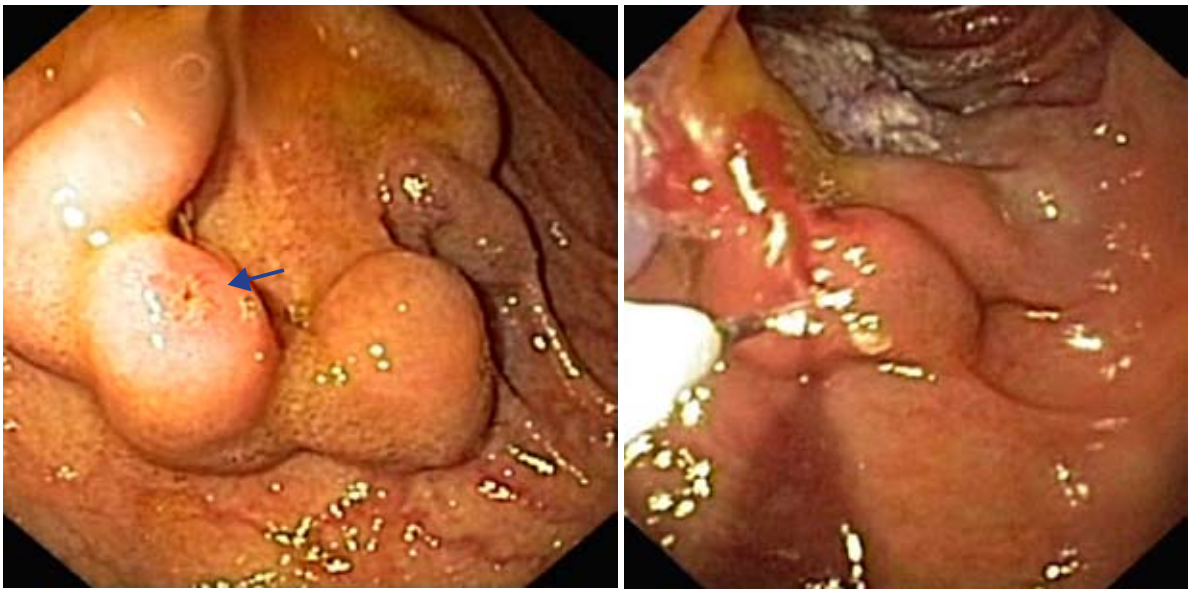
Part 3
Small and Large Intestine

Case 1

Roongruedee Chaiteerakij, MD.
Rungsun Reknimitr, MD.

A 45-year-old male patient with underlying alcoholic cirrhosis came to the emergency department due to massive hematemesis. He underwent gastroduodenoscopy.

The figure was shown below.



The finding was tortuous dilated submucosal vessels with red spot surrounding with small whitish patch on top (white nipple sign(arrow)) at the second part of duodenum.

The diagnosis was duodenal varices with stigmata of recent bleeding, **"**white nipple sign**"** which is a plug of platelet fibrin on varix and the patient was treated with tissue adhesive n-butyl-2-cyanoacrylate (Histoacryl) injection.

Differential diagnosis was submucosal folds of the duodenum and dilated veins.

Discussion

Gastroesophageal varices are commonly found in patients with portal hypertension, however, varices located at other sites called ectopic varices can be found which are usually in small intestine and colon. Duodenal varix is often detected in the second and third part of duodenum and caused by retroperitoneal porto-systemic shunt due to portal vein obstruction. The collateral vessels originate from pancreatoduodenal vein and drain to inferior vena cava via retroperitoneal vein[1]. Bleeding duodenal varices rarely occur since these tend to have a smaller diameter and shorter length than esophageal varices, moreover, they are usually located deeper and mainly on the serosa of the duodenum[1], in contrast to the submucosal position of esophageal varices. However, it can also be in submucosal layer, as noted in this case, and cause massive and life-threatening bleeding.

The physicians should be aware the possibility of bleeding ectopic varices in patients with portal hypertension presenting with gastrointestinal hemorrhage of unknown origin and the bleeding duodenal varices should be considered in cases of hemorrhage from duodenum although the duodenal ulcer is more often the correct diagnosis.

