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Case report

Mitral stenosis due to rheumatic heart disease - A rare cause of massive hemoptysis

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ABSTRACT

Severe mitral valve stenosis caused by rheumatic heart disease presenting initially as massive hemoptysis has become a rare occurrence in contemporary western medicine. Massive hemoptysis can be due to multiple disease processes including airway diseases such as bronchiectasis, pulmonary parenchymal disease of infectious or autoimmune etiology, pulmonary AVM's, hematologic disorders, and numerous drugs and iatrogenic injuries. It is less associated with congestion from rheumatic heart disease due to the earlier detection and subsequent management of cardiac valve disease preventing the sequela of more severe disease.

We describe a case of a 59 year-old woman with hemoptysis, who was found to have severe mitral stenosis consistent with rheumatic heart disease. We demonstrate the appearance of pulmonary venous congestion can be seen on bronchoscopic examination in severe mitral stenosis and discuss the significance of the Wilkins score to help guide management.

1. Introduction

While hemoptysis is a well-known and documented sequela of mitral stenosis (MS), its occurrence in contemporary western medicine has become rare [1,2]. Rheumatic heart disease (RHD) is the result of an exaggerated immune response to specific bacterial epitopes in a susceptible host [3]. Chronic RHD affecting the mitral valve apparatus progresses over years to decades and causes a number of pathologic changes, affecting the mitral valve apparatus, which are diagnostic for rheumatic valve disease [4,5]: fusion of the leaflet commissures; thickening, fibrosis, and calcification of the leaflet cusps [6]; and thickening, fusion, and shortening of the chordae tendineae.

According to the WHO, the estimated global prevalence of rheumatic heart disease (RHD) is 15.6 million cases. Of those identified cases, 79% originated in less developed countries, with an estimated prevalence in those countries of 2.5–3.2 cases per 1000 all-age population vs. 0.3 cases per 1000 population in developed countries [7]. The incidence rate of rheumatic fever is < or = 10/100,000 per year in America and Western Europe, while a higher incidence (> 10/100,000) was documented in Eastern Europe, Middle East (highest), Asia and Australasia [8]. The prevalence of Rheumatic Fever, RHD and their mortality rates varied widely between countries and between population groups within the same country; globally, about 2% of deaths from cardiovascular diseases are related to rheumatic heart disease [9].

Despite an apparent fall in incidence over time, Acute Rheumatic Fever (ARF) incidence rates remain relatively high in non-Western countries [8]. In developed countries, the progression of disease is more indolent and manifests at older ages (above 50 years); clinically detected RHD is most commonly diagnosed in individuals aged 20 to 50 years with nearly two-thirds of cases occurring in females [10]. Therefore, the incidence of hemoptysis as the presenting symptom of RHD without associated shortness of breath, chest pain or signs of hypervolemia is limited to case reports [1]. Hemoptysis as a separate entity occurs in around 10% of patients with chronic lung disease [11] and is found in ca. 0.1% of all outpatients [12] and almost 0.2% of all inpatients [13] each year.

The decline in hemoptysis as a presenting symptom of RHD is likely due to the dramatic decline in its prevalence in developed nations and the earlier detection, and subsequent management, of valvular disease preventing the sequela of more severe disease. The work up of hemoptysis is described along with the evaluation of MS.

2. Case report

A 59-year-old African American female with a history of pulmonary tuberculosis treated twenty-one years ago, active tobacco use, and untreated hepatitis C presented with three days of coughing up frank red blood. This was preceded by a month of viral-like upper respiratory

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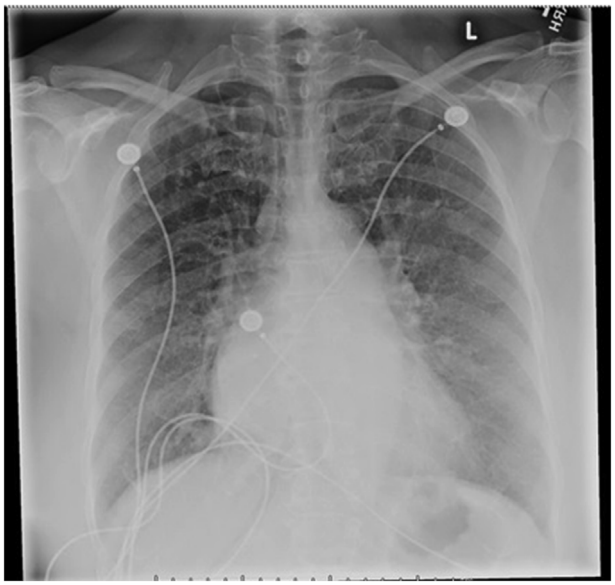

