

CASE REPORT

Pericardial tamponade caused by a perforating atherosclerotic ulcer of the ascending aorta

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Abstract - We present the case of a patient with pericardial tamponade caused by a rare complication of atherosclerosis. An eighty-year-old male suffered a witnessed cardiac arrest due to pulseless electrical activity (PEA). Transthoracic echocardiography showed a pericardial effusion of 1.5 to 2 cm. Emergency subcostal pericardiocentesis drained approximately 15 ml of sanguinolent fluid. Subsequent CT scan of the thorax showed a penetrating atherosclerotic ulcer (PAU) of the proximal ascending aorta with signs of active bleeding into the pericardium and right pleural cavity. The patient was diagnosed with a pericardial tamponade and haemothorax due to a perforated PAU of the ascending aorta, resulting in PEA. Although a PAU of the descending aorta and distal aortic arch can be treated by endovascular repair (EVAR), open thoracic surgery remains the treatment of choice for lesions located in the ascending aorta or proximal mid-aortic arch.

Keywords - Cardiac tamponade, penetrating atherosclerotic ulcer, CT scan, endovascular repair, surgical repair, atherosclerotic disease.

Introduction

Atherosclerotic disease affects roughly 30–40% of the population and accounts for 41% of all deaths. Apart from the acute coronary syndromes, classic aortic dissections and symptomatic aortic aneurysms, few other atherosclerotic complications are considered as causes in acute life-threatening situations. However, in an increasingly elderly population combined with the age-related increase in atherosclerotic disease, even the more rare complications of atherosclerosis are likely to be seen more frequently. We present the case of a patient who died from a rare complication of atherosclerotic disease, a perforating atherosclerotic ulcer of the aorta.

Case

We present the case of an eighty-year-old male, who suffered a witnessed cardiac arrest during transfer by ambulance from the emergency room of another hospital to our ICU. The patient presented with a history of three consecutive days of progressive dyspnoea, non-productive cough and a mild fever, without chest pain of any kind and no signs of myocardial ischaemia on his ECG. Chest X-ray showed a small consolidation in the right lower lobe. His medical history revealed a history of COPD, hypertension, atrial fibrillation, peripheral atherosclerotic stenotic vascular disease (Fontaine class II) and occlusive coronary artery disease with a recent hospital admission for unstable angina pectoris. Medication use included sotalol, phenprocoumon, several anti-hypertensive medications, a statin and ipratropium bromide inhalations.

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Based on the clinical presentation, the initial diagnosis was sepsis due to pneumonia with respiratory failure. The patient received broad-spectrum antibiotics (cefuroxime and gentamicin), was intubated, mechanically ventilated and a central venous line for fluid and vasopressor therapy was inserted. Due to a shortage of ICU beds at the referring centre, the patient was transferred to our hospital.

During transport the patient developed acute hypotension and bradycardia, followed by pulseless electrical activity (PEA). Resuscitation was initiated immediately. On arriving at our hospital, the patient was receiving CPR, had no palpable peripheral pulse, no measurable blood pressure, no recorded oxygen saturation or capnography, but normal bilateral breath sounds on manual ventilation. ECG showed a narrow complex bradycardia and CPR was continued according to PEA protocol. The cardiologist performed a transthoracic echocardiography (TTE) which revealed no signs of right ventricular enlargement or elevated right side pressures, but did show a pericardial effusion of 1.5 to 2 cm. Emergency subcostal pericardiocentesis drained approximately 15 ml of sanguinolent liquid. After an additional CPR cycle ROSC occurred with a very low output state and a SaO₂ of 90%. Arterial blood pressure measurements showed systolic blood pressures of 60–70 mm Hg and inotropic (dobutamine) and vasopressor medication (norepinephrine) were started. Total time to ROSC was approximately 45 minutes.

Post-resuscitation the haemoglobin content had dropped from 7.0 mmol/L to 3.6 mmol/L. During an initial screening for other causes of blood loss and shock, abdominal ultrasound (FAST screening) showed a previously unknown abdominal aortic aneurysm of approximately 5 cm, without signs of leakage. The chest X-ray (Figure 1 B) showed an enlarged superior mediastinum with left apical cap, suggestive of mediastinal haemorrhage, and a diffuse whitening of the right hemithorax due to pleural

effusion. There were no signs of pneumothorax.