Although, there is no consensus in treatment for bleeding duodenal varix due to the rarity of this entity, endoscopic procedure including band ligation, sclerotherapy[2], tissue adhesive injection[3] or clipping should be considered as the first line therapy. Surgical procedures, such as duodenal resection or extrahepatic portosystemic shunts[4], or interventional radiologic therapy with transileocolic vein obliteration or balloon-occluded retrograde transvenous obliteration[5] are alternative treatment to stop bleeding after failure of endoscopic therapy.

References

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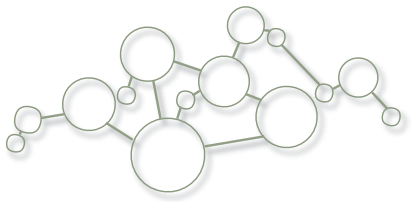
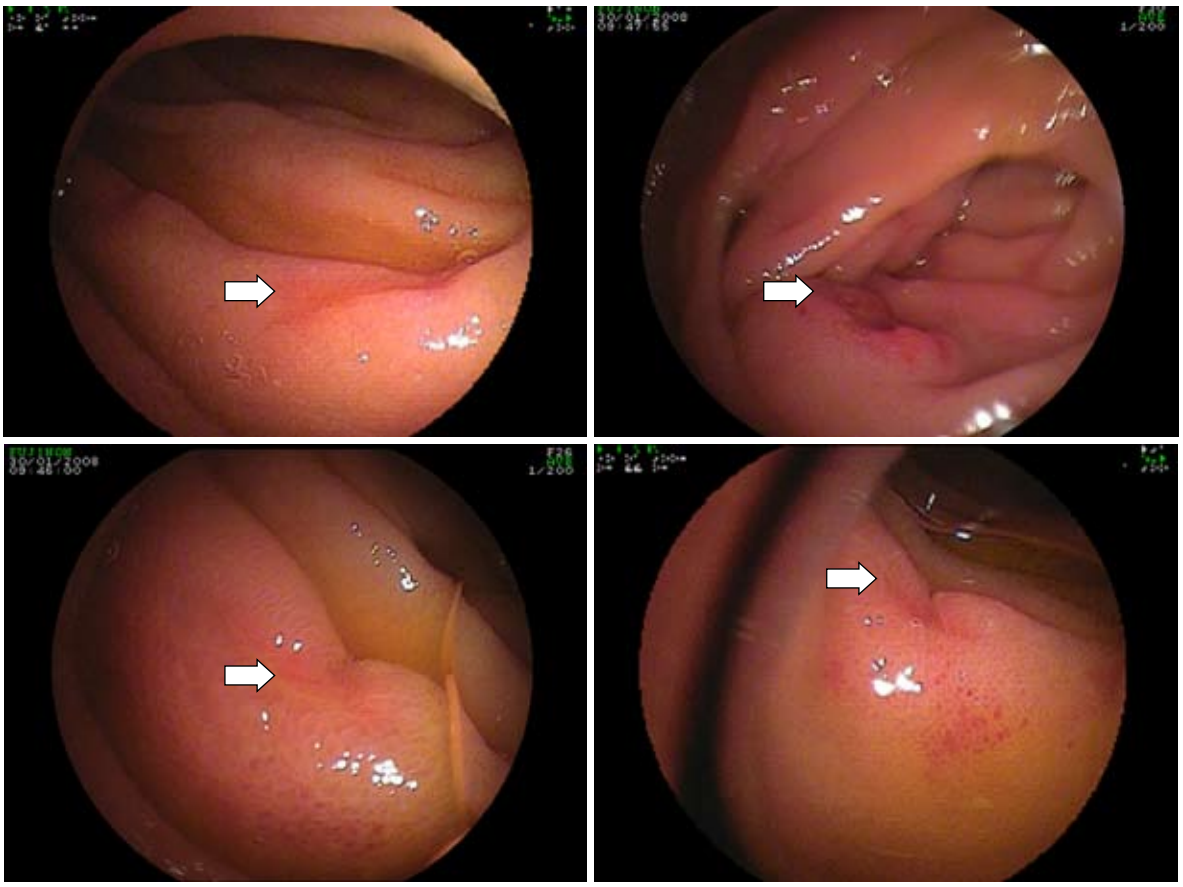


Case 2

Sombat Treeprasertsuk, MD.
Varocha Mahachai, MD.

