Case Report

Acute Pancreatitis After Acute Aortic Dissection: Report of a Case and Review of the Literature

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ABSTRACT
Acute aortic dissection (AAD) is an uncommon clinical picture with a high mortality rate. Early diagnosis and treatment are important in decreasing the mortality. Classically, AAD is characterized by a severe sudden onset pain of the chest, back, and abdomen. However, the patients with AAD may admit with a variety of different clinical findings as due to level of dissection in the aorta. In this study, our aim is to emphasize that the AAD rarely will cause gastrointestinal symptoms. In this paper, we reported a case of ADD caused acute pancreatitis, presenting in emergency department with abdominal pain.

Key words: Acute pancreatitis, aortic dissection, gastrointestinal complication

ÖZET
Akut aort diseksiyonu sonrası gelen akut pankreatit bir olgunun sunumu ve literatürün gözden geçirilmesi


Anahtar kelimeler: Akut pankreatit, aort diseksiyonu, gastrointestinal komplikasyon

Acute aortic dissection (AAD) is relatively rare and is often fatal disease caused by a tear in the aortic intima (1). The presenting symptoms and signs of aortic dissection are so myriad and nonspecific that dissection may be overlooked initially in up to 40% of cases (2). Thus the patients with AAD may present to emergency department with very different complications in relation with affected area. It is clear that the frequency of AAD induced complications is according to localizations involved of the aorta. However, the number of patients presenting with AAD induced pancreatitis is uncommon in the literature.

Major gastrointestinal complications such as gastrointestinal hemorrhage, dysphagia, acute abdomen, bowel obstruction, and ischemic colitis have been referred to occur after aortic dissection in the published English literature (3-5). This case report describes a patient with AAD induced pancreatitis, who was initially presented with abdominal pain.

CASE REPORT

A 52-year-old male was admitted to our emergency service with complaints of suddenly started severe epigastric pain and nausea.

The patient said that the pain was very severe and had spreaded towards his chest and left arm.

He had a history of hypertension diagnosed 4 years ago and regulated by antihypertensive drugs. On physical examination, the abdomen was tender in all areas. Blood pressure was 110/70 mmHg; pulse rate, 60/minute; and respiration rate, 20/minute. The main laboratory findings were: WBC count, 11900/uL (3580–11070); serum amylase, 2290 U/L (28–100); serum lipase, 2777 U/L (13–60); total bilirubin, 1,9 mg/dL (0,1–1,5); serum AST 309 U/L (8–46 U/L), and serum ALT 171 U/L (7–46) while other findings were within normal limits. In radiological examination, widening in mediastinum was seen on chest X-ray (Figure 1).

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Figure 1. The chest roentgenogram of the patient shows the widening in mediastinum. In addition with the widening of descending aorta was seen.

Thoracic and abdominal computed tomography that was performed to rule out AD showed an intimal flap which was separated the aortic lumen into two parts which were extended from the proximal descending aorta to the truncus celiac and the superior mesenteric arteries (Figure 2A, 2B).

There were a increasing in the size of the pancreas, heterogeneity in the parenchyma of the pancreas, and increased density in peripancreatic tissue with fat. After clinical and radiological evaluation, type III aortic dissection and acute pancreatitis were diagnosed and medical treatment was administered in the Emergency Department. Then the patient was transferred to the cardiovascular surgery department for further medical treatment.

The laboratory abnormalities returned to normal levels 7 days after admission (Table 1). The patient was clinically stable as long as his hospitalization period. The patient was discharged from the hospital according to the consideration and suggestion of cardiovascular surgeons.

Figure 2a. This axial CT scan at the level of main pulmonary artery shows normal appearance of ascending aorta but in the other hand the widening of descending aorta with divided aortic lumen by an intimal flap.

Figure 2b. This axial CT scan at the level of the mesenteric artery shows intimal flap dividing the aortic lumen and reaching to the superior mesenteric artery. In the same time CT scan shows left and right anterior pararenal fluid density, and around the pancreatic tail due to pancreatitis.

