Title Page

Transpleural systemic artery-to-pulmonary artery communications in the absence of chronic inflammatory lung disease. A case-series and review of the literature.

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Highlights:

- Transpleural systemic-to-pulmonary artery shunts in the absence of chronic inflammatory lung disease are usually related to previous trauma, intervention or sepsis.
- Congenital transpleural systemic-to-pulmonary artery shunts are rare.
- These shunts are seldom symptomatic in the absence of chronic lung disease.

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Keywords: Pulmonary Arteries; Bronchial Arteries; Vascular Malformations; Lung; Thoracic imaging.

Abbreviations:

CT: Computed tomography

VATS: Video assisted thoracoscopic surgery

CXR: Chest X-ray

AVM: Arteriovenous malformation

PAVM: Pulmonary arteriovenous malformation

HHT: Hereditary hemorrhagic telangiectasia

TB: Tuberculosis

Abstract:

Aim: To describe the causes and CT and angiographic appearances of transpleural systemic-artery-to-pulmonary artery shunts in patients without chronic inflammatory lung disease and determine their best management.

Materials and Methods: All patients referred to a tertiary referral unit between January 2009 and January 2020 in whom a diagnosis of a systemic-to-pulmonary artery communication without underlying chronic inflammatory lung disease was subsequently made have been included in this report. Medical records and imaging findings were retrospectively reviewed.

Results: Ten patients (M:F = 7:3; median age 42 years [22 – 70 years] with systemic artery-to-pulmonary artery shunts without chronic inflammatory lung disease were identified. Five were misdiagnosed as having a pulmonary arteriovenous malformation and had been referred for embolization. In six patients there was either a history of accidental or iatrogenic thoracic trauma or of inflammatory disease involving the pleura and in two patients, in whom a previous medical history could not be obtained, there were CT features suggesting previous pleural inflammatory disease. Two shunts were thought to be congenital. All individuals were asymptomatic other than one with localised thoracic discomfort that dated from the time of surgery. All patients have been managed conservatively and have remained well with a median follow-up of 4.5 years [1 – 11.3].

Conclusions: Localized transpleural systemic artery-to-pulmonary artery shunts in the absence of chronic inflammatory lung disease are usually related to previous thoracic trauma/intervention or abdominal or pulmonary sepsis involving a pleural or diaphragmatic surface. Congenital shunts are rare. Our experience and much of the literature supports conservative management.

Introduction:

Bronchial artery to pulmonary artery communications measuring up to 500 μm exist in normal lung and can be demonstrated by microangiography [1, 2] as well as on catheter angiography of normal bronchial arteries in living subjects. These anastomoses increase in size in certain pathological states, in particular chronic inflammatory lung disease, resulting in retrograde filling and reversed flow within peripheral pulmonary artery branches and sometimes within whole lung segments [3,4]. Friable pulmonary vessels within the diseased lung are thus exposed to systemic artery pressure and this may result in hemoptysis, which can be life-threatening [3,4]. This systemic artery-to-pulmonary artery shunting is a common finding on bronchial artery angiograms performed in this group of patients presenting with hemoptysis [4]. Although there are no published papers that specifically address a relationship between the size of these communications or the degree of pulmonary artery opacification with the severity of haemoptysis, our own experience and that of others suggests that such a relationship does not exist. The very presence of a visible systemic artery-to-pulmonary shunt on angiography is, however, a helpful pointer to the likely source of bleeding [3]. Pseudoaneurysmal change within the friable peripheral pulmonary artery branches may be seen in as many as 10% of these individuals [3] and is often a sign of particularly severe disease.

It is well recognized that in those individuals in whom the chronic inflammatory process involves the pleura, there is likely to be a non-bronchial systemic arterial supply to the diseased lung from intercostal arteries, thoracic branches of the subclavian and axillary arteries and diaphragmatic branches of the inferior phrenic arteries [5]. These vessels contribute a supply to the diseased lung via transpleural systemic artery-to-pulmonary artery anastomoses and reversed flow within peripheral pulmonary arterial

branches is commonly visualized when these arteries are studied at angiography (Figure 1).

Unlike the bronchial artery-to-pulmonary artery anastomoses that can be demonstrated in normal individuals, it is felt that these transpleural communications represent new vessels that have developed within areas of chronic inflammation as such anastomoses have not been documented in normal tissues. It is less well recognized that transpleural systemic artery-to-pulmonary artery anastomoses causing a variable degree of shunting may also develop in response to focal pleural trauma or inflammation without underlying pulmonary parenchymal disease. These are often incidental findings on thoracic CT studies performed for other reasons and may be mistaken for pulmonary or systemic arteriovenous malformations [6]. The significance of these abnormalities is unclear, and their optimal management is not known. This article serves to highlight the imaging findings of these transpleural anastomoses and to discuss their best management.

Materials and Methods:

The medical records and imaging findings of all patients referred over an eleven-year period between January 2009 and January 2020 to a tertiary referral unit specializing in pulmonary vascular anomalies, in whom a diagnosis was made of a systemic artery-to-pulmonary artery shunt in the absence of chronic inflammatory lung disease, were retrospectively reviewed. Patient demographic data and clinical histories, including possible etiological factors accounting for the transpleural anastomoses and their subsequent management and follow-up, were obtained from the hospital medical records or the patient's general practitioner. Both the referral hospital imaging and

any subsequent investigations required to confirm the diagnosis were reviewed. Descriptive statistics used throughout. The results are presented as mean or median and range.

Ethics approval was granted and informed consent waived prior to commencing this retrospective study.

Results:

Ten individuals were diagnosed as having a transpleural systemic artery-to-pulmonary artery shunt (Male: Female ratio = 7:3; median age 40 years [range 23-70 years]). In all but four patients there was either a history of accidental or iatrogenic thoracic trauma or of inflammatory disease involving the pleura or diaphragm. All were asymptomatic other than one individual with localized thoracic discomfort that dated from the time of thoracic surgery. In five patients an erroneous diagnosis of a pulmonary arteriovenous malformation had been made and they had been referred specifically for embolization.

Presentation:

In nine patients the thoracic vascular abnormality was an incidental finding on chest imaging performed for unrelated reasons. One individual sought medical advice because of persistent discomfort related to a right lower thoracoscopic surgical and chest drain wound and was noted to have a bruit at this site on clinical examination.

Etiology:

Two patients had undergone transpleural intervention (intercostal chest drains, video assisted thoracoscopic surgery (VATS) and/or pleurodesis). Two others had a history of previous severe chest infections, one of whom also had a concomitant pulmonary

embolus. Two patients had a history of previous abdominal surgery complicated by a subphrenic abscess, in one of whom the abscess fistulated. In two patients whose imaging was sent for review, no previous medical details could be obtained from the referring hospital, but both individuals had CT features of pleural thickening likely secondary to infection, pulmonary embolic disease or thoracic intervention. In two patients, both of whom had large apical transpleural shunts, there was no history of accidental or iatrogenic trauma, pulmonary sepsis or pulmonary embolic disease.

Imaging:

Contrast enhanced thoracic CT – aortic phase in four patients, pulmonary arterial in four and portal venous in two – demonstrated an abnormality in all individuals. Eight were right sided; five were supplied by ipsilateral inferior phrenic and intercostal arteries; two by inferior phrenic artery branches alone; two by intercostal arteries alone; and one by ipsilateral thoracic branches of the subclavian and axillary arteries. Plain CXR appearances were variable and non-specific although in some there was evidence of localized pleural thickening at the site of the shunt (Figure 2a, 4a) or lobulated soft tissue densities due to prominent pulmonary arterial branches (Figures 3a, 5c). CT findings consisted of:

- A nodular or more diffuse soft tissue opacity in the lung periphery abutting the pleura or attached to it via linear strands and continuous with one or more peripheral pulmonary artery branches (Figures 3b,c, 4a-c, 5a,b);
- Hypertrophy of intercostal or other systemic arteries which were best visualized on CT images acquired during an aortic phase of contrast medium enhancement (Figures 2b, 4b,c);

- The direction of flow through the systemic artery-to-pulmonary artery anastomoses could be appreciated on CT in five patients. In four of these individuals, images obtained during the aortic phase of contrast medium enhancement demonstrated increased density of peripheral pulmonary artery branches when compared with more central vessels due to their systemic arterial supply (Figures 2c, 4c). In one previously reported patient [6], in whom the CT had been performed during the pulmonary arterial phase of contrast medium enhancement, a 'negative' filling defect was present in the peripheral pulmonary artery due to retrograde filling with unopacified blood via the transpleural shunt, which was mistaken for a pulmonary embolus.

Three patients underwent diagnostic angiography. Aortography and selective intercostal artery angiograms were performed in the single patient with localized symptoms related to the site of thoracoscopy to assess if there was a role for embolization. Embolization was not performed due to numerous small transpleural anastomoses that were present and the recognition that this procedure would not be curative. One of the patients in whom the erroneous diagnosis of a pulmonary AVM had been made, underwent pulmonary arteriography with a view to its embolization. The true nature of this abnormality was recognized when retrograde flow within a pulmonary artery branch was demonstrated on this study; this patient has been previously reported [6]. One patient underwent angiography to assess the anatomy of the transpleural communications at the left lung apex to determine if there was a role for surgery or embolization. Neither was performed due to the complex anatomy that was present and the fact that the patient was asymptomatic. The angiographic findings mirrored those seen on CT (Figures 5b and 6c-e).:

- Hypertrophy of systemic arterial branches supplying the transpleural anastomoses;
- Numerous small transpleural anastomotic vessels;
- Retrograde filling of pulmonary arterial branches in the adjacent lung.

Follow-up:

All patients have been managed conservatively and have remained well with a median follow-up of 4.5 years (1 – 11.3 years). None has suffered from hemoptysis or developed other symptoms. The localized chest wall discomfort reported by one patient at presentation has resolved. In eight individuals who have undergone follow-up CT imaging between 0.2 and 7.3 years (median 2 years) after presentation, there has been no significant change in the imaging findings.

Discussion:

Bronchial artery-to-pulmonary artery and transpleural non-bronchial systemic artery-to-pulmonary artery anastomoses have been recognized for very many years and have received much attention in the literature [1-8]. These are most frequently related to chronic pulmonary and/or pleural inflammatory disease, especially bronchiectasis and/or chronic tuberculosis, and may be associated with hemoptysis, which can be life-threatening. Similar large anastomoses occur in patients with various forms of cyanotic congenital heart disease [8]. A large systemic arterial supply to an area of lung in the absence of chronic inflammatory lung disease or congenital cardiac anomalies is, however, much less common and may be congenital or acquired.

