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Postradiofrequency ablation inflammatory pseudotumor associated with pulmonary venoocclusive disease: case report and review of the literature

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ABSTRACT

Radiofrequency ablation of pulmonary veins is a common therapeutic intervention for atrial fibrillation. Pulmonary vein stenosis and venoocclusive disease are recognized complications, but the spectrum of pathologies postablation have not been previously reviewed. A recent case at our hospital showed a left hilar soft tissue mass in association with superior pulmonary vein stenosis in a patient 4 years postablation. On resection, this proved to be an inflammatory pseudotumor composed of myofibroblasts in an organizing pneumonia-type pattern with adjacent dendriform ossifications. Pulmonary venoocclusive change was a prominent feature. Literature on the histopathology of postradiofrequency ablation complications is limited. The severity of vascular pathology appears to increase with the postablation interval. Although pulmonary vascular changes are the most common late finding, fibroinflammatory changes including pulmonary pseudotumor formation, attributable to thermal injury, should be considered in the differential diagnosis of these cases.

1. Introduction

Radiofrequency ablation of pulmonary veins is an increasingly common therapeutic intervention for medically refractory atrial fibrillation that targets the proximal myocardial tissue beyond the pulmonary vein ostia [1]. Pulmonary vein stenosis and subsequent pulmonary venoocclusive disease are recognized but uncommon complications of this procedure [2]. Associated pulmonary inflammatory pseudotumor formation necessitating surgical resection has not been previously reported.

2. Materials and methods

Two surgical resections from our institution were reviewed by light microscopy, one of pseudotumor formation with associated pulmonary vein stenosis (current case). A literature search was performed for the following terms: "histology," "pathology," and "histopathology" each with "pulmonary vein radiofrequency ablation atrial fibrillation." Pathologic features of the Massachusetts General Hospital cases were compared with those in existing reports.

3. Results

3.1. Index case clinical history

A 66-year-old man with chronic obstructive pulmonary disease and hypertension had refractory atrial fibrillation that required radiofrequency ablation. The left upper, left middle, and right upper pulmonary veins were isolated and ablated. The procedure was complicated by hemopericardium and cardiac tamponade requiring emergency pericardiocentesis. He recovered and remained in sinus rhythm.

Four years later, he experienced the onset of hemoptysis that was small in volume but frequent, associated with a 40-lb unintentional weight loss over 2 months. Computed tomographic scan of the chest showed a 3.5-cm ill-defined left hilar soft tissue mass extending into the mediastinum and associated with stenosis of the left superior pulmonary vein, thickening of the left upper lobe bronchus, peribronchial lymph adenopathy, and ground glass opacities (Fig. 1). Multiple calcified pulmonary and splenic nodules suggestive of prior granulomatous disease were also identified. Positron emission tomography of the mass showed moderate signal uptake of indeterminate etiology, and ventilation-perfusion scintigraphy demonstrated marked V/Q mismatch to the left upper lobe.

New mild peripheral blood cytopenias were noted, and a diagnosis of immunoglobulin A multiple myeloma was established by bone

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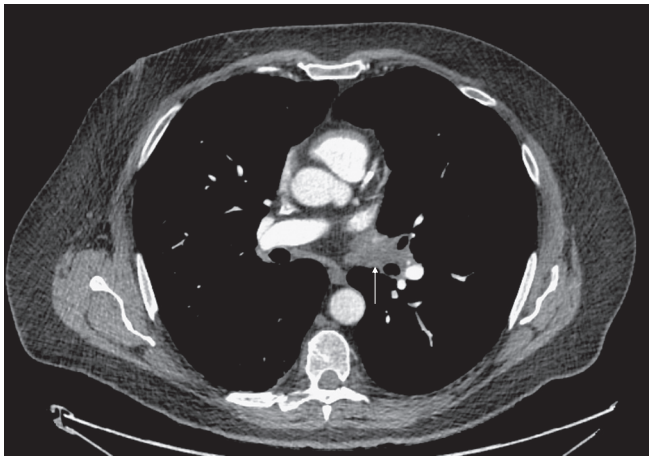

Fig. 1. Computed tomography (soft tissue window) demonstrates an ill-defined left hilar mass surrounding the left pulmonary vein (arrow).

marrow biopsy. Hemoptysis persisted, and treatment of the myeloma was deferred. Bronchoscopy showed diffuse vascularity of the left upper lobe bronchial mucosa. Endobronchial biopsy was nondiagnostic and complicated by bleeding. A decision was made to proceed to left upper lobectomy. On gross examination, an ill-defined 3.5-cm tumor was present adjacent to the hilum and surrounded by subcentimeter calcified nodules.

