Pericardiophrenic artery embolisation for control of massive haemoptysis

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ABSTRACT

Non-bronchial systemic arteries, apart from normal and anomalous bronchial arteries, may be a source of massive haemoptysis in a chronically inflamed lung via transpleural anastomoses. Transcatheter embolisation is an established therapeutic method of choice in the management of massive haemoptysis. We report embolisation of a hypertrophied pleural branch of the pericardiophrenic artery for the management of massive haemoptysis in a 61-year-old woman. Initial computed tomography chest imaging showed peribronchial thickening and subpleural scarring in the lingula lobe, with ground-glass changes secondary to haemoptysis. Angiography demonstrated a hypertrophied branch of the left pericardiophrenic artery supplying an abnormal bunch of vessels in the lingula and anastomosing with the homolateral inferior phrenic artery. This was successfully embolised with gel foam. The left internal thoracic artery was later embolised in order to control the repeat haemoptysis. A brief anatomical review of the source of massive haemoptysis, anatomy of the internal thoracic and pericardiophrenic arteries and the clinical implications are discussed.

Keywords: bronchial embolisation, haemoptysis, inferior phrenic artery, internal thoracic artery, pericardiophrenic artery
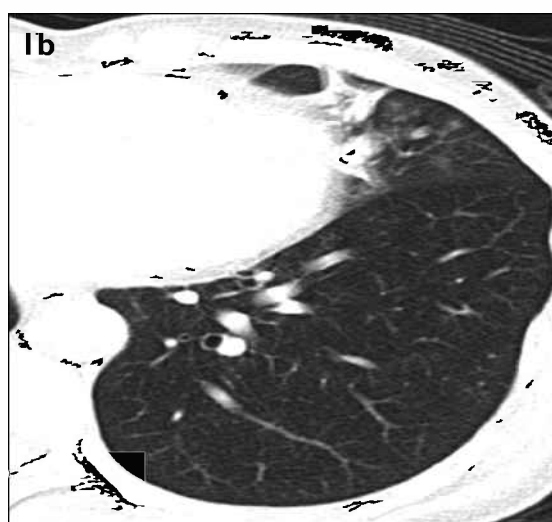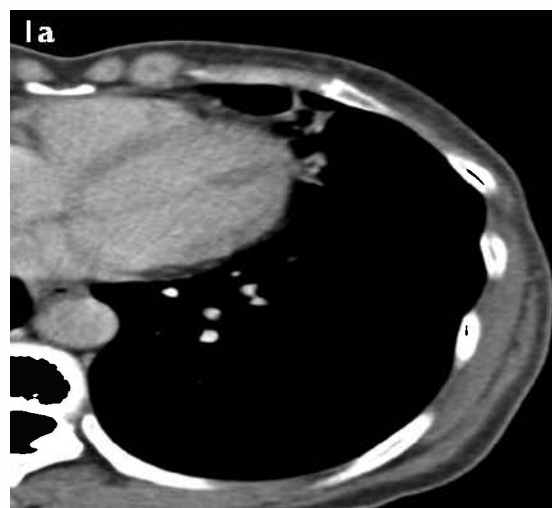
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Fig. 1 CT images of the thorax in the (a) mediastinal and (b) lung window settings show peribronchial thickening, subpleural scarring and ground-glass changes in the lingula.

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INTRODUCTION

Bronchial artery embolisation is an established therapeutic method of choice in the management of massive haemoptysis (expectoration of > 240 ml blood in 24 hours).[1] In most patients, the bronchial arteries, rather than the pulmonary arteries, are the source of massive haemoptysis. Many non-bronchial systemic arteries may also supply the chronically inflamed lung through transpleural collaterals, depending on the anatomic locations of the pulmonary parenchymal lesions and the associated pleural thickening and adhesions.[1] The internal thoracic artery is an important but unusual systemic feeder vessel supplying the bleeding area.[2] The feeder vessels may arise either directly from the internal thoracic artery or from any of its branches.[2]

