Massive haemoptysis: an unusual connection

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DESCRIPTION

A 60-year-old man presented with a 4-day history of small volume haemoptysis. His medical history revealed a pericardiectomy with pericardial window creation for severe constrictive pericarditis secondary to seropositive rheumatoid arthritis in 1997 and pulmonary tuberculosis aged 7, with a treated recurrence in 2005. He was a lifelong smoker of 20 cigarettes per day. He lived independently. His regular medication included prednisolone 10 mg and meloxicam.

On examination, he looked unwell. His heart rate was 140, respiratory rate 22 and oxygen saturations 96% on air. Auscultation revealed crepitations at the left lung base. He had 500 mL of brisk haemoptysis. His haemoglobin fell from 85 to 73 g/L. Inflammatory markers were raised (C reactive protein 169 mg/L, white cell count 14.7×10⁹/L, neutrophil count 12.9×10⁹/L), prothrombin time ratio was mildly raised (1.4), platelet count was normal (260×10⁹/L). He received intravenous antibiotics, tranexamic acid and two units of red blood cells. The haemoptysis slowed and stopped within 48 hours.

A chest X-ray showed extensive pericardial calcification and left lung consolidation. A CT pulmonary angiogram showed focal mid-anterolateral myocardial rupture with contrast entering a lateral mid-wall false aneurysm from the left ventricle (figure 1A,B). There was consolidation adjacent to the aneurysm suggesting haemorrhage into the lung (figure 1A,B). Cardiac MRI (C-MRI) confirmed a 2 cm lateral wall false aneurysm without evidence of myocardial infarction (figure 2).

The patient was referred for consideration of cardiac surgery, but the risk was thought to be high. Percutaneous closure was contemplated but it was thought that the orifice of the aneurysm was very near the papillary muscle and so a device might become entangled in the chordae. The patient remained well with no further haemoptysis and was discharged with conservative management.

A CT thorax 6 weeks later showed the left ventricular contrast extravasation was now absent and the left ventricular pseudoaneurysm had resolved (figure 1C,D). A fistulous connection between the airway and pericardium, previously obscured by haemorrhagic consolidation, was now evident. Bronchoscopy excluded any abnormality. Cultures for tuberculosis were negative.

DISCUSSION

Pseudoaneurysms of the left ventricle are usually a late complication of myocardial infarction. Rarely
they can be caused by cardiac surgery, trauma and pericardial infections including tuberculosis.\(^1\)\(^2\) Rupture can be precipitated by infection weakening the pseudoaneurysm sac.\(^1\)\(^2\)\(^3\)\(^4\) In such cases, blood is normally contained within the pericardium resulting in tamponade. However, if the pericardium is breached or is partially absent, as in our patient, haemoptysis can occur with formation of a ventricular-bronchial fistula. It is likely that most patients who develop such a fistula die rapidly. There are a few case reports of successful surgical management.\(^5\)\(^6\)\(^7\)\(^8\)

In our patient, the pseudoaneurysm was likely a long-term complication of his pericardiectomy. Bronchoscopy found no evidence of \textit{Mycobacterium tuberculosis}. C-MRI and angiogram revealed no old myocardial infarction. When the pseudoaneurysm ruptured, the absence of normal pericardium allowed blood to enter the left lung creating a pericardial-bronchial fistula. The patient’s survival must have been due to fortuitous haemostasis and plugging off the orifice of the pseudoaneurysm.

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\textbf{REFERENCES}