

# Multiple penetrating aortic ulcers and rupture of superior mesenteric artery branch presenting with symptoms similar to unstable angina

## A case report

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### Abstract

**Rationale:** Penetrating aortic ulcer (PAU) and rupture of a superior mesenteric artery branch is a rare but potentially life-threatening condition.

**Patient concerns:** We describe a case of 73-year-old man was brought to our Emergency Department for intermittent back pain.

**Diagnoses:** The final diagnoses are PAU (Stanford B) and rupture of a branch of the superior mesenteric artery.

**Interventions:** Two covered stents were placed in the thoracic aorta and the right external iliac artery, and the superior mesenteric artery branch was embolized. The patient subsequently underwent exploratory laparotomy, where 6000 to 7000mL of intra-abdominal hematoma was evacuated.

**Outcomes:** After the operation, the patient recovered smoothly and was discharged 20 days later. During 3-year follow-up, the patient did not develop any pain or discomfort.

**Lessons:** Acute aortic syndrome (AAS) and acute coronary syndrome (ACS) may be difficult to distinguish, particularly for elderly patients with extensive atherosclerotic disease. Antithrombotic agent administration should be carefully considered.

**Abbreviations:** AAS = acute aortic syndrome, ACS = acute coronary syndrome, AD = aortic dissection, CT = computerized tomography, CTA = computerized tomography angiography, DSA = digital subtraction angiography, ECG = electrocardiogram, IMH = intramural hematoma, PAU = penetrating aortic ulcers, TIMI = thrombolysis in myocardial infarction.

**Keywords:** acute coronary syndrome, intraperitoneal hemorrhage, penetrating ulcer of the aorta

## 1. Introduction

Penetrating aortic ulcer (PAU) is a rare disease manifested by atherosclerotic plaque with ulceration which disrupts the internal elastic lamina of the aorta. PAU may lead to chest or back pain and electrocardiogram (ECG) changes or elevated troponin

levels. So it is most likely misdiagnosed with acute coronary syndrome (ACS).<sup>[1–3]</sup> Here, we report a case of multiple penetrating aortic ulcers complicated by superior mesenteric artery branch rupture which was initially diagnosed with unstable angina.

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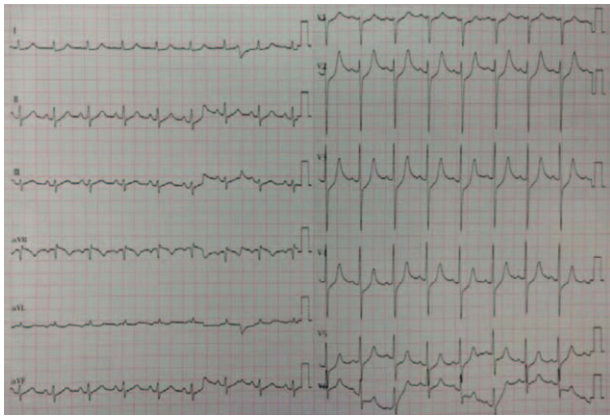
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## 2. Case report

A 73-year-old man was brought to the emergency department of our hospital for intermittent back pain. The patient was well until approximately 12 days before admission, when he felt sharp pain in the left back, which disappeared spontaneously. However, the symptom worsened 6 days later, with a Visual Analogue Scale (VAS) pain score of 8/10. He had no previous medical history and no known allergies. There was no family history of vascular disease. Clinical examination revealed normal blood pressure (110/75 mm Hg) without a significant pulse differential in the arms. ECG showed T-wave depression in V1–V6 (Fig. 1). The levels of cardiac troponin and D-dimer were 0.12 ng/mL (reference <0.04 ng/mL) and 1313 ng/mL (reference <230 ng/mL), respectively. Echocardiography indicated moderate aortic regurgitation with no wall motion abnormality. Emergency coronary angiography showed mild stenosis (30%) with normal coronary blood flow (Fig. 2). He was given dual antiplatelet therapy and subsequently discharged. However, his left back pain still continued.