Fig. 1. Single view CXR revealing fibronodular changes in both apices without infiltrates or effusions.

symptoms, chills, unintentional weight loss and night sweats. She denied productive cough, lower extremity swelling, shortness of breath or chest pain. She was an active smoker of 0.25 packs per day for forty-five years (11.25 pack year smoking history) with additional alcohol use estimating four beers per day since she was a teenager. She denied any illicit drug use.

Upon presentation she was in no acute distress with normal mental status. Vitals on admission revealed a temperature of 98.2°F, heart Rate of 89 bpm, blood pressure of 144/80 mmHg, respiratory rate of 16 while saturating 96% breathing ambient air. There was a prominent S1, split S2 with prominent P2, and a decrescendo-crescendo diastolic murmur loudest at the apex. Pulmonary exam revealed bibasilar crackles. Her abdomen was soft, non-tender and without organomegaly. There was no cyanosis or clubbing of the extremities.

Complete blood count revealed: WBC of 6.2, Hb of 12.9, Hct of 40.1, PLT of 318. PT was 11.9 seconds, INR was 1.1. Hepatic panel was normal with an AST of 27 U/L, ALT of 23 U/L, Alk phos of 60 U/L & Total Bilirubin of 0.9 mg/dL.

A chest x-ray (CXR) (Fig. 1) revealed fibronodular changes in both apices without infiltrates or effusions. A chest computer tomography angiogram (CTA) (Fig. 2) revealed extensive ground glass opacities,

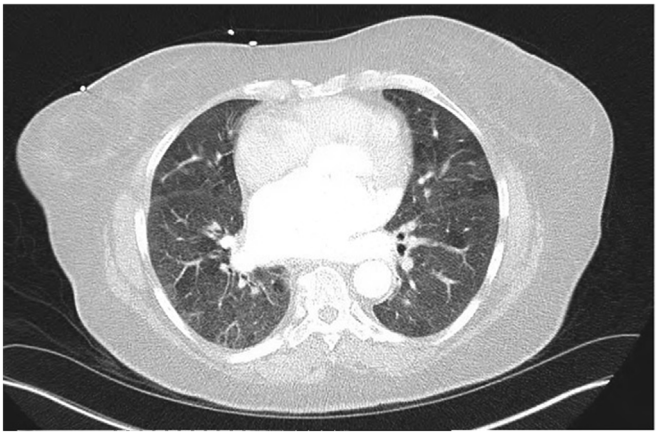

Fig. 2. CTA of the chest demonstrating extensive ground glass opacities with bilateral calcified pulmonary nodules most pronounced in the right upper lobe, and a dilated left atrium. No pulmonary embolism or AVM was seen.

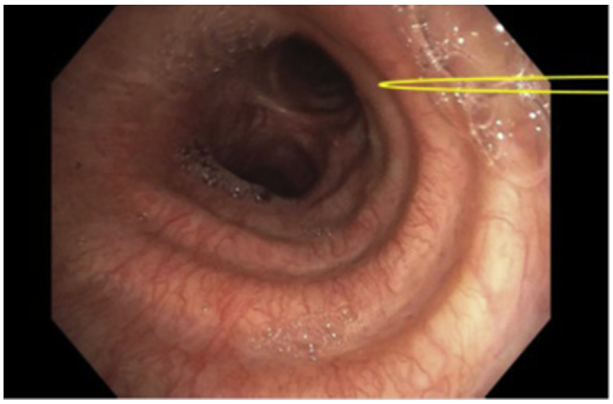

Fig. 3. Bronