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Penetrating Atherosclerotic Ulcer with Elevated Troponin in A Patient with Old Myocardial Infarction: A Case Report

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Abstract: Penetrating atherosclerotic ulcer (PAU), an uncommon etiology of acute aortic syndrome (AAS), is a potential cause of chest pain seen in emergency departments. As PAU may lead to electrocardiogram (ECG) changes or rarely, elevated troponin levels, it is most likely misdiagnosed as acute coronary syndrome (ACS). Hence, individuals with PAU may be offered potentially life-threatening treatment. This paper reports a case of a 81-year-old male who presented with intermittent chest pain with a history of old inferior myocardial infarction and stent placement in the left circumflex coronary artery (LCX) three years ago. Initially, he was diagnosed with non-ST-elevation myocardial infarction (NSTEMI) based on abnormal ECG changes and raised troponin I. However, emergency coronary angiography (CAG) showed no restenosis in the left circumflex coronary artery (LCX) but with mild stenosis in the left anterior descending artery (LAD) and right coronary artery (RCA). Computed tomographic angiography (CTA) of the whole aorta showed multiple atherosclerotic plaques with penetrating atherosclerotic ulcer in the aortic arch and descending aorta. Endovascular aortic repair with Ankura II covered stent was performed. This case study reminds us that it is clinically difficult to distinguish PAU from ACS. Upon excluding ACS from the diagnosis, we should take into consideration of PAU, especially in elderly patients with positive cTnI.

Keywords: Penetrating atherosclerotic ulcer; Non-ST-elevation myocardial infarction; Elevated troponin; Acute aortic syndrome

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1. Introduction

Penetrating atherosclerotic ulcer (PAU) of the aorta was first put forward by Shennan Schumacher [1] and defined by Stanson, et al. [2] as an atherosclerotic lesion with ulceration that penetrates the internal elastic lamina and promotes hematoma formation within the media of the aorta. With the advent of multi-slice spiral computed tomographic angiography (MSCTA), PAU has been increasingly recognized in clinical practice, especially in recent years. Due to its overlapping symptoms of chest pain or back pain with acute coronary syndrome (ACS), it is rarely diagnosed in emergency departments. Moreover, PAU may occasionally cause ECG changes or elevated troponin levels in certain patients, leading to a misdiagnosis of non-ST-elevation myocardial infarction (NSTEMI) along with delayed diagnosis and treatment. To our knowledge, there are very few studies or in-depth case reports regarding the relationship between PAU and troponin evaluation. In this paper, we report a case of PAU, occurring in a patient with old myocardial infarction (OMI), who was initially diagnosed with NSTEMI in view of ECG changes and elevated troponin.

2. Case discussion

An 81-year-old elderly male presented with intermittent chest pain for 6 months. The chest pain was at the anterior chest wall, around the cardiac area, and it worsened two days ago, with a Visual Analogue Scale (VAS) pain score of 6/10, but the pain improved with rest. He denied any shortness of breath, palpitation, or cough. He had a history of old inferior myocardial infarction and stent placement in the left circumflex coronary artery (LCX) three years ago. Other than that, he has been suffering from hypertension for the past 13 years. His medication consisted of aspirin, atorvastatin calcium tablets, clopidogrel hydrogen sulfate tablets (took it for one year after stenting), benazepril, and metoprolol. He had no smoking history, but with a history of light drinking. There was no family history of hypertension or coronary heart disease (CHD). After admission to Henan Provincial Chest Hospital, he had mildly elevated blood pressure of 146/85 mmHg with a pulse rate of 75 beats per minute. There were no abnormal findings through physical examination; his lungs were clear, his heart rhythm was regular, with normal cardiac sound, and blood pressure was the same in the left and right arm. Electrocardiogram (ECG) showed abnormal Q waves in lead II and III as well as T-wave inversion in lead I and aVL (**Figure 1**).