We present a case of massive haemoptysis from a non-bronchial systemic circulation to the diseased lung from a hypertrophied pleural branch of the pericardiophrenic artery, which arises from the internal thoracic artery. In addition, the anastomosis of this collateral supply with the homolateral inferior phrenic artery was also demonstrated on angiography. This artery was super selectively catheterised and embolised. We also include
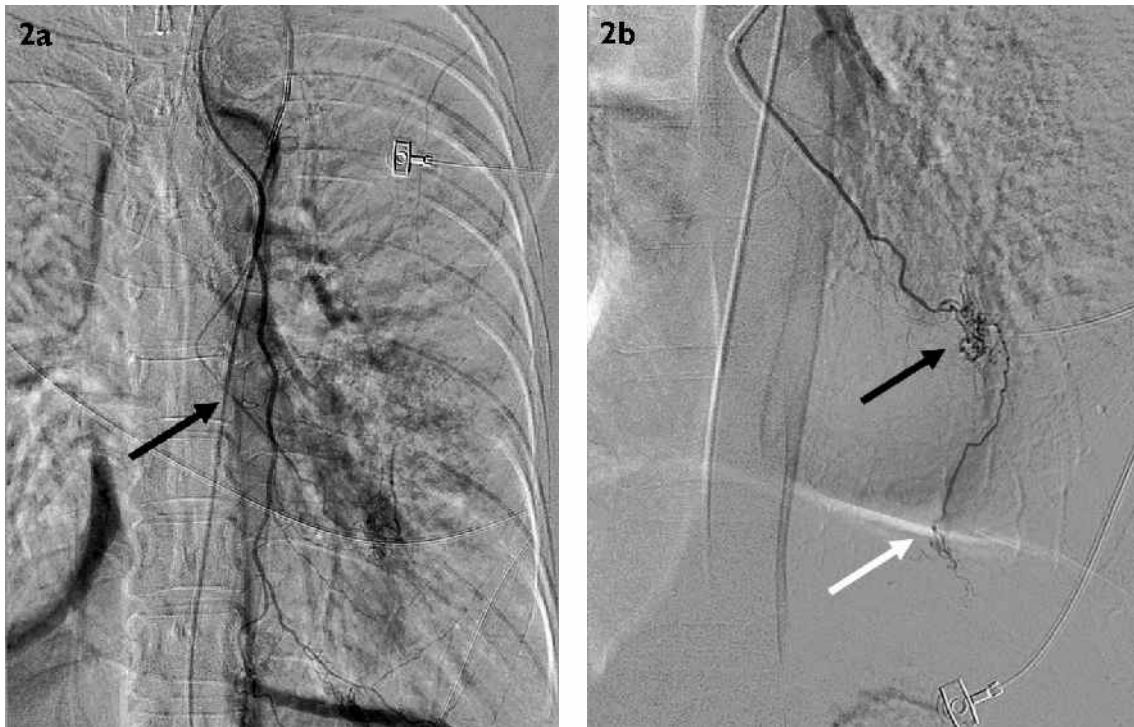
Fig. 2 Left internal thoracic artery angiograms show (a) the pericardiophrenic artery (arrow); and (b) abnormal vascularity (black arrow) and communication with the left inferior phrenic artery (white arrow) after super-selective catheterisation of the left pericardiophrenic artery.

a brief anatomical and pathophysiological review of the source of massive haemoptysis and the anatomy of the internal thoracic artery and pericardiophrenic artery.

CASE REPORT

A 61-year-old woman presented with a history of intermittent cough that lasted for a few months and a single episode of haemoptysis. Her medical history included hypertension and hyperlipidaemia. Initial chest radiograph did not reveal any significant findings. Computed tomography (CT) imaging of the thorax revealed subsegmental collapse/consolidation at the inferior lingula segment, with mild dilatation and peribronchial thickening of the lingular bronchi and subpleural scarring. The lesion in the lingula was abutting the anterior chest wall (Fig. 1). There were also patchy ground-glass changes in the superior lingular segment and left lower lobe, which represented blood in the alveoli secondary to haemoptysis.

The patient had another episode of massive haemoptysis after an interval of three days, and was hospitalised. She was then referred for emergency bronchial artery embolisation. Thoracic aortogram and selective bronchial angiograms revealed no abnormal vascularity. In the presence of a normal bronchial circulation and selective left subclavian, anterior intercostal and left internal thoracic angiograms were performed to determine the source of the haemoptysis.

