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Anterior Cord Syndrome after Embolization for Malignant Hemoptysis

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Abstract

Keywords

► interventional radiology
► complication
► hemoptysis
► paralysis
► spinal cord

Interventional radiology plays an integral role in the management of massive and recurrent submassive hemoptysis. Risks of bronchial artery embolization (BAE) are well described and include spinal ischemia and paralysis, most often related to nontarget embolization of the artery of Adamkiewicz or other large radiculomedullary artery supplying the anterior spinal artery. There is increasing literature regarding spinal infarction following BAE when arterial supply to the spinal cord was not evident. The existence of unrecognized patient comorbidities may further contribute to procedural risks.

Case Report

A 45-year-old female with stage IV adenocarcinoma of the lung was referred to interventional radiology (IR) clinic for bronchial artery embolization (BAE) for recurrent hemoptysis. One month earlier, she underwent bronchoscopy by interventional pulmonology (IP) for the treatment of her hemoptysis, with argon plasma coagulation and flexible cryoprobe for tissue and clot extraction and hemostasis with recanalization of the left mainstem bronchus. She experienced clinical improvement for 2 weeks, at which time her hemoptysis recurred, although less severe.

She was seen in IR clinic where she reported increasing daily productive cough with blood tinged sputum; however, the symptoms were inconsistent with massive hemoptysis. Her most recent CT of the chest was notable for extensive mediastinal and hilar lymphadenopathy and a 5.9 × 4.6 × 4.8 cm left infrahilar pulmonary mass without bronchial artery enlargement. After discussion of risks and benefits of the procedure, the patient deferred treatment, pending worsening of hemoptysis.

Six weeks later, her hemoptysis worsened to daily and submassive, with recurrent anemia, and radiographic progression of disease, including enlargement of the left infra-

hilar mass and complete obstruction of the left mainstem bronchus. The patient wished to proceed with a joint IP and IR procedure under general anesthesia.

The patient was induced and intubated by the anesthesiologist without incident. The interventional pulmonologist performed bronchoscopy, tumor debulking, and left mainstem bronchus recanalization with a core catheter monopolar radiofrequency device. Right common femoral artery access was obtained with ultrasound guidance. A thoracic aortogram was performed followed by selective catheterization of multiple descending thoracic aorta branches using a 5-Fr Mikaelson catheter (AngioDynamics, Latham, NY). A single right bronchial-intercostal arterial trunk supplying the right lung was identified, without evidence of tumoral arterial supply. Bronchial arterial supply from the aorta to the left lung was not identified. Several left intercostal arteries with bronchial communications were identified, supplying tumor and surrounding lung from left T4, T5, and T6–T7 intercostal arterial trunk.

Selective digital subtraction angiography (DSA) via the 5-Fr catheter positioned in the ostium was sequentially performed and images reviewed. DSA performed at T5 demonstrated pulmonary parenchymal staining and focal tumor staining (►**Fig. 1a**). A 2.4-Fr microcatheter (Progreat; Terumo Medical
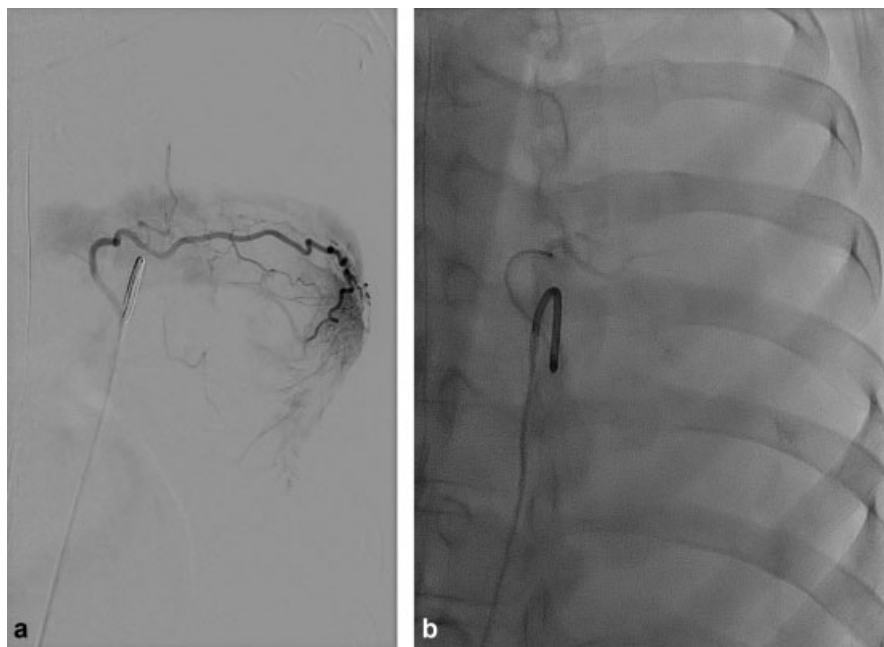
Fig. 1 T5 intercostal artery angiography: (**a**) tumor staining proximally and distal pulmonary parenchymal perfusion; (**b**) postembolization stasis with resolution of tumor staining and distal perfusion.

