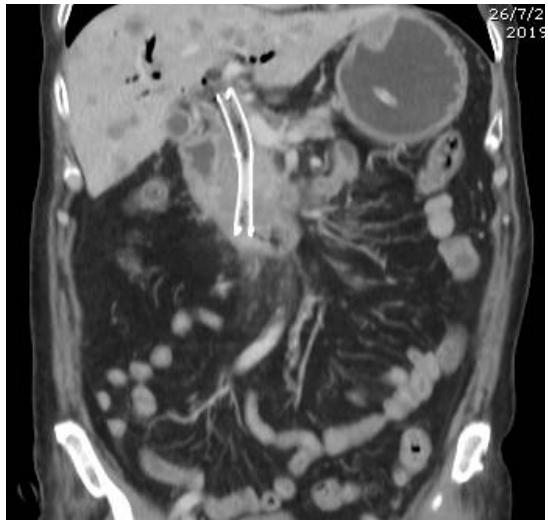

Fig 1. This figure demonstrated a biliary self-expandable metal stent (SEMS) located across pancreatic mass obstructing distal common bile duct. Air seen in both left and right intrahepatic bile ducts confirm patency of the SEMS

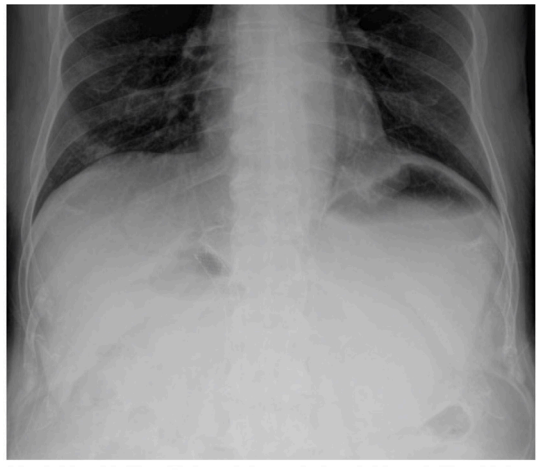

Fig 2. This figure demonstrated plenty amount of gastric content and a biliary self-expandable metal stent (SEMS) in place. Given clinical history of persistent symptom of vomiting with positive splashing sign, this film was suggestive for gastric outlet obstruction

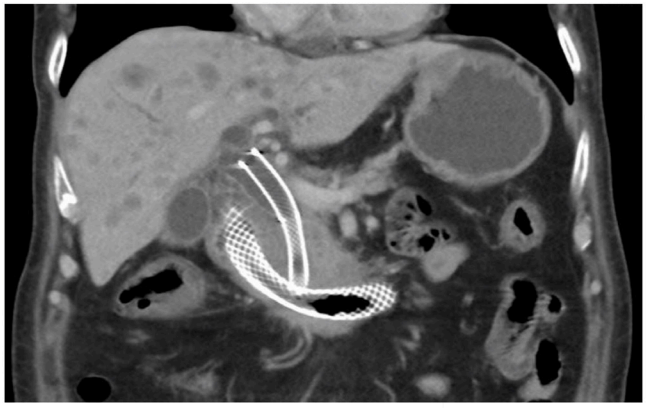

Fig 3. This figure demonstrated a biliary self-expandable metal stent and a duodenal stent in place. Both stents were in proper position to resolve problems of biliary and duodenal obstruction

DIAGNOSIS

Combined biliary and duodenal obstruction from pancreatic adenocarcinoma

DISCUSSION

Pancreatic cancer is one of the most common causes of GOO with incidence of 15-20% from literature.[1] A retrospective study of 36 patients with malignant GOO treated with endoscopy found that 86% of patients required single stent at the initial therapy. Fifty-three per cent was nil per os before treatment, and only 3% was still nil per os after treatment. Twenty-five per cent required re-intervention. Forty-four per cent had concomitant or subsequent biliary obstruction.[2] In this current case, the patient started his symptom with biliary obstruction followed by duodenal obstruction and eventually successfully treated with duodenal stenting. Surgical bypass is generally effective with operation related mortality rate lower than 10% despite relatively high rate of morbidity at one third of cases. Surgical bypass is hence practically preserved for patients whose anticipated survival time is longer than 6 months.[1]

In patients with combined biliary and duodenal obstruction, prognosis is poorer than patients with biliary obstruction alone. Management of such kind of patients are more complex. A study of 64 patients with combined biliary and duodenal obstruction done by Mutignani, M et al. showed that biliary obstruction occurred before concurrently, and after onset of duodenal obstruction in 46, 14, and 4 patients, respectively with success rate of combined endoscopic stenting at 100%, 86%, and 100%, respectively. Location of duodenal obstruction was proximal (n=31), adjacent (n=25), and distal to the papilla (n=8). Early and late complication occurred at 6% and 16%, respectively. Median survival time was 81 days (range 2-447 days).[3] Mutignani, M et al. has classified level of duodenal obstruction in relationship to biliary obstruction as Type I; stenosis occurs at the level of the duodenal bulb or upper duodenal genu but without involvement of the papilla. Type II; stenosis affects the second part of the duodenum with involvement of the major papilla. Type III; stenosis involves the third part of the duodenum distal to and without involvement of the major papilla.[3] In this current case, the patient had combined biliary and duodenal obstruction with Type I stenosis and was successfully treated with double stenting.

A multicenter, randomized trial from The Netherlands compared results of stent placement versus gastro-jejunostomy (GJJ) for management of GOO in 21 and 18 patients, respectively. Median time to improve food intake was more rapid in patients underwent stenting than GJJ; 8 versus 5 days, P<0.001, respectively, but time of long-term relief was shorter for stenting; 72 versus 50 days, P=0.05, respectively. Major complication occurred more in stenting than GJJ group; 6 in 4 patients versus 0; P=0.02, respectively, as well as recurrent obstructive symptoms (8 in 5 patients versus 1 in 1 patient; P=0.02), respectively, and re-intervention (10 in 7 patients versus 2 in 2 patients; P<0.01), respectively. No differences in median survival (stent: 56 versus GJJ: 78 days) and quality of life. Given the fact that that stenting provided faster response but GJJ provided better long-term results, patients with life expectancy longer than 2 months should receive GJJ rather than duodenal stenting.[4] In this current case, as expected survival time of the patient was shorter than 2 months, we then decided to place a duodenal stent rather than surgical bypass.

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Hepatocellular Carcinoma Invading Duodenum

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CASE HISTORY

A 63-year-old male with compensated alcoholic cirrhosis and recurrent hepatocellular carcinoma (HCC) presented with passing melena for 2 days. The physical examination showed stable vital signs, pale conjunctivae and massive ascites. Nasogastric lavage revealed neither coffee-ground nor bile content. His hemoglobin level was at 11 g/dL.

EGD was performed and showed a 3 cm bulging subepithelial lesion with multifocal inflammations at posterior wall of the duodenal bulb with spontaneous blood oozing. There was a clean based ulcer at the opposite site of subepithelial lesion (Fig 1). Subsequently, CT scan of abdomen with contrast revealed the HCC occupying left, right and caudate lobe of liver invading the posterior aspect of pylorus and duodenal bulb (Fig 2). The extensive tumor thrombus in right, left, and main portal vein was noted (Fig 3). Due to advanced stage of HCC and the poor performance status of the patient, the aim of treatment was best supportive care.

DIAGNOSIS

HCC invading to duodenal bulb presented with upper gastrointestinal hemorrhage

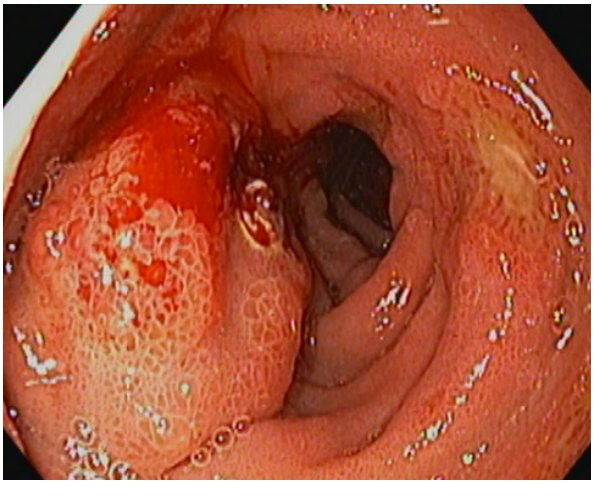

Fig 1. Subepithelial lesion with blood oozing at posterior wall of duodenal bulb.

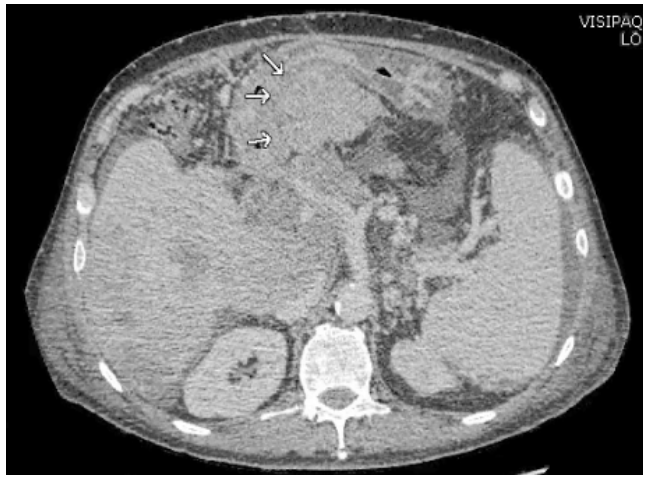

Fig 2. HCC invade wall of duodenal bulb (white arrows)

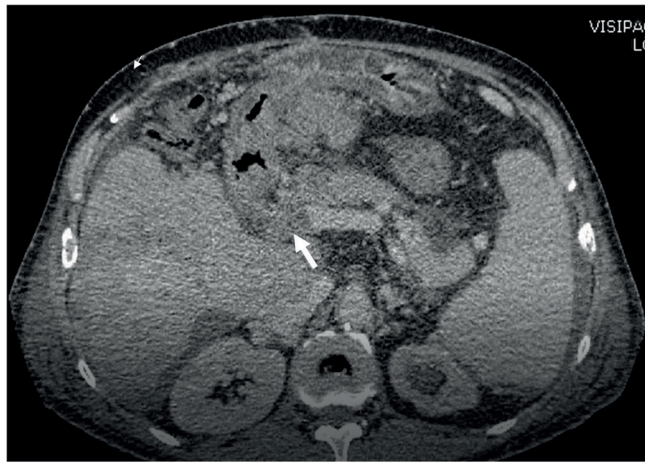

Fig 3. Tumor thrombus at main portal vein (white arrow)

DISCUSSION

Tumor bleeding is not a common cause of upper gastrointestinal hemorrhage (UGIH) which the reported incidence was at 3.7% .[1] The most common cancer presented with UGIH is gastric cancer (73 %), followed by esophageal cancer (16%) and malignancy at duodenum (11%)[2] which are mostly from direct invasion or metastasis.[3] Hepatocellular carcinoma(HCC) invading duodenum with UGIH is rare (only 0.5-2% of overall HCC cases).[4]

The characteristics of HCC with duodenal invasion include 1) tumor size more than 5 cm (67-100%), 2) tumor location at right lobe (30-75%) and 3) portal vein invasion (25-100%).[4-6] Half of patients (56%) with UGIH had active bleeding during EGD of which most lesions was ulcerative lesion for 81%.[4] Treatment option depends on the staging of primary cancer.[4] The prognosis is poor with overall survival ranging from 3 weeks to 4 months.[4-6]

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Duodenal Varices

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CASE HISTORY

A 41-year-old male, a heavy alcohol drinker, presented with a 1-day history of hematochezia. At the emergency room, he developed hypovolemic shock, and the nasogastric lavage showed fresh blood. Physical examination revealed evidence of chronic liver disease, including spider nevi, palmar erythema, and parotid gland enlargement. After initial resuscitation, he underwent upper endoscopy, which showed esophageal varices without bleeding stigmata, and fresh blood was seen in the duodenal bulb and second part of duodenum. Nonetheless, the bleeding site could not be identified. Push enteroscopy was undertaken, which demonstrated an actively bleeding duodenal varix with white nipple sign in the third part of the duodenum. Histoacryl was injected, and hemostasis was achieved (Fig 1). He received five days of intravenous octreotide, and subsequent non-selective beta-blocker to achieve optimal heart rate. He had no recurrent bleeding after six months of follow-up.

