Intramural Duodenal Hematoma after Endoscopic Therapy for a Bleeding Duodenal Ulcer in a Patient with Liver Cirrhosis

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Abstract

We report a case of intestinal obstruction due to intramural hematoma of the duodenum following therapeutic endoscopy for a bleeding duodenal ulcer in a patient with liver cirrhosis. A 44-year-old man was admitted to our hospital with severe epigastralgia, nausea and tarry stool. Two years previously he had undergone endoscopic sclerotherapy for esophageal varices caused by alcoholic liver cirrhosis. Endoscopy revealed an open ulcer with a bleeding vessel in the duodenal bulb, and sclerotherapy was performed by clipping the vessel and injecting 20 ml of 0.2% epinephrine. His platelet count was 3.5×10^4/μl. Twelve hours later, he again developed epigastralgia and hypotension. Emergency computed tomography and ultrasonography revealed an intramural hematoma, 15×18 cm in diameter, at the dorsal and lateral duodenum. Endoscopy and upper gastrointestinal series revealed severe stenosis of the duodenal lumen caused by intramural hematoma. He received parenteral feeding for 22 days and within 8 weeks the hematoma was gradually absorbed using conservative management. Intramural duodenal hematoma may be diagnosed as a complication of the endoscopic procedure in a patient with a bleeding tendency, such as liver cirrhosis.

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Key words: intramural duodenal hematoma, endoscopic therapy, liver cirrhosis

Introduction

Intramural hematoma of the duodenum is an uncommon condition, which develops after blunt abdominal trauma (1–3). It is also, and less commonly, reported as a complication of anticoagulant therapy, blood dyscrasia, pancreatic disease and collagen vascular disease (1–6). Intramural hematoma of the duodenum has been reported as a complication of diagnostic or therapeutic endoscopy (7–15). We report a case of intramural hematoma of the duodenum after clipping of a bleeding duodenal ulcer and endoscopic injection of epinephrine in a patient with liver cirrhosis, and review reports of iatrogenic intramural hematoma of the duodenum.

Case Report

A 44-year-old man with a four-year history of liver cirrhosis was admitted to our hospital on January 2, 2003 with fever, epigastralgia and tarry stool. He had been a heavy drinker for over 24 years, and two years previously he underwent endoscopic sclerotherapy for esophageal varices. He had not taken non-steroidal anti-inflammatory drug just before hospitalization. On admission, a physical examination revealed epigastric tenderness but no jaundice, anemia, ascites or hepatosplenomegaly. His temperature was 38.7°C, pulse rate 111 per minute, and blood pressure 133/66 mmHg. Relevant laboratory test results were as follows: hemoglobin 11.7 g/dl; leukocytes count 5,100/mm³; platelet count 35,000/mm³; prothrombin time 128%; partial thromboplastin time 33 seconds; serum albumin 4.1 g/dl; total bilirubin 2.7 mg/dl; serum aspartate transaminase (AST) 213 U/l; serum alanine transaminase (ALT) 111 U/l; and total cholesterol 77 mg/dl. Hepatitis B surface (HBs) antigen, hepatitis B core antibody and HCV antibody were all negative. The severity of cirrhosis was therefore classified as Child-Pugh grade A and the etiology suggested an alcohol cause, not virus. The fever on admission was caused by type A influenza. Gastrointestinal endoscopy revealed an open ulcer at the superior
A total of 20 ml of 0.2% epinephrine was injected locally and the bleeding vessel was clipped. The gastrointestinal endoscopy also revealed the existence of reflux esophagitis and gastric erosion, and absence of recurrent esophageal varices. The urease test was positive for Helicobacter pylori infection.

He developed severe epigastralgia and hypotension (70 mmHg of systolic blood pressure) approximately 12 hours after the endoscopic treatment. Emergency abdominal computed tomography and ultrasonography revealed a huge hematoma at the dorsal and lateral duodenal wall, 15x18 cm in diameter (Fig. 1). Furthermore, the hemoglobin concentration had decreased to 7.8 g/dl and the serum amylase level had increased from 60 IU/l on admission to 1,441 IU/l (Table 1). Endoscopic examination revealed nearly complete exclusion of the lumen of the second portion of the duodenum by the hematoma. According to these findings, diagnosis of acute pancreatitis and duodenal obstruction due to duodenal intramural hematoma was made. The patient was treated with intravenous administration of H2 blocker. In addition, he was conservatively managed with continuous nasogastric suction and total parenteral nutrition. The serum amylase level decreased to 90 IU/l after five days. After twenty-two days an upper gastrointestinal series revealed a severe stenosis of the second portion of the duodenum with a fistula formation from the oral side of the stenotic lumen to the intramural hematoma (Fig. 2). It was thought that the fistula was formed by the increased internal pressure of the hematoma. Subsequently, the hematoma gradually decreased.

