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Case report

Mechanical intravascular hemolysis after percutaneous closure of perioaortal pseudoaneurysm

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Introduction

Intravascular mechanical haemolysis may complicate the implantation of foreign materials into the bloodstream. This complication has been described most frequently with valve replacements, cardiac patches, or mechanical circulatory support devices. It appears to be exceptional in association with the implantation of occluders. In this work, we present a case of mechanical haemolysis induced by an Amplatzer occluder following the occlusion of a peri-aortic pseudoaneurysm.

Case report

The patient was a 45-year old male with a positive family history (his father died at the age of 36 due to myocardial infarction and his mother died at the age of 67 from a ruptured

aortic aneurysm), a former smoker treated for arterial hypertension and dyslipidaemia.

In 2014, he underwent selective coronarography at our institution for angina pectoris and was found to have severe involvement of three coronary arteries – a tight stenosis of the mid-section of the anterior interventricular branch (RIA) and chronic collateralised occlusion of the circumflex branch and the right coronary artery. The echocardiogram demonstrated good systolic function of both ventricles with no signs of serious valve disease. Significant dilation of the ascending aorta (53 mm, index 22 mm/m²) was the only incidental finding. Based on the examination results, the patient was indicated to undergo surgical re-vascularisation and ascending aorta replacement. In the same year, he underwent a quadruple coronary artery bypass using both mammary arteries (the left mammary artery onto the anterior interventricular branch and the right connected to the left coronary artery and distally to the ramus diagonalis and ramus intermedius). The right coronary artery was re-vascularised using a vein graft, which was connected to the ascending aorta replacement (Gelweave 26 mm prosthesis). The surgical procedure itself and the following postoperative course were uncomplicated.

One year after surgery, the patient began complaining of dyspnoea on exertion and developed a new heart murmur. A trans-oesophageal echocardiogram and a CT of the heart revealed the presence of a peri-aortic, or rather, a peri-prosthetic pseudoaneurysm (66 mm × 60 mm) communicating across a defect within the prosthesis (8 mm × 7 mm) at the site of the venous bypass insertion into the right atrium (Figs. 1 and 2). A Doppler examination showed the presence of a left to right shunt, at least of intermediate significance. This

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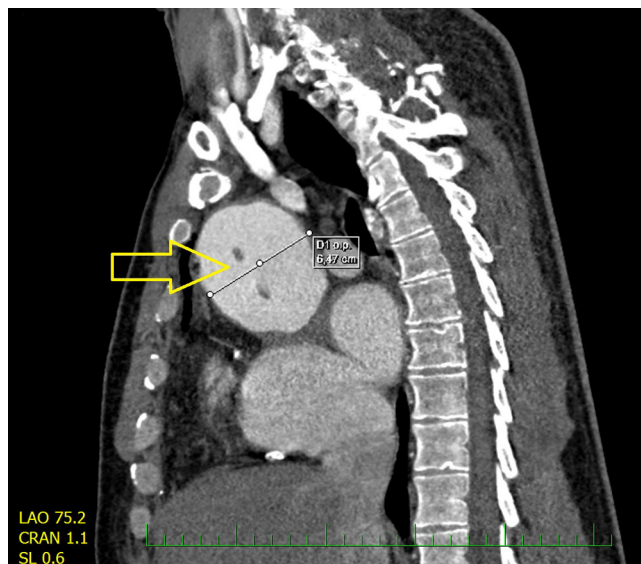


Fig. 1 – CT image of the peri-aortic pseudoaneurysm (yellow arrow).

corresponded to the right ventricle dilation (basal dimension 45 mm) and borderline systolic function with signs of pulmonary hypertension at rest (estimated systolic pressure in the pulmonary artery was 55 mm Hg).