Congenital

Three congenital anomalies are recognized:

- The first is one of the various types of pulmonary sequestration or malinosculation [7, 9, 10] in which a large systemic artery arises from the thoracic or upper abdominal aorta (or less commonly from a visceral arterial branch) and supplies a portion of lung that has a normal bronchial anatomy and normal pulmonary venous drainage. This 'normal' lung may or may not have a normal pulmonary artery supply. There is, however, no communication between this anomalous systemic artery and the pulmonary artery branches and, therefore, no systemic artery-to-pulmonary artery shunt.
- The second is a congenital bronchial artery-to-pulmonary artery malformation in which branches of an hypertrophied bronchial artery communicate directly with central pulmonary artery branches within a segment of otherwise normal lung. Such malformations are very rare and may occur sporadically or be associated with hereditary hemorrhagic telangiectasia (HHT), an autosomal dominant condition which is much more commonly associated with pulmonary arteriovenous malformations (PAVMs). These congenital communications in HHT should not be confused with the acquired systemic arterial supply to a PAVM that may occur after its embolization. These bronchial artery-to-pulmonary artery malformations may be asymptomatic or be a cause of hemoptysis [11].
- The third is a pleural systemic artery-to-pulmonary artery vascular malformation [12 -14].

The first two of these anomalies lie outside the scope of this article and will not be considered further; the congenital transpleural AVM will be discussed later in this article.

Acquired

Acquired transpleural systemic artery-to-pulmonary artery shunts in the absence of chronic pulmonary inflammatory disease or complex congenital cardiac anomalies are considered rare and most publications describing these abnormalities are case reports of single patients [12-19]. Accidental or iatrogenic trauma, pulmonary sepsis involving the adjacent pleura and pulmonary embolic disease are the most frequent etiological factors as was the case in our own series reported here. The largest series reported to date of these acquired transpleural anastomoses is from the cardiology literature [20]. Guler at et evaluated 561 patients in whom left internal mammary artery cardiac bypass surgery had been performed and found five individuals with transpleural anastomoses between branches of the left internal mammary artery and pulmonary vessels. These anastomoses are recognized as developing due to inadvertent disruption of the visceral pleura during surgery inciting a localized inflammatory response in the same way as those that develop at other sites in the thorax related to trauma and/or infection.

Congenital versus acquired

The vast majority of transpleural systemic artery-to-pulmonary artery communications in the absence of chronic inflammatory lung disease are acquired although the causative insult may only be recognized on careful review of the patient's previous medical history, which may have occurred many years before diagnosis of the shunt.

Of the eight patients in this sereies with acquired lesions, the history of the causative accidental or iatrogenic chest trauma or pulmonary sepsis was not recalled by the patient at the time of initial assessment in five and was only elicited on subsequent direct questioning after the diagnosis of a transpleural shunt had been made. Of those cases reported in the literature as being congenital, several are likely to have been acquired based upon their imaging findings and/or the description of dense pleural adhesions at subsequent surgery [21-23].

In the absence of a history of thoracic trauma or sepsis, however, as was the case in two of the patients reported here, a congenital vascular malformation must be considered. In addition to the lack of previous pulmonary disease, there were other differences between the lesions seen in these two patients and those present in patients with acquired shunts:

- Both involved the apical pleura whilst the others involved the lung bases;
- There was an absence of pleural thickening;
- There was more prominent dilatation of peripheral pulmonary artery branches close to the transpleural shunting, presumably reflecting the longstanding nature of the abnormality;
- In one patient there were several small thin-walled pulmonary cysts in the periphery of the upper lobe close to the transpleural shunts which did not have a characteristic appearance of previous pulmonary infection and may, therefore, have been developmental (Figures 6a,b).

Management

In most of the early case reports of transpleural shunts, surgical correction was attempted despite the fact that the majority of patients were asymptomatic [15, 21, 24].

The rationale for intervention has subsequently been questioned with the recognition firstly, that most, if not all of these shunts remain asymptomatic on long-term follow-up and secondly, that attempts to obliterate transpleural shunting by surgery or embolization is often not possible and may be associated with significant complications [12, 14, 19, 25]. Our own series, in which all patients have been successfully managed conservatively despite the presence of dramatic shunting in some, serves to illustrate that these lesions are best left alone. The increasing use of CT means that these acquired transpleural systemic artery-to pulmonary communications are likely to be encountered more frequently.

This study is not without limitations, which include the small sample size and the relatively short median imaging follow-up of 2 years, although the median clinical follow-up was 4.5 years. Furthermore, the majority of patients included in this study were referred for treatment, which may have led to a selection bias. It is unlikely however that other patients with these shunts that were not referred for treatment had complications as our unit is the regional referral center for the management of hemoptysis.

In conclusion, focal areas of transpleural systemic artery-to-pulmonary artery shunting in the absence of chronic inflammatory lung disease are usually due to previous pleural inflammation or accidental or iatrogenic trauma although some may be congenital. They are almost always incidental findings on thoracic or upper abdominal imaging performed for another reason. Close scrutiny of their appearances will allow avoidance of an erroneous diagnosis of a pulmonary arteriovenous malformation. This series represents the largest to date and provides further evidence that these

lesions appear to remain asymptomatic on long-term follow-up without intervention and can be managed conservatively.

