Spontaneous intramural duodenal haematoma with transient biliary obstruction and acute cholecystitis

Basir N, Chong V H

ABSTRACT

Intramural duodenal haematoma (IDH) is uncommon and usually presents with upper gastrointestinal bleeding. Trauma, anticoagulant therapy, blood coagulation disorders and endoscopic interventions have been reported to cause IDH. IDH secondary to antiplatelet therapy has not been previously reported in the literature. We report IDH secondary to aspirin therapy that was associated with transient obstructive jaundice and acute cholecystitis in a 47-year-old woman. The patient was successfully managed with conservative management.

Keywords: aspirin, gastrointestinal haemorrhage, haematoma, obstructive jaundice

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INTRODUCTION

Intramural haematoma of the gastrointestinal tract, including intramural duodenal haematoma (IDH), is uncommon and mostly traumatic in nature.[1-3] Spontaneous IDH usually occurs in association with anticoagulant therapy,[4-7] but that of antiplatelet therapy has not been previously reported. We report a case of spontaneous IDH secondary to aspirin therapy that was complicated by transient biliary obstruction and acute cholecystitis.

CASE REPORT

A 47-year-old woman from a neighbouring country presented with a one-week history of abdominal pain and an episode of haematemesis (approximately 50 ml). Her background medical history included hypertension, type 2 diabetes mellitus, cerebrovascular accident with minimal residual right-sided hemiparesis and inactive systemic lupus erythematosus (SLE). The patient did not have any history of gastrointestinal complaints prior to her presentation. Her medications included amlodipine, perindropril, metformin, glicazide and soluble aspirin. Aspirin was only started one month ago.

Physical examination revealed tachycardia at 120 beats per minute, but the patient was normotensive. She had mild right hypochondrium tenderness without guarding. Per rectal examination was normal. The rest of the examinations were unremarkable. Laboratory investigations showed normocytic normochromic anaemia (Hb 11.4, normal range [NR] 12.0–16.0 gm/dL), mild renal impairment (serum urea 7.6, NR 2.9–7.1 mmol/L), a creatinine level of 147 (NR 53–115) μmol/L and a cholestatic liver profile (serum alkaline phosphatase 295 [NR 38–126] U/L, serum gammaglutamyl transferase 88 [NR 7–64] U/L and serum bilirubin 107 [NR 3.4–17.1] μmol/L). The coagulation profile and serum amylase were normal. An urgent ultrasonography showed a slightly thickened gallbladder with pericholecystic fluid. No stones were noted and the bile ducts were normal. The working diagnoses were acute cholecystitis and probable Mallory-Weiss tear of the oesophagus. The patient was started on oral omeprazole, intravenous fluid and intravenous antibiotics (cefuroxime 750 mg twice daily and metronidazole 500 mg thrice daily). As she was haemodynamically stable with no further haematemesis, upper gastrointestinal endoscopy was deferred due to the patient's cost constraints. There were no signs and symptoms to suggest active lupus.

The patient remained asymptomatic in the first 24 hours but later developed fever and another episode of haemetamesis. Her serum haemoglobin level dropped by 3 gm and her coagulation profiles became slightly deranged (prothrombin time 14.7 [NR 10.5–13.5] seconds and international normalised ratio 1.4 [NR 0.8–1.2]). Interestingly, a repeat liver profile showed improvement. An upper gastrointestinal endoscopy showed a submucosal mass with ulceration and an underlying clot (Fig. 1). Mild oozing was observed around the clot, and it was injected with 5 ml of adrenaline (1 mg in 1/10,000 dilution). An urgent computed tomography (CT) imaging showed a distended first part of the duodenum and a mass with air pockets extending from the duodenal bulb to the third part of the duodenum (Fig. 2). The lumen was obliterated. These findings were consistent with an IDH and also indicated

Gastroenterology Unit, Department of Medicine, Raja Isteri Pengiran Anak Saleha Hospital, Bandar Seri Begawan, BA 1710, Brunei Darussalam

Basir N, MBBS
Medical Officer

Chong VH, MRCP, FRCP, FAMS
Consultant

Correspondence to:
Dr Chong Vui Heng
Tel: (673) 877 8218
Fax: (673) 224 2690
Email: chongvuih@yahoo.co.uk

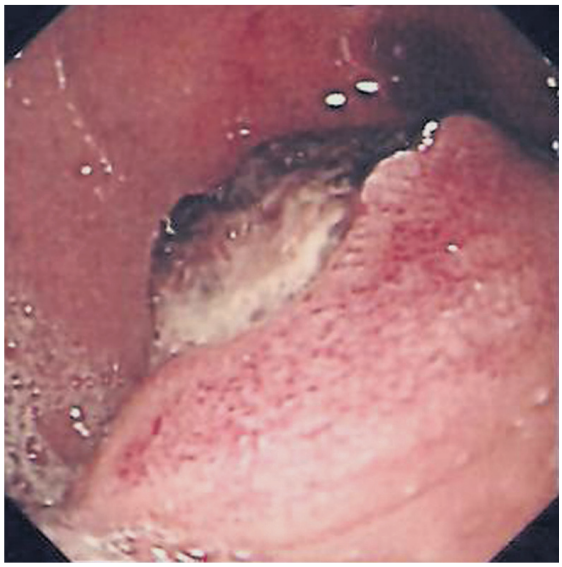

Fig. 1 Endoscopic image of the duodenal mass shows a blood clot protruding from the mucosa breech.