DIAGNOSIS

Bleeding duodenal varix

DISCUSSION

Ectopic varices are described as the dilated portosystemic collateral veins located in unusual sites other than the gastroesophageal region.[1] Bleeding ectopic varices accounts for 1-5% of all variceal bleeding.[2] Seventeen percent of ectopic varices occur at the duodenum.[3] Common underlying causes of duodenal varices are cirrhosis, the previous history of esophageal varices ligation, and extrahepatic portal hypertension.[4] Hematemesis and hematochezia are the common presentations, however obscure gastrointestinal bleeding and iron deficiency anemia have been reported.

Endoscopy is the best diagnostic tool for duodenal varices characterized by the appearance of dilated tubular structures at the duodenum. The presence of red wale or white nipple sign is indicative of high bleeding risk. Push enteroscopy, balloon-assisted enteroscopy, and computed tomography (CT) angiography can be useful for diagnosis.[5]

Recommendations for the management of duodenal varices are based on case series. Somatostatin or its analog intravenous infusion for 3-5 days may be beneficial, and

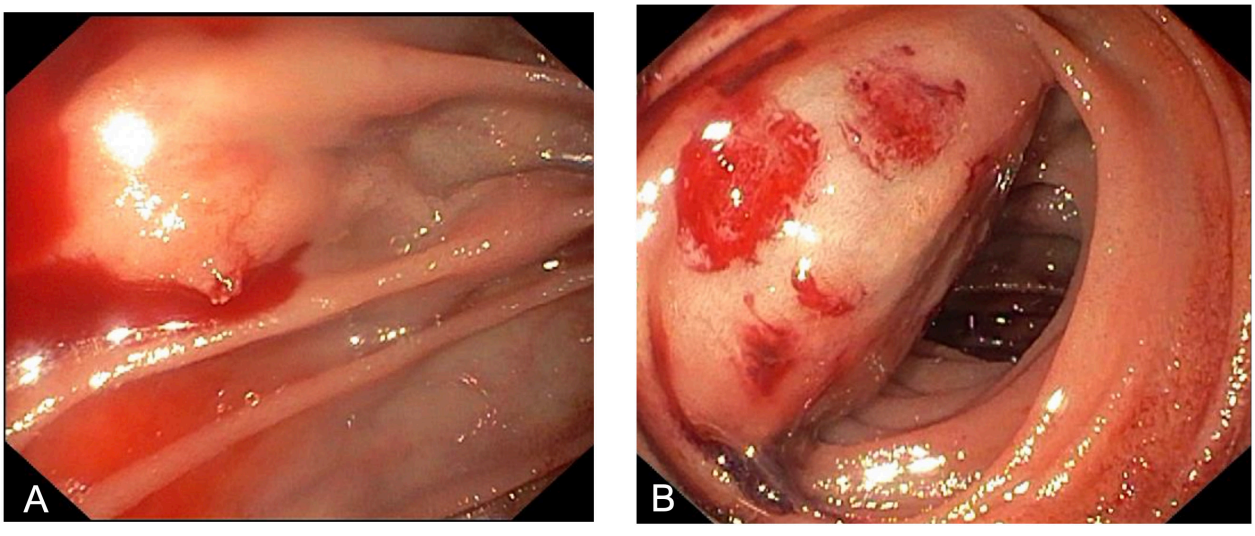

Fig 1. A. Bleeding duodenal varix at the third part of duodenum B. Successful hemostasis after histoacryl injection

endoscopic interventions are the first therapeutic modality for hemostasis. Endoscopic band ligation, sclerotherapy injection, and clipping are reported to have reasonably good technical success in several studies.[6] Angiographic interventions and surgical treatment can be considered in patients who failed endoscopic therapy.

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Cytomegalovirus Ileitis

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CASE HISTORY

A 45-year-old Thai woman was referred from a community hospital due to persistent watery diarrhea for 3 weeks despite receiving oral antibiotics. She also had intermittent low-grade fever and mild colicky abdominal pain. Her underlying disease was acquired immune deficiency syndrome (AIDS) of which the presentation was *Pneumocystis jiroveci* pneumonia 3 months before her diarrhea. Current medications were 3TC, TDF, EFV and prophylactic drugs for opportunistic infections (Azithromycin, Co-trimoxazole and Fluconazole). Physical examination revealed only low-grade fever without lymphadenopathy or hepatosplenomegaly. Initial septic work up showed red blood cells 0-1 cells/HPF, white blood cells 0-1 cell/HPF in stool without parasites. Stool culture yielded no growth. Since she was not responsive to ceftriaxone, metronidazole and vancomycin, colonoscopy was performed one week later which showed severe inflamed mucosa with edema at the ileocecal (IC) valve (Fig 1). No patulous IC valve was found. The mucosa of the terminal ileum was circumferentially covered with whitish exudate and multiple severely inflamed ulcers (Fig 2). Biopsies were done at inflamed ulcers and were sent for histopathology and special laboratory tests. Histopathology showed eosinophilic intranuclear inclusion bodies with eosinophilic to basophilic intracytoplasmic inclusion bodies in the tissue from the terminal ileum and IC valve, consistent with Cytomegalovirus (CMV) infection (Fig 3). Acid fast stain (AFB), modified AFB, the polymerase chain reaction for TB and non-tuberculous mycobacteria (NTM) were negative. Culture for TB or NTM yielded no growth.

DIAGNOSIS

CMV ileitis

DISCUSSION

CMV causes infectious disease in various parts of gastrointestinal tract particularly in the esophagus and the colon. It is commonly found in the immunocompromised hosts such as AIDS, organ transplantation, hematologic malignancy, cancer therapy and steroid usage.[1] Common clinical manifestations of CMV colitis in HIV-infected patients are diarrhea, fever and weight loss.[2] CMV colitis

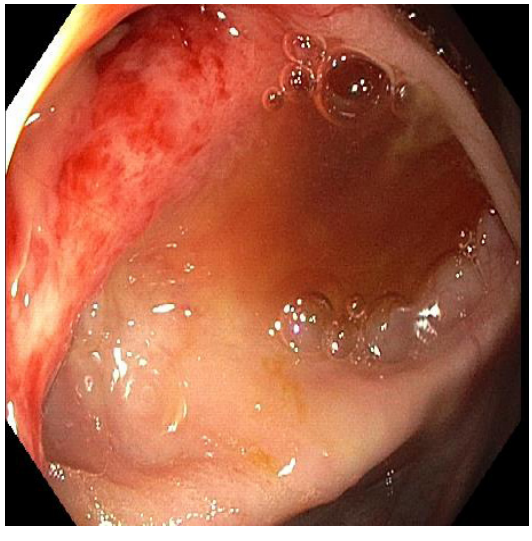

Fig 1. Colonoscopy showed severe inflamed mucosa with edema at ileocecal valve

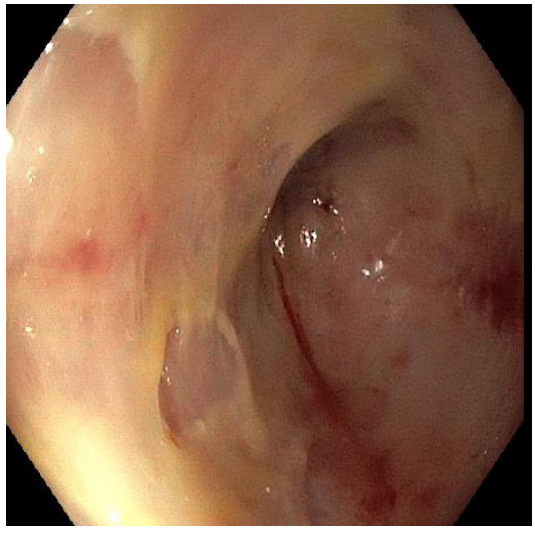

Fig 2. Colonoscopy revealed circumferential whitish exudative covering mucosa at terminal ileum with some area of inflamed mucosa

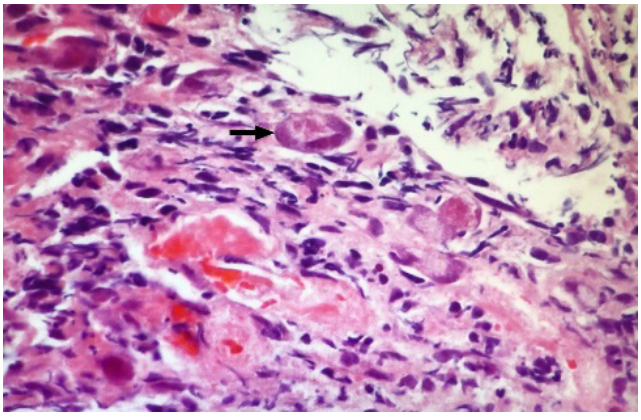

Fig 3. Histopathology showed intranuclear inclusion bodies with eosinophilic to basophilic intracytoplasmic inclusion bodies (black arrow)

is one of AIDS defining illnesses, accounting for about 25%.[2] Half of HIV patients who were diagnosed as having CMV colitis had homosexuality and mean CD4 was 127.92/mm^3.[3] The endoscopic findings of CMV colitis in patients without inflammatory bowel disease are usually non-specific; ulcer could be commonly found in 70% especially the well-demarcated small ulcer and the semi lunate ulcer.[4] Other findings include cobblestone appearance, tumor and polyp like lesions.[4] CMV can be also found on normal colonic mucosa.[2] Hematoxylin-and-eosin staining shows typical viral inclusion. Gold standard for definite diagnosis is the identification of CMV specific immunohistochemistry in tissue biopsy.[5] Differential diagnoses are other infectious colitis (TB colitis), drug-induced colitis and colon cancer. Ganciclovir is the mainstay of its treatment.

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Appendiceal Inversion

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CASE HISTORY

A 50-year-old female, with history of total abdominal hysterectomy, bilateral salpingo-oophorectomy and appendectomy about 7 years ago, presented for screening colonoscopy. Physical examination was unremarkable. Colonoscopy showed a 0.5 x 2 cm, long tubular structure at appendiceal orifice (Fig 1). The consistency of this structure was soft and the overlying mucosa including surface pattern appeared unremarkable under white light and blue laser imaging (BLI). Appendiceal inversion was diagnosed. However, CT scan of lower abdomen and endoscopic ultrasound (EUS) were performed to exclude appendiceal tumors. CT scan showed a small tubular structure within the cecal lumen below the ileocecal valve (Fig 2). EUS using a radial echoendoscope (EG-580 UR, Fujifilm, Tokyo, Japan) showed target-like appearance which contained 5 distinct layers compatible with appendix. There was no tumor identified.

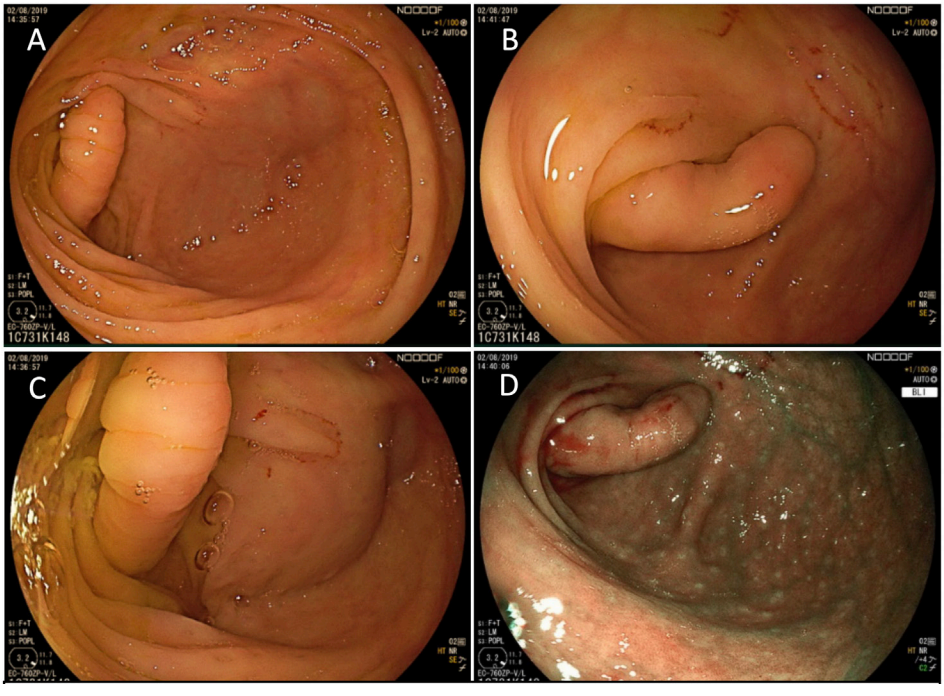

Fig 1. A-C : Colonoscopy showed tubular-like lesion size 0.5 x 2 cm at appendiceal orifice, normal overlying mucosa, soft consistency. **D** : BLI showed similar surface pattern with surrounding mucosa.