Figures:

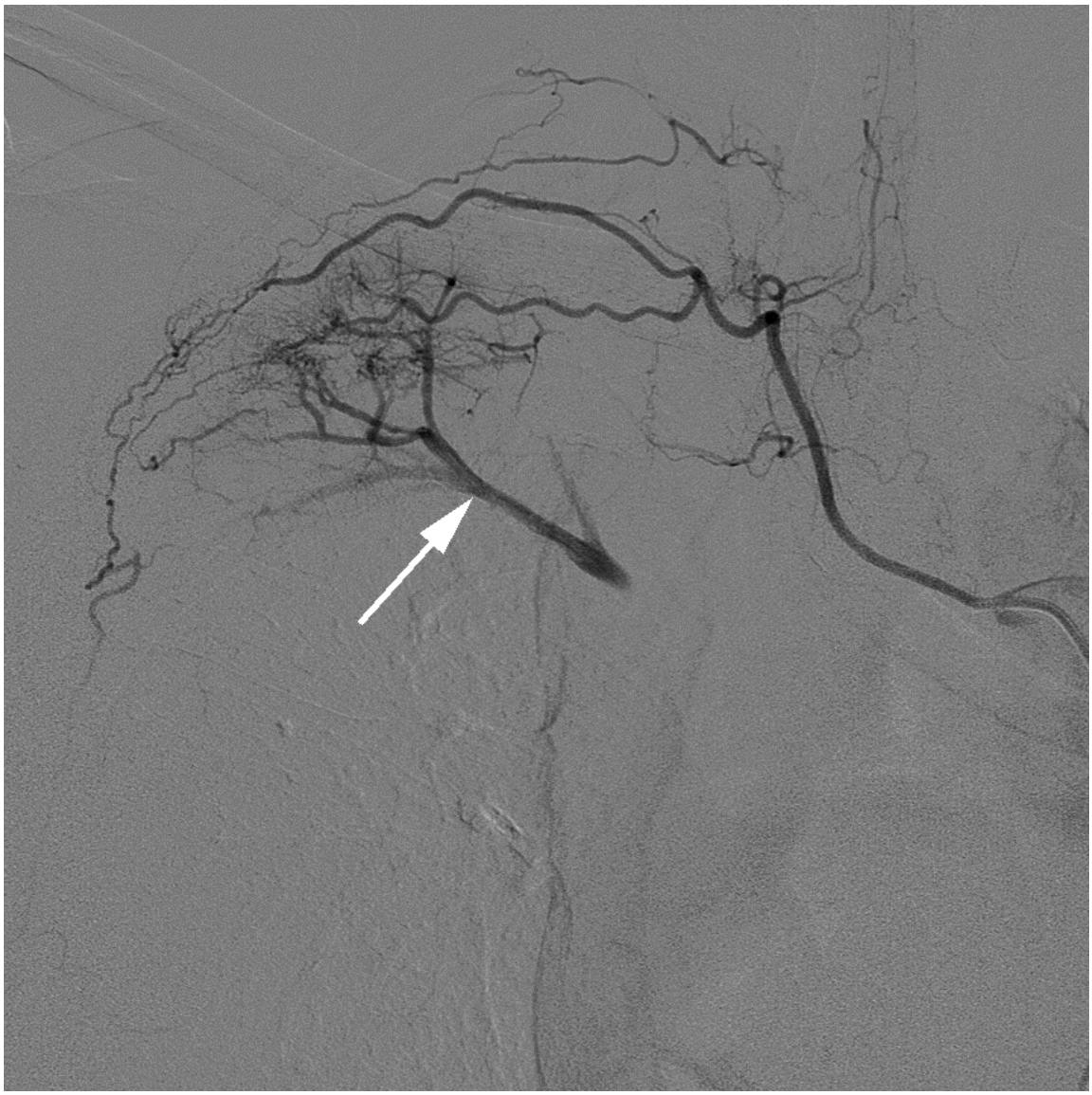


Figure 1. Selective right third intercostal digital subtraction arteriogram in patient with hemoptysis due to previous TB demonstrating retrograde filling of right upper lobe pulmonary artery branch (arrow) via numerous transpleural intercostal artery-to-pulmonary artery communications

