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Diffuse Pulmonary Meningotheliomatosis Diagnosed Via Transbronchial Cryobiopsy

To the Editor:

A 61-year-old woman with a prior history of extensive cigarette smoking and vaping had been followed for 3 years due to the presence of diffuse upper lobe predominant centrilobular and perifissural micronodules which had been stable on computed tomography (CT) imaging for the last 3 years (Figs. 1A, B). However, the recent development of a nonproductive cough with nonexertional dyspnea and an isolated decline in diffusing capacity for carbon monoxide on pulmonary function testing prompted a concern for progression of an underlying interstitial lung disease. Transbronchial cryobiopsy was chosen as the diagnostic procedure given clinical suspicion for hypersensitivity pneumonitis and concerns with obtaining an adequate diagnostic sample via traditional forceps biopsy.

The patient underwent rigid bronchoscopy, with cryobiopsy performed twice in the right upper lobe (RUL) and twice in the right lower lobe (RLL), under fluoroscopic guidance and with an in-situ occlusion balloon. Freezing time was 6 seconds using a 1.9 mm cryoprobe. Bleeding was minimal and no pneumothorax occurred. Cytology and cultures from a bronchoalveolar lavage of the left upper lobe was unremarkable. Cryobiopsy specimens had an average cross-sectional area of 51 mm^2. The cryobiopsy specimens in the RUL were notable for 3 separate foci of pulmonary meningothelial-like nodules (Figs. 1C–E). Specimens from the RLL were unremarkable. Immunohistochemical stains showed the lesional cells to be positive for progesterone receptor and epithelial membrane antigen, and negative for pan-cytokeratin, synaptophysin, smooth muscle actin and HMB-45. The remainder of the RUL and RLL specimens demonstrated normal alveolar tissue. The imaging findings and pathology were consistent with a diagnosis of diffuse pulmonary meningotheliomatosis (DPM). The patient was counseled on the benign nature of DPM, with plans to undergo further workup of her abnormal diffusing capacity, including evaluation for pulmonary vascular disease.

Minute pulmonary meningothelial-like nodules (MPMNs) were first described as chemodectomas (with features akin to the chemoreceptor cells of the carotid body)[1] before subsequent studies demonstrated immunohistochemical features consistent with cells of meningothelial origin.[2] MPMNs are small (100μm to 5 mm), randomly distributed lesions (though with a peripheral tendency) and can very occasionally be cystic; although an upper lobe predominance has been reported as in our case, this is not universal.[2,3] The differential diagnosis of the imaging findings in DPM includes miliary tuberculosis, diffuse micrometastatic disease, sarcoidosis, amyloidosis, and hypersensitivity pneumonitis; however unlike in DPM, these other diagnoses do not remain stable during longterm followup imaging. On pathology, MPMNs do not appear to be distributed in an airway- or vessel-centric pattern, and stain positive for epithelial membrane antigen, vimentin and progesterone receptor.[4] MPMNs have a female predilection, are most commonly discovered in the sixth decade of life, and are not associated with smoking. The etiology of these lesions remains unclear; they appear to be a reactive proliferative process possibly from minute vascular occlusion, with associated conditions including pulmonary thromboembolism[5] and metastatic malignancy.[6] MPMNs are considered benign, although genetic evaluation of micronodules from DPM suggest that polyclonal variation in these lesions could represent a transitional phase to neoplastic proliferation,[7] and a single case report notes the incidence of a primary pulmonary meningioma associated with MPMNs in the same pathologic specimen.[8]

As they are a common incidental finding in lung tissue (13.8% in surgical lung biopsy specimens resected for other reasons),[4] MPMNs are considered an entity of little clinical significance; however in DPM, these nodules present

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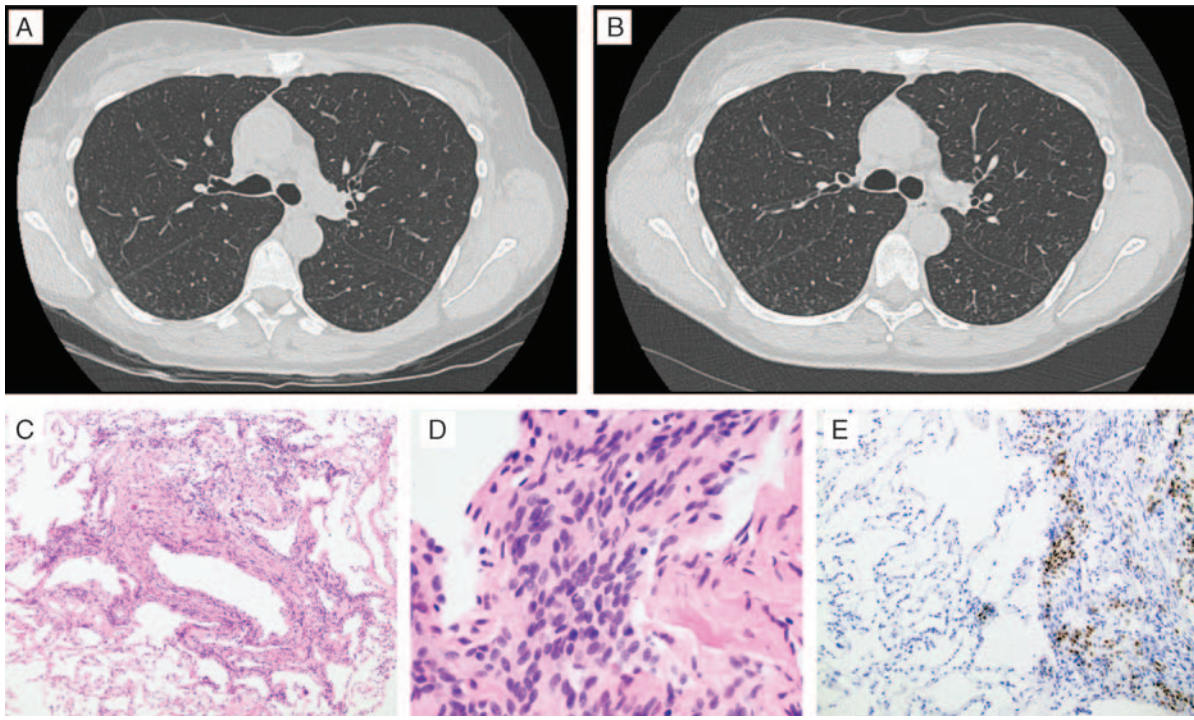

