Superior Mesenteric Artery Syndrome Caused by Huge Mycotic Abdominal Aortic Aneurysm

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Abstract

A 92-year-old man who had been hospitalized for dementia developed sudden-onset bilious vomiting accompanied by a fever of 40°C. Physical examination revealed an 8 cm diameter pulsatile mass in the upper abdomen. Computed tomography of the abdomen demonstrated a huge infrarenal saccular aneurysm with a lobulated appearance. We considered this to be a mycotic abdominal aortic aneurysm compressing the third portion of the duodenum and causing proximal duodenal dilatation and superior mesenteric artery (SMA) syndrome.

Key words: mycotic aneurysm, superior mesenteric artery (SMA) syndrome intermittent shaking chill, bilious vomiting, pulsatile abdominal mass

Introduction

Although widespread use of antibiotics has decreased the incidence of mycotic aneurysms, they still account for up to 2.6% of all abdominal aortic aneurysms with a 75% frequency of life-threatening hemorrhage and overall mortality of 67% (1, 2). A few authors have reported superior mesenteric artery syndrome (SMA syndrome) caused by non-mycotic arteriosclerotic aortic aneurysm (3-7), but to our knowledge, none have reported mycotic aneurysm as the cause. We present a case of SMA syndrome caused by a huge infrarenal abdominal mycotic aneurysm.

Case Report

A 92-year-old man who had been hospitalized for dementia for one year suddenly developed bilious vomiting (Day 0). He had been in good health, with a past medical history of a permanent cardiac pacemaker for complete atrioven-

tricular block and a colostomy after surgery for colon cancer. Shortly before the episode, he had developed repeated aspiration and worsening productive cough. On physical examination, his consciousness was impaired to level I-2 on the Japan Coma Scale; blood pressure was 120/72 mmHg and pulse rate 78 beats/min. He had a high fever of 40°C, his respiratory rate was 20 breaths/minute with oxygen saturation of 97% on ambient air; and there were no abnormal findings in his abdomen. Laboratory data on day 3 showed an elevated white blood cell count (WBC) and C-reactive protein (CRP) level. Although auscultation and X-ray of the chest were normal, we suspected a respiratory tract infection caused by aspiration because of the preceding aspiration and respiratory symptoms. He was treated with nasogastric aspiration and intravenous ampicillin/sulbactam (6 g/day), and his inflammatory markers improved from Day 3 to Day 12 (WBC decreased from 11,400/μL to 7,800/μL and CRP from 23 mg/dL to 7.9 mg/dL). Laboratory data on day 12 showed anemia, hypoproteinemia, elevation of lactate dehydrogenase (LDH) and mildly impaired renal function (Ta-
Table 1. Laboratory Data

<table>
<thead>
<tr>
<th>Variable</th>
<th>Day12</th>
<th>Day25</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC (/µL)</td>
<td>7,800</td>
<td>6,200</td>
</tr>
<tr>
<td>Differential count (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stab</td>
<td>19</td>
<td>20</td>
</tr>
<tr>
<td>Seg</td>
<td>60</td>
<td></td>
</tr>
<tr>
<td>Lympho</td>
<td>13</td>
<td>15</td>
</tr>
<tr>
<td>Eosino</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>RBC (X10^4/µL)</td>
<td>280</td>
<td>269</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>24.1</td>
<td>22.6</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>7.5</td>
<td>7.1</td>
</tr>
<tr>
<td>Platelet (/µL)</td>
<td>41.2</td>
<td>14.4</td>
</tr>
<tr>
<td>Na (mEq/L)</td>
<td>149</td>
<td>141</td>
</tr>
<tr>
<td>K (mEq/L)</td>
<td>3.3</td>
<td>2.9</td>
</tr>
<tr>
<td>Cl (mEq/L)</td>
<td>97</td>
<td>90</td>
</tr>
<tr>
<td>BUN (mg/dL)</td>
<td>28</td>
<td>22</td>
</tr>
<tr>
<td>Creatinine (mg/dL)</td>
<td>1.5</td>
<td>1.6</td>
</tr>
<tr>
<td>Total Bilirubin (mg/dL)</td>
<td>1.0</td>
<td>0.4</td>
</tr>
<tr>
<td>GOT (IU/L)</td>
<td>31</td>
<td>20</td>
</tr>
<tr>
<td>GPT (IU/L)</td>
<td>40</td>
<td>13</td>
</tr>
<tr>
<td>LDH (IU/L)</td>
<td>829</td>
<td>640</td>
</tr>
<tr>
<td>Total Protein (g/dL)</td>
<td>5.4</td>
<td>5.1</td>
</tr>
<tr>
<td>Albumin (g/dL)</td>
<td>1.8</td>
<td>1.6</td>
</tr>
<tr>
<td>CRP (mg/dL)</td>
<td>7.9</td>
<td>12.3</td>
</tr>
</tbody>
</table>

Despite the improvement in inflammatory markers, the amount of bilious fluid drained from the nasogastric tube gradually increased to up to 3 L/day, and on Day 15 examination of the upper abdomen revealed a pulsatile mass, 8 cm in diameter, with cutaneous edema. Around the same time, he also developed intermittent chills.