A CT scan was made to determine the cause of the pericardial tamponade and the mediastinal enlargement. This showed a blush of contrast emanating from the ascending aorta, originating from a perforated penetrating atherosclerotic ulcer (PAU) immediately above the right coronary cusp (Figures 1 D and F). There was a large pericardial and pleural effusion, most probably blood. Furthermore, the CT showed an abdominal aneurysm (without signs of rupture), a sternal fracture, and several rib fractures caused by mechanical chest compressions.

After consulting the cardiothoracic surgeon and taking into account the long period of insufficient circulation, the persisting haemodynamic instability, the pre-existent co-morbid conditions and the age of the patient, we refrained from an emergency open thoracic surgical procedure. Endovascular treatment was not considered an option since the perforated PAU was located in the ascending aorta. In accordance with the wishes of the immediate family, treatment was suspended and the patient died from cardiac tamponade and haemorrhage.

Figure 1. A 6-month old chest X-ray on file, B showing the chest X-ray post-resuscitation. C and E are CT cross-sections at different levels before intravenous contrast. D and F showing corresponding cross sections to C and E, after intravenous contrast. Arrows: 1 - mediastinal enlargement; 2 - pleural effusion; 3 - i.v. contrast "blush" emanating from the aorta; 4 - pericardial effusion; 5 - extravasation of contrast medium.



Discussion

A PAU of the aorta is one of the acute aortic syndromes (AAS) and was first described in 1934 by Shennan. In 1986 it was defined by Stanson as an ulceration of atheromatous plaques through the internal elastic lamina into the media or deeply into the adventitia. Other aortic syndromes, such as the intramural haematoma (IMH) and the acute aortic dissection have been studied in far more detail. Over the last 20 years, coinciding with the development of multidetector CT scans (MD-CT), its radiological diagnosis has been made with increasing certainty. On CT scans a PAU can be seen with a local IMH, or as a perforation of the whole aortic wall with or without contained haemorrhage.

In patients presenting with signs suggesting aortic dissection, PAUs are found in 2.3% to 11% of cases [1-4]. However, the true incidence is unknown since probably many patients remain asymptomatic. Most PAUs are located in the descending thoracic aorta [5], of which 26% are located in the proximal third, 41% in the mid-third, and 15% in the distal descending aorta. PAU in the mid-arch and the distal arch account for 7% and 11%, respectively. The incidence of PAU of the ascending aorta appears to be much lower. Two retrospective studies found that the incidence varied between 4.6% and 13% of patients with a PAU [6,7].

The presenting symptom of a PAU is often sudden onset of severe back and/or chest pain, thereby having much overlap with other AAS and acute coronary syndromes (ACS). A more chronic course of PAU may present with chronic back pain in the absence of musculoskeletal disorders. Chronic pain is thought to be due to continued erosion of the ulcer (into the adventitia), progression to aortic dissection, or aneurysm formation. Two-thirds of AAS patients are male and many of the co-morbid conditions that are often seen in PAU patients, such as advanced age [1], smoking, hypertension, COPD, coronary artery disease and peripheral arterial obstructive disease, are also related to atherosclerotic disease.

A PAU is an intimal disease which is thought to originate in the thickened intima with chronic atherosclerotic changes, then penetrate the internal elastic lamina into the aortic media with subsequent development of a saccular- or pseudo aneurysm with the risk of rupture [8]. In contrast, the classic intramural haematomas and aortic dissections are caused by media degeneration and haemorrhage from the vaso vasorum in the diseased medial layer itself. Figure 2 illustrates the possible progression of a PAU, to an intramural haematoma and aortic dissection or a transmural perforation.

The diagnosis is established either by CT scan, magnetic resonance imaging (MRI), or transoesophageal echocardiography (TEE). Conventional radiology is of limited or no use in the diagnosis of non-ruptured or non-perforated PAU. The overall sensitivity of conventional chest X-rays for all AAS is poor (64%), and even less (47%) if the AAS is confined to the ascending aorta [9]. Conventional angiography has commonly been replaced by CT scan, MRI and TEE.

Well-established therapeutic options exist for classic aortic dissection and IMH. However, the optimal treatment of PAU re-

mains unclear. Recommendations from the European Society of Cardiology [10] and the review by Cho [11] suggest that asymptomatic patients with distal disease should receive medical treatment including adequate blood pressure control, statin therapy, modification of cardiovascular risk factors and be closely monitored with serial imaging. Patients with penetration of the aortic wall, thoracic aorta deterioration on serial imaging, or persisting pain should be scheduled for repair. Some authors consider medical management for a PAU located in the ascending aorta ineffective and it was suggested that PAUs of the ascending aorta carry a higher risk of rupture and therefore early surgical intervention is indicated.