FIGURE 1. Computed tomography demonstrated stable diffuse micronodular disease, 3 years prior (A) and at time of referral for biopsy (B). Histopathologic evaluation of the upper lobe transbronchial cryobiopsy specimens demonstrated multiple parenchymal nodules measuring ~2 mm in diameter (C, H&E stain, ×100 original magnification). Higher power view showed the bland lesional cells to have round to oval nuclei, arranged in a nested pattern within the alveolar septa (D, H&E stain, ×600 original magnification). Immunohistochemical stains demonstrated positivity for progesterone receptor (E, ×200 original magnification) and epithelial membrane antigen, but were negative for pan-cytokeratin and synaptophysin, ruling out carcinoid tumorlets, and negative for smooth muscle actin and HMB-45, ruling out lymphangioleiomyomatosis (not shown).

diffusely throughout the lung tissue and may be associated with symptoms such as cough, dyspnea and exercise intolerance, as well as a restrictive deficit on pulmonary function testing.[9] A direct connection between symptoms and the pathology is often unclear (as in our case), and no specific treatment has been described. Given that their presentation may mimic more concerning disease processes such as lymphangitic carcinomatosis or diffuse granulomatous processes, lung biopsy is recommended. To our knowledge, this is the first case report of the diagnosis of DPM being made via transbronchial cryobiopsy. Multiple groups have demonstrated MPMNs on transbronchial forceps biopsy[10]; however as single MPMNs can be an incidental finding, the larger samples of intact, contiguous tissue obtained by cryobiopsy (50 mm^2, as compared with 3 to 5 mm^2 for traditional forceps biopsies) may offer an advantage over traditional forceps biopsy in confirming DPM and avoiding a non-diagnostic sample.

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An Unexpected Journey of a Loose Screw

To the Editor:

Airway perforation as a complication of an anterior cervical discectomy and fusion is extremely rare, with only one case reported in the literature.[1] Rare airway surgical complications highlight the opportunity for interdisciplinary collaboration between subspecialties. Although clinically practiced, sparse literature exists regarding multidisciplinary airway management of spine surgery complications. Here, we describe a case of an airway complication from a cervical fusion plate and the interdisciplinary approach that was used to successfully treat the tracheal perforation.

In this report, we present a case of a 34-year-old female with history of intravenous drug use who presented with sepsis from Methicillin-resistant *Staphylococcus aureus* bacteremia and extensive epidural abscess along the cervical, thoracic and lumbar spine. During her complex hospital course, she received antibiotics and underwent extensive surgical debridement, cervical intervertebral device placement, and anterior cervical fusion with cervical plate (Stryker) and screws. After discharge for rehabilitation, a chest radiograph was obtained due to persistent cough and a foreign body was found (Fig. 1A). Interventional pulmonology (IP) was consulted for foreign body removal. Because of the foreign body appearing to be a metal screw and the recent surgical history of the patient, a cervical spine and chest computed tomography (CT) scan was performed. The CT scans revealed concern for cervical spine instability (Fig. 1C), tracheal perforation (Figs. 1C, D) and a screw located in the right lower lobe. A multidisciplinary team of orthopedic surgery, IP and otorhinolaryngology (ENT) planned and participated in the operation. Based on multidisciplinary discussion, cervical spine stabilization was considered the first priority, which required a stable airway and endotracheal intubation. Because the extent of tracheal injury was unknown, IP and ENT collaborated to design multiple scenarios and treatment strategies for airway management. Due to cervical spine instability, rigid bronchoscopy requiring neck hyperextension was contraindicated. After laryngeal mask airway placement, flexible bronchoscopy (FB) visualized the tracheal perforation on the posterior membrane of the proximal trachea (Figs. 1E, F) without evidence of air leak and concluded a temporary airway stent was not required. The small size and proximal location of the injury allowed for FB-guided placement of endotracheal tube distal to the tracheal injury to avoid potential expansion of the tracheal defect with the inflated endotracheal tube cuff. The orthopedic team removed the cervical hardware and 3 remaining screws. ENT examination of the trachea after hardware removal found no air leak, but sternocleidomastoid flap was performed for tracheal perforation support (Fig. 1G). A halo vest was applied for temporary stabilization. IP retrieved the fourth screw using a 2.0 mm flexible jaw forceps through FB (Fig. 1B). The patient remained intubated for an additional 5 days on zero positive end-expiratory pressure. After 1 week in a halo-vest, the patient underwent a posterior cervical-thoracic fusion. Follow up FB showed appropriate tracheal healing. On 5-year follow-up, the patient is without respiratory/airway concerns.

To our knowledge this is the second case of an anterior cervical fusion plate system causing airway perforation[1] and the first to cause hardware migration to the lung. This case, although not requiring complex airway management strategies, highlights the need for multidisciplinary planning and intraoperative decision-making regarding tracheal perforation management. Cardillo and colleagues classified tracheal lacerations into 4 levels with specific management options. Level 1 injury involves the tracheal mucosa and submucosa. Level 2 involves the muscular wall and is associated with air leak. Level 3 is

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