On the day of admission, the patient suddenly lost consciousness upon drinking water. His son held him in a sitting position



**Figure 1.** The ECG of the patient at first admission. ECG = electrocardiogram.

and he regained consciousness 5 minutes later. Emergency medical services were called immediately. On arrival at our emergency department, approximately 20 minutes after the onset of symptoms, the patient was alert and responsive. On examination, his blood pressure was 42/32 mm Hg, pulse rate was 120 beats/min, respiratory rate was 22/min, and body temperature was 37.6°C. No bruits were heard in the carotid, subclavian, or femoral arteries. Cardiac examination revealed normal heart sounds and no murmurs. The lungs were clear. The abdomen was distended with positive shifting dullness, and diffuse periumbilical and lower abdominal tenderness without a pulsatile mass. The remainder of the examination was normal. Blood test revealed severe anaemia and disturbance in blood coagulation (Table 1). ECG showed sinus tachycardia. Dopamine and erythrocyte suspension were administered to achieve systolic pressures between 90 and 100 mm Hg. Abdominal ultrasonography revealed a large amount of ascites. Cranial computed tomography (CT) revealed no abnormalities. CT of the chest showed dilatation of the aortic arch and descending aorta. Aortic CT angiography (CTA) revealed the following: There were multiple plaques in the ascending aorta and aortic arch. The niche sign protruded in the descending aorta and the proximal end of the abdominal aorta (Fig. 3). Further diagnostic abdominocentesis showed nonclotting blood. The patient was taken urgently

**Table 1**

**Results of blood test.**

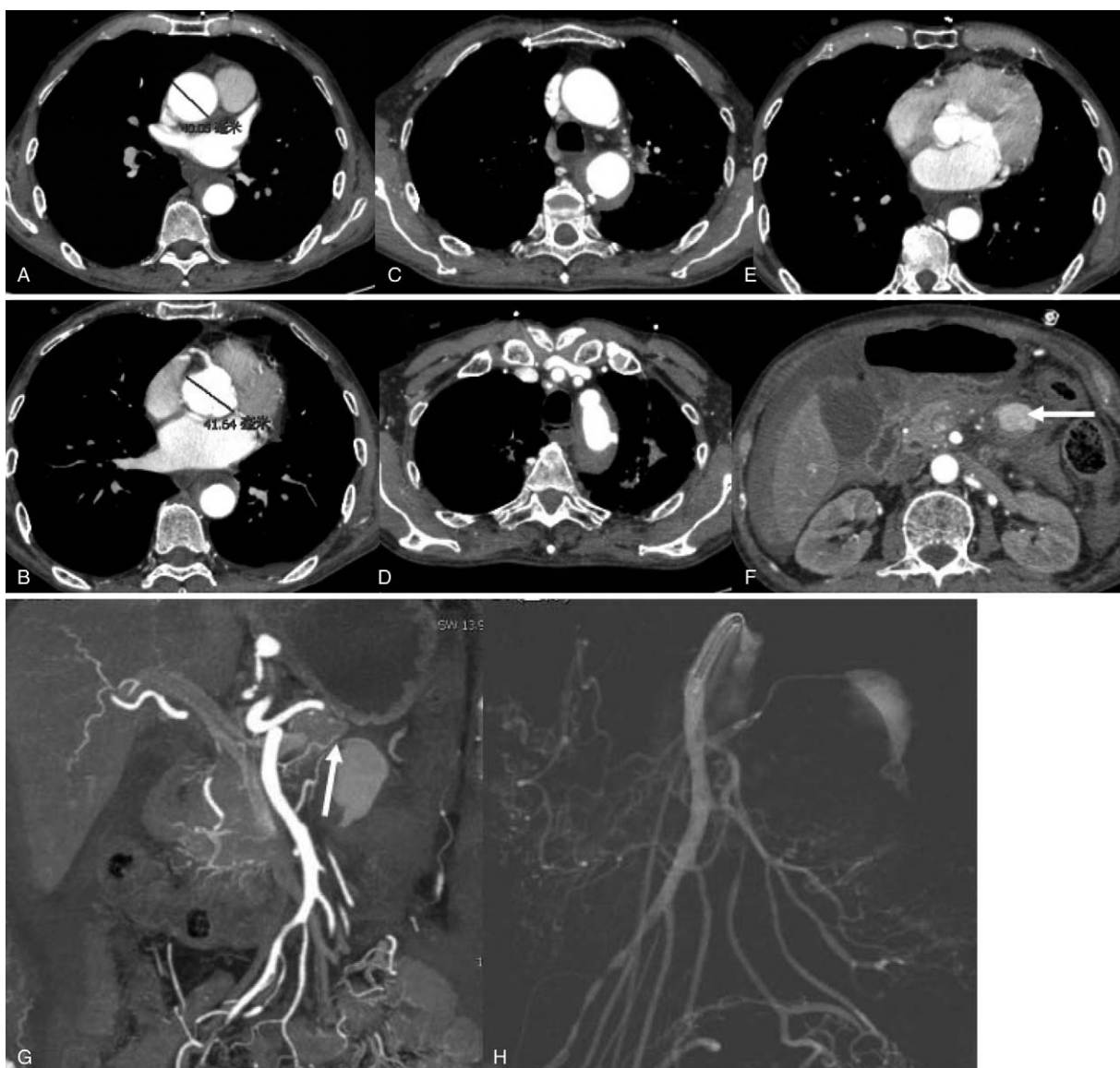
Blood test	Result	Reference range
Arterial blood gas analysis		
PH	7.43	7.35–7.45
pCO <sub>2</sub> , mm Hg	40	35–45
pO <sub>2</sub> , mm Hg	198	80–100
Standard bicarbonate, mmol/L	26.5	22–27
Lactate acid, mmol/L	1.1	0–1
Blood routine (vein)		
White blood cell (/L)	10.51	4–10
Neutrophil, %	90.2	50–70
Hemoglobin, g/L	51	120–160
Hematocrit, %	16.32	40–50
Platelet (/L)	344	100–300
Biochemical analysis (Vein)		
K <sup>+</sup> , mmol/L	4.4	3.5–5.5
Na <sup>+</sup> , mmol/L	136	135–145
Cl <sup>-</sup> , mmol/L	106	96–106
Total serum protein, g/L	50	60–80
Serum albumin, g/L	18	35–50
Coagulation analysis (Vein)		
PT	20.3	11–15
APTT, s	>180	28.0–43.5
PTA	47%	80%–120%
INR	1.72	0.85–1.5