![Figure 1](image1.png)

**Figure 1.** Abdominal computed tomography reveals a huge hematoma in the lower part outside the clipping (arrow) at the duodenal bulb (A), stenosis of the lumen (arrow) of the descending duodenum (B) and the largest part of hematoma (arrow) 15x18 cm in diameter (C).

![Figure 2](image2.png)

**Figure 2.** An upper gastrointestinal series 22 days later demonstrate that the intramural hematoma pressed the second portion of the duodenum from the outside (arrowheads) and is connected to the oral side of the stenotic lumen by a fistula formation (arrow). Contrast media is depicted in the duodenum lumen and the hematoma.

### Table 1. Laboratory Data on Admission and One Day after Endoscopic Procedure

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Jan 2, 2003 on admission</th>
<th>Jan 5, 2003 1 day after endoscopic procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>TBil</td>
<td>2.7 mg/dl</td>
<td>0.8</td>
</tr>
<tr>
<td>AST</td>
<td>213 U/l</td>
<td>80</td>
</tr>
<tr>
<td>ALT</td>
<td>111 U/l</td>
<td>52</td>
</tr>
<tr>
<td>Amylase</td>
<td>60 IU/l</td>
<td>1,441</td>
</tr>
<tr>
<td>Hb</td>
<td>11.7 g/dl</td>
<td>7.8</td>
</tr>
<tr>
<td>Platelet</td>
<td>35,000 /mm³</td>
<td>40,000</td>
</tr>
</tbody>
</table>
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in size on both ultrasonography and computed tomography (Fig. 3). Total parenteral nutrition was changed on the 22nd day by an oral intake of meal. An upper gastrointestinal series 8 weeks later showed that the duodenal stenosis had almost completely disappeared and the fistula no longer existed (Fig. 4).

Discussion

Intramural hematoma of the duodenum is a complication of the blunt trauma to the abdomen. More than 80% of cases have a history of abdominal trauma, and over 60% of these are reported in children under 15 years old (1–3). Intramural hematoma is most commonly observed in the duodenal wall of the gastrointestinal tract. The descending portion of duodenum is located in front of the vertebral columns and fixed by retroperitoneum. It has a rich submucosal vascular supply (1). The duodenum is thus easily injured by external force. Nontraumatic intramural hematomas of the duodenum are reported to develop for other reasons such as anticoagulant therapy, blood dyscrasia, pancreatic diseases, ruptured aneurysms of the intestinal artery and endoscopic procedure for duodenal mucosa (1–15).

To our knowledge, 16 cases of nontraumatic intramural hematoma of the duodenum have been reported as a complication of endoscopic biopsy or sclerotherapy (8–15). As shown in Table 2, six cases of duodenal hematomas are reported after endoscopic therapy for bleeding ulcers (13–15). Four patients had a coagulation disorder, such as liver cirrhosis, HELLP syndrome or end-stage renal failure. In the present patient, severe thrombocytopenia associated with liver cirrhosis was present.

In all therapeutic modalities for bleeding ulcers, local injection of epinephrine, polidocanol and fibrin tissue adhesive onto the mucosa was most commonly used. Rohrer et al reported that the local injection method causes tissue damage in variable degrees, possibly leading to the development of intramural hematoma (14). Epinephrin was used for hemostasis in all cases. It would appear, however, that epinephrine does not cause duodenum hematoma more easily, as it is used for most hemostasis treatment of hemorrhagic ulcers.

The amount of epinephrine used for the present patient, 20 ml, was greater than the reported cases. It was considered, therefore, that a large amount of epinephrine may cause a patient with liver cirrhosis and thrombocytopenia to develop intramural hematoma more easily.

Four patients suffered from the complication of pancreatitis, probably due to the compression of the papilla of Vater by hematoma. Jones et al (1) reported that serum amylase levels elevated in 16.4% among 116 cases, probably because of direct pancreatic trauma and/or ampullary obstruction by hematoma. In the present case, serum amylase levels elevated to 1,441 IU/l and decreased to the normal range on the sixth day.

Intramural hematoma of the duodenum is mainly treated with surgical procedure or conservative therapy. Until the

Figure 3. Abdominal computed tomography 8 weeks later shows the deceased hematoma (arrow).