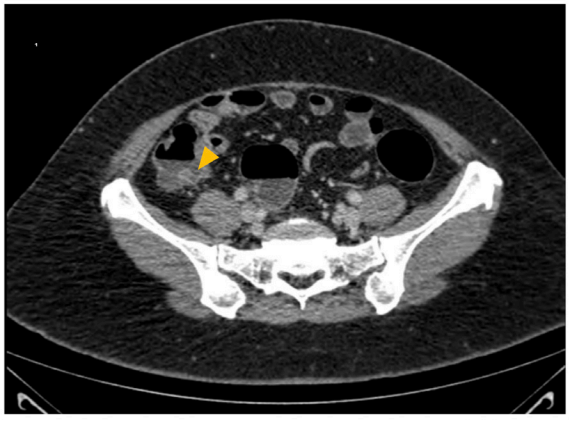

Fig 2. CT lower abdomen showed a small tubular structure within the cecal lumen below level of the IC valve (yellow arrow)

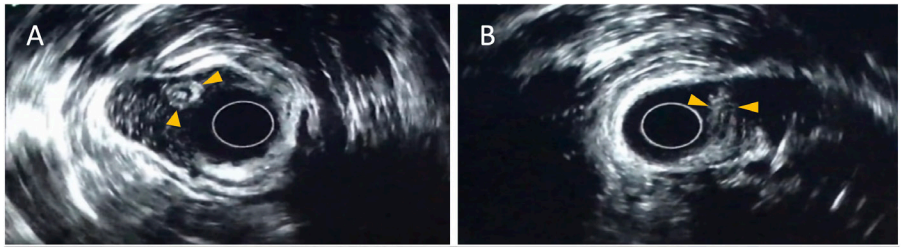

Fig 3. Radial EUS showed multiple internal hypo-hyperechoic layers with target-like appearance (axial view – Fig 3A), tubular structure consists of 5 distinct layers (saggital view – Fig 3B), Consistent with appendiceal inversion (yellow arrows)

DIAGNOSIS

Appendiceal inversion

DISCUSSION

Appendiceal inversion (AI) describes as an invagination of the vermiform appendix into the cecal lumen. AI should be recognized and differentiated from other cecal pathologies such as polyps or neoplasms.[1] Patients with AI were predominantly women in 5th decade of life.[2] Majority of reported AI were found after open appendectomy with traditional inversion-ligation technique, however asymptomatic AI without prior history of appendectomy has been reported.[1] The gross appearance of AI can be recognized during colonoscopy. The mucosal appearance of AI was indistinct from the surrounding colon mucosa without neoplastic mucosal/pit pattern. CT scan of the abdomen was used to confirm an absence of underneath appendiceal tumors. Endoscopic ultrasound examinations either by a forward-viewing echoendoscope or a miniprobe inserting through the colonoscope provided a detail of wall-layer of the AI, which normally appeared as a 5-layer pattern similar to colon mucosa, and can be used to exclude possible underlying tumor or pathology. Reversal or resection of the AI should be avoided, because of its benign nature and a risk of peritonitis associated with surgical management.[1]

One of differential diagnosis is appendiceal intussusception. Although, intussusception of the appendix was infrequent and had an incidence of 0.01% among patients undergoing appendectomy. Presences of intramural or intraluminal lesions such as parasites, fecoliths, foreign bodies, neoplasms (polyps, mucoceles, adenocarcinomas, carcinoid tumors), hypertrophic lymphoid follicles, mucoceles, endometrial implants and postinflammatory scarsproduces acted as a leading point to intussusception. The treatment of choice in appendiceal intussesception is surgical removal of the appendix either by laparotomy or laparoscopically.[3]

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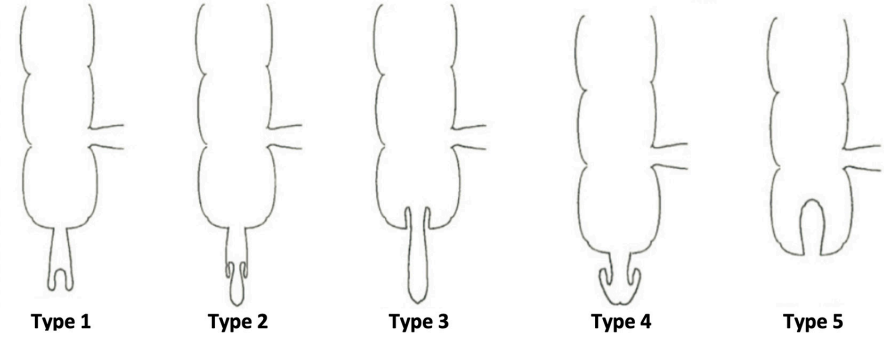

Type I: The tip of the appendix forms the intussusceptum and is invaginated into the proximal appendix, which is the intussuscipiens
Type II: The invagination starts at some point along the length of the appendix. The intussuscipiens is the adjacent tissue
Type III: The invagination starts at the junction of the appendix and caecum. The caecum is the intussuscipiens
Type IV: This is retrograde intussusception, where the proximal appendix is invaginated into the distal appendix

Fig 4. Classifications of appendiceal intussusception (modified from Varsamis N, et al, 2012)

Appendiceal Endometriosis

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CASE HISTORY

A 48 years old female with history of iron deficiency anemia due to myoma uteri with hypermenorrhea presented with one-year history of abdominal discomfort and bloating. She denied history of melena, hematemesis, or hematochezia. Physical examination showed stable vital signs, mild pale conjunctiva with unremarkable abdominal and rectal examination including normal stool. Her gynecological examination revealed left ovarian cyst which was planned for surgical removal. The initial investigation showed white blood cell count of 4950 × 106/L with neutrophil predominance, hemoglobin of 12.6 g/dL, platelet count of 217000 × 106/L and INR of 1.06. Blood test for kidney and liver function were within normal limit. She underwent colonoscopy as a part of work up for anemia and chronic abdominal pain. The colon appeared normal, except for the mass found in cecum (Fig 1). The appendix was inverted and enlarged from the underlying subepithelial mass. Mucosal biopsy was done which revealed chronic appendicitis. She then undergone CT scan of the abdomen, which revealed appendiceal intussusception associated with intraluminal enhancing soft tissue mass occupying the entire appendix, measured about 1.5 cm in diameter and 3.3 cm in length (Fig 2 & 3). Surgical consultation was obtained regarding appendiceal neoplasm. Right hemicolectomy with left salpingo-oophorectomy was performed. The intraoperative and postoperative courses were uneventful. Histology revealed endometrial glands and stroma located along submucosa to subserosa area with some area of hemorrhage (Fig 4), diagnostic of endometriosis.

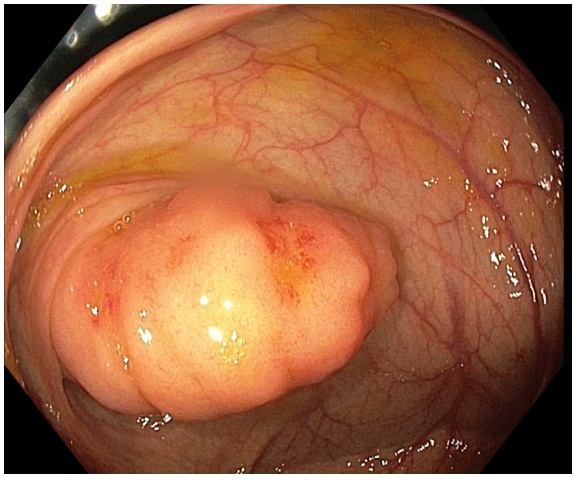

Fig 1. Inverted and enlarged appendix with subepithelial mass

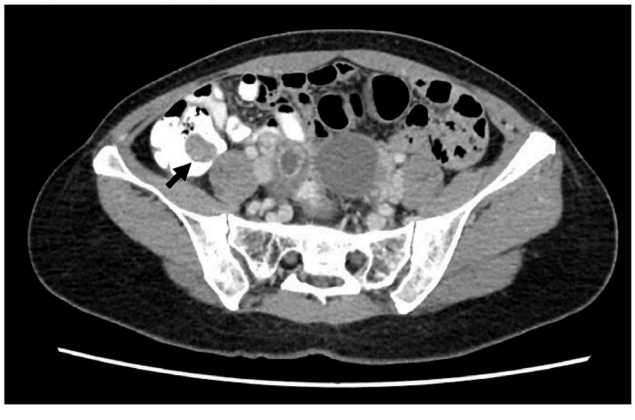

Fig 2. Appendiceal intussusception associated with intraluminal enhancing soft tissue mass occupying entire appendix (arrow)

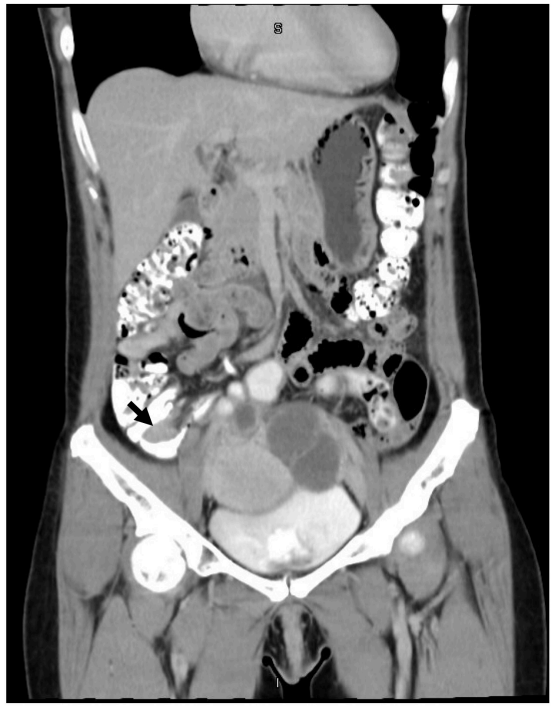

Fig 3. Appendiceal intussusception associated with intraluminal enhancing soft tissue mass occupying entire appendix (arrow)

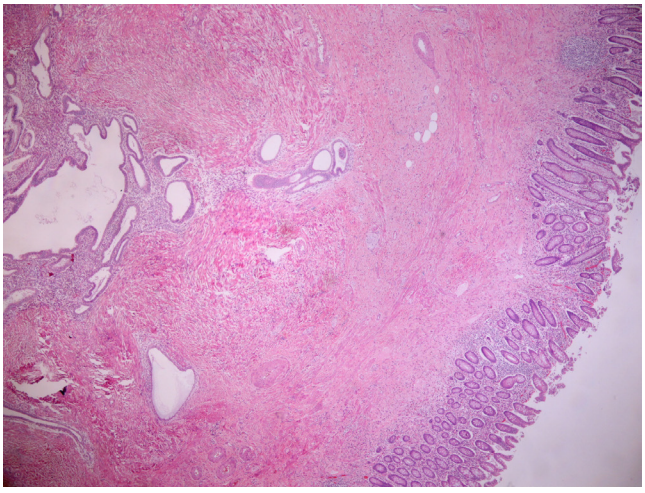

Fig 4. Endometrial glands and stroma located along submucosa to subserosa area with some area of hemorrhage

DIAGNOSIS

Appendiceal endometriosis

DISCUSSION

Appendiceal inversion or intussusception is an uncommon finding, with an estimated prevalence of 0.01%. It affects predominantly adult women, most commonly in their forties. This can be resulted from abnormal peristalsis secondary to irritation/inflammation, as well as predisposing anatomic factors including a mobile mesoappendix, a large appendiceal orifice, or an underlying mass. Pathological findings of the mass include endometriosis, adenomas, adenocarcinoma, and mucinous neoplasms.[1-3] Due to the possibility of a malignant etiology, most patients were proceeded to either appendectomy, cecectomy, or right hemicolecotomy.[4] In this case the history of ovarian cyst was a clue for diagnosis since it could be endometriotic cyst. Endometriosis is one of the leading causes of chronic pelvic pain in women. The functioning endometrial tissue located outside the uterus can affect various intraabdominal organs including the appendix. Apart from the uncommon site, the wide spectrum of clinical presentation in endometriosis of the appendix ranging from asymptomatic to severe recurrent abdominal pain, making it difficult to diagnose.