The traditional open surgical treatment is replacement of the diseased aorta, which depending upon location of the PAU (ascending aorta, arch or descending aorta), is associated with a mortality of approximately 5% to 20% [8,11-16], and considerable morbidity. Considering the common multiple medical comorbid conditions, endovascular stent-graft repair may be a more suitable choice. Reviews of stent grafting in patients with acute presentation of PAU of the descending aorta demonstrated a success rate of 92% to 100% [8,12]. This could be achieved with an intervention related mortality of 12% [12,17].

However, the use of endovascular repair in a PAU of the ascending aorta has not been studied extensively. In none of the reported cases endovascular repair was applied.

Stenting close to the aortic valve, coronary ostia and carotid arteries could result in valvular damage, myocardial and cerebral

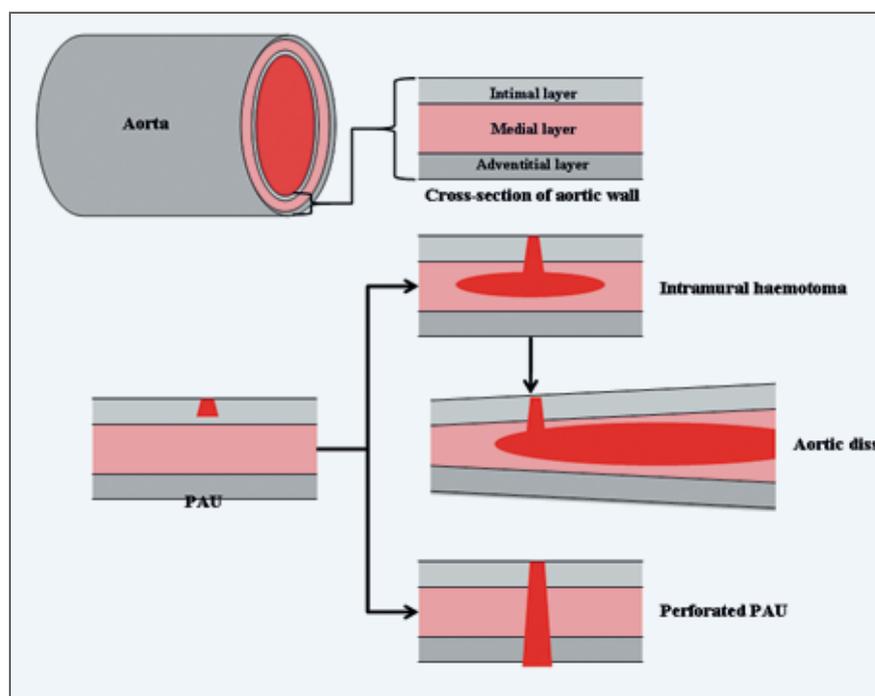
infarction and occlusion of the major branching arteries. While endovascular treatment of a PAU of the descending aorta and distal aortic arch (endovascular aortic repair or EVAR) has proven to be an excellent therapeutic option, it is unsuitable for lesions located in the ascending aorta or proximal-mid aortic arch.

Conclusion

As illustrated by this case, PAU is an AAS and can lead to the development of an IMH, aortic dissection and/or perforation, requiring emergency treatment. PAU is an intimal disease and occurs mostly in patients of advanced age and with advanced atherosclerotic disease. PAU is probably under-diagnosed as it is often an accidental finding in asymptomatic patients. The increasing use of multi-detector CT scans and the increasing number of elderly patients suffering from atherosclerotic disease, means that PAU will probably be diagnosed more often. Hence, this will result in an increasing group of patients in need of intervention or careful follow-up.

Medical treatment is suitable for stable patients with lesions distal to the aortic arch. Conventional open thoracic surgical treatment carries a high risk of mortality and morbidity. Endovascular treatment of a PAU of the descending aorta and distal aortic arch (EVAR) has proven to be an excellent therapeutic option. However, to date conventional open thoracic surgery remains the treatment of choice for lesions located in the ascending aorta or proximal-mid aortic arch.

Figure 2. Suggested progression of a PAU, to an intramural haematoma and aortic dissection or a transmural perforation.



Abbreviations

COPD	Chronic obstructive pulmonary disease
SaO ₂	Arterial oxygen saturation
ROSC	Return of spontaneous circulation
PAU	Penetrating atheromatous ulcer
PEA	Pulseless electrical activity
AAS	Acute aortic syndromes
IMH	Intramural haematoma
MD-CT	Multi-detector CT scan
PIT	Primary intimal tear
MRI	Magnetic resonance imaging
TEE	Transoesophageal echocardiography

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