Intramural Duodenal Hematoma and Hemoperitoneum in Anticoagulant Therapy following Upper Gastrointestinal Endoscopy

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Introduction

Intramural hematoma of the duodenum is an uncommon but not rare entity. It is usually caused by blunt abdominal trauma [1], and occurs mainly in young men and children, with 82% of the patients being younger than 30 years [2]. Sometimes this lesion occurs in patients with pancreatic disease [3], coagulopathy [4], postendoscopic biopsy [5], or anticoagulant therapy [6]. Nevertheless, intramural duodenal hematoma and hemoperitoneum as complications following nontherapeutic upper gastrointestinal (UGI) endoscopy without biopsies in a patient on anticoagulant therapy has not been reported previously. The use of oral anticoagulants is gradually increasing among patients who have pulmonary embolism, deep venous thrombosis, prosthetic values, or persistent atrial fibrillation [7]. Hemorrhagic complications have been reported to occur in 10–30% of patients following prolonged oral anticoagulant administrations, and the incidence of intramural hematoma of the intestine in the setting of anticoagulation is 1 in 2,500 patients [8]. A single hematoma has been reported in 85% of patients, and multiple hematomas in 15% [9]. The jejunum was the most common site of the hematoma (69%), followed by the ileum (38%) and duodenum (23%) [9].
We describe a 74-year-old female on anticoagulant therapy, who developed an intramural hematoma 2 days after UGI endoscopy.

Case Report

A 74-year-old female patient was sent to the Emergency Department of Kaohsiung Medical University Hospital, Kaohsiung, Taiwan, ROC, because of a sudden onset of abdominal pain, nausea and vomiting. She had previously been healthy except for a past history of atrial fibrillation and ischemic heart disease, and had been taking warfarin (JH1) for 2 years. Following her complaint of upper abdominal pain for several days, fiberoptic UGI endoscopy (GIF-Q260; Olympus Medical System Corp., Tokyo, Japan) was performed by a gastroenterologist 2 days prior to this episode. Only superficial gastritis was noted and no endoscopic biopsy was taken. Nausea, postprandial vomiting, and lower abdominal pain developed 2 days after endoscopy and she was re-admitted for further management.

On general physical examination, the patient’s abdomen was mildly distended. Tenderness without muscle guarding was found over the lower quadrant of the abdominal wall. Her vital signs were stable. The laboratory data did not reveal any abnormality except a slightly lowered hemoglobin level of 10.4 g/dl. Her baseline hemoglobin level before this episode of symptoms was 12.5 g/dl; the prothrombin time (PT) and partial thromboplastin time (PPT) were 33.5 and 42.2 s, respectively. The international normalized ratio (INR) value was 2.7 (normal range = 1.5–2.5). She developed hypovolemic shock on the second day of admission and the blood pressure dropped to 74/38 mm Hg. Her hemoglobin fell to 7.7 g/dl; the PT, PPT and INR values rose to 34.1 s, 116.3 s and 7.6, respectively. After fluid resuscitation, blood transfusion with three units of packed red blood cells and correction of coagulopathy with fresh frozen plasma and parenteral administration of vitamin K, an abdominal computed tomography (CT) scan was carried out. The CT scan demonstrated an intramural duodenal hematoma of high attenuation, as an annular wall thickening of the second, third, and fourth portions of the duodenum with some free fluid collection in the bilateral pararenal spaces (fig. 1). Warfarin sodium was stopped immediately and nasogastric decompression was instituted. Total parenteral nutrition was also started for nutritional support during this period. Nine days later, an UGI study revealed the lumen of duodenum to be patent; therefore, the patient resumed oral intake 10 days after this nonoperative conservative management. Finally, she was discharged uneventfully, after 15 days of hospitalization. The resolution of hematoma was demonstrated by follow-up CT scans 1 month after onset (fig. 2).

Discussion

Though most duodenal intramural hematomas result from oral anticoagulant toxicity [6, 8, 9], they may also occur in patients from the performance of duodenal biopsies or injections. Our patient developed intramural duodenal hematoma and hemoperitoneum most probably due to anticoagulant therapy (warfarin) toxicity as
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Well as complications of UGI endoscopy. Between 1965 and 2004, there was no report of a clinical presentation of duodenal hematoma and hemoperitoneum as complications of anticoagulant therapy and diagnostic UGI endoscopy without any mucosal biopsy taken.

Intramural hematoma of the duodenum is usually accompanied by nausea, vomiting, and abdominal pain which varies from mild and vague to acute [1–9]. Abdominal pain is generally the most frequent initial symptom [10]. The presence of hemoperitoneum in our patient probably resulted from a large intramural duodenal hematoma that leaked into the peritoneum. Duodenal intramural hematoma should be considered in any patient with abdominal pain, nausea and vomiting who is receiving long-term anticoagulation therapy, particularly following diagnostic or therapeutic UGI endoscopy. Additionally, some patients can develop acute pancreatitis or biliary obstruction [3, 11]. Routine laboratory tests in such cases should include PT, PPT, and INR. Early non-invasive diagnosis can be made using UGI series and CT scans, and the latter is considered as the imaging modality of choice and may be helpful in differentiating duodenal perforation from hematoma without perforation [9, 10]. The treatment for this condition, as in our case, is best approached conservatively, since operative treatment is associated with a high complication rate and longer hospitalization [1, 12, 13]. Once the diagnosis is established and generalized peritonitis or transmural perforation ruled out by appropriate imaging studies, it is suggested that the patient be placed on nasogastric suction and parenteral hyperalimentation. Surgical intervention should be reserved for those with prolonged obstruction or those who have evidence of perforation or peritonitis. Most patients with significant resolution of the obstruction are allowed to resume oral intake within 2 weeks [5, 11]. Our patient was successfully managed with conservative treatment despite her associated hemoperitoneum and hypovolemic shock.

Conclusion

This case shows the possibility of development of an intramural duodenal hematoma in patients on anticoagulant therapy, without biopsies being taken.

References


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