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Malignant Duodeno-Colic Fistula

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CASE HISTORY

A 90-year-old female without underlying disease presented with poor intake, generalized abdominal pain, significant weight loss (13 kg in 1 year), and watery diarrhea for 3 weeks. Physical examination showed cachexia and mildly pale conjunctivae, otherwise was unremarkable. Her stool examination was unremarkable. Colonoscopy was performed and revealed an ulcerative mass with friable mucosa covering with exudate about 8 cm in the length. This mass had two fistulous tracts connected to 1st and 2nd part of the duodenum. The remaining colon was unremarkable. Histopathology from the mass biopsy showed moderately differentiated adenocarcinoma. CT whole abdomen with contrast showed lobulated cavitary mass size 8x8x10 cm at hepatic flexure obliterated 1st and 2nd part of duodenum with fistulous tract along 1st part duodenum.

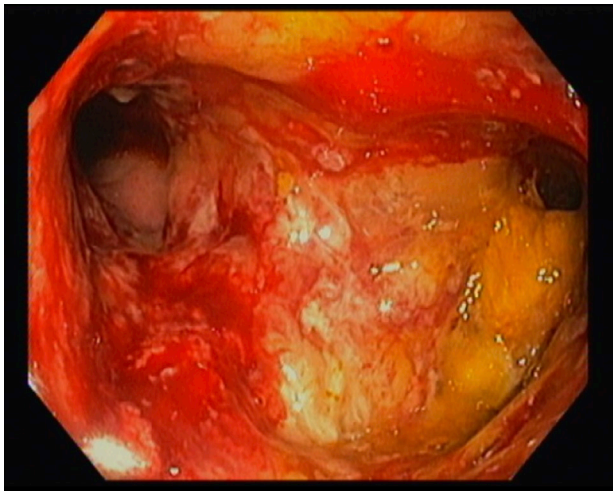


Fig 1. Two fistulous tract connect to 1st part and 2nd part duodenum

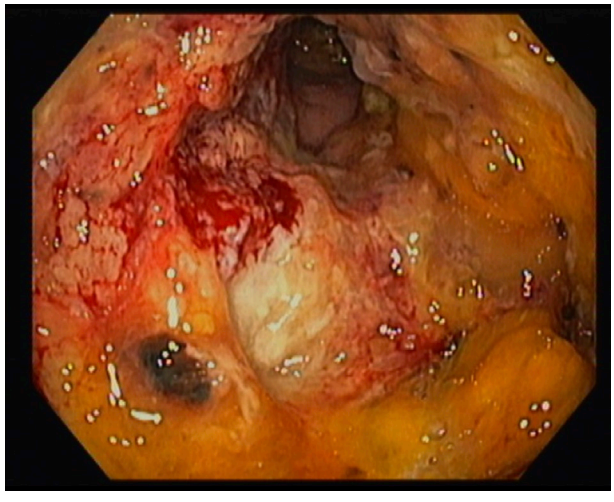
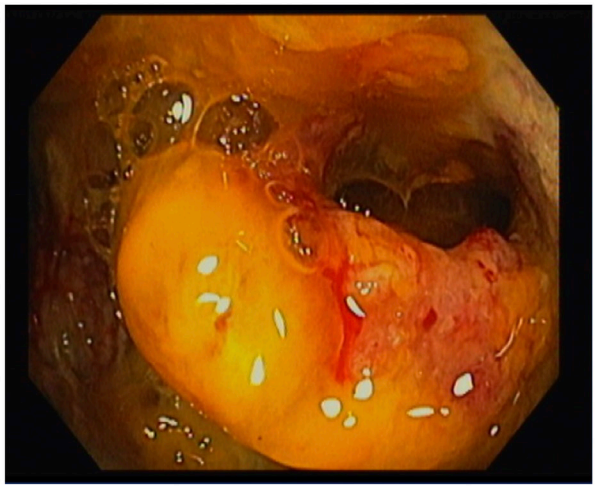
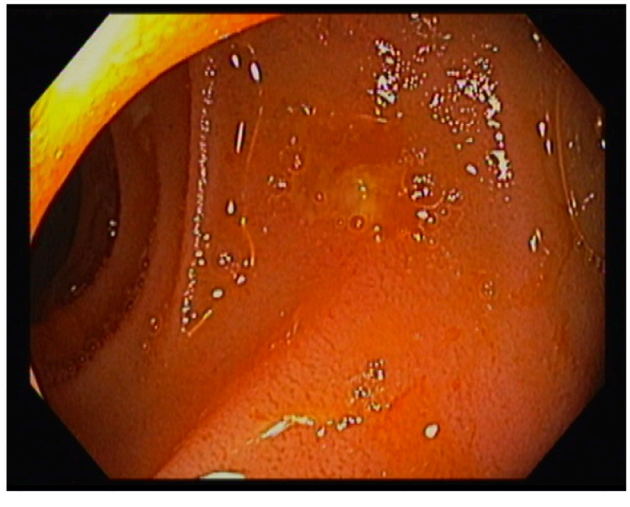
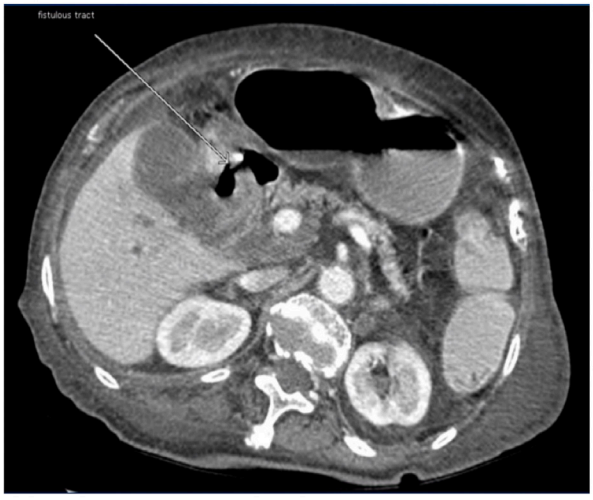


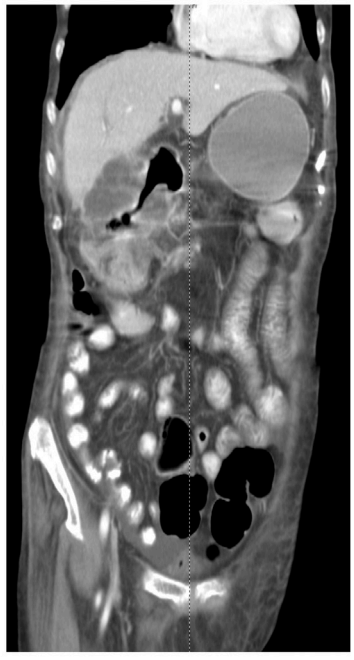
Fig 2. Fistulous tract to 1st part duodenum





Figs 3&4. Fistulous tract to 2nd part duodenum





Figs 5&6. CT whole abdomen with contrast showed lobulated cavitary mass size 8x8x10 cm at hepatic flexure obliterated 1st and 2nd part of duodenum with fistulous tract along 1st part duodenum.

DIAGNOSIS

Locally advanced adenocarcinoma of colon with fistulous tracts between hepatic flexure of colon and duodenum

DISCUSSION

Incidence of malignant duodeno-colic fistula has been estimated to be 1 in 900 of colorectal carcinomas.[1] The duodeno-colic fistulas are most frequently found between proximal transverse colon and duodenum due to anatomical proximity of these structures. Common causes of malignant duodeno-colic fistula are carcinomas of colon, duodenal, and gallbladder.[2] Clinical features of malignant duodeno-colic fistula are diarrhea (80%), weight loss (72%), pain (58%), feculent vomiting (42%), palpable abdominal mass (38%), and undigested food in stool (17%).[3] Surgical modalities for management of malignant duodeno-colic fistula include of right hemicolectomy with partial duodenectomy and primary closure of duodenal wall defect, right hemicolectomy with partial duodenectomy and patching of duodenal defect using an intestinal loop, right hemicolectomy with pancreaticoduodenectomy, or ileo-transverse colostomy with gastrojejunostomy.[3] Survival of patients with malignant duodeno-colic fistula is usually less than 12 months when treated with palliative ileo-transverse colostomy with gastrojejunostomy.[4]

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Rectal Neuroendocrine Tumor

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CASE HISTORY

An otherwise healthy 47-year-old woman presented with left lower abdominal pain for three months. Her initial workup was significant for a positive fecal occult blood test and a hemoglobin level of 9 g/dL. She underwent colonoscopy, which showed a yellowish, smooth surface, round, and firmed 1 cm subepithelial lesion at the lower rectum (Fig 1). Radial endoscopic ultrasonography (EUS) revealed a 0.7x0.8 cm. homogenous hypoechoic lesion arising from the muscularis mucosae of the rectal wall (Fig 2). Endoscopic mucosal resection (EMR) was performed, and the tissue pathology demonstrated a well-differentiated neuroendocrine tumor.

DIAGNOSIS

Rectal neuroendocrine tumor

DISCUSSION

Neuroendocrine tumors (NETs) of the colon and rectum are derived from the enterochromaffin cells of the gut. The clinical presentation of NETs can be nonspecific and often depends on the site of origin. About half of all rectal NETs are diagnosed at the time of routine lower endoscopy. Symptoms are similar to those of patients with rectal adenocarcinoma, including bleeding, pelvic/rectal discomfort, and a change in bowel habits. The carcinoid syndrome is rarely seen with rectal NETs.[1-3] Endoscopic

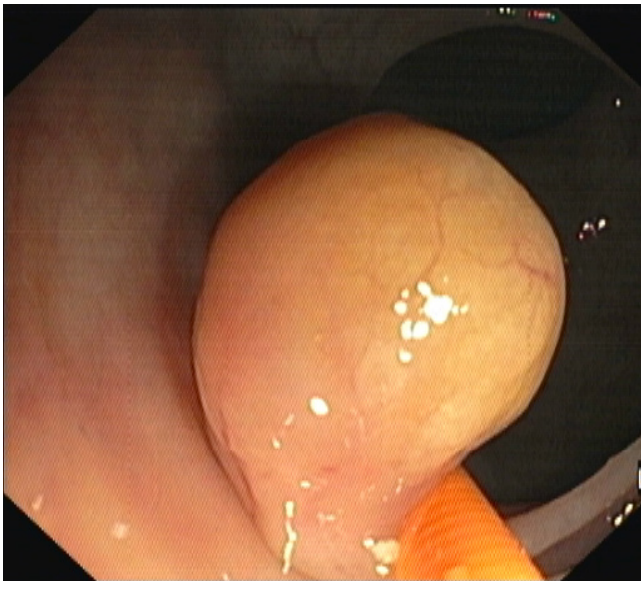

Fig 1. Endoscopic image of the subepithelial lesion at the lower rectum

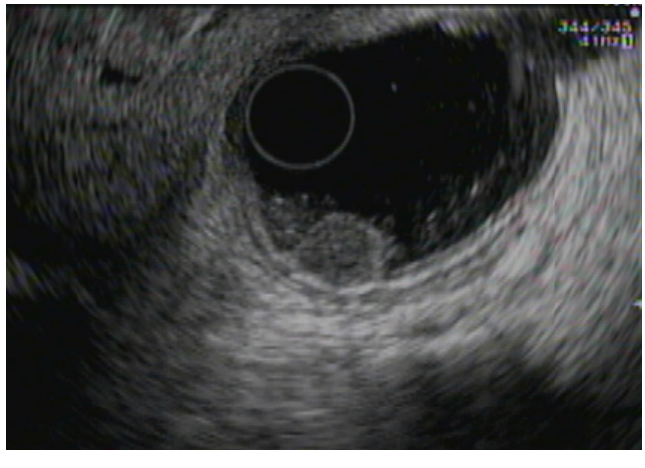

Fig 2. Radial endoscopic ultrasound image: a 0.7x0.8 cm homogenous hypoechoic lesion arising from the muscularis mucosae

findings usually show smooth, round, polypoid lesion with normal overlying mucosa within 5–10 cm from the anal verge. EUS is recommended to assess tumor size, depth of invasion, and the presence of lymph node involvement to determine the appropriate treatment.[4] Complete resection is an essential indicator of curative treatment for rectal NETs. Endoscopic resection has the role for rectal NETs ≤10 mm with no risk factors for metastasis; however the best endoscopic technique has not yet been identified.[5]

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Rectal Gastrointestinal Stromal Tumor

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CASE HISTORY

A 34-year-old male presented with rectal pain after defecation and sense of incomplete evacuation for 3 months. Sigmoidoscopy showed a rectal subepithelial mass located at 5 cm from the anal verge. Rectal endoscopic ultrasound (EUS) was performed with a linear-array echoendoscope (GF-UCT140AL5, Olympus Medical System Corp., Japan). It revealed a heterogeneous hypoechoic mass measuring about 54 x 38 mm in diameter (Fig 1). The mass originated from the 4th layer of rectal wall. The EUS also demonstrated a perirectal hypoechoic lymph node measuring about 13 x 11 mm in diameter (Fig 2). EUS-guided fine needle biopsy (FNB) was performed once with a blunt-tipped 20-gauge needle (EchoTip Procore; ECHO-HD-3-20-C; Wilson-Cook Endoscopy, Winston-Salem, NC, USA) (Fig 3). The specimen consisted of a piece of dark brown elongated tissue about 1.0 cm in the length. Cytopathology showed few fragments of spindle cells proliferation in fibro-collagenous stroma. The immunohistochemical staining reported the positive stain of CD117, CD34, and DOG1 which consistent with gastrointestinal stromal tumor.