Histologic examination demonstrated an inflammatory pseudotumor (Fig. 2A) composed of myofibroblasts in an organizing pneumonia-type pattern (Fig. 2B) that were immunopositive for smooth muscle actin (Fig. 2B inset) and negative for anaplastic lymphoma kinase-1 (not shown). The adjacent lung showed multiple dendriform ossifications (Fig. 2C). A single microscopic focus of granulomatous inflammation with giant cells was identified, but no organisms were isolated (not shown). The surrounding lung showed extensive pulmonary venoocclusive disease with fibromuscular thickening and patchy obliteration of venules (Fig. 2D), paraseptal hemosiderosis, and grade 2 to 3/6 pulmonary hypertensive arteriopathy.

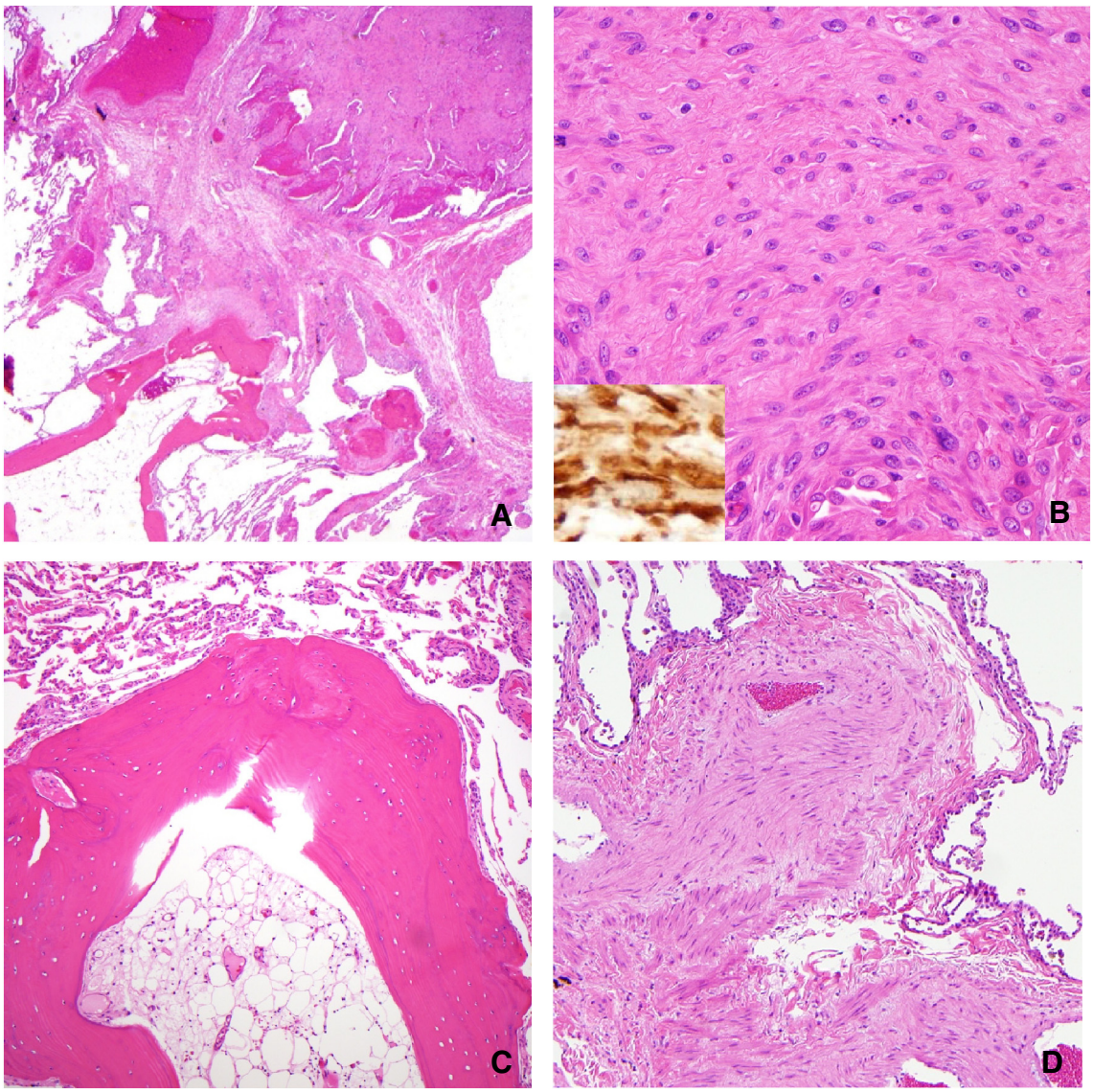

Fig. 2. Low-power view of mass lesion (A). Central dense proliferation of benign myofibroblasts in an organizing pneumonia pattern immunopositive for smooth muscle actin (inset) (B). Peripheral nodular heterotopic ossification (C). Fibromuscular thickening of numerous venules, a histologic correlate of venoocclusive disease (D).