<table>
<thead>
<tr>
<th>Variable</th>
<th>First Hospital Day</th>
<th>Seventh Hospital Day</th>
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<tbody>
<tr>
<td>Hemocrit (%)</td>
<td>40.2</td>
<td>32.7</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>13.9</td>
<td>10.8</td>
</tr>
<tr>
<td>White cells (thousand/uL)</td>
<td>11.9</td>
<td>13.3</td>
</tr>
<tr>
<td>Differential count (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neutrophils</td>
<td>84.9</td>
<td>78</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>9.7</td>
<td>11.6</td>
</tr>
<tr>
<td>Platelets (thousand/uL)</td>
<td>145</td>
<td>180</td>
</tr>
<tr>
<td>Mean corpuscular volume (fl)</td>
<td>89.1</td>
<td>90.9</td>
</tr>
<tr>
<td>Prothrombin time (10-14 sec)</td>
<td>12.5</td>
<td>11.5</td>
</tr>
<tr>
<td>Partial-thromboplastin time (25-35 sec)</td>
<td>20.4</td>
<td>20.2</td>
</tr>
<tr>
<td>Sodium (mEq/L)</td>
<td>133</td>
<td>145</td>
</tr>
<tr>
<td>Potassium (mEq/L)</td>
<td>5.4</td>
<td>4.2</td>
</tr>
<tr>
<td>Chloride (mEq/L)</td>
<td>96</td>
<td>109</td>
</tr>
<tr>
<td>Serum amylase (28–100 U/L)</td>
<td>2290</td>
<td>60</td>
</tr>
<tr>
<td>Serum lipase (13–60 U/L)</td>
<td>2777</td>
<td>62</td>
</tr>
<tr>
<td>Total bilirubin (0.1–1.5 mg/dL)</td>
<td>1.9</td>
<td>1.5</td>
</tr>
<tr>
<td>Serum AST (8–46 U/L)</td>
<td>309</td>
<td>48.1</td>
</tr>
<tr>
<td>Serum ALT (7–46 U/L)</td>
<td>171</td>
<td>35.6</td>
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DISCUSSION

AAD is an important disease, although it is rare, which may lead to several complications according to localizations involved. Complications often occur randomly, and the outcome is often fatal (5). Some complications such as myocardial infarction, stroke and hemiparesis were reported previously in the literature (6-8). To our knowledge, acute pancreatitis as a form of presentation of AAD is unusual. Pombo et al and Goff et al reported a case of pancreatitis following AAD (9, 10). Our case is the third report, in which AP is secondary to acute AAD.

Generally, acute pancreatitis (AP) may occur in the postoperative period of various surgical procedures such as abdominal aortic aneurysm repair, and cardiac surgery (11, 12). In these cases, systemic and regional hypoperfusion, atheromatous emboli to arteries supplying the pancreas, and direct trauma to the pancreas during the operation from surgical dissection have been distinguished in the causes of acute pancreatitis. Similarly, AAD induced acute pancreatitis may be associated with ischemia. We believe that in our patient, an ischemia secondary to hypoperfusion may be responsible for the AP.

Elevated serum amylase and/or lipase, particularly when their serum levels are more than three times of normal, are diagnostic for acute pancreatitis (13).

The increased serum levels of these enzymes may be related to an ischemic pancreatic disease. Thus, in the absence of any pancreatic disease, increased enzyme levels may point to aortic dissection.

As mentioned above, the probable pathological mechanism is ischemia of pancreas as a result of obstruction of arterials supplying the pancreas. When there is ischemia in the pancreas, amylase and lipase enter the bloodstream directly or via the lymphatic. In result, serum amylase and lipase levels rise and contribute to the biochemical diagnosis of acute pancreatitis (14).

In summary, acute pancreatitis following acute aortic dissections is uncommon, but its true incidence may be higher than those reported here. The diagnosis of aortic dissection should be considered in a patient presenting with unexplained gastrointestinal symptoms, especially those associated with acute pancreatitis.

REFERENCES


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