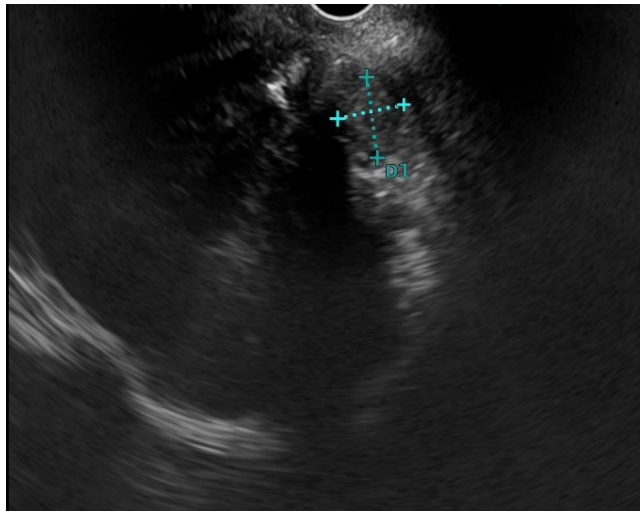

Fig 2. An EUS image from a linear-array echoendoscope (GF-UCT140AL5, Olympus Medical System Corp., Japan) demonstrated a perirectal hypoechoic lymph node.

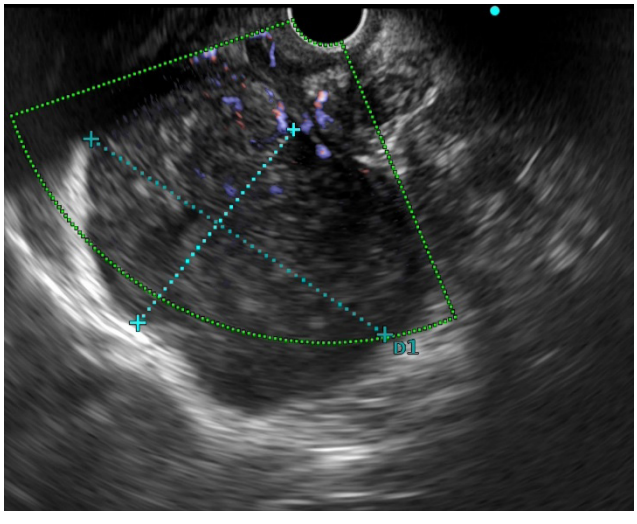

Fig 1. An EUS image from a linear-array echoendoscope (GF-UCT140AL5, Olympus Medical System Corp., Japan) demonstrated a heterogeneous hypoechoic mass originating from the 4th layer of rectal wall.

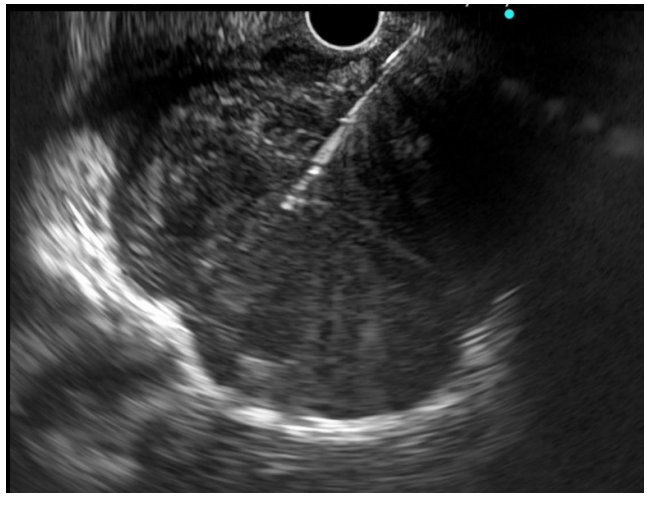

Fig 3. Endoscopic ultrasound-guided fine-needle biopsy was performed with a 20-gauge needle (EchoTip Procore; ECHO-HD-3-20-C; Wilson-Cook Endoscopy, Winston-Salem, NC, USA), the tip of the needle was presented in the middle of the lesion.

DIAGNOSIS

Rectal gastrointestinal stromal tumor

DISCUSSION

Gastrointestinal stromal tumors (GISTs) account a small number of malignant tumors in gastrointestinal (GI) tract, but they are the cause of 80% GI mesenchymal neoplasms. GISTs can occur in anywhere along the GI tract as the stomach (60%), jejunum and ileum (30%), duodenum (4%–5%), rectum (4%), colon and appendix (1%–2%), and esophagus (<1%).[1,2] Clinical presentations of GISTs depend on their locations and size. GISTs are usually asymptomatic until they progress to advanced stages which may causes various symptoms including gastrointestinal bleeding and anemia, early satiety, abdominal distension/discomfort and/or pain due to tumor compression.[1]

Most GISTs usually are incidentally diagnosed by endoscopy as a subepithelial lesions (SELs) and the tissue which is from a standard endoscopic forceps mucosal biopsy will not be enough and adequate for pathological diagnosis or determining the prognosis of GISTs.[3] Contrast-enhanced computed tomography (CT) and/or EUS is suggested for GISTs larger than 2 cm to evaluate whole images of tumor and high-risk features. EUS-guided tissue diagnosis is widely used to confirm preoperative workup of the lesions.[3] A recent meta-analysis including 6 randomized controlled trials with 669 patients which compared EUS-FNB versus EUS-guided fine needle aspiration (EUS-FNA) showed superiority of EUS-FNB over EUS-FNA for adequacy of samples (94.9% versus 80.6%, P = 0.007), and diagnostic accuracy (OR, 4.10; 95% CI, 2.48-6.79; P < .0001) with decreasing the number of passes in EUS-FNB group.[4] Besides diagnostic yield, EUS-guided tissue acquisition can obtain the specimens which are enough for histological diagnosis and immunohistochemistry staining even from difficult positions due to the anatomy. These information are very important for prognosis of GISTs.[5]

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Colitis Cystica Profunda

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CASE HISTORY

A 24-year-old Thai man without underlying disease presented with recurrent rectal bleeding for 7 years. He had been suffering from constipation, but digital evacuation was denied. Physical examination and basic laboratory tests were unremarkable. Multiple sessile polypoid lesions including three shallow rectal ulcers at 3-5 cm from anal verge were detected at colonoscopy (Fig 1-3). Subsequently EUS was done which showed heterogenous echogenicity and thickening of rectal mucosa containing multiple intra-mucosal cysts, maximal diameter of the largest cyst was 1.24 cm. A questionable disruption of muscularis propria was detected (Fig 4-5). Piecemeal polypectomy was done and pathological diagnosis was colitis cystica profunda (CCP).

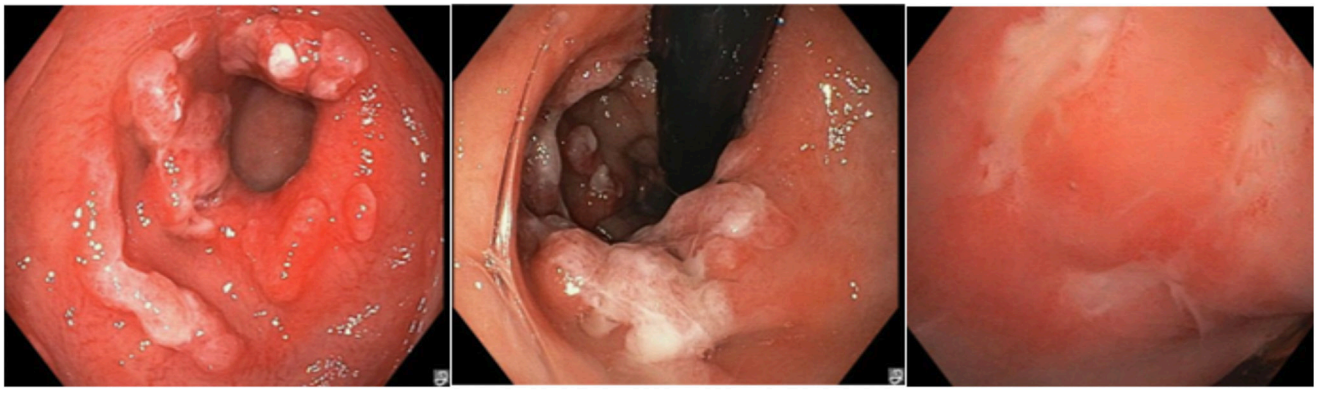

Fig 1&2. Colonoscopy showed multiple sessile, poly lesions in the rectum
Fig 3. Colonoscopy showed three shallow rectal ulers at 3-5 cm from anal verge

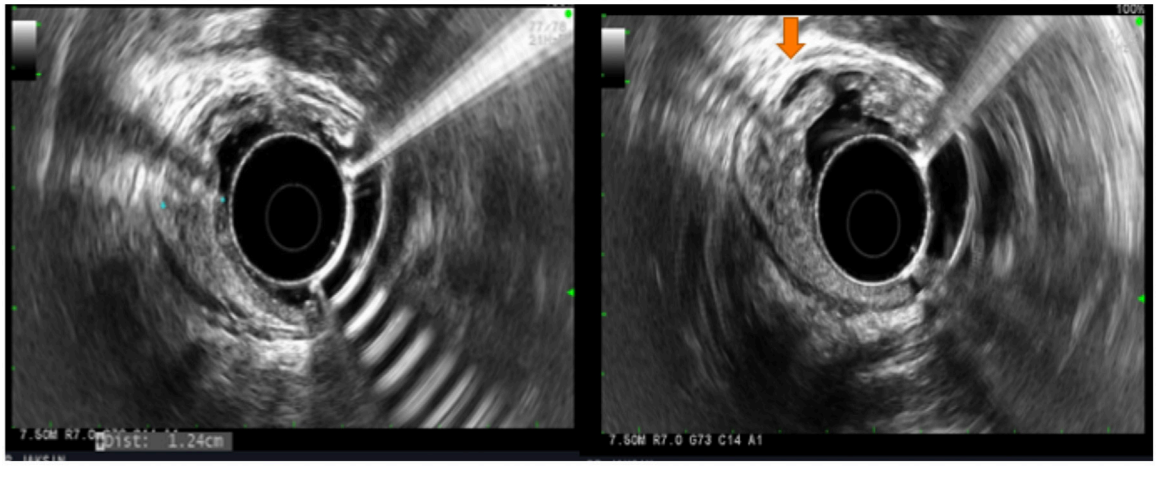

Fig 4&5. EUS showed thickening rectal mucosa with heterogeneous echogenicity containing Multiple cysts. A questionable disruption of muscularis propria was detected (arrow)

DIAGNOSIS

Colitis cystica profunda

DISCUSSION

Colitis cystica profunda (CCP) is an uncommon in clinical practice, it may be the sole lesion in some cases but may present with other concomitant conditions such as IBD[1] or colon cancer.[2] CCP can also mimic adenomatous polyp or malignancy.[3] Common symptoms pertaining to CCP include rectal bleeding, mucus discharge, tenesmus, proctalgia fugax, altered bowel habits and a long existing constipation.[4] CCP can be either localized form or diffuse form involving a variable length of the rectal mucosa or colon. The etiology of CCP is unclear[5,6] however it associated with rectal prolapse in approximately 50% of cases.[7] The endoscopic appearances are not unique leading to missed diagnosis as polyp or malignancy.[5,7] The shallow ulcers detected by colonoscopy in this patient may represent solitary ulcer of rectum which was postulated to be analogous syndrome as CCP caused by rectal prolapse.[8] EUS examinations were reported with findings of cystic lesions in submucosa or spongy or thicken mucosa with muscularis propria intact.[2,7,9] The EUS in this patient was consistent with other reports but a questionable disruption of muscularis propria which may represent fibrous septum.[7] Treatments of CCP are re-education of bowel habits to avoid straining, administration of fiber and laxative and biofeedback. In patients with CCP associated rectal prolapse, surgical correction for rectal prolapse should be considered.[10]

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IgG-4 Related Sclerosing Cholangitis

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CASE HISTORY

A 47-year-old man presented with progressive jaundice, pruritus, and weight loss for two months. He has a history of a left orbital mass and lacrimal gland enlargement for the past two years. The liver function test showed a total serum bilirubin of 27.6 mg/dL, alkaline phosphatase of 338 U/L. Computed tomography (CT) showed diffuse intrahepatic duct (IHD) dilation, mildly dilated common hepatic duct (CHD), and thickening of the common bile duct (CBD), suspicious for distal CBD stricture. Periductal soft tissue thickening of the entire course of the extrahepatic bile duct was also observed, raising concern of distal cholangiocarcinoma. The pancreas appeared enlarged diffusely.