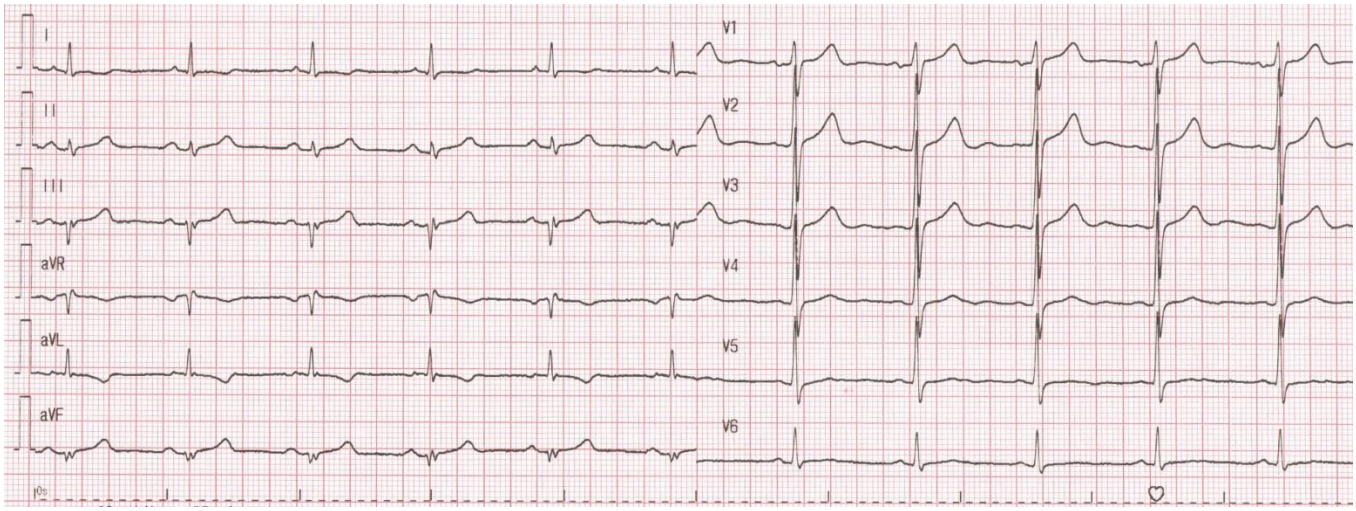

Figure 1. ECG demonstrating abnormal Q waves in lead II and III as well as T-wave inversion in lead I and aVL.

The level of high-sensitivity cardiac troponin I (hs-cTnI) was 0.172 ng/mL (reference < 0.08 ng/mL), which was slightly raised. Other laboratory investigations, such as routine blood test, liver function test, renal function and electrolyte test, coagulation function test, NT-pro BNP, as well as d-Dimer, showed normal values. Transthoracic echocardiography (TTE) showed enlarged left atrium, incrassated ventricular septum, and broadened ascending aorta, without wall motion abnormalities. The patient was initially diagnosis with NSTEMI based on abnormal ECG changes and raised troponin I. In addition, he had a history of OMI. Low molecule heparin and dual antiplatelet therapy were administered before an emergency coronary angiography (CAG). However, CAG showed no restenosis in the left main (LM) coronary artery and LCX, but mild stenosis in the left anterior descending (LAD) and right coronary artery (RCA). It also showed Thrombolysis in Myocardial Infarction (TIMI) grade 3 flow in those three coronary arteries (**Figure 2**).

