

Haemorrhagic shock and spontaneous haemothorax

J. Heidt^{1,*}, X.Y.D Beele², K.P. van Lienden³, R. van Raalte¹

¹Department of Intensive Care, Tergooi Hospital, Hilversum, the Netherlands, ²Department of Radiology, Tergooi Hospital, Hilversum, the Netherlands, ³Department of Interventional Radiology, Academic Medical Centre, Amsterdam, the Netherlands, *corresponding author: tel.: +31(0)88-7531753, fax: +31(0)88-7531289, e-mail: jheidt@tergooi.nl

CASE REPORT

A 75-year-old female presented to the emergency department with dyspnoea, pain between the scapulae and hypotension. She had no remarkable medical history and only used a non-steroidal anti-inflammatory drug for lower back pain. There was no recent history of cough, fever or haemoptysis.

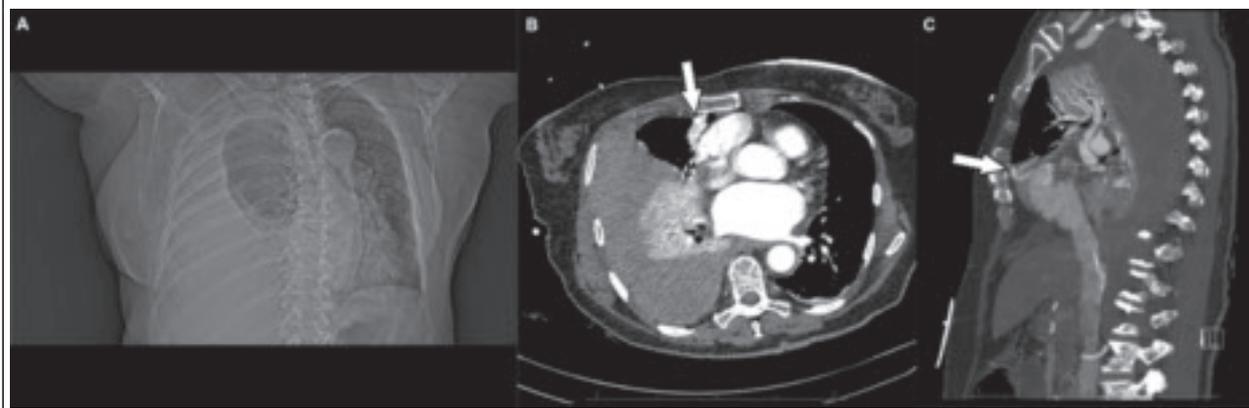
Under suspicion of an aortic dissection she was transferred to the radiology department for a CT scan where she lost consciousness and developed pulseless electrical activity. Cardiopulmonary resuscitation (CPR) was initiated and due to right-sided diminished lung sounds and the suggestion of a tension haemo/pneumothorax on the preparation CT scan a needle thoracocentesis was performed, which drained blood. After ten minutes of CPR she regained spontaneous circulation. She was mechanically ventilated and transferred

to the intensive care unit for further resuscitation. Laboratory results showed a decreased haemoglobin level of 3.7 mmol/l, a haemorrhagic coagulopathy (platelets $105 \times 10^9/l$, prothrombin time 24.8 seconds and an activated partial thromboplastin time of 82 seconds) and severe combined acidosis (pH 6.76, pO₂ 16.7 kPa, pCO₂ 8.2 kPa on 1.0 fraction of inspired oxygen, lactate 16.2 mmol/l). Transfusion and correction of the coagulopathy was carried out. A chest drain was placed, which produced blood. After stabilisation a second CT scan was performed (*figure 1*).

WHAT IS YOUR DIAGNOSIS?

See page 337 for the answer to this photo quiz.