The patient underwent endoscopic ultrasonography (EUS), which revealed pancreatic parenchymal changes, including heterogeneity of parenchyma with hyperechoic foci, hyperechoic strands, lobularity, and a hypoechoic rim surrounding the pancreas. CBD had symmetrical, smooth, and diffuse thickening of the wall. The wall thickness measured 4 mm (Fig 1). Endoscopic retrograde cholangiography (ERC) was performed and showed a long segment stricture of distal CBD measuring about 2 cm with a relative CHD dilatation (7-8 mm in diameter), irregular cystic duct wall, normal filling of gallbladder, and multifocal irregular strictures of both right and left IHDs without pre-stenotic dilation (Fig 2). The findings were consistent with sclerosing cholangitis. A 10 French x 7 cm endobiliary stent was placed due to severe cholestatic jaundice. Additional laboratory showed elevated serum IgG-4 level of 1,080 mg/dL. He was treated with prednisolone with the improvement of symptoms liver function test after two weeks of treatment.

DIAGNOSIS

Autoimmune pancreatitis (AIP) and type 2b IgG-4 related sclerosing cholangitis (IgG-4 SC)

DISCUSSION

IgG-4 SC is one of the several diseases associated with autoimmune pancreatitis. The diagnosis is made from a combination of imaging, serology, histopathology, evidence of other organ involvements, and response to steroid treatment.[1] In type 2 IgG-4 SC, the stenosis is distributed at distal bile duct and intrahepatic bile duct level. This type is subclassified into two types; type 2a is accompanied with pre-stenotic dilation, while type 2b is characterized as the stricture without pre-stenotic

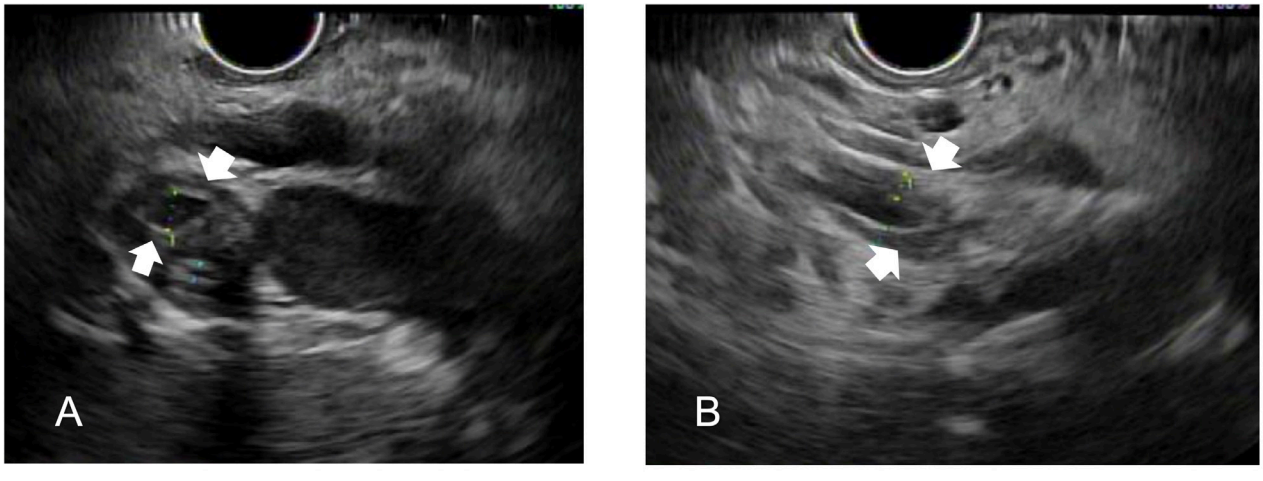

Fig 1. A and B, Linear endoscopic ultrasound showed symmetrical, smooth and diffuse thickening of common bile duct wall (white arrows).

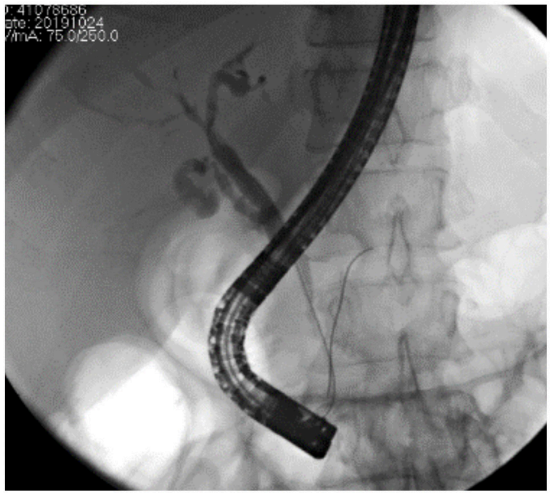

Fig 2. Cholangiogram demonstrates a 2 cm distal CBD stricture, and multifocal, irregular bilateral intrahepatic duct strictures without pre-stenotic dilation.

dilation, reduced ductal arborization, which should be discriminated from "pruned tree" appearance in primary sclerosing cholangitis (PSC).[2] An elevation of the IgG-4 level is seen up to 90 % of cases.[3]

EUS may be useful to differentiate IgG-4 related cholangiopathy from distal cholangiocarcinoma (CCA). Du et al. reported that wall thickening was found more frequently in IgG-4 SC, compared to CCA. In contrast, CCA had more mass occupying lesions than IgG-4 related disease.[4] Naitoh et al. reported that the thickened bile duct wall detected by ultrasonography, CT-scan, EUS, and intraductal ultrasonography (IDUS) involves circular-symmetrical thickening, smooth outer and inner margins, and a homogenous internal echo at the stenotic area.[5] IgG-4 SC improves rapidly with steroid therapy, which can be initiated without ERC or biliary drainage in cases of mild jaundice, without cholangitis, and patients with whom the diagnosis is definite. The pathological examination for biliary stricture is not essential.[6,7]

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Distal Cholangiocarcinoma Associated Choledochal Cyst Type II

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CASE HISTORY

A 74-year-old man presented with painless progressive jaundice and significant weight loss for one month. His underlying conditions included hypertension and coronary artery disease. Physical examination revealed icteric sclera and palpable gallbladder. Blood chemistry was significant for direct hyperbilirubinemia and elevation of alkaline phosphatase. Abdominal computed tomography (CT) scan showed diffuse dilatation of bilateral intrahepatic ducts (IHDs), common hepatic duct (CHD), and common bile duct (CBD) without definitive cause of obstruction. The patient underwent endoscopic ultrasound (EUS), which showed a fusiform dilatation of the proximal CBD measuring 1.7 cm in diameter (Fig 1) with a smooth tapering of the mid and distal CBD. The distal CBD had a connection to a 1.7 x 1.5 cm round anechoic area, suggestive of a cystic lesion locating just above the ampulla (Fig 2). The cyst did not compress upon the CBD, and therefore was not the cause of CBD obstruction. Subsequent endoscopic retrograde cholangiopancreatography (ERCP) demonstrated a very short stricture of distal CBD with fusiform dilatation of upstream bile duct. The IHDs were dilated bilaterally. A round 1.2 cm cavity was observed during contrast injection of the distal CBD, suggesting a cyst or diverticulum with the distal CBD connection (Fig 3). Brush cytology of distal CBD was obtained, and a 7 French x 5 cm plastic stent was placed. The EUS and ERCP findings suggested choledochal cyst type II, and the cytology revealed adenocarcinoma. The patient underwent a pyloric resecting pancreaticoduodenectomy (PrPD), and surgical pathology confirmed the diagnosis.

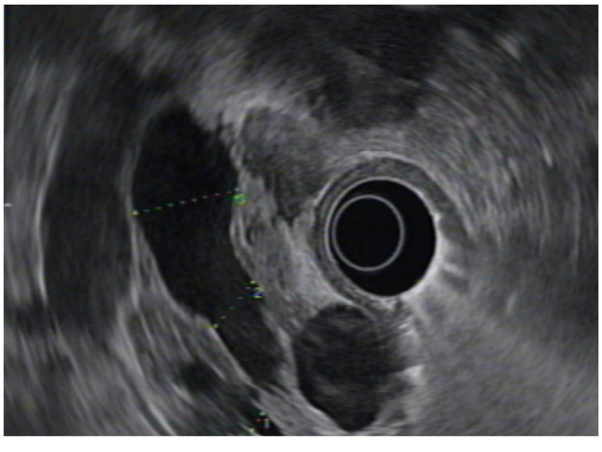

Fig 1. A fusiform dilatation of the proximal CBD measuring 1.7 cm in diameter with a smooth tapering of the mid and distal CBD.

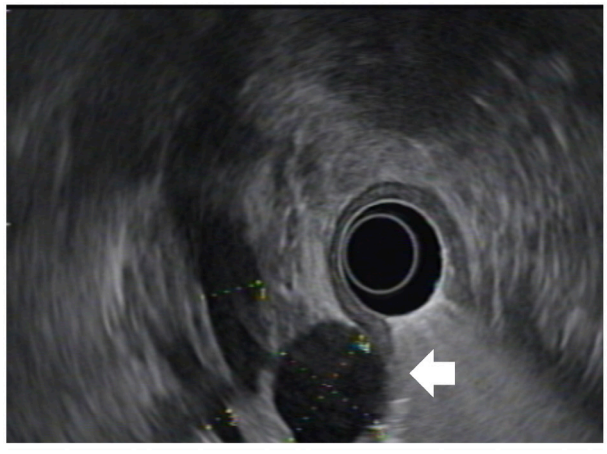

Fig 2. The distal CBD had a connection to a 1.7 x 1.5 cm round anechoic area consistent with cyst.

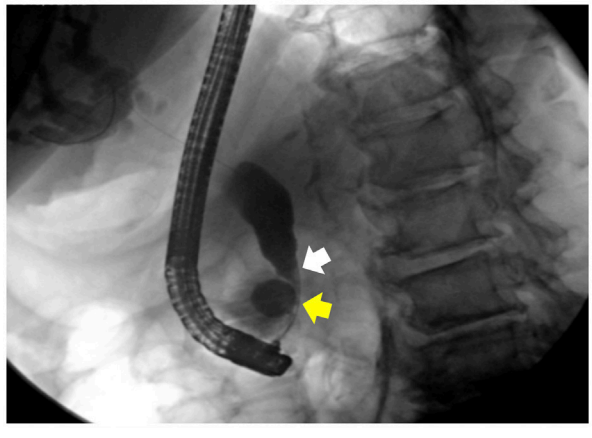

Fig 3. Cholangiogram revealed a dilated common bile duct, smooth tapering and short segment stricture of the distal CBD (white arrow) with contrast filling a 1.2 cm cavity connecting to the distal CBD (yellow arrow).

DIAGNOSIS

Choledochal cyst type II with distal cholangiocarcinoma

DISCUSSION

Choledochal cysts are congenital anomalies of the biliary tract manifesting as cystic dilatation of the extrahepatic or intrahepatic bile ducts. Extrahepatic supraduodenal diverticulum, choledochal cyst type II, per the Todani classification, is a rare subtype, accounting for less than 6% of all reported cysts.[1] Choledochal cysts increase the risk of biliary tract cancer ranging from 6 - 30%.[1,2] Todani et al. reported biliary malignancy in 68% of patients with type I choledochal cyst, 21% of type IV, and only 5% in type II.[3] A study from Korea found no patients diagnosed with distal cholangiocarcinoma in choledochal type II.[4] We, herein, report a rare case of distal cholangiocarcinoma in a patient with choledochal cyst type II.