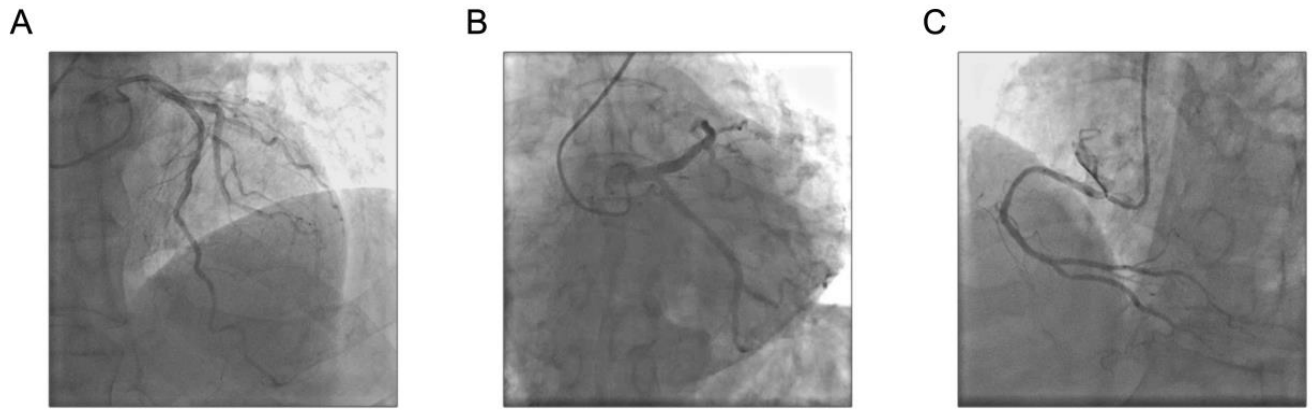

Figure 2. Coronary angiography; **A**: Mild stenosis in left anterior descending (LAD); **B**: No identifiable atherosclerotic plaque in left main (LM) and left circumflex coronary artery (LCX); **C**: Mild stenosis in right coronary artery (RCA)

We excluded other diseases that may show positive hs-cTnI, such as acute pulmonary embolism, acute aortic syndrome, Takotsuba syndrome, and myocarditis. Notably, there was a significant report of dilated ascending aorta through clinical ultrasound, which provided important value for further diagnosis. Computed tomographic angiography (CTA) of the whole aorta was done, which showed multiple atherosclerotic plaques with penetrating atherosclerotic ulcer in the aortic arch and descending aorta (**Figure 3**).

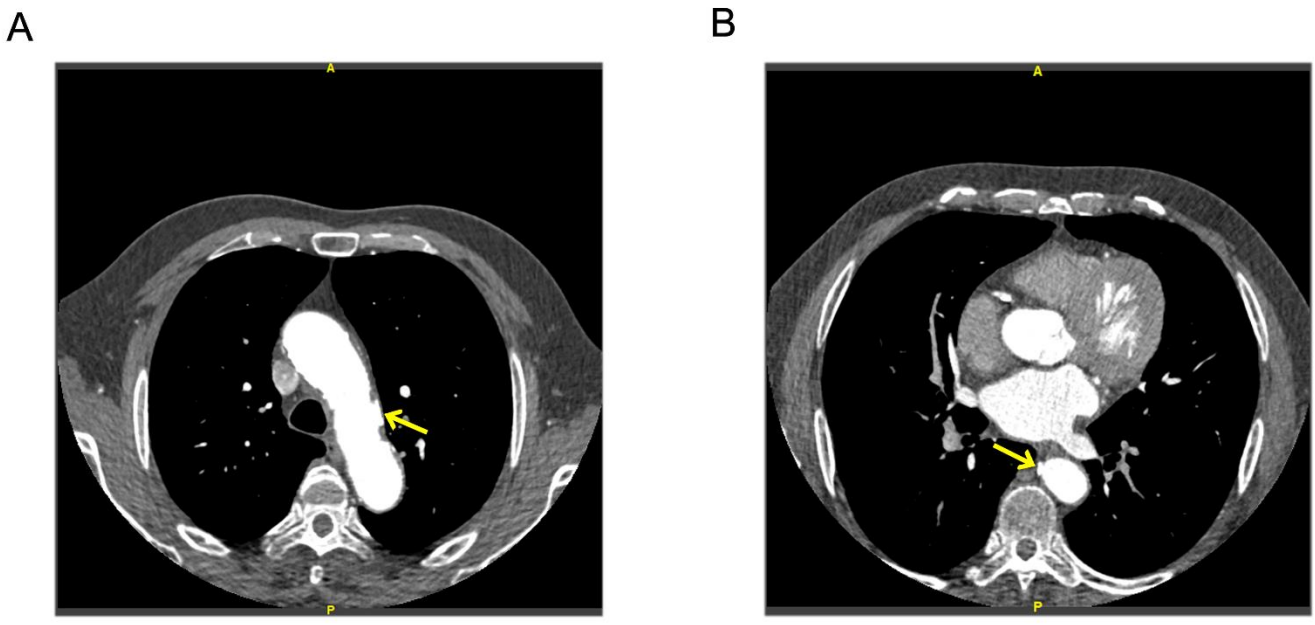

Figure 3. CT angiography of the aorta; **A**: PAU in the aortic arch; **B**: PAU in the descending aorta

With the CTA findings, PAU may be considered as the cause of chest pain and elevated hs-cTnI. In order to prevent bleeding or aortic rupture, low molecular heparin and dual antiplatelet were discontinued immediately. Then, endovascular aortic repair with Ankura II covered stent was performed. The surgery was successful, and the chest pain resolved. The patient was cured and discharged without recurrence on postoperative day 8. In this case, the final diagnosis was PAU (Stanford B).