APTT=activated partial thromboplastin time, Cl=chloride ion, INR=international normalized ratio, K<sup>+</sup>=potassium ion, Na=sodium ion, pCO<sub>2</sub>=partial pressure of carbon dioxide, PH=pondus hydrogenii, pO<sub>2</sub>=oxygen partial pressure, PT=prothrombin time, PTA=prothrombin time activity.

for an operation. In the digital subtraction angiography (DSA), penetrating ulcers were found in the descending aorta and the abdominal aorta, and extravasation was noted from a branch of the superior mesenteric artery (Fig. 3). Two covered stents were placed in the thoracic aorta and the right external iliac artery. Meanwhile, the superior mesenteric artery branch was embolized. The patient subsequently underwent exploratory laparotomy, where 6000 to 7000 mL of intra-abdominal hematoma was evacuated. The surgery was successful, and the pain disappeared. He was ultimately discharged with the following diagnosis: PAU (Stanford B) and rupture of a branch of the superior mesenteric artery. During 3-year follow-up, the patient did not develop any pain or discomfort.



**Figure 2.** Coronary angiography. Coronary angiography demonstrated no identifiable atherosclerotic plaque in the left main (LM), left anterior descending (LAD), and left circumflex (LCX) arteries (A and B); minimal plaque in the right coronary artery (RCA) (C). Flow of the 3 coronary arteries reached the Thrombolysis in Myocardial Infarction (TIMI). Diagnosis: coronary artery atherosclerosis.



**Figure 3.** Arterial aortic augmentation computed tomography angiography (CTA) and digital subtraction angiography (DSA). CT angiography of the abdominal and thoracic aorta demonstrated multiple ulcers; dilated ascending aorta (A and B); penetrating aortic ulcer (PAU) on the descending aorta (C); PAU on the aortic arch (D); peritoneal fluid (E); the arrow indicated leakage of contrast medium (F and G); DSA showed hemorrhage from the superior mesenteric artery (H).