A 55 year-old female present with obscure GI bleeding and referred for investigations. She got EGD and colonoscopy last week from private hospital and showed normal study. Physical examination showed stable vital signs with low hemoglobin level which dropped from13 to 8 gm/dl within 2 weeks. Stool occult blood also showed positive. He had history of diabetes and hypertension and on ASA for primary prevention of cardiovascular event. Double balloon endoscopy was done and shown as figure 1-4



Double balloon endoscopic findings showed multiple small ulcer at 3rd to 4th part of duodenum. There was one ulcer showed swelling, erythematous and clean based. There was no any other mucosal lesion or intestinal bloody content (downward from oral rout in about 200 cm. estimated from 10 times of DBE insertion). The final diagnosis was small bowel ulcer may be related to aspirin.

Discussion

Non-steroidal anti-inflammatory drugs (NSAIDs) is one of the major cause of gastrointestinal complication. NSAIDs enteropathy is a common condition but may be under diagnosed due to the facility to get diagnosis. A high level of clinical suspicion in NSAIDs users who have occult GI blood loss, hypo-albuminemia, obstructive GI symptoms and history of long-term NSAIDs use (>6 months) is important to get the diagnosis. Three hit hypothesis of small bowel injury by NSAIDs composed of the following step: firstly NSAIDs which is the lipid-soluble weak acids may interact with surface membrane phospholipids and cause direct damage of enterocyte mitochondria by uncoupling oxidative phosphorylation. This step may be reversed by metronidazole, which can reduced mitochondrial oxygen consumption. Secondly, after mitochondrial damage occurred, it leaded to intracellular energy depletion, flux of calcium and secondary generation of free radicals. Consequently, the breakdown of intercellular integrity occurred and increased intestinal permeability. Lastly, enterocyte was more vulnerable to damage by intraluminal intestinal contents for example bile, food, bacteria and enzymes[1-3]. Treatment with avoidance of NSAIDS is recommended and PPIs may protect against NSAID enteropathy. PPI may be helpful by antisecretory actions, anti-inflammatory, anti-oxidative mechanism and other protective mechanisms[4]. Other treatment with antibiotics such as metronidazole, sulphasalazine is under investigations and need more evidences for clinical use[5-6].

References

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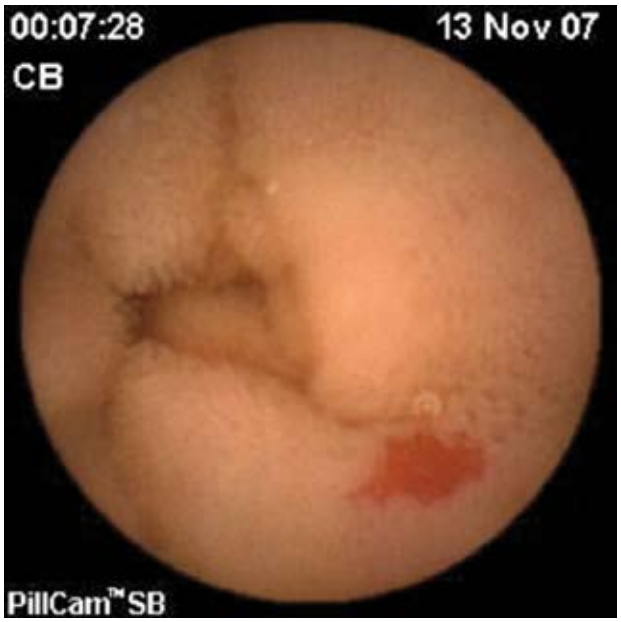
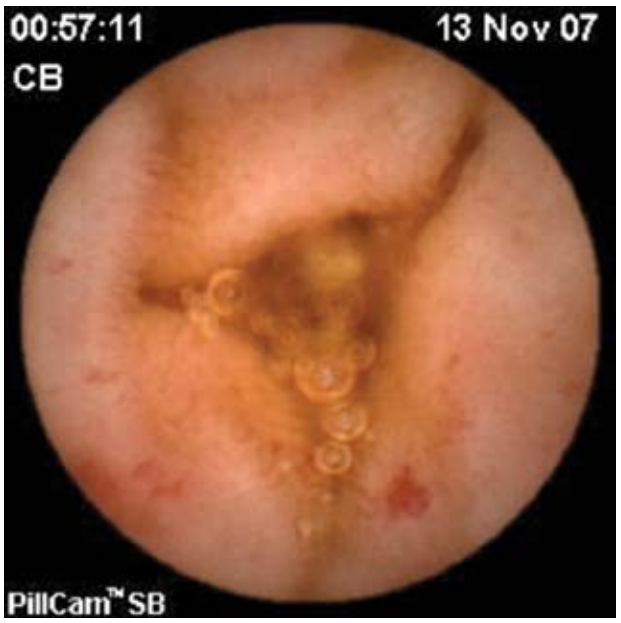
Case 3