Figure 4. An upper gastrointestinal series 8 weeks later shows that the duodenal stenosis had almost completely disappeared and the fistula was not present.
Intramural Duodenal Hematoma after Endoscopy

Table 2. Intramural Hematoma after Endoscopic Therapy for Bleeding Duodenal Ulcers

<table>
<thead>
<tr>
<th>Author</th>
<th>Patient’s age (years)</th>
<th>Underlying disease</th>
<th>Location of the ulcer</th>
<th>Therapy for bleeding ulcer</th>
<th>Volume injected (ml)</th>
<th>Latency</th>
<th>Size of hematoma (cm)</th>
<th>Complication</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sadry (13)</td>
<td>Adult/F</td>
<td>Acute renal failure on hemodialysis due to interstitial nephritis</td>
<td>Second portion of the bulb</td>
<td>Epinephrine and aethoxysklerol</td>
<td>12</td>
<td>The next day</td>
<td>4</td>
<td>Jaundice, Necrotizing pancreatitis</td>
<td>Conservative</td>
<td>Died after 2 weeks due to necrotizing pancreatitis</td>
</tr>
<tr>
<td>Rohrer (14)</td>
<td>22/F</td>
<td>HELLP syndrome</td>
<td>Interior wall of the bulb</td>
<td>Epinephrine and polidocanol</td>
<td>12</td>
<td>Within 24 hrs</td>
<td>8x10x13</td>
<td>Spontaneous absorption</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rohrer (14)</td>
<td>51/M</td>
<td>Alcoholic liver cirrhosis (Plt 40,000/ml)</td>
<td>Posterior wall of the bulb</td>
<td>Epinephrine and tissucol</td>
<td>7</td>
<td>Within 24 hrs</td>
<td>5x10</td>
<td>Pancreatitis</td>
<td>Conservative</td>
<td>Spontaneous absorption</td>
</tr>
<tr>
<td>Rohrer (14)</td>
<td>61/M</td>
<td>End-stage renal failure</td>
<td>Interior wall of the bulb</td>
<td>Epinephrine and laser</td>
<td>8</td>
<td>24 hours later</td>
<td>4x8</td>
<td>Pancreatitis</td>
<td>Conservative</td>
<td>Spontaneous absorption</td>
</tr>
<tr>
<td>Rohrer (14)</td>
<td>78/M</td>
<td>End-stage renal failure, Goodpasture’s syndrome</td>
<td>Posterior wall of the bulb</td>
<td>Epinephrine and tissucol</td>
<td>24</td>
<td>48 hours later</td>
<td>2x2</td>
<td>Pancreatitis</td>
<td>Gastroctomy for bleeding ulcer</td>
<td>Died 20 days after laparotomy for sepsis</td>
</tr>
<tr>
<td>Han (15)</td>
<td>37/M</td>
<td>None</td>
<td>Anterior wall of the bulb</td>
<td>Epinephrine</td>
<td>Not described</td>
<td>A few hours later</td>
<td>Not described</td>
<td>Pancreatitis</td>
<td>Laparoscopic drainage of the hematoma</td>
<td>Improved 11 days later</td>
</tr>
<tr>
<td>Present case</td>
<td>44/M</td>
<td>Alcoholic liver cirrhosis (Plt 35,000/ml)</td>
<td>Superior wall of the bulb</td>
<td>Epinephrine and clipping</td>
<td>20</td>
<td>12 hours later</td>
<td>15x18</td>
<td>Pancreatitis</td>
<td>Conservative</td>
<td>Spontaneous absorption</td>
</tr>
</tbody>
</table>


References
4) Fingerhut A, Rouffet F, Eugene C, Fendler JP, Hillion D, Ronat R., early 1970’s, most patients were treated surgically, usually by the excision of the hematoma and/or bypass surgery (1–3). However, recent reports support the non-operative treatment associated with nasogastric suction and central hyperalimentation (4, 16). The abundant blood supply of the duodenal wall is expected to absorb the hematoma promptly, and postoperative complications including intestinal ileus, pancreatitis, and wound healing problems may be avoided (16). Although the present patient’s hematoma was the largest in size (15×18 cm) among the reported cases, with conservative treatment, the size reduced steadily and the duodenal stenosis was almost completely back to normal within 8 weeks.

In summary, we reported a case of intramural hematoma of the duodenum following endoscopic ethanol injection for a bleeding duodenal ulcer. After endoscopy, careful follow-up monitoring is needed for a patient with liver cirrhosis or a coagulation disorder.

References