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Direct Endoscopic Necrosectomy

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CASE HISTORY

A 66-year-old male presented with symptomatic pancreatic wall-off necrosis (WON). EUS-guided drainage was scheduled. After the lesion was identified by EUS, a 19-G FNA needle was punctured into the WON cavity, the inner stylet was withdrawn, a 0.035 Jag-wire (Boston Scientific, Natick, MA) was inserted through the needle and coiled within the WON cavity; under concomitant fluoroscopic guidance. The puncture tract was then dilated with coaxial dilators 6-Fr cystotome followed by an 8-mm Hurricane balloon (Boston Scientific, Natick, MA). A 16 x 20 lumen apposing metal stent (LAMS) (SPAXUS; Taewoong Medical, Gyeonggi-do, South Korea) was placed from WON cavity to the stomach (Fig 1). After 24 hours, a plain abdominal x-ray showed inadequate expansion of the LAMS lumen. We then dilated the LAMS by a 15-mm CRE™ balloon (Boston Scientific, Natick MA) (Fig 2). Direct endoscopic necrosectomy (DEN) was subsequently performed for 30 minutes with a 13-mm snare (Boston Scientific, Natick, MA, USA) (Fig 3). One hundred milliliters of hydrogen peroxide (H_2O_2) diluted with water was used for chemical debridement as an adjunct to mechanical debridement during DEN.

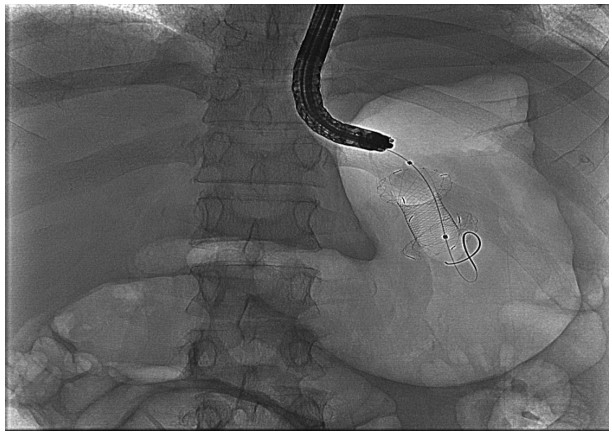

Fig 2. The dilation of the lumen-apposing-metal stent (LAMS) with a 15-mm CRE™ balloon (Boston Scientific, Natick MA). The LAMS was dilated until disappearance of waist shape of the LAMS.

TECHNIQUE

EUS-guided drainage of WON plus direct endoscopic necrosectomy

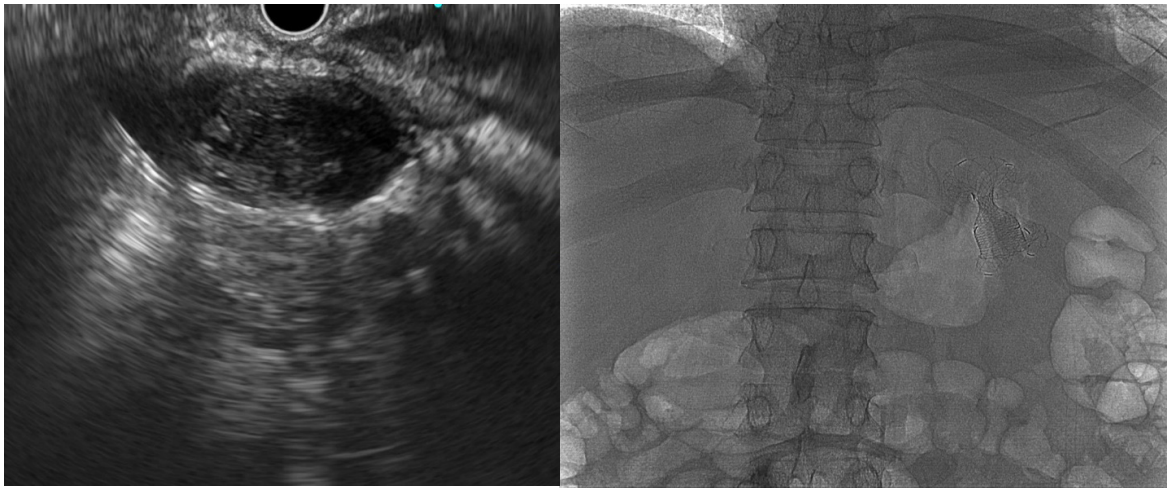

Fig 1. EUS-guided drainage of pancreatic wall-off necrosis by a 16 x 20 mm lumen apposing metal stent (SPAXUS; Taewoong Medical, Gyeonggi-do, South Korea).

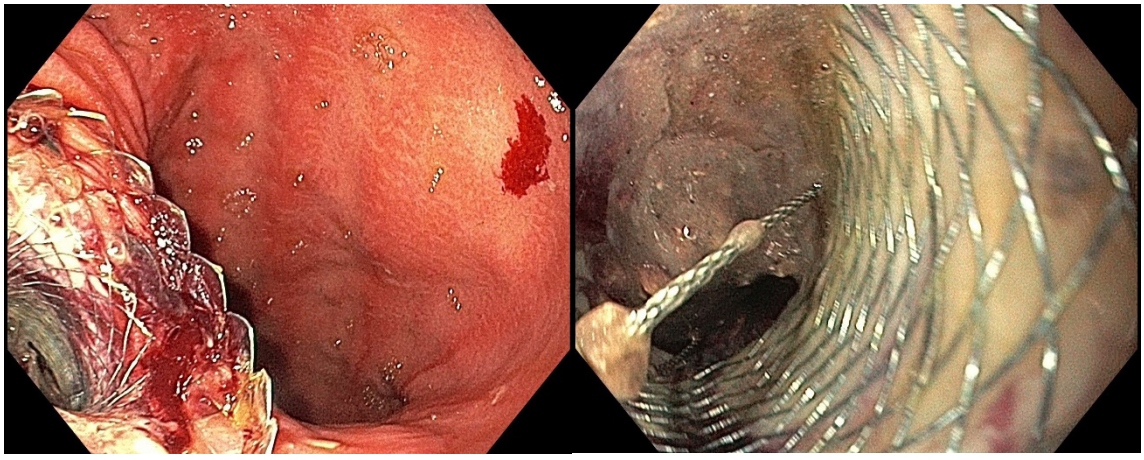

Fig 3. The lumen-apposing-metal stent (LAMS) was properly placed. Direct endoscopic necrosectomy was performed with a 13-mm snare (Boston Scientific, Natick, MA, USA)

DISCUSSION

EUS-guided drainage of pancreatic fluid collection (EUS-PFC) has comparable efficacy and safety with surgical cystogastrostomy. However, patients who underwent EUS-PFC had a significantly shorter median length of hospital stay and lower mean costs.[1] The first successful series of DEN was reported in 2000. The early results showed that DEN had faster recovery, less adverse events and traumatic than those in surgical necrosectomy.[2] DEN consists of debridement of WON using a gastroscope that is inserted directly into the collection via the stomach or duodenum through the cyst-gastrostomy or cyst-duodenostomy fistula or LAMS tract. The tract is sometimes dilated by balloon to enable passage of the endoscope and then the necrotic debris is removed from the WON and pulled back into the lumen using a variety of endoscopic tools such as snare, dormia basket and retrieval nets. Irrigation helps to loosen solid material partially adherent to the wall and small debris may also be removed by suctioning. Successful resolution rate of WON was reached to 88% in patients who underwent DEN compared to 45% who underwent standard drainage alone. The adverse events of DEN were limitation and comparable with standard drainage.[3] A systematic review covering 14 studies reported the clinical successful rates of DEN ranged from 75% to 91%. Complications of this technique included perforation, air embolism, and bleeding, which occurs in 3% – 21% of patients.[4]

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Adrenal Gland Tumor

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CASE HISTORY

A 71-year-old female presented with adreno-corticotrophic hormone (ACTH) dependent Cushing's syndrome. Magnetic resonance imaging showed multiple liver cysts, bilateral adrenal enlargement, no pancreatic lesion, and no dilatation of the bile duct. PET scan showed an increase PET uptake at head and uncinate process of pancreas. EUS was performed with a linear-array echoendoscope (EG3870UTK, Pentax Corporation, Tokyo, Japan) to examine the pancreas and demonstrate adrenal gland tumor; the patient was in the left lateral position and sedated with propofol under protocol of moderate sedation. EUS examination revealed unremarkable pancreas including head and uncinate process of the pancreas. We successfully endosonographically demonstrated the left adrenal gland tumor. The demonstrated left adrenal tumor was a bulky homogeneous mass with a typical character of "seagull" of the left adrenal gland. The mass measured 42x27 mm in diameter (Fig 1). We started to examine from the gastroesophageal junction by rotating the echoendoscope in clockwise direction and the aorta is identified. From this important landmark, we advanced gently the echoendoscope to see celiac trunk take-off and continued to rotate in clockwise torque. We can identify the left adrenal gland which is between the aorta and upper pole of left kidney during this examination (Fig 1). In this case, endoscopic ultrasound (EUS) demonstrated a homogeneous hypoechoic mass which located at left adrenal position measuring about 42 x 27mm in diameter as shown in Fig 2. The adrenal gland mass had a "seagull" shape as shown in Fig 3. EUS also imaged the whole of normal pancreatic parenchyma from the head of pancreas to tail of pancreas.

DIAGNOSIS

Adrenal gland tumor

DISCUSSION

The right adrenal gland is located at the upper pole of the kidney and correlates with the inferior vena cava, the crus of the diaphragm. The margin of the liver is the landmark to identify these structures. The typical echogenic of adrenal gland is bright, dark and bright echo lines. The thickness of adrenal gland is about 5 ±

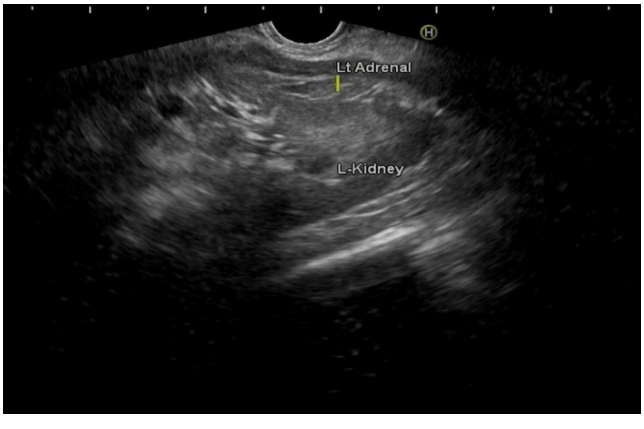

Fig 1. Demonstrated the location of left adrenal gland and left kidney by a linear-array echoendoscope (EG3870UTK, Pentax Corporation, Tokyo, Japan)

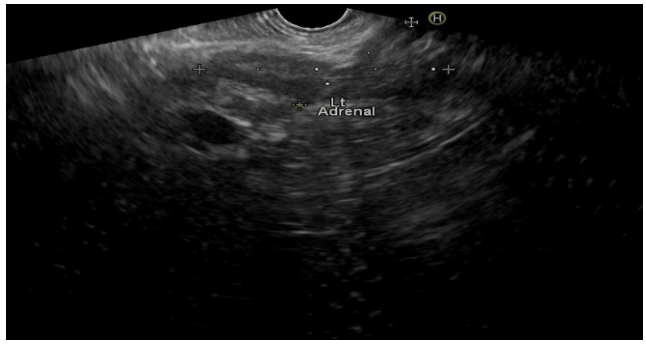

Fig 2. Demonstrated a homogeneous hypoechoic mass 27 x 42mm located at the position of left adrenal gland by a linear-array echoendoscope (EG3870UTK, Pentax Corporation, Tokyo, Japan)

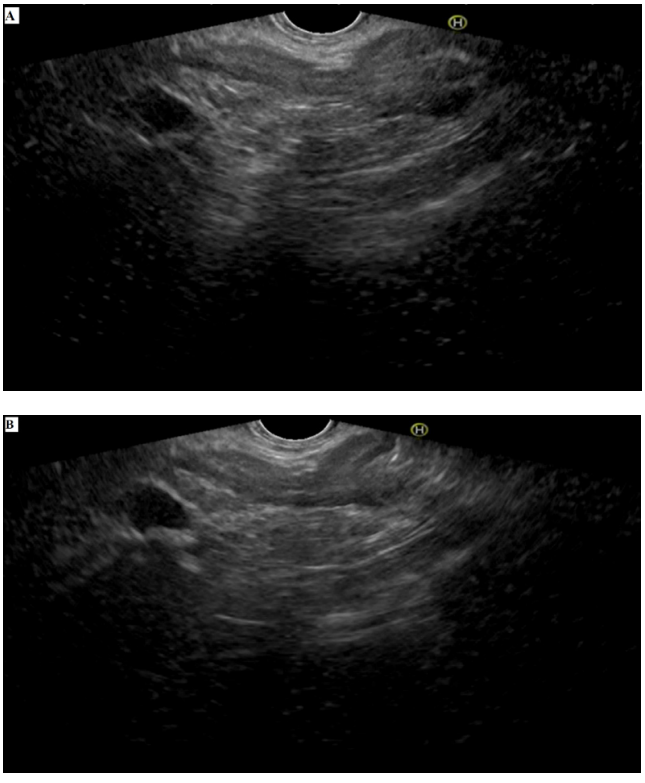

Fig 3. Demonstrated left adrenal gland mass. (A) B-mode. (B) B-mode with dTHI by a linear-array echoendoscope (EG3870UTK, Pentax Corporation, Tokyo, Japan).