Corporation, Somerset, NJ) was then coaxially advanced further into left T5 intercostal artery and embolization was performed with slow, small aliquot injections of 300 to 500 μm polyvinyl alcohol (PVA) particles (Cook Medical, Bloomington, IN) with intermittent angiography to assess flow. After reaching embolization endpoint (►**Fig. 1b**), the microcatheter was flushed with normal saline and removed, followed by double flush of the 5-Fr catheter. This process was repeated with coaxial technique at the left T6–T7 intercostal artery trunk, after DSA demonstration of tumor staining and bronchopulmonary shunting (►**Fig. 2a–d**), and at the left T4 intercostal artery, where angiography demonstrated bronchopulmonary shunting (►**Fig. 3a, b**). Neither the artery of Adamkiewicz nor other anterior spinal artery (ASA) supply was identified. Hemostasis of the right common femoral artery was obtained with an arterial closure device (StarClose; Abbott Vascular, Santa Clara, CA). The interventional pulmonologist then returned to perform a postembolization diagnostic bronchoscopy, which revealed a patent left-side tracheobronchial tree up to the subsegmental level with no overt bleeding visualized. Patient was extubated and transferred to the recovery area.

Approximately 2 hours later, after awakening in the recovery area, the patient complained of not being able to move her legs, but was found to have intact sensation and rectal tone. MRI of the thoracic and lumbar spine was obtained approximately 4 hours after the end of the procedure, showing no abnormal thoracic or lumbar cord signal to suggest infarction. Neurosurgery was consulted. The patient was transferred to the ICU, where a lumbar drain was placed for perfusion. Intravenous steroids and strict blood pressure control were initiated. Two days later, the patient had persistent flaccid paralysis of her legs and a repeat MRI of the entire spine was performed. MRI was notable for interval development of edema along the anterior horns of the spinal

cord from C5–C6 to T4–T5 (►**Fig. 4a–c**), consistent with a cord infarction. Of note, a disc osteophyte complex compressing the thecal sac was present at C6–C7 (►**Fig. 4a**). Her neurological exam was notable for a sensory level at the umbilicus, corresponding to around T4–T5, flaccid paralysis in the lower extremities, and subjective paresthesia of the left hand which she reported unchanged from preprocedure. No motor abnormality of the upper extremities was present. The patient spent 1 week in the hospital following the procedure and was then discharged to a skilled nursing facility with persistent lower extremity paralysis and resolution of hemoptysis.