Nathaya Tangmankongworakoon, MD.
Sombat Treeprasertsuk, MD.

A 90 year-old female present with hematochezia and got EGD and colonoscopy last 2 week from private hospital and showed normal study. Wireless capsule endoscopy (WCE) was performed and showed as figures:





WCE showed multiple abnormal vascular lesions in small intestine vary in size and shape. The final diagnosis is angiodysplasias of small intestine.

Discussion

A vascular lesion, angiodysplasia may present with active hemorrhage in about 60% and some patients present with recurrent GI bleeding[1]. Most common site of this lesion is in colon but it found in small intestine in 15-20% of angiodysplasia. Wireless capsule endoscopy (WCE) has become a valuable tool for the detection of this lesion. The diagnostic yield of WCE for obscure gastrointestinal (GI) bleeding has been established of 60% and angiodysplasia was one of the most common diagnoses[2]. New instrument such as double-balloon enteroscopy (DBE) is also a useful, safe and well-tolerated method with a high diagnostic and therapeutic impact for angiodysplasia[3]. The diagnostic efficacy of both WCE (65%) and DBE (53%) was not significantly different. However, DBE has a better role of therapeutic aspect[4]. Rebleeding after endoscopic intervention is one of the major concern and may occurred in about 20% of GI bleeding patients caused by angiodysplasias[5]. For the pharmacologic therapy, there was a study showed that Thalidomide had a potent inhibitor of angiogenesis in experimental models. It might regulated vessel growth or had antiangiogenic effect. Its dosage of 100 mg. daily, for 3 months with mean follow-up of 3 years showed substantial improvements in the number, size, and color intensity of angiodysplasias but it needed more information before using in clinical practice[6].