1 mm (range 4–8 mm) in size.[1] Given the left adrenal gland is located between the upper pole of the left kidney and pancreatic tail, to demonstrate the left adrenal gland, ones may use one of these following techniques. For the first technique, the echoendoscope was placed in the position that endosonographer could show celiac axis, then endosonographer rotated the scope clockwise until the left adrenal gland is shown. For second technique, the endosonographer started by rotating the echoendoscope probe from neck of the pancreas toward tail of the pancreas. Once, the left kidney is identified, the endosonographer pull the echoendoscope backward until the echoendoscope reached upper border of the left kidney. Around this area, the endosonographer gradually rotate echoendoscope gently, the left adrenal gland will be then visualized.[2-4]

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Endoscopic Ultrasound Guided Fine Needle Biopsy (EUS-FNB) In Diagnosing Gastric Mantle Cell Lymphoma

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CASE HISTORY

A 63 year-old man presented with a history of abdominal pain. Physical examination was unremarkable, there were no abdominal mass or palpable lymph nodes. On upper GI endoscopy, there is a 2-cm protruding-ulcerative sub-epithelial mass noted at the anterior body of the stomach. (Fig 1) Endoscopic ultrasound (EUS) was performed (Fujifilm SU-1, linear echoendoscope) and revealed a heterogenous hypoechoeic mass measuring 2.4 cm x 2.8 cm in diameter, the mass originates from the muscularis mucosa layer. (Fig 2) An endoscopic biopsy forceps was used to obtain tissue at the mucosa of the lesion with 4 core tissues obtained. At the same time, EUS guided FNB was performed using a 25G FNB needle (Boston Scientific, Acquire) with 2 successful passes on both the gastric lesion and a regional lymph node. (Fig 3) There were no complications resulting from both procedures. The histology result obtained from the forceps biopsy was negative for malignancy. However, the gastric and lymph node biopsy shows proliferation of small-cleaved mature lymphoid cells. (Fig 4A) These lymphoid cells stain CD20, cyclin D1 and Bcl2. CD3, CD5, CD43, CD10, and Bcl6 immunohistochemistry is negative. (Fig 4B&C) The Ki-67 index is approximately 30%. These findings were in consistent with Mantle Cell Lymphoma of classic type. The patient was subsequently referred for chemotherapy.

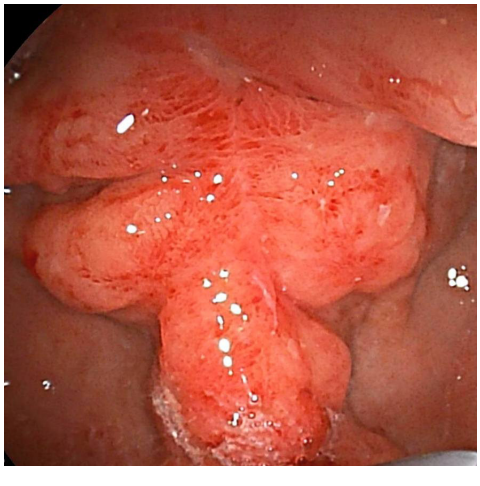

Fig 1. Protruding, ulcerative anterior gastric wall mass with large prominent gastric fold.

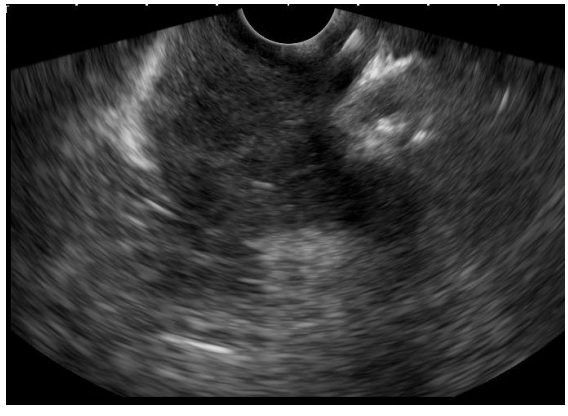

Fig 2. Heterogenous, hypoechoeic gastric mass arising from the 2nd (muscularis mucosa) layer

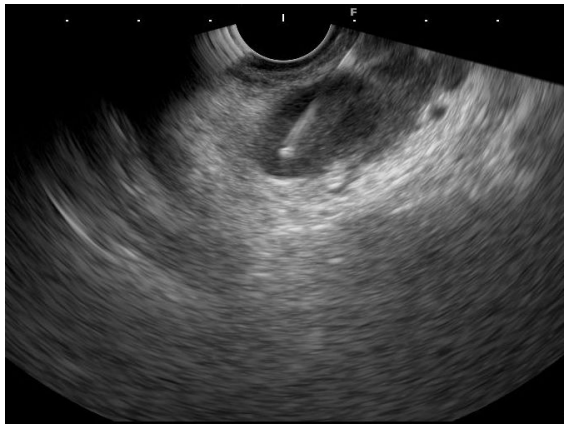

Fig 3. EUS guided FNB of a lymph node.

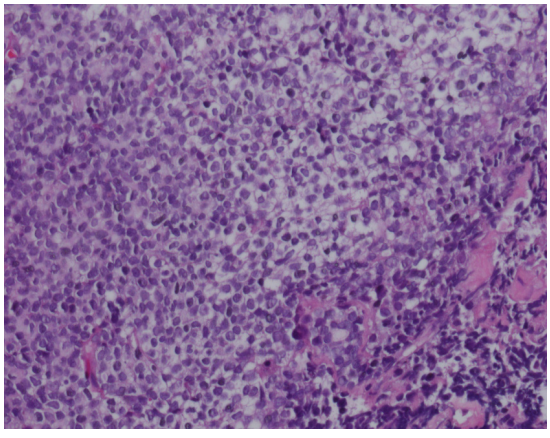

Fig 4A: Gastric tissue showing proliferation of small-cleaved mature lymphoid cells

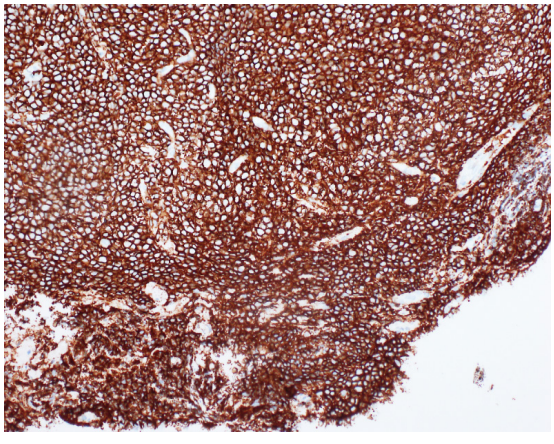

Fig 4B. Immunohistochemistry of gastric lymphoid cells showing positive CD20

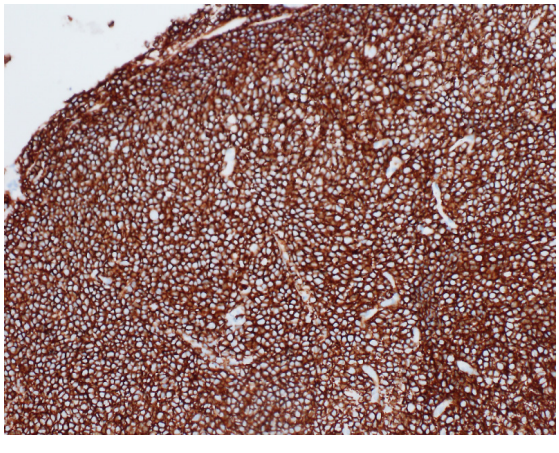

Fig 4C. Immunohistochemistry of gastric lymphoid cells showing positive Cyclin D1.

DIAGNOSIS

EUS-FNB in diagnosing gastric Mantle cell lymphoma

DISCUSSION

Mantle cell lymphoma (MCL) is a rare type of aggressive lymphoma. It represents 4% of US and 7-9% of Europe of all lymphomas. Diagnosis is often made histologically from biopsies of lymph nodes, bone marrow or blood which exhibits typical immunohistochemical findings.[1] Gastrointestinal tract (GI) involvement lymphomas are not uncommon. It was thought that the incidence ranges from 15% to 30%. Consequently, upper GI involvement occurs in 28 to 44% and lower GI occurs in 72 to 88% of all cases.[2,3] While endoscopic forceps biopsies of gastric lesions are preferred, EUS fine needle biopsy (FNB) provides a reasonable alternative method of diagnosis if the aforementioned yield was negative. Here we describe a case of mantle cell lymphoma diagnosed on EUS FNB of a gastric lesion found on endoscopy. The actual incidence of gastric MCL is still unknown today. Historically, Multiple Lymphomatous Polyposis (MLP) is the most common endoscopic manifestation of GI tract lymphoma.[4] Iwamuro et al. reported that amongst gastric lesions of mantle cell lymphomas found on endoscopy, 26.9% were superficial type; 23.1% of protruded type; 23.1% of fold thickening type; 23.1% were ulcerative type and 3.8% were combined type (protruded and ulcerative).[5] The role of EUS in evaluating GI tract subepithelial lesions provides excellent details in evaluating the origin of tumor based on delineating the histologic layers of the GI tract, therefore the likely pathology (GIST, Lymphoma, NET, external compression of tumour etc).[6] Additionally, it provides an excellent modality in loco regional staging of primary gastric lymphomas.[6,7] On endosonographic imaging of the gastric wall for gastric lymphomas, it may appear more homogenous with enlargement of gastric folds and preservation of mucosa compared to other subepithelial lesions.[8] These lesions may arise from either 2nd, 3rd or 4th layer of the gastric wall.[6] To date, there are no pathognomonic endosonographic features that is diagnostic of MCL, hence diagnosis is largely dependent on histology. With that in mind, together with the ability of tissue acquisition (FNB), EUS has been the preferred imaging modality for such condition in recent times. EUS FNB of unknown lympadenopathies has been shown to have a high diagnostic accuracy of up to 98%, furthermore it is able to classify lymphomas base on the WHO classifications of up to 88%.[9] In addition to that, EUS FNB of specific gastric lesions are sensitive and specific with a diagnostic accuracy of up to 89%.[10] EUS FNB is also highly advantageous in differentiating high grade and low grade lymphomas. In a retrospective study, 24 patients with a diagnosis of lymphoma whom underwent EUS-FNA/B was able to correctly identify B cell monoclonality in 95% of cases and correctly classify lymphomas in 73% of them.[11] It is worth to note that EUS FNB provides a good salvage

modality in obtaining a tissue diagnosis in gastric lesions with negative histological result by endoscopic forceps biopsy. A retrospective study reported that 29 out of 62 (43%) of patients underwent EUS FNA of gastric lesions had unsuccessful, negative tissue diagnosis by endoscopic forceps biopsy.[10] There may also be promising evidence in using EUS to target appropriate sites of infiltrating gastric tumours for deep, large endoscopic biopsies with bite-on-bite biopsies.[12] In summary, gastric MCL is rare amongst gastric lymphomas. Although EUS FNB is useful in the diagnosis of gastric lesions such as this compare to endoscopic forceps biopsies, more studies will be required to investigate the optimal choice of tissue acquisition comparing both modalities.

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