Anatomy

The ASA is formed by anterior spinal branches arising from the vertebral arteries at the level of the foramen magnum.[1,2] The ASA varies in caliber through its course, and is largest in the lumbosacral region and smallest in the thoracic region, where it is considered vulnerable to ischemia. Each level of the spinal column contains a radicular artery which supplies the nerve roots and the dura.[3] At some levels, the radicular artery is enlarged and maintains its embryonic connection to the ASA, and is then called a radiculomedullary artery. However, due to embryologic regression, most spinal segmental arteries do not supply the spinal cord itself. Rather than being a single, continuous vessel, the ASA is made of multiple anastomotic networks but can be discontinuous.[4] The implication is that if a large radiculomedullary vessel is occluded, adequate collateral flow from the more cranial portions of the ASA cannot be counted on to supply sufficient blood flow.[4] When a segmental vessel is occluded, the greatest damage is located at the level of entrance of the medullary artery, but changes are seen both above and below the cord.[4]
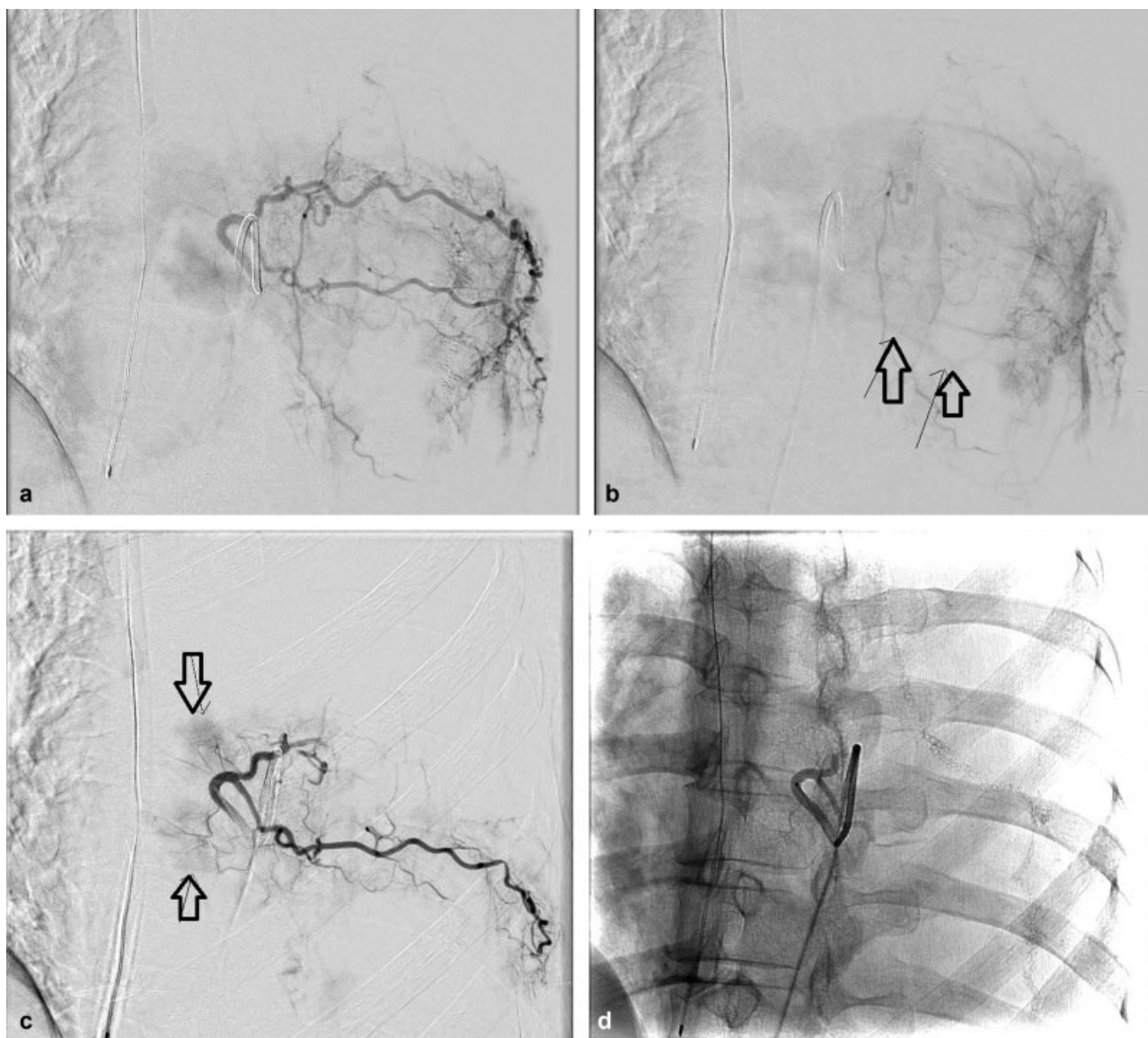
Fig. 2 T6–T7 intercostal trunk angiography: (**a**) pulmonary perfusion; (**b**) pulmonary venous return (*arrows*); (**c**) angiography after partial embolization demonstrates increased tumor blush (*arrows*); (**d**) postembolization stasis with resolution of tumor staining, pulmonary perfusion, and pulmonary venous return.