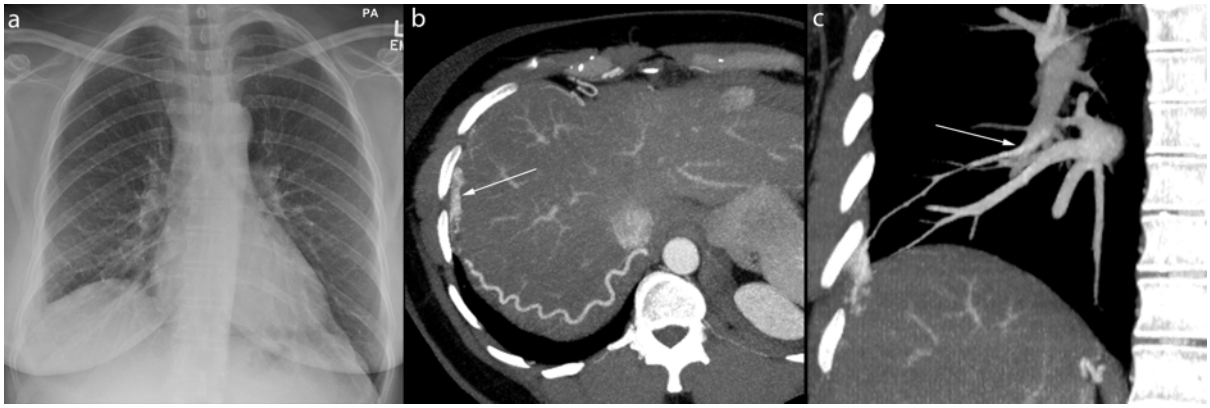


Figure 2. 44-year-old woman with incidental finding of right basal systemic artery-to-pulmonary artery shunting secondary to previous chest infection several years previously. **a.** Frontal chest radiograph demonstrating pleural thickening in the right lateral costophrenic angle. **b.** Axial maximal intensity projection CT image obtained during the aortic phase of contrast medium enhancement demonstrating hypertrophy of the right inferior phrenic artery supplying an area of thickened pleura (arrow) in the right lateral costophrenic angle. **c.** Coronal maximum intensity projection CT image demonstrating retrograde filling of a pulmonary arterial branch via transpleural anastomoses. Note the increased enhancement of this pulmonary arterial branch (arrow) compared with the main right descending pulmonary artery due to its systemic arterial supply.

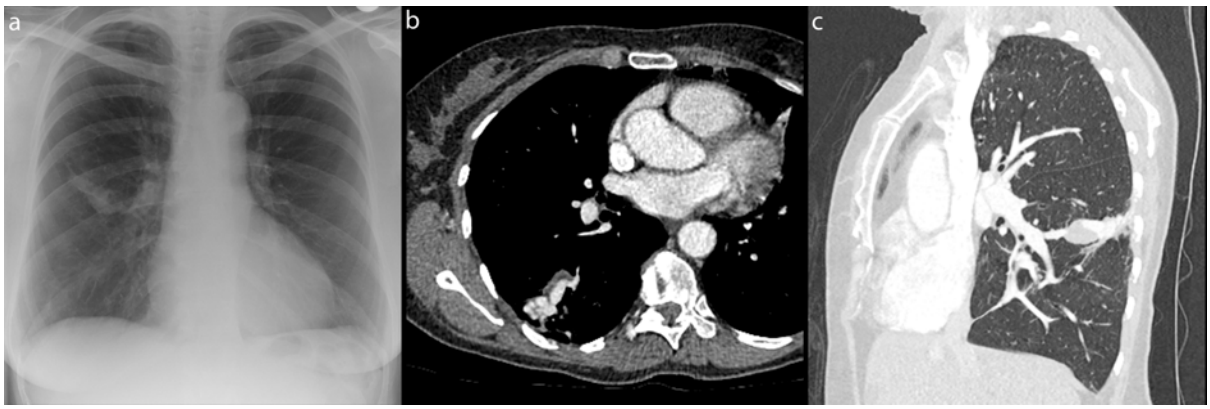


Figure 3. Transpleural systemic artery-to-pulmonary artery shunting in 66-year-old lady secondary to a peripartum lung abscess more than 20 years previously. **a.** Frontal

chest radiograph demonstrating lobulated soft tissue opacity projected over the right mid-zone. **b.** Axial CT image demonstrating dilated pulmonary arterial branches in the periphery of the right lower lobe with adjacent parenchymal strands extending to the pleural surface. **c.** Sagittal contrast enhanced CT image demonstrating dilated pulmonary arterial branches in the periphery of the superior segment of the right lower lobe with adjacent pleural thickening parenchymal scarring. The soft tissue component adjacent to the dilated pulmonary artery branch represents post-inflammatory scarring.

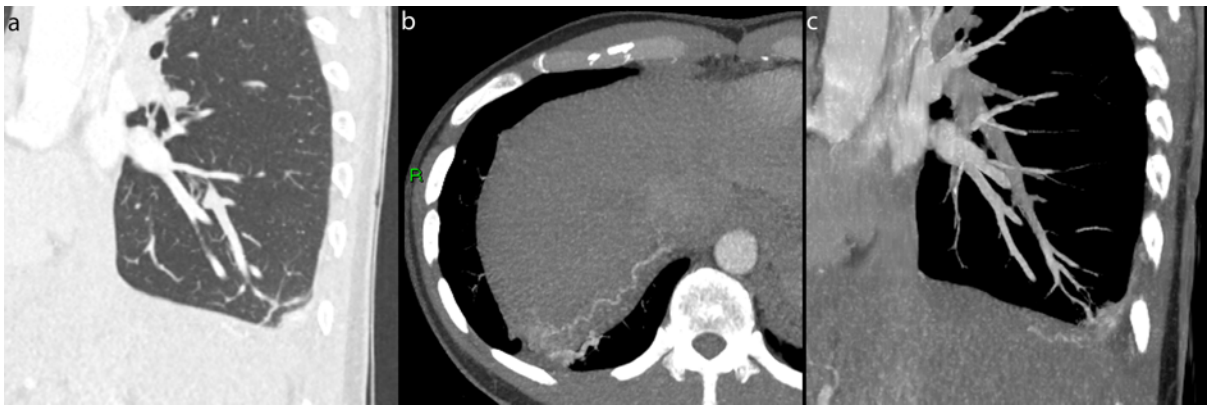


Figure 4. 48-year-old man with a history of right sided pulmonary embolic disease and a chest infection several years previously. Incidental finding of right basal transpleural systemic artery-to-pulmonary artery shunt on imaging performed for another reason. **a.** Parasagittal CT image demonstrates an area of pleural thickening and adjacent pulmonary parenchymal stranding in the right posterior costophrenic recess intimately related to peripheral pulmonary arterial branches. **b.** Axial maximum intensity projection CT image through the lung bases demonstrates right inferior phrenic artery hypertrophy and retrograde peripheral pulmonary artery branch contrast medium enhancement through the area of pleural thickening. **c.** Parasagittal maximum intensity projection CT image demonstrating increased enhancement of the peripheral

portion of pulmonary arterial branch relative to its more central portion due to the systemic arterial supply.