Left internal thoracic angiogram revealed a hypertrophied pleural branch of the pericardiophrenic artery, which was coursing obliquely in an inferolateral direction (Fig. 2a). This hypertrophied artery was supplying an abnormal bunch of vessels in the lingula anteriorly, corresponding to the parenchymal lesions seen on the CT image. This anomalous vessel was also anastomosing directly with the homolateral inferior phrenic artery (Fig. 2b). The pericardiophrenic artery was super-selectively catheterised with a microcatheter and successfully embolised using gel foam suspension. At our institute, temporary agents are used so that vascular access is available in cases of recurrent bleed. Gel foam suspension, being a temporary embolic agent, is preferred over permanent embolic agents such as glue and metallic coils. Polyvinyl alcohol particles may cause necrosis in the embolised organ and therefore, this agent was specifically avoided. Embolisation of the internal mammary artery was being performed in our patient, and there was a presence of communication to the phrenic artery and its branch below the diaphragm. In order to avoid the complication of distal organs embolisation caused by a permanent embolic agent, a gel foam suspension was used.

However, the patient had another episode of haemoptysis the following day. Repeat thoracic
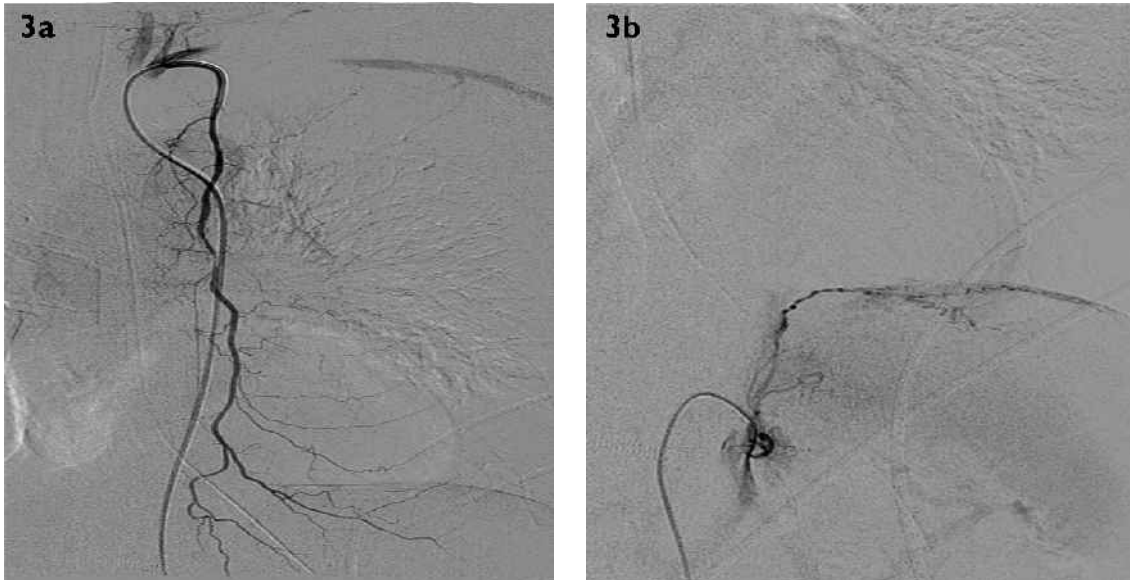
Fig. 3 (a) Left internal thoracic; and (b) inferior phrenic angiograms performed after the embolisation of the pericardiophrenic artery and repeat haemoptysis show no abnormal vascularity or staining.

arteriography (Fig. 3a) was performed, but no active contrast extravasation suggestive of bleeding was demonstrated. A check angiogram of the left inferior phrenic artery did not reveal any abnormal vascularity or staining, thus confirming successful embolisation of the pericardiophrenic artery performed earlier (Fig. 3b). Due to repeat haemoptysis and the absence of any identifiable source of bleeding, we decided to embolise the left internal thoracic artery; we suspected that angiographically occult small feeders may be the source of the haemoptysis. This was successfully performed with gel foam suspension. The patient tolerated the procedure well with no immediate complications, and her haemoptysis resolved. She experienced no further episodes of haemoptysis for the last 18 months of follow-up.