Physical exam reveals weakness below the level of occlusion, and loss of pain and temperature at or below the level of occlusion.[5]

At the cervical level, the origin of supplementary arterial supply to the ASA is variable, but is generally believed to arise above the aortic arch from either the vertebral or subclavian arteries.[3] In the thoracic spine, the spinal segmental arteries arise from the intercostal arteries.[3] When present, radiculo-medullary arterial feeder branches to the ASA supply the spinal cord below the point of entry.[2] It is reported that the spinal cord is supplied by, on average, seven or eight anterior radiculomedullary arteries, which vary in the levels from which they arise, and that radiculomedullary arteries do not frequently occur between C8 and T2.[3] The larger, more significant radiculomedullary arteries are angiographically identifiable and have been reported to vary in size from 340 to 1,122 μm.[4] The largest and most well-known radiculo-medullary artery is the artery of Adamkiewicz, also known as the great anterior radiculomedullary artery (GARMA), which supplies the lower two-thirds of the spinal cord. The GARMA originates between T9 and T12 in 75%, between T5 and T8 in 15%, and between L1 and L2 in 10% of the population and measures on average 872 μm in adults.[4] It is on the left in 70 to 80% of people.[3] Like other radiculomedullary arteries, it has an angiographic appearance classically described as a hairpin turn with subsequent communication with the ASA which courses along the midline of the spine.

Discussion

Owing to the development of lower extremity paralysis within the immediate postprocedural period, nontarget embolization of an occult radiculomedullary artery, specifically the artery of Adamkiewicz, was suspected, although no
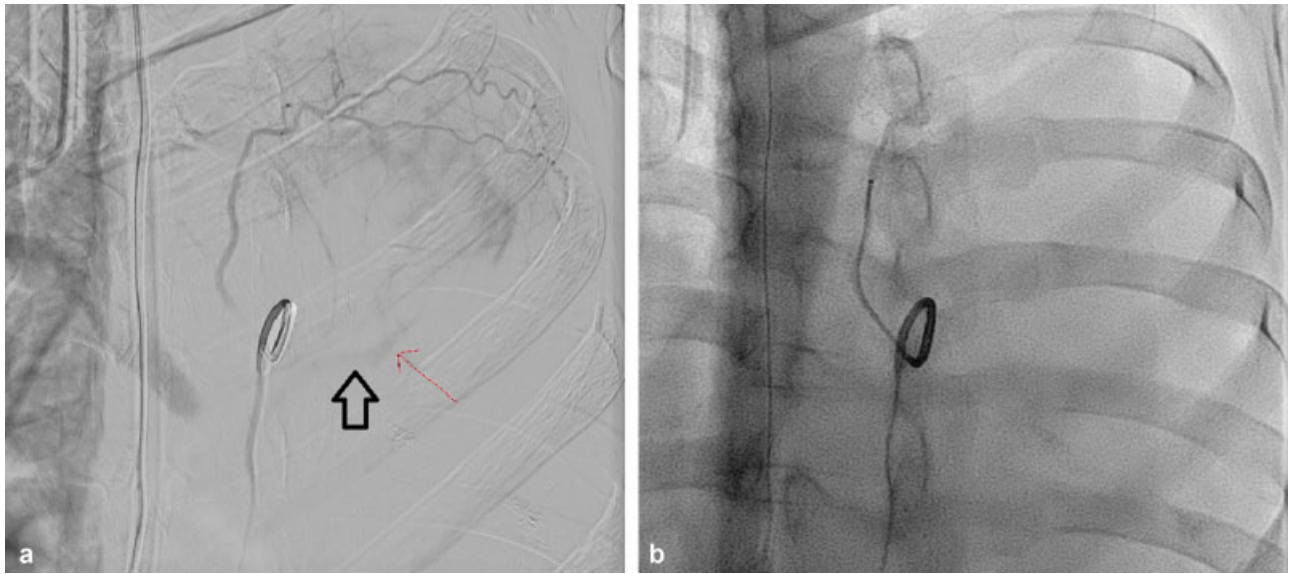
Fig. 3 T4 intercostal artery angiography: (**a**) pulmonary parenchymal perfusion and shunting to the pulmonary vein (*arrow*); (**b**) postembolization stasis with resolution of pulmonary perfusion and shunting.
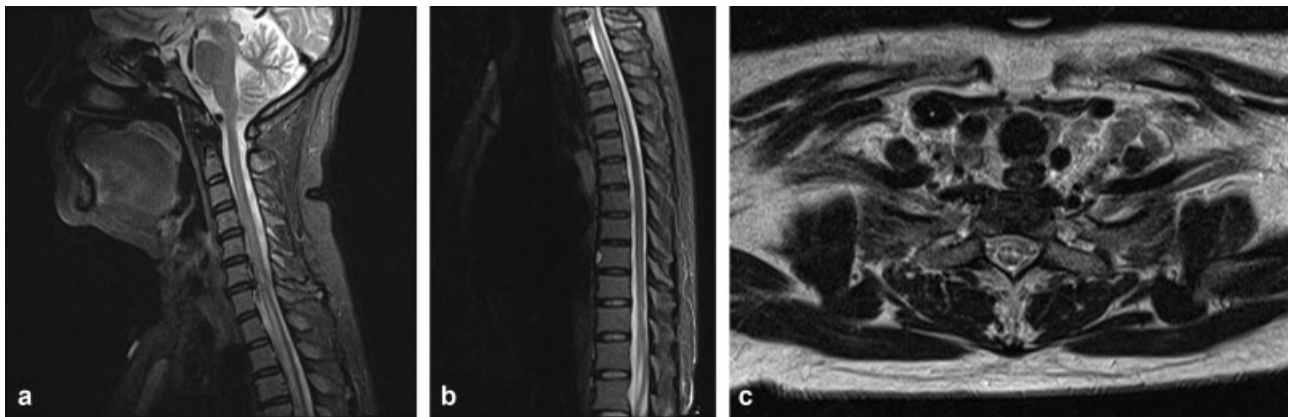