Contrast-enhanced abdominal computed tomography (CT) on Day 19 demonstrated a huge infrarenal saccular aneurysm with a lobulated appearance. The lesion compressed and severely obstructed the third portion of the duodenum and the superior mesenteric artery (SMA), and we supposed that this had caused SMA syndrome (Fig. 1A). The amount of fluid aspirated from the nasogastric tube was reduced by changing to the left lateral decubitus position, which is a typical symptom of SMA syndrome. In addition, although no organisms were isolated from 3 sequential sets of blood cultures, CT showed an abscess compressing the left iliopsoas muscle (Fig. 1A-C). This suggested that the abscess had originated from the left-side wall of the aortic aneurysm, generated aortitis, and expanded contiguously to the left iliopsoas muscle. Abdominal CT performed one year previously showed no abnormalities in the abdominal aorta (Fig. 1D).

We considered that the SMA syndrome was caused by the huge infrarenal aneurysm, which had several characteristics of mycotic aneurysms: 1) rapid growth, 2) lack of calcium deposition in the aneurismal outer wall of the aorta, 3) multilobular appearance, 4) saccular configuration, and 5) peri-aortic soft tissue mass. Therefore, the antimicrobials were changed to intravenous meropenem (1.5 g/day) and vancomycin (1 g/day) to target pathogens likely to cause mycotic aneurysm such as Pseudomonas aeruginosa and methicillin-resistant Staphylococcus aureus.

During the next two weeks, his conscious level (JCS; I-2) and general condition were stable, as were the inflammatory markers (WBC count, 6,200/µL; CRP, 12.3 mg/dL at day 25) (Table 1). However, he continued to have intermittent shaking chills with a fever of 38°C, and the pulsatile mass in the upper abdomen enlarged to 10 cm. On Day 37, the nasogastric aspirate became blood-stained/rust-colored, suggesting bleeding originating from a gastroduodenal ulcer or a fistula between the stenotic portion of the digestive tract and the mycotic aneurysm. On day 39, the patient developed shock, consciousness level deteriorated further to JCS III-3, and he died on day 40.

Discussion

Osler described the infected aortic aneurysm, or so-called mycotic aneurysm, in 1885 (8). These lesions represent less than 3% of all aortic aneurysms (1, 9). Back pain, a palpable and pulsatile abdominal mass, fever, and bacteremia are well-known clues to the clinical diagnosis (10, 11). Non-mycotic arteriosclerotic abdominal aortic aneurysms are reported to increase in size at a mean rate of 0.2 to 0.5 cm per year. In the present case, the aneurysm rapidly expanded to 8-10 cm in diameter and was accompanied by abscess formation and intermittent shaking chills, findings consistent with bacteremia (2) and mycotic aneurysm. Therefore, although no organisms were isolated in repeated blood cultures, we considered the case to be of infectious origin. Although Salmonella and Staphylococcus species are the pathogens commonly isolated from blood or organ cultures (9), the frequency of positive blood or tissue cultures in patients with mycotic aortic aneurysms is no more than 60 to 70% (1, 12).
Rokitansky first described vascular compression of the
duodenum, sometimes termed SMA syndrome, arteriomes-
tenteric duodenal compression, or Wilkie’s syndrome (13).
The syndrome occurs when the aortomesenteric angle di-
minishes to 6 to 16 degrees, and reported risk factors in-
clude conditions such as severe wasting diseases, prolonged
bed rest, anorexia nervosa, malabsorption, and use of a body
cast.

Although our patient had none of the cited risks for SMA
syndrome, his bilious vomiting was ameliorated by left lat-
eral decubitus positioning, a typical symptom of SMA syn-
drome (5). Other findings, including a pulsatile, rapidly ex-
panding abdominal mass, pyrexia refractory to antimicrobial
treatment, and intermittent shaking chills suggested the co-
existence of a mycotic aneurysm. Therefore, we consider
that our case demonstrates a novel mechanism of SMA syn-
drome, in which the precursor was mycotic aneurysm. Al-
though a few cases of SMA syndrome caused by non-
mycotic arteriosclerotic aortic aneurysms have been reported
(5-7, 14, 15), ours appears to be the first case attributable to
a mycotic abdominal aortic aneurysm.

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