DISCUSSION

Systemic circulation, mainly of the bronchial arteries, is the primary source of bleeding in patients with massive haemoptysis. Bronchial arteries may have a normal or anomalous origin. Bronchial arteries arising outside the T5–T6 thoracic aorta level are considered anomalous.[1] Anomalous bronchial arteries can arise from the thoracic or abdominal aorta, brachiocephalic and subclavian arteries or their branches, intercostal arteries, coeliac artery branches and phrenic arteries.[1] They are identified as bronchial vessels, as they cross the lung hilus and parallel the course of the bronchi once within the thorax.[1]

Non-bronchial systemic collateral arteries may contribute to nearly 5% cases of massive haemoptysis[1]

They are different from anomalous bronchial arteries in that they are not congenital and develop during the process of various disease.[1] The course of these arteries is not parallel to those of the bronchi, and they may pass through pulmonary ligaments or adherent pleura.[1] The non-bronchial systemic collaterals can arise as branches of intercostal arteries, the thyrocervical trunk, internal thoracic artery, thoracodorsal artery, lateral thoracic arteries, other branches of subclavian artery and even intra-abdominal arteries such as the inferior phrenic artery, left gastric artery and aortic branches.[1,3] The most important non-bronchial systemic collateral source of haemoptysis is the subclavian artery and its branches (most commonly, the internal thoracic artery) for upper lobe bleeding and the inferior phrenic artery for lower lobe bleeding.[4]

The internal thoracic artery usually arises from the inferior border of the first portion of the subclavian artery, about 2 cm from the clavicle, opposite the thyrocervical trunk.[5] It descends behind the cartilages of the upper six ribs at a distance of about 1.25 cm from the margin of the sternum.[5] At the level of the sixth intercostal space, the internal thoracic artery terminates by dividing into the musculophrenic (lateral branch) and superior epigastric artery (medial branch).[5] Variations in the origin as well as in the terminal division of the internal thoracic arteries have been described in the literature.[6]

The internal thoracic artery is the source for a number of branches, namely pericardiophrenic, mediastinal, thymic, bronchial, sternal, anterior intercostals, perforating, lateral costal, musculophrenic and superior epigastric.[5] The pericardiophrenic artery is a long,

slender and somewhat tortuous vessel that usually arises from the internal thoracic artery just after the latter vessel has entered the thorax.[5] It courses obliquely downward and slightly posterior between the pleura and the pericardium, sending small branches to these structures.[5] It accompanies the phrenic nerve to the diaphragm until it anastomoses with the musculophrenic and the superior and inferior phrenic arteries.[5] Variations in the origin of the pericardiophrenic artery have also been described.[6]

There are well-established cases of internal thoracic artery being the primary source of bleeding in patients with massive haemoptysis in the English literature.[2,7-10] Jardin and Remy noted that among their 11 patients whose internal thoracic artery was the source of bleeding, in patients with haemoptysis, the collateral vessels were most frequently involved; these vessels originated from the proximal area and on the medial side of the internal thoracic artery and were related to the pericardiophrenic branches.[2] However, the exact number of such cases was not reported.

In our case, thoracic CT imaging helped to localise the possible origin of haemoptysis to the lingula, and hence, a selective left subclavian angiogram was performed in the presence of normal thoracic and selective bronchial angiograms. This revealed a hypertrophied vessel in relation to the left internal thoracic artery supplying the lingula. Further super-selective catheterisation of the internal thoracic artery confirmed this to be a hypertrophied pleural branch of the pericardiophrenic artery, which was super-selectively catheterised and embolised. This hypertrophied pleural branch of the pericardiophrenic artery was also anastomosing directly with the homolateral inferior phrenic artery.

It is important to recognise that non-bronchial systemic collateral arteries may be a source of haemoptysis. Super-selective catheterisation of smaller vessels such as the pericardiophrenic artery is now possible with the available microcatheters. This can help preserve the internal thoracic artery which may be of implication in future cardiac surgeries. However, in our case, an embolisation of the internal thoracic artery was performed due to an episode of recurrent haemoptysis following pericardiophrenic embolisation. Also, the possibility of collateral circulation between the pericardiophrenic artery and the inferior phrenic artery should be borne in mind, and embolisation of the inferior phrenic artery may also be performed to control haemoptysis, if required.

It is important to be aware of the anatomy of non-bronchial systemic collaterals. A thorough search for a non-bronchial systemic arterial supply should be made during angiography in patients with haemoptysis when no bronchial artery source is identified. This is essential as there may be an early recurrent bleeding after successful embolisation of the bronchial artery in the event of a missed non-bronchial systemic artery at initial angiography.

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