中文題目：上消化道內視鏡止血後延遲之腸壁內出血 – 一病例系列報告

英文題目：Delayed Intramural Duodenal Hematoma Following Endoscopic Hemostasis - A Case Series

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Background*:* Intramural duodenal hematoma (IDH) is a fatal condition of gastrointestinal bleeding into the duodenal wall, which is a complication of mainly abdominal blunt trauma, usually occurring in young male populations. However, IDH caused by endoscopic treatment is rare but often life-threatening and has been reported to complicate pancreatitis, biliary sepsis, and bowel obstruction. Infrequent case reports have been published worldwide, and the direct cause leading to this complication has been debated, arguably either due to the patient's underlying coagulopathy or due to iatrogenic reasons. In reported literature, IDH is usually found within a few days after endoscopic intervention due to related symptoms presented, preferably abdominal pain, vomiting, or fever. In this case series, we present three cases of delayed onset of IDH after endoscopic hemostasis, in which IDH was discovered more than one week following the endoscopic intervention.

Case: Case one was an 87-year-old man with a past history of atrial fibrillation and receives anticoagulant Apixaban 5mg twice a day for many years. He received endoscopic exam due to presentation of tarry stool for three days, revealing an active bleeding duodenal ulcer at the duodenal bulb, Forrest classification 1b. Hypertonic saline with epinephrine (HSE) injection 16ml was administered along with argon plasma coagulation (APC) for hemostasis. On the eighth day after endoscopic hemostasis, fever was noted with dyspnea. Blood lab analysis suggested obstructive jaundice complicating biliary sepsis, and abdominal computed tomography (CT) revealed a large IDH measuring 11cm*8.5cm and extending to the right pararenal space. Antibiotic treatment was prescribed, and the patient was discharged on the fifth week of admission with physical condition returning to baseline status.

Case two was a 75-year-old man with a past history of coronary artery disease (two-vessel-disease) post stenting and under dual antiplatelet therapy with aspirin 100mg and clopidogrel 75mg daily. Panendoscopy was arranged due to tarry stool and abdominal pain for a day, which revealed an exposed vessel with active spurting bleeding over a diverticulum at the second portion of the duodenum, Forrest classification Ib. HSE 16ml and two hemoclips were applied for hemostasis. Thirteen days after hemostasis the patient brought to the emergency department complaining of abdominal pain. Computed tomography revealed an IDH combined with abscess formation measuring 11cm*8.2cm around the inferior portion of periduodenal space and extending to pararenal space. He was discharged four weeks later after receiving antibiotic treatment.

Case three was a 79-year-old man with a past history of coronary artery disease (two-vessel-disease) under daily Aspirin 100mg with chronic kidney disease, stage 3B. He received

emergent thoracic endovascular aneurysm repair (TEVAR) due to abdominal aortic aneurysm rupture, which hematemesis was noted two weeks after TEVAR. Upper endoscopy suggested bleeder at the second portion of the duodenum, and 10 c.c. of HSE was administered. Seven days following endoscopy shock status was presented. Blood lab analysis suggested internal bleeding, sepsis and obstructive jaundice. Abdominal CT revealed a large hematoma favoring IDH, measuring 9.8cm x 6.1cm, at retroperitoneum. Partial duodenectomy with Billroth II reconstruction were performed to attempt hemostasis, however he died of internal bleeding 10 days later following surgery.

Discussion: We report three cases of delayed onset of intramural duodenal hematoma after endoscopic hemostasis. What is notable is that all three patients reported all have a past history of cardiovascular diseases and are under either anticoagulants or antiplatelets; they are at an advanced age; and the latency period of intramural duodenal hematoma was over a week.

Estimated occurrence of IDH following endoscopic hemostasis were less than 0.1% according to previous literature. Over 80% of IDH is caused by blunt trauma, followed by bleeding disorders (coagulopathies, chronic renal diseases, or blood dyscrasias) and complications of anticoagulation therapy. However, in the few case reports published regarding IDH following endoscopic hemostasis, the onset time of IDH often occurred within a few days after hemostasis intervention, highlighting the uniqueness of our reported cases.

Intervention methods for gastrointestinal bleeding usually feature the use of hypertonic saline, epinephrine injection or sclerotherapy, while others report uses of composite hemostasis methods combining hemoclipping or application of fibrin glue. Common complications of IDH include jaundice, biliary sepsis and pancreatitis due to intra-abdominal hematoma compressing the biliary duct; and conservative management is often suggested in treating IDH when compared to invasive methods to avoid additional surgical trauma, or when the hematoma progresses. The admission period ranges from days to several weeks, depending on the patient's clinical condition. Most published cases report several weeks of admission owing to conservative treatment and the high mortality rates in IDH, which the patient often requires close observation and careful treatment.

The subject of this study is to point out the prolonged onset of IDH with its complications in our reported patients. Submural bleeding might continue to occur after endoscopic hemostasis, which would be indolent in the few consecutive days and not observed by endoscopy. Due to successful hemostasis in the intestinal lumen, the bleeding might not present as typical gastrointestinal bleeding, but rather as an intramural hematoma or even bleeding into the intra-abdominal spaces. Since the intramural hematoma is not formed immediately, the time that took to present clinical symptoms obstructing the bile duct and diagnosis would increase, thus the delayed presentation of IDH. The patients in this study were all at advanced age, which might play a role in atypical presentation of clinical symptoms that contribute to the longer latent period.

We presume that patients with certain underlying factors will increase the risk of developing delayed IDH, i.e., liver cirrhosis, impaired renal function or with coagulopathy disorders. In addition, patients who have been taking anti-coagulopathy medications are of greater risk as well. Although routine follow-up endoscopy is not necessary, we suggest a careful survey in patients at high-risk for delayed IDH.

Conclusion: Delayed IDH occurring more than one week later usually presents itself with gastrointestinal symptoms including nausea, vomiting, obstructive jaundice, pancreatitis or shock. This case series serves as an important reminder for clinicians to be alert of these warning signs and take necessary actions when they occur.