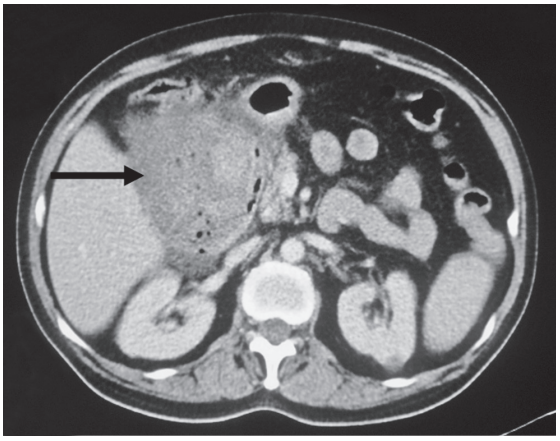

Fig. 2 Axial CT image shows intramural duodenal haematoma with air pockets (arrow) and obliteration of the duodenal lumen.

a thickened gallbladder with pericholecystic oedema. No gallstones were seen in the gallbladder.

A surgical consult was obtained. As the patient was haemodynamically stable, conservative management was continued. She was transfused with a total of eight units of packed cell and four units of fresh frozen plasma. She was kept nil per oral and was continued on intravenous acid suppression (omeprazole 40 mg twice daily) and antibiotics. A repeat oesophagogastroduodenoscopy on the fifth day showed a duodenal ulcer with a haematoma at the ulcer base. Distally, the lumen was partially compressed and a punctum with slow oozing of stale blood was seen. *Helicobacter (H.) pylori* testing was negative. The patient recovered and was discharged 18 days later. She was seen once post discharge and had remained well. The patient then returned to her home country for further management.

DISCUSSION

Spontaneous intramural haematoma is rare and most commonly associated with anticoagulation therapy.[4-6] It is estimated that one in 2,500 patients on anticoagulation therapy per year (0.04% per year) is at risk of this complication.[4] The most commonly affected site in anticoagulation-associated intramural haematoma is the jejunum (64%–69%) followed by the ileum (26%–38%) and duodenum (10%–23%).[4,8] This is in contrast to traumatic intramural haematoma where the duodenum is most commonly affected. Traumatic IDH occurs mainly in children, usually after abdominal trauma such as falls and bicycle handlebar accidents. The clinical presentations, diagnosis and management are generally similar for spontaneous and trauma-associated IDH. The only difference is the need to manage any associated injuries in trauma-associated haematoma, while the correction of coagulopathy is required in spontaneous haematoma.

Common presentations of IDH include upper abdominal pain with or without haematemesis, malaena, nausea and vomiting.[3-6] Interestingly, IDH complicated by obstructive jaundice, acute pancreatitis and perforation with peritonitis have also been reported.[3,9] The diagnosis of IDH can be made by endoscopy, and features of a bluish submucosal mass with or without ulcerations or clot should raise the suspicion of IDH. However, as seen in our case, the diagnosis of IDH can be missed, especially if the endoscopist is unfamiliar with it. Currently, contrast-enhanced CT is reported to be the best imaging modality for diagnosing IDH.[1]

The management of IDH consists of haemodynamic support such as blood transfusion, and the correction of coagulation abnormalities for coagulopathy-associated IDH. Acid suppression is also important, especially if there are ulcerations or erosions. In fact, most cases of IDH can be successfully managed with conservative management alone.[3-6] However, in some cases, other interventions, including surgery and guided drainage, may be required if the bleeding or obstructive symptoms do not resolve.[10,11] Complete resolution of IDH usually occurs within three weeks after the onset, varying from ten days to three months.[12]

Apart from anticoagulation therapy, associations with heparin therapy, haemostatic disorders, ulcers and acute pancreatitis have also been reported to be associated with spontaneous intramural haematoma.[7,13-15] However, to our knowledge, while IDH in association with aspirin therapy has not been previously reported, antiplatelets-

induced oesophageal haematoma has been reported in the literature.[16]

In our patient, the only risk factor was aspirin therapy. She did not have any history of dyspepsia, peptic ulcer disease, gallstones disease or any abdominal trauma, and her coagulation profile was normal on admission. It is very likely that the incident had started with an ulcer in our patient. Erosions into the submucosal vessels and bleeding between the mucosa and the submucosal plane will lead to intramural haematoma. The ulcer is likely to be aspirin-related, especially since our patient was negative for *H. pylori* and only complained of dyspepsia after starting on aspirin therapy. However, testing for *H. pylori* was only based on the rapid urease test, which is known to be associated with low sensitivity in our local setting.[17] The presence of platelet dysfunction secondary to aspirin was probably an important contributing factor. In our case, we believed that the transient biliary obstructions were due to the IDH, as both ultrasonography and CT imaging did not show any gallstones or other biliary abnormalities. The improvement in the liver profile of our patient after the second haematemesis may have been due to the evacuation of the haematoma, which was sufficient to result in the relief of biliary obstruction. However, it would have been useful to have performed a repeat testing for *H. pylori* and repeat imaging after recovery, as biliary stones or sludge can be missed. Although SLE causing vasculitis and thrombocytopenia was another possibility in our patient, it was very unlikely as the SLE was not active.

In conclusion, our case highlights a rare but important complication of antiplatelet therapy. As more patients are now being prescribed antiplatelet therapy, the number of complications may increase, and it is important that clinicians are aware of such rare complications.

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