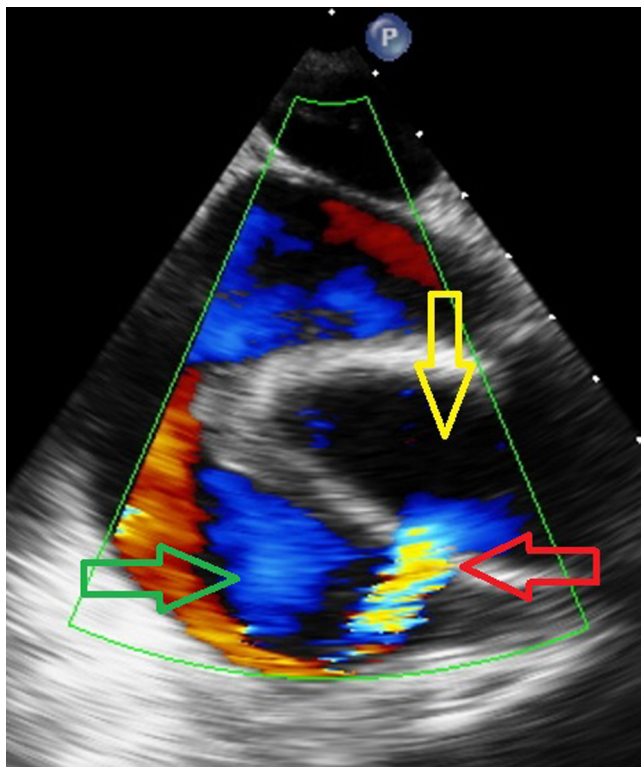


Fig. 2 – Echocardiography image of the blood flow (black arrow) between the ascending aorta prosthesis (yellow arrow) and the pseudoaneurysm (green arrow). The prosthesis defect is located at the site of the original venous bypass insertion.

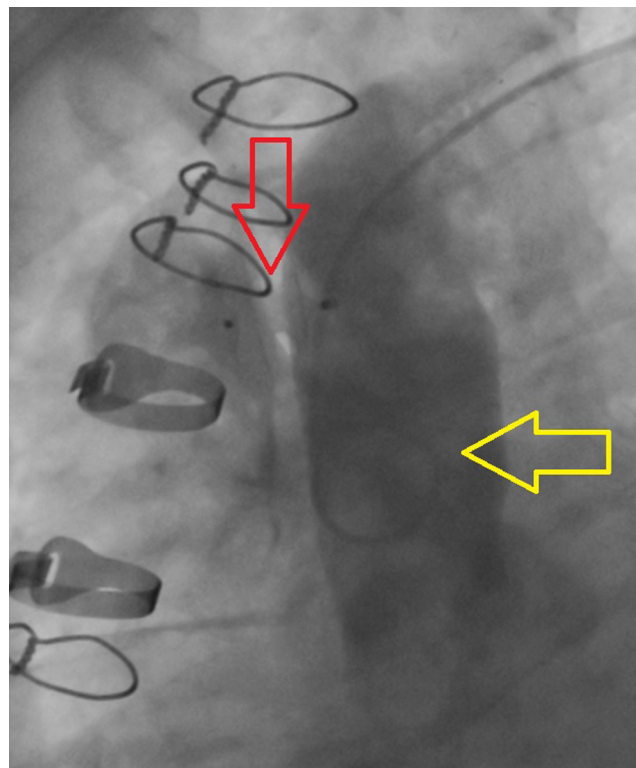


Fig. 3 – Angiography image of the ascending aorta prosthesis (yellow arrow) and Amplatzer occluder (red arrow) implanted into the neck of the pseudoaneurysm at the site of the prosthesis defect.

With the aim of closing the pseudoaneurysm via catheterisation, the patient underwent an invasive examination which verified the patency of the arterial bypasses and the occlusion of the venous bypass. A communication with the peri-aortic space was visible on the ascending aorta prosthesis, 5–6 cm from the aortic valve. This pseudoaneurysm was then probed using a 5 F RCB (Right coronary bypass) catheter and restrictive filling was demonstrated (systolic, diastolic, and mean pressure within the pseudoaneurysm cavity was 33/19/21 mm Hg with a corresponding pressure of 164/77/111 mm Hg in the aorta). An Amplatzer septal occluder 10 mm in diameter was then inserted into the neck of the pseudoaneurysm, thus closing it (Fig. 3).

The following day, the patient developed haematuria and jaundice. The laboratory results demonstrated an elevated bilirubin (total 73 $\mu\text{mol/l}$, unconjugated 46 $\mu\text{mol/l}$), AST (2.23 $\mu\text{kat/l}$) and lactate dehydrogenase (33.8 $\mu\text{kat/l}$). The blood count showed a fall in haemoglobin (from 135 g/l to 110 g/l) as well as a decrease in platelet count (from 140 to 95 $\times 10^9/l$). By contrast, there was a rise in plasma free haemoglobin (1178 mg/l). Echocardiography demonstrated turbulent flow across the occluder into the pseudoaneurysm (Fig. 4). These findings confirmed the diagnosis of acute mechanical haemolysis on the Amplatzer occluder.

Once hydration was started (3000 ml normal saline/day) and clopidogrel was discontinued to accelerate thrombotization of the pseudoaneurysm, the patient's clinical condition improved gradually and the jaundice and haematuria

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