Fig. 4 MRI spine: (**a**) sagittal cervical STIR image demonstrates large disc–osteophyte complex at C6–C7 with high-intensity cord signal abnormality from C5 to C6 to the thoracic spine; (**b**) sagittal thoracic STIR image demonstrates high-intensity cord signal abnormality from cervical spine to T4–T5; (**c**) axial T2-weighted image at C7 demonstrates increased signal intensity within the anterior horns.

radiculomedullary artery was identified on careful review of images during or after the procedure. The subsequent MRI findings were more complex. Cord signal abnormality unexpectedly extended from C5–C6 to T4–T5, with a large disc osteophyte complex at C6–C7. Normal cord signal was present throughout the majority of the thoracic cord and the entirety of the lumber spinal cord.

The highest level of intervention was T4. As radiculomedullary arterial branches to the ASA supply the spinal cord below the point of entry,[2] nontarget embolization from the T4 intercostal artery through an occult radiculomedullary artery to the ASA would be expected to result in cord signal abnormality below that level as well as above, but not asymmetrically above that level.

Could the Disc Osteophyte Complex at C5–C6 Have Played a Role?

The identification of a disc osteophyte complex at C6–C7 compressing the thecal sac just below the most cephalad

level of edema at C5–C6 on postprocedural MRI was unexpected and raises the question of whether the disc osteophyte complex contributed to cord infarction. Unfortunately, no preprocedural cervical spine imaging was available for this patient for comparison. Preexisting spinal stenosis could have contributed to spinal cord injury due to narrower spinal canal cross-sectional area. Median disc protrusions causing vascular compression resulting in an acute ASA syndrome have been reported.[6] In the reported cases, there was no evidence of preexisting cord compression, and the ischemic symptoms developed after an inciting movement.[6] Development of paraplegia secondary to an acute cervical disc herniation in a patient with unrecognized cervical spine posterior disc disease after anesthesia has been reported.[7] Theoretical mechanisms were felt to be loss of supporting muscle tone protecting the spine due to pharmacologic paralysis during anesthesia, neck hyperextension during intubation and positioning, and bucking and agitation.[7] The patient suffered paraplegia with retained use of the

upper extremities despite cervical disc herniation, as in our case. Moreover, as with our patient, preexisting intermittent numbness in an upper extremity was only identified retrospectively.