3. Discussion

To our knowledge, cardiac troponin I (cTnI) and T (cTnT) are components of the contractile apparatus of myocardial cells and are expressed almost exclusively by the heart. They are of great value in the diagnosis of ACS. The latest guideline from the European Society of Cardiology (ESC) recommended that myocardial injury should be defined as the detection of an elevated cTn value above the 99th percentile upper reference limit (URL) [3]. Above all, an acute myocardial infarction (AMI) can be clinically diagnosed by myocardial injury and ECG changes together. In this case, the patient with OMI was initially diagnosed with NSTEMI based on ischemic ECG manifestations and elevated cTnI. He received anticoagulant and antiplatelet therapy, and subsequently underwent emergency CAG. However, the strange thing was that no abrupt coronary closure occurred; he only had mild to moderate stenosis. After ruling out other diseases associated with increased cTn value, the patient was diagnosed with PAU through CTA of the whole aorta.

PAU is an uncommon etiology of acute aortic syndrome (AAS), which includes aortic dissection (AD) and intramural hematoma (IMH). The epidemiological significance of PAU is that it accounts for 2% to 7% of AAS [4]. To our knowledge, there are very few studies or in-depth case reports on the relationship between AAS and troponin evaluation, and even less of that on PAU. To date, only one large clinical trial has investigated the mechanism of troponin release in AAS patients, including coronary dissection, interference between flap and coronary ostia, acute left ventricle pressure overload, and shock [5]. Nevertheless, up to now, no study has determined the possible mechanism of elevated troponin in PAU.

PAU is caused by an atherosclerotic plaque penetrating through the internal elastic lamina into the aortic media. It can further evolve into a saccular aneurysm or an IMH or rupture by penetrating through all three layers of the wall. A study showed that the risk of aortic rupture is higher in patients with PAU (40%) compared to those with type-A (7%) and type-B dissections (4.0%) [6]. Due to these potential risks, it is crucial to have an accurate diagnosis and treatment for PAU. In this case, the patient was initially diagnosed with NSTEMI in view of the ECG changes and elevated troponin. The patient was treated with antithrombotics and anticoagulants, which was rather dangerous. Fortunately, these drugs were withdrawn immediately after an emergency coronary angiography that showed no occlusion in the original stent at the LCX and other coronary arteries. In order to avoid potential risks, a rapid and accurate diagnosis of PAU is extremely necessary.

Some studies have shown that the common features of patients affected by PAU include aging, male, tobacco smoking, hypertension, coronary artery disease, chronic obstructive pulmonary disease, and concurrent abdominal aneurysm [7-9]. However, imaging is required for a definitive diagnosis of PAU. There are three commonly used diagnostic imaging techniques for PAU in modern medicine, including CT angiography, magnetic resonance imaging (MRI), and TTE. However, in the 2014 ESC guidelines on the diagnosis and treatment of aortic diseases, CTA has been recommended as the best choice for diagnosing PAU considering its excellent diagnostic accuracy, widespread availability, easier patient access and tolerance, short duration for image acquisition and processing, and more importantly, enabling rapid collection of key information for decision-making in clinical settings [10]. Meanwhile, it is not only used for determining the size, range, location, and classification of PAU, but also to devise clinical treatment strategies.

4. Conclusion

In this paper, a rare case of PAU with raised cTn, positive ECG changes, and mild to moderate stenosis in three coronary arteries through coronary angiography was presented. This case reminds us that it is clinically difficult to distinguish PAU from ACS. Therefore, in order to prevent mortality and life-threatening complications, we should take into consideration of PAU after excluding ACS, especially in elderly patients with positive cTnI.

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Disclosure statement

The authors declare no conflict of interest.

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