References

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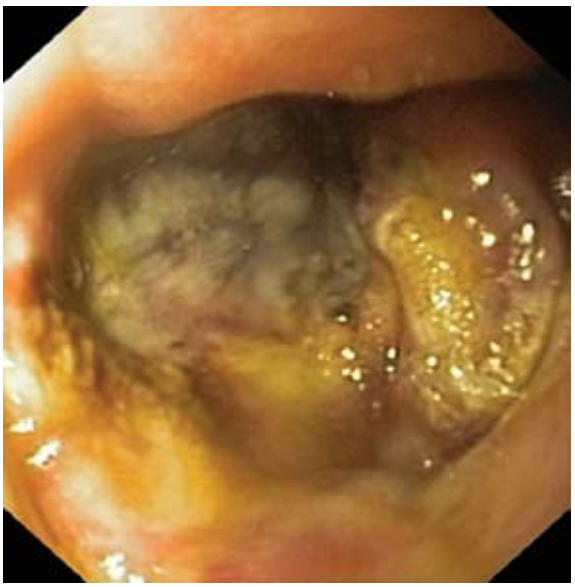
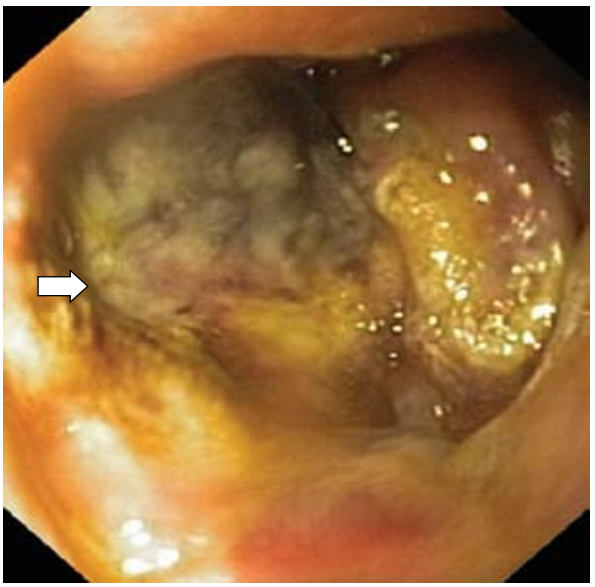
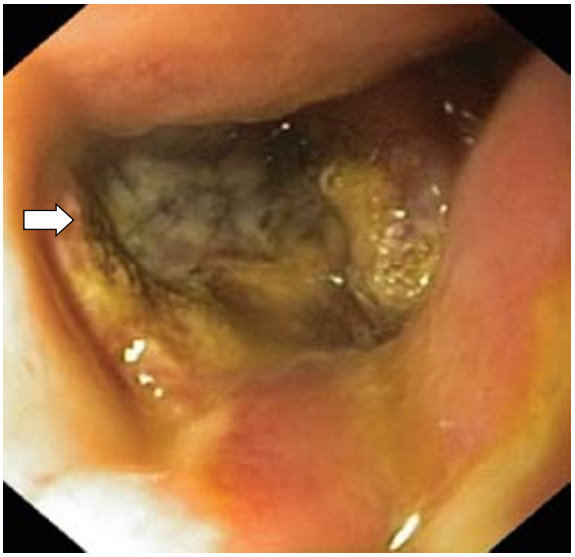
Case 4

Nathavut Sirimontaporn, MD.
Sombat Treeprasertsuk, MD.

A 75 years old male, admit at coronary care unit because of severe ischemic cardiomyopathy. During admission, he developed cardiogenic shock with ischemic stroke. Then, vasopressor and aspirin were given, and his clinical symptom was improved . Few days later, he passed bright red blood stool, and sigmoidoscopy was performed and shown as pictures.







Sigmoidoscopic findings : Rectum : large rectal ulcer with friability mucosa size 3x3 cm. at 5 cm. above anal verge with 2-3 hemorrhagic spot. Argon plasma coagulation was applied at lesion without immediate complication.

Discussion

Acute hemorrhagic rectal ulcer syndrome (AHRUS) is characterized by sudden onset, painless, and massive hemorrhage from rectal ulcer(s) in patients with serious underlying illnesses. It was first reported in Japan[1]. The diagnosed based on the following criteria 1) sudden onset of painless, massive rectal bleeding; 2) serious underlying disorders; 3) presence of ulcerations with ongoing bleeding or stigmata of recent bleeding in the rectum, as confirmed by colonoscopy; and 4) stool or biopsy cultures were negative[2]. The colonoscopic findings or rectal ulcers usually appeared as round, geographical, or Dieulafoy-like ulcer without significant surrounding inflammation, exudates, or ecchymosis. Histopathologic findings of rectal ulcer(s) showed evidence of necrosis with denudation of covering epithelium, hemorrhage, and multiple thrombi in the vessels of the epithelium and underlying stroma. Differential diagnosis of AHRUS includes solitary rectal ulcer syndrome (SRUS) which often occurs in young adults with a previous history of constipation, self-digitation. Stercoral ulcer which chronic constipation always precedes the occurrence of the disease, and sometimes residual solid fecal mass can be found. Infectious rectal ulcers is important to be excluded by stool examination or rectal biopsy or cultures. AHRUS can be treated by local cauterization, local injection with hemostasis agents, or suture ligation. However, the management of underlying disease and hemodynamic stabilization will ease and relief this problems.

References

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