Figure 1. A) Preparation CT scan, prior to CPR and chest drain placement, showing extensive pleural fluid with compression of the right lung and mediastinal shift; B) Transverse view of a contrasted CT scan showing extensive haemothorax (pleural fluid with Hounsfield Units +55, compatible with blood) with compression of the right lung and mediastinal shift. In the collapsed middle lobe a hyperdense structure is visible (subpleural, parasternal right, arrow); C) Saggital reconstruction of a contrasted CT scan showing an extensive haemothorax with compression of the right lung. A hyperdense structure is visible (arrow)



DIAGNOSIS

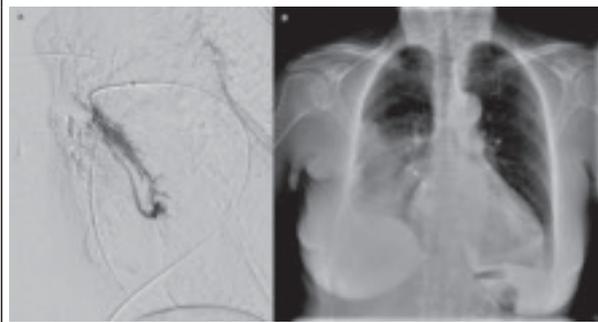
Causes of nontraumatic haemothorax include pneumothorax, coagulopathy, neoplasms, pleural endometriosis and vascular anomalies.¹ In our patient the CT scan revealed a haemothorax and a hyperdense structure matching a pulmonary arteriovenous malformation (PAVM).

PAVMs are abnormal connections between the pulmonary artery and vein, resulting in a low resistance right-to-left shunt. Most PAVMs are hereditary and occur in hereditary haemorrhagic telangiectasia, an uncommon autosomal dominant disorder associated with progressive development of vascular malformations. PAVMs may also be idiopathic, occur as a result of trauma and infection (e.g. schistosomiasis), or be secondary to hepatopulmonary syndrome in cirrhosis and bidirectional cavopulmonary shunting.^{2,3}

Spontaneous haemothorax as a complication of a PAVM is rare and massive haemothorax with mediastinal shift has rarely been reported.^{1,4} Incidence of haemothorax and haemoptysis in PAVMs varies from 1-8%.⁵ Pregnant women are predisposed to rupture of a PAVM, especially in the last trimester. This is thought to be due to increased cardiac output and hormonal effects on the blood vessels, causing increase in size of the PAVM.⁶

The diagnostic tool of choice to determine the underlying cause of the haemothorax is multidetector CT angiography. Treatment of choice for PAVMs is transcatheter embolotherapy ('coiling'). If coiling is unsuccessful an exploratory thoracotomy should be performed.³ One could argue immediate thoracocentesis with chest drains, as the haemothorax may tamponade the bleeding. Some authors suggest definitive thoracocentesis should be delayed until embolisation has been performed.¹ Our patient underwent a transcatheter embolisation and a video-assisted thoracoscopy for removal of the haematoma the day after (*figure 2*). No underlying cause for the PAVM could be found and she was diagnosed having an idiopathic PAVM.

Figure 2. A) Image during angiography, showing the catheter positioned in the arterial feeder of the PAVM and outflow through the pulmonary vein. B) Chest X-ray, performed after the transcatheter embolisation and thoracoscopic removal of the haematoma



Although rarely seen, a PAVM as underlying cause of a spontaneous haemothorax should always be considered. CT angiography is the investigation of choice to detect a PAVM.

REFERENCES

1. Berg AM, Amirbekian S, Mojibian H, et al. Hemothorax due rupture of pulmonary arteriovenous malformation. *Chest*. 2010;137:705-7.
2. Khursid I, Downie GH. Pulmonary arteriovenous malformation. *Postgrad Med J*. 2002;78:191-7.
3. Cartin-Ceba R, Swanson KL, Krowka MJ. Pulmonary arteriovenous malformations. *Chest*. 2013;144:1033-44.
4. Khan AA, Hunt I, Hamdane K, et al. Massive pulmonary arteriovenous malformation presenting with tamponading haemothorax. *BMJ Case Rep*. 2009;2009:bcr2006071852. Epub 2009 Feb 18.
5. Ference BA, Shannon TM, White RI, et al. Life-threatening pulmonary hemorrhage with pulmonary arteriovenous malformations and hereditary hemorrhagic telangiectasia. *Chest*. 1994;106:1387-90.
6. Gershon AS, Faughnan ME, Chon KS, et al. Transcatheter embolotherapy of maternal pulmonary arteriovenous malformations during pregnancy. *Chest*. 2001;119:470-7.