The timeline for this case is shown in Figure 4.

### 3. Discussion

Penetrating aortic ulcers (PAU), aortic dissection (AD), and intramural hematoma (IMH) are the 3 most serious diseases involving the aorta which are described as acute aortic syndromes (AAS). AD is frequently confused with acute coronary syndrome (ACS),<sup>[1-5]</sup> but PAU misdiagnosed as ACS is relatively rare.<sup>[6]</sup> To our knowledge, only 1 case has been reported (Table 2).<sup>[2-6]</sup> In this case, the patient was admitted to the hospital because of intermittent back pain and was initially diagnosed as ACS rather than AAS. The rate of misdiagnosis doubles in the presence of ECG changes or elevated troponin levels. Unfortunately, the patient in our case inappropriately received antithrombotics. Antithrombotic agent administration has also been associated with increased

hemorrhagic pericardial fluid, hemorrhagic pleural effusion, and hemodynamic instability.<sup>[1]</sup>

Given that our patient had prominent back pain, aortic regurgitation, and elevated D-dimer levels, we should have immediately considered the probability of AAS. CTA may be lifesaving and should have been done prior to primary intervention. Our patient reported syncope subsequently and hemodynamic instability due to the intra-abdominal hemorrhage, which was later confirmed by ultrasound and CT. Existing coagulopathies or anticoagulation may also induce spontaneous intraperitoneal hemorrhage and a trend toward greater in-hospital mortality. The rupture of PAU has previously been reported in the literature. Tittle et al<sup>[7]</sup> reported 26 PAUs and found that rupture was present in 10 (38%) cases. Nathan et al<sup>[8]</sup> reviewed 388 cases of PAU from January 2003 to June 2009 and found complications of PAU, including rupture in up to 16 (4.1%) cases. We also considered the possibility of this