Figure 5. Large systemic artery-to-pulmonary artery shunt in 32-year-old man with a history of recurrent spontaneous pneumothoraces treated by video-assisted thoracoscopic talc pleurodesis several years previously.

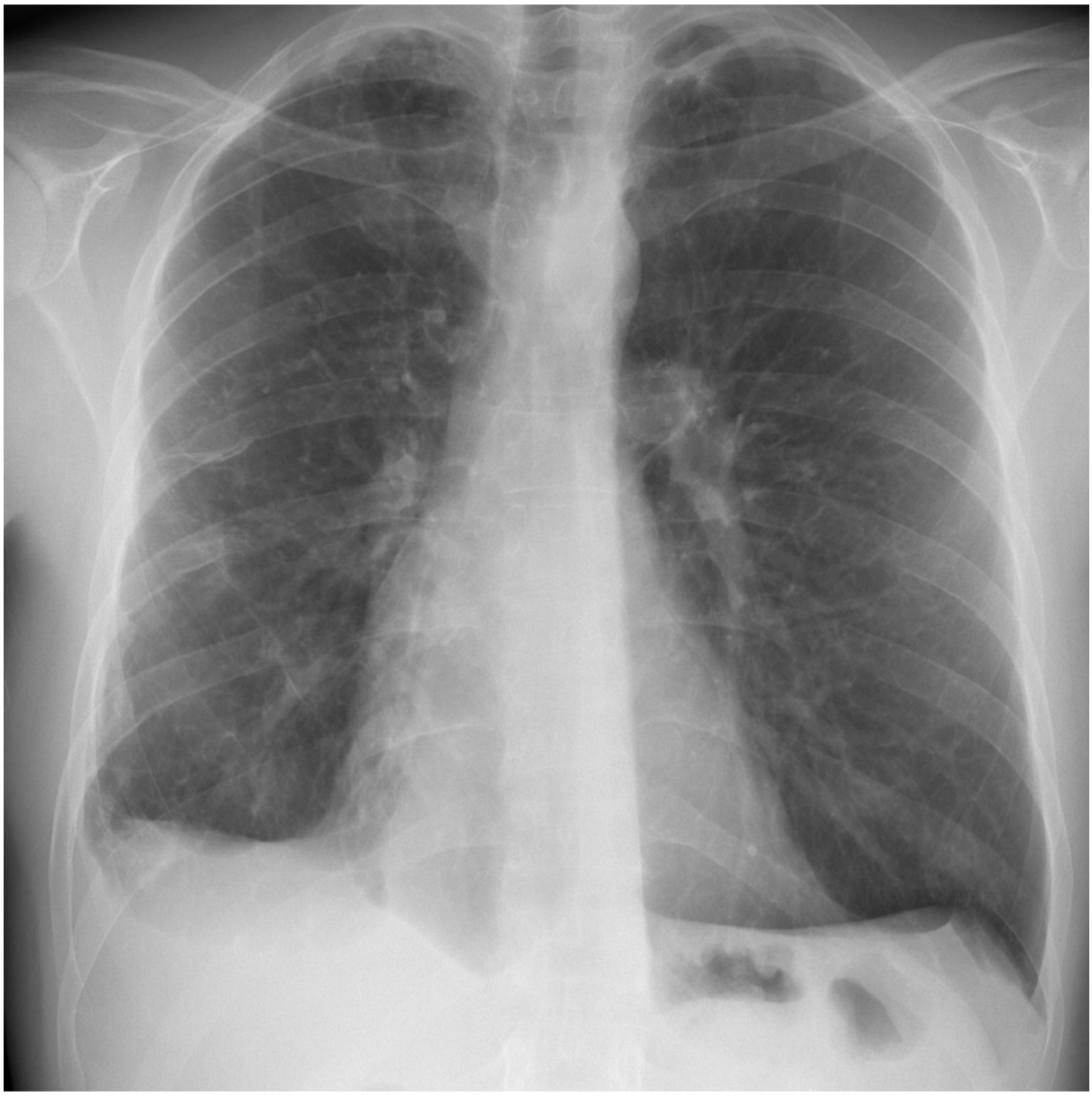
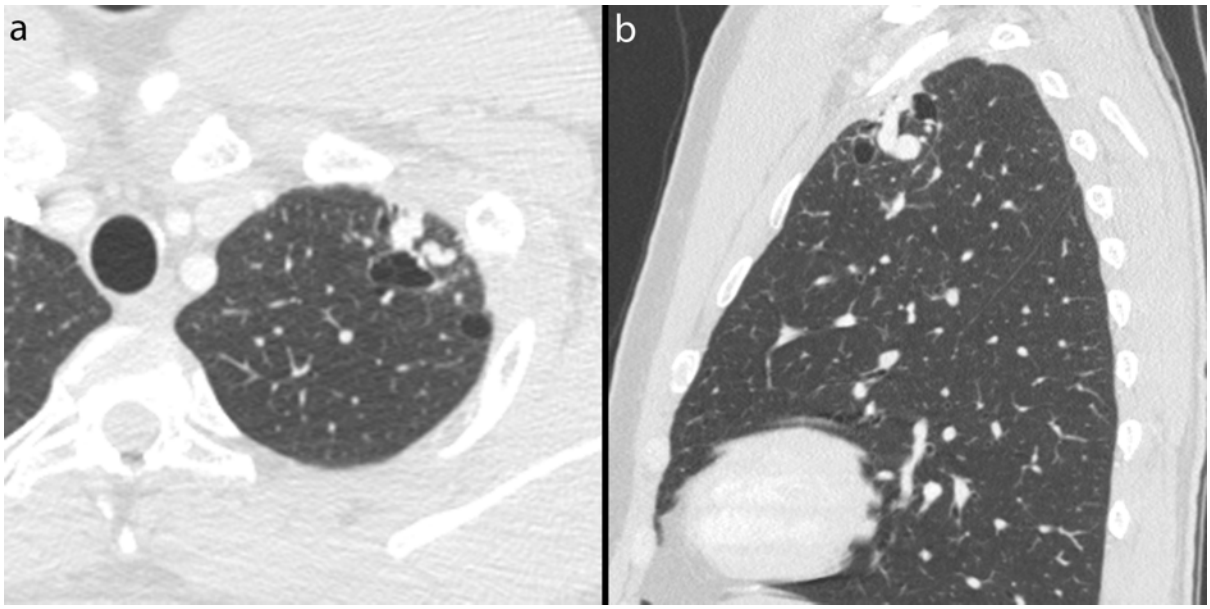