Despite intubation and bronchoscopic intervention, exaggerated movements of the cervical spine were not believed to have occurred during the combined IP and IR case. The patient had been intubated without incident for multiple procedures and surgery in the previous 2 years, including rigid bronchoscopy which can require significant neck extension and was not used in the combined IP–IR case. Airway assessment revealed full view of the glottis and intubation was easily performed with standard equipment and minimal neck extension. The patient remained supine, and the neck remained neutral for the remainder of the case. Extubation was uneventful without excessive coughing or bucking.

Hypothetically, disc osteophyte compression of the cord, and the ASA, could have resulted in reversal of arterial flow. While the arterial supply to the cord is cranial-caudal, and radiculomedullary arterial supply to the ASA perfuses the cord below the point of entry, it is proposed that compression of the ASA at the C6–C7 level could have resulted in retrograde flow cephalad. Such reversal of arterial flow could account for the exclusive presence of cord edema at and above the highest level of arterial intervention if the cause of cord infarction was nontarget arterial embolization of the ASA via an occult radiculomedullary artery arising from an intercostal artery.

Did Embolization for Malignant Hemoptysis Increase Procedure Risk?

BAE was historically described for the treatment of benign conditions such as cystic fibrosis and tuberculosis.[8] Bronchial and intercostal arteries are most frequently interrogated and treated for massive hemoptysis. Enlarged and tortuous bronchial arteries are classically described imaging characteristics for benign etiologies of massive hemoptysis, but in malignant causes these classic findings range from 32.5 to 72% only.[8–11] BAE has a reported incidence of spinal artery ischemia between 1.4 and 6.5%.[1] In this patient, direct bronchial artery originating from the aorta to the left lung was not present, but collateral flow from multiple intercostal arteries to arteries supplying tumor and lung parenchyma was present. While both tumor blush and systemic arterial to pulmonary venous shunting were identified, the arteries supplying the lung mass were not hypertrophied.

It is logical that embolization of non-hypertrophied arteries would lead to quicker stasis and earlier redirection of blood flow. Wang and colleagues reported one case of spinal cord infarction in their series of 30 patients undergoing BAE for hemoptysis of malignant origin.[8] In that case, T3 and T4 intercostal arteries were embolized with 100 to 300 μm Embospheres (Biosphere Medical, Rockland, MA). The spinal artery was not demonstrated angiographically. It was not specified whether arterial enlargement was present in that patient. Postprocedural MRI demonstrated hyperintense signal in the cord at T4. Chen et al compared BAE for malignant versus benign etiologies and found no significant difference between the two in terms of complication rate.[9]

Could Hypoperfusion Have Played a Role?

In addition to mechanical insult, arterial insufficiency is a known mechanism of cord infarction.[4] The mean arterial pressure (MAP) is a factor in spinal cord perfusion, and the average MAP ranges from 70 to 100 mm Hg. Most patients who experience arterial hypotension, even for prolonged durations, do not develop spinal cord injury.[10] It has been proposed that the small percentage of patients who suffer neurologic injury from arterial hypotension have less physiologic reserve and require higher arterial pressures to maintain spinal autoregulation.[10] In this case, MAP was only below normal for a short interval during the initial bronchoscopic portion of the procedure and was otherwise maintained at or above normal. It is unlikely that hypoperfusion contributed to infarction of the spinal cord.

Conclusions

While nontarget embolization of an undetected thoracic radiculomedullary artery seems probable in this unfortunate case, it does not explain the asymmetric cord insult involving the cervical spine and the upper thoracic spine. We hypothesize that the cervical disc osteophyte complex contributed in some manner to the infarction, despite uneventful intubation and procedural positioning, whether by direct compression and infarction or by ASA compression, resulting in retrograde flow to the cervical spine. This case differs from our standard approach to BAE in that it was performed under general anesthesia and in conjunction with endobronchial intervention. It is impossible to know whether the patient's outcome would have differed had the BAE been performed alone under moderate sedation. The risks of BAE, including paralysis, stroke, and death, were carefully reviewed with the patient both in clinic when the procedure was deferred and upon her return when she wished to proceed. The occurrence of spinal infarction despite standard precautions and procedural technique and the unexpected finding of significant cervical spinal stenosis with adjacent cord injury demonstrate the importance of thorough patient counseling prior to any intervention, as every intervention carries known, and potentially unforeseeable, risks.

Disclosures
The authors report no relevant financial disclosures.

Conflict of Interest
None.

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