# SOUTHWEST JOURNAL of PULMONARY & CRITICAL CARE

# Journal of the Arizona, New Mexico, Colorado and California Thoracic Societies <u>www.swjpcc.com</u>

Repeat Episodes of Massive Hemoptysis Due to an Anomalous Origin of the Right Bronchial Artery in a Patient with a History of Coccidioidomycosis

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### Abstract

Anomalous bronchial arteries originate outside the space bound by the T5 and T6 vertebrae at the major bronchi. Here, we highlight a case of a 37-year-old man with a past medical history of coccidioidomycosis and who presented with massive hemoptysis. A bronchial angiogram showed the patient had a right bronchial artery originating anomalously from the left subclavian artery. The patient ultimately underwent a bronchial artery embolization, after which he achieved symptomatic remission.

#### Introduction

Hemoptysis from primary coccioidomycosis is unusual and should prompt a search for other causes (1). These could include bronchitis, malignancy, or rarely, a fungus ball. Anomalous bronchial arteries have

origins outside the space bound by the T5 and T6 vertebrae at the level of the major bronchi (2). Bronchial artery embolization is the standard treatment for patients with ruptured anomalous bronchial arteries and resultant hemoptysis (3). Here, we present a unique case of a 37year-old male with a past medical history of coccidioidomycosis and previous episodes of massive hemoptysis who was found to have an anomalous right bronchial artery originating in his left subclavian artery. Symptomatic remission was achieved with bronchial artery embolization. To our knowledge, this is the only reported case of a patient with a history coccidioidomycosis and a ruptured anomalous right bronchial artery that was successfully treated with bronchial artery embolization.

# Case Presentation

Our patient is a 37-year-old man with a past medical history significant for coccidioidomycosis (resolved nine years prior) and previous episodes of massive hemoptysis who presented to our emergency room with multiple episodes of hemoptysis over the course of one day. On admission, he reported a five-pack year smoking history. He denied hematemesis, dyspnea, and angina, a history venous thromboembolism and alcohol and recreational drug use. In the emergency department, the patient was afebrile, his blood pressure was 177/119 mmHg, heart rate was 96 beats/min, respiratory rate was 16 breaths/minute, and his oxygen saturation was 95% on room air. The patient's physical exam revealed diffuse rales throughout the right lung and decreased breath sounds in the right lower lobe. The remainder of the patient's physical exam was negative for acute abnormalities. His lab values on admission were significant only for an elevated D-dimer at 1.28 mcg/mL; his hemoglobin was 14.2 gm/dL and his INR was 0.93 sec/mL. His chest radiograph showed ill-defined patchy parenchymal densities over the bilateral lower lobes (Figure 1).

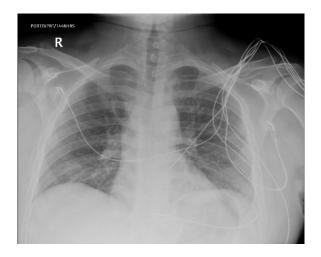


Figure 1. Chest x-ray reveals ill-defined patchy parenchymal densities over the lower lobes suggest evolving multifocal pneumonia or atypical viral pneumonia.

He experienced a witnessed episode of hemoptysis, expectorating 300 cc's of blood, prompting an emergent bronchoscopy. During the bronchoscopy, bloody secretions were noted to in his right lower lobe. A five centimeter dark red gelatinous material was removed and sent for pathology studies alongside bronchoalveolar lavage washings. Two mL's of 2% epinephrine were administered, after which no active oozing was noted. The patient was then intubated for airway protection and admitted to the intensive care unit.

A repeat chest radiograph revealed opacification throughout the right lung with evidence of volume loss (Figure 2).



Figure 2. Chest x-ray showing interval development of opacification throughout the right lung with evidence of volume loss including rightward mediastinal shift. The left lung is clear.

The patient was empirically treated for atypical pneumonia with azithromycin, ceftriaxone, dexamethasone, and albuterol breathing treatments. A computed tomography angiogram (CTA) of the chest with contrast showed multifocal flocculent and nodular infiltrate posterolateral aspect right lower lobe as well as mild mucous plugging and bronchial edema. Bronchial angiography confirmed the branching of the right bronchial artery from the left subclavian artery (Figure 3) and evidence of shunting to the right lower lobe (Figure 4).



Figure 3. Bronchial angiography prior to embolization- right bronchial artery directly arising from the left subclavian artery and is unusually large in caliber.

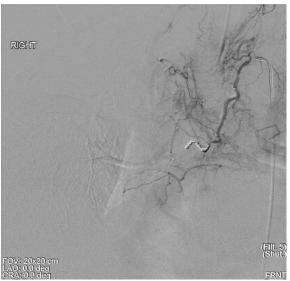


Figure 4. Bronchial angiography confirms opacification of the right lower lobe.

After the aberrant artery was confirmed on bronchial angiogram, the patient underwent a right bronchial artery embolization. He was subsequently extubated. Pathology and bronchoalveolar lavage studies revealed blood; the patient's infectious and autoimmune work-up were entirely negative. He was discharged home with self-care. To date, the patient has only experienced one episode of hemoptysis status-post embolization.

## Discussion

Differential diagnoses for massive hemoptysis include pulmonary infections, such as coccidioidomycosis, invasive aspergillosis and Mycobacterium tuberculosis, and cardiovascular causes, including anomalous origin of bronchial arteries. A thorough diagnostic evaluation is needed to identify the causative underlying pathology, site of bleeding, and vascular anatomy, so that the appropriate treatment can be initiated (3). Common origins of the bronchial arteries include the inferior aortic arch. distal descending thoracic aorta, subclavian artery, brachiocephalic trunk, thyrocervical trunk and coronary artery (5). A bronchial angiogram was pivotal in the evaluation of the anatomy of the bronchial arteries in our patient's case, as it allowed for the optimal artery embolization due to the identification of an anomalous artery early in his treatment course.

The bronchial arteries can become dilated and tortuous due to chronic inflammatory diseases such as bronchiectasis, coccidioidomycosis and tuberculosis, and are prone to vascular remodeling; rendering them fragile (6). The new collateral vessels have thin walls, making them prone to rupture and bleeding. In our patient's case, chronic inflammation related to his prior coccidioidomycosis infection contributed to the remodeling of his anomalous right bronchial artery, rendering it prone to rupture and therefore the likely culprit of his massive hemoptysis.

# Conclusion

Overall, this case emphasizes the importance of recognizing the fragility of anomalous bronchial arteries. A history of previous episodes of hemoptysis can alert clinicians to the possibility of a congenital abnormality exacerbated by subsequent infection.

### References

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