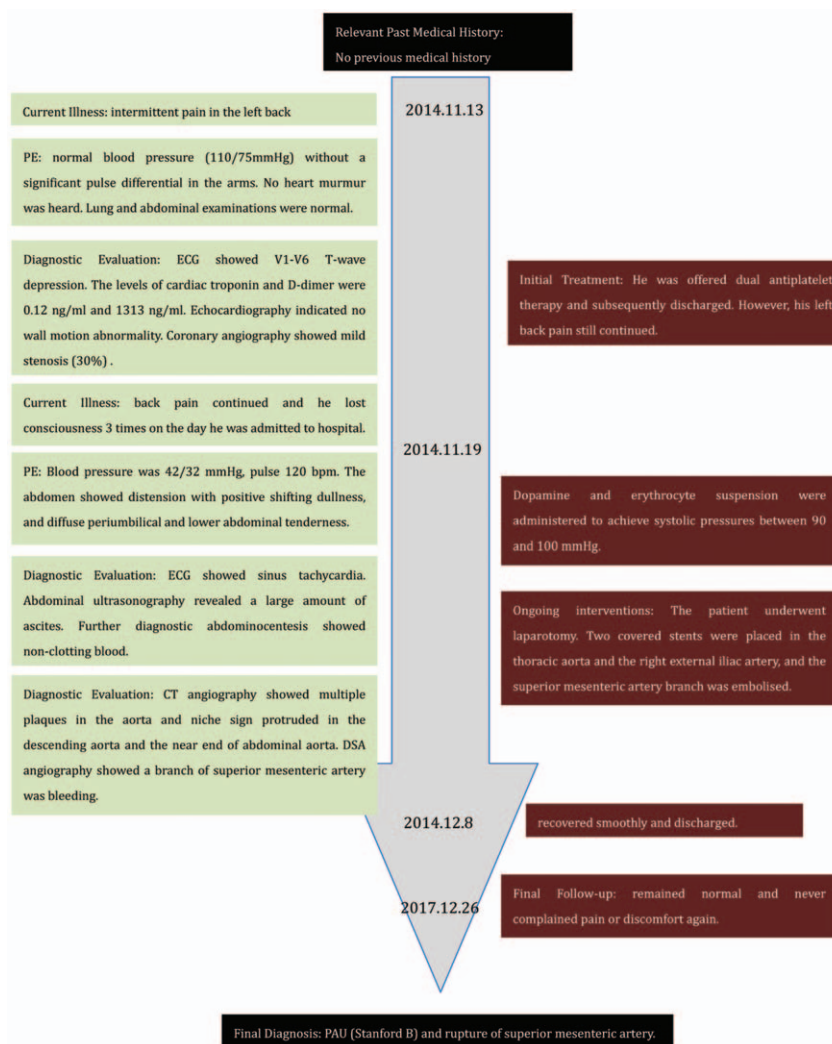


Figure 4. The timeline for this case.

complication when the patient manifested with hypovolemic shock and intraabdominal hemorrhage.

However, the cause of intraabdominal hemorrhage was unexpected after CTA was performed. In fact, a large amount of intraperitoneal hemorrhage caused by spontaneous rupture of a

branch of the superior mesenteric artery, such as in this case, is relatively rare. The causes of rupture of a superior mesenteric artery branch include aneurysm, vascular malformation, atherosclerotic spontaneous rupture, inflammation and infection, fibromuscular dysplasia, mycotic embolization, congenital anomalies, spontane-

Table 2

Summary of reported cases of acute aortic syndrome (AAS) misdiagnosed as acute coronary syndrome (ACS).

Authors	Age, y	Sex	Type of AAS	Initial symptom	Initial diagnosis
Bilge et al <sup>[6]</sup>	80	Male	PAU	Chest pain radiating to the back	Acute inferior myocardial infarction
	55	Male	AD	Not described	AMI
Choi et al <sup>[2]</sup>	33	Male	AD	Not described	AMI
	61	Female	AD	Not described	AMI
Tang et al <sup>[3]</sup>	61	Male	AD	Not described	AMI
	76	Female	AD	Not described	AMI
	71	Male	AD	Not described	AMI
Hawatmeh et al <sup>[4]</sup>	56	Male	AD	Severe retrosternal chest pain	Acute inferior myocardial infarction
	60	Male	AD	Tearing, substernal pain and was radiating to the neck and shoulders	Acute inferior myocardial infarction
Tolefac et al <sup>[5]</sup>	53	Male	AD	Severe and intractable retrosternal chest pain	AMI

AD=aortic dissection, AMI=acute myocardial infarction, PAU=penetrating aortic ulcers.

ous dissection, collagen vascular disease, and various autoimmune disorders.<sup>[9]</sup> There was no change in the size of the arterial aneurysm and vascular malformation during the DSA. According to the severe calcification observed in the patient, we believe that atherosclerotic spontaneous rupture of the superior mesenteric artery branch was the direct cause of the internal bleeding.<sup>[10]</sup> Ruptured mesenteric branch aneurysms could traditionally be treated successfully with aggressive surgical intervention, according to recent studies.<sup>[11,12]</sup> PAU might be complicated by intramural hematoma (IMH) formation, saccular pseudoaneurysm development, and rupture.<sup>[8]</sup> However, we believe this is the first case reported to have PAU complicated with rupture of a branch of the superior mesenteric artery in the literature.

In conclusion, we present a rare case of PAU (Stanford B) and rupture of the superior mesenteric artery. This rare case had a favorable outcome following open surgery. It also reminds us that AAS and ACS may be difficult to be distinguished from each other clinically, and that an urgent diagnosis and a subsequent treatment strategy are necessary. AAS should be taken into consideration in similar cases, particularly for elderly patients with extensive atherosclerotic disease, in order to prevent mortality and life-threatening complications.

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### Author contributions

SZ and RL collected the patient's clinical data and wrote the article. They contributed equally to the article. GZ designed the report.

**Conceptualization:** Suqiao Zhang, Guoqiang Zhang.

**Data curation:** Suqiao Zhang, Rui Lian.

**Formal analysis:** Suqiao Zhang.

**Funding acquisition:** Rui Lian.

**Methodology:** Rui Lian.

**Writing – original draft:** Suqiao Zhang.

**Writing – review & editing:** Suqiao Zhang.

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