Figure 5a. Frontal chest radiograph demonstrates right basal pleural thickening.

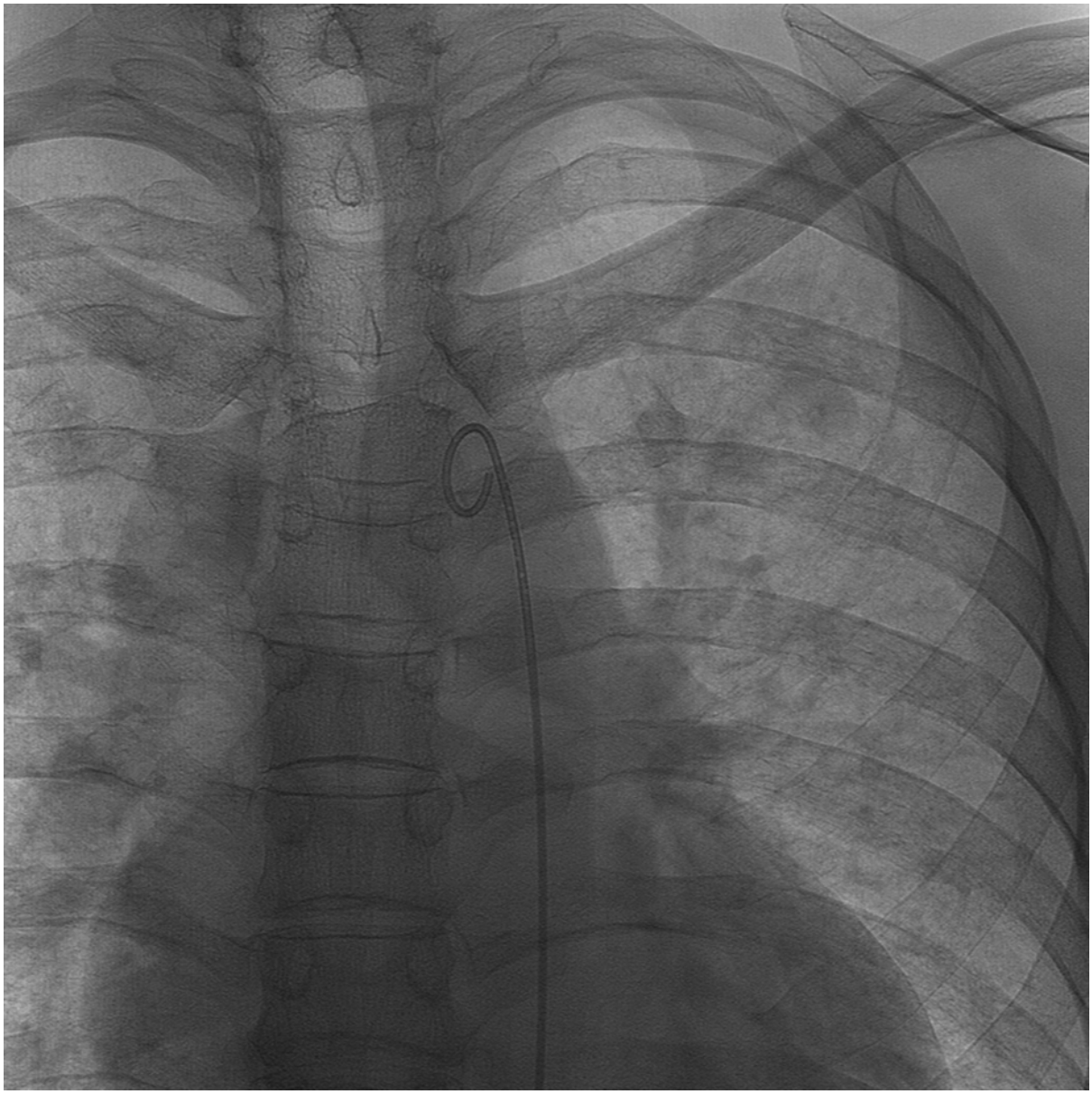


Figure 5b. Right lower intercostal artery angiogram using a digital subtraction technique demonstrates hypertrophy of the intercostal artery and numerous tiny transpleural vessels communicating with dilated peripheral right lower lobe pulmonary artery branches that are filling retrogradely into the midzone (arrows). Adjacent intercostal arteries (not shown) were also hypertrophied and contributed to transpleural shunting.

Figure 6. 37-year-old man with presumed congenital transpleural systemic artery-to-pulmonary artery shunting.



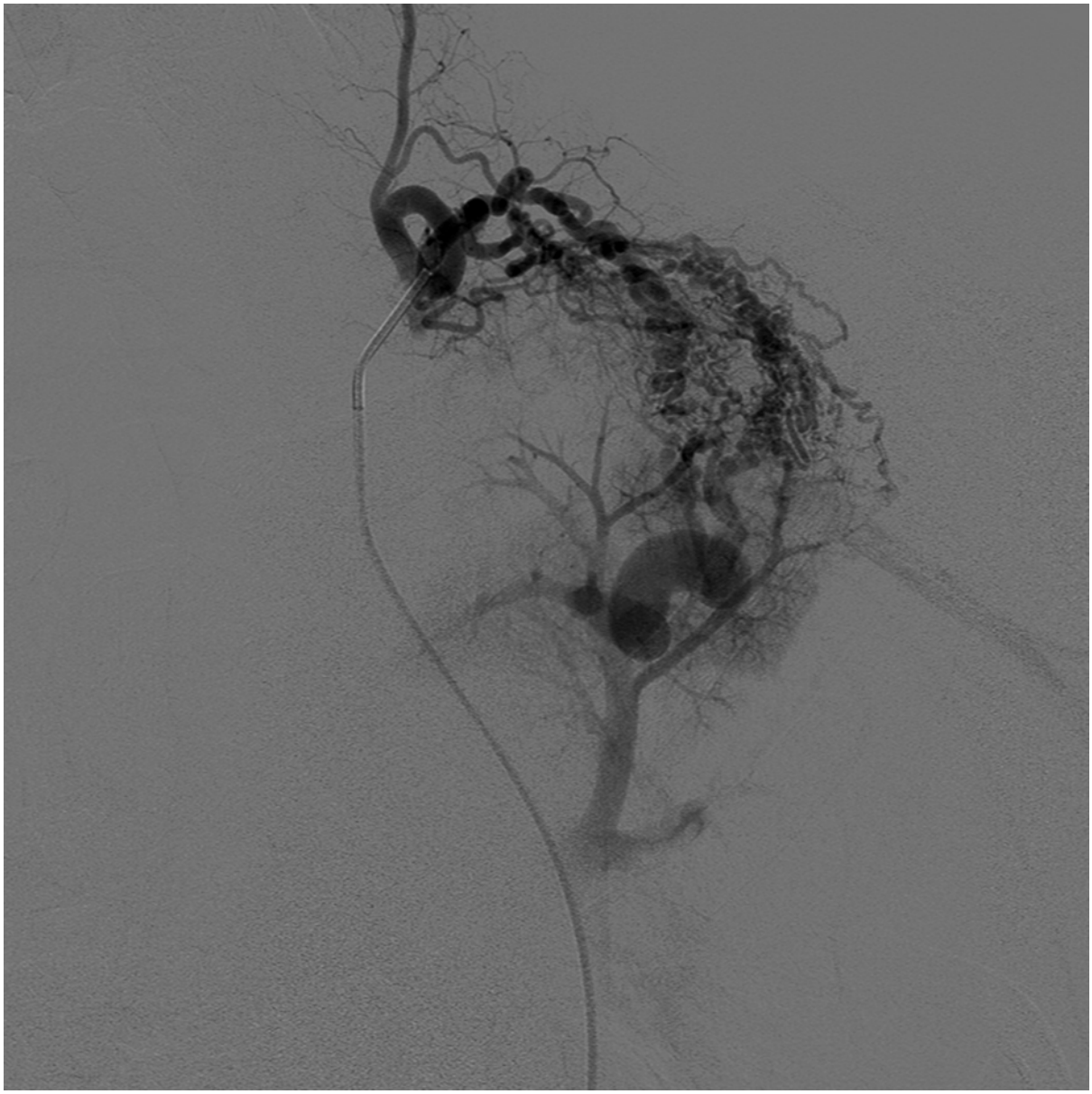
a. Axial CT image through the lung apices demonstrates dilatation of peripheral pulmonary arterial branches immediately adjacent to the pleural surface. Several small thin-walled cysts were also present. **b.** Sagittal CT image demonstrating the peripheral pulmonary artery dilatation and surrounding pulmonary parenchymal cysts that were not present elsewhere within either lung.



c. Control film from digital subtraction aortogram which shows some subtle soft tissue nodules in the left upper zone.



d. Aortogram demonstrates marked transpleural systemic artery-to-pulmonary artery shunting at the left lung apex supplied by numerous small branches of the left axillary, costocervical and internal mammary arteries. A dilated pulmonary arterial branch (arrow) fills retrogradely to the left hilum.



e. Selective left costocervical trunk arteriogram better demonstrates the numerous transpleural anastomoses and the retrograde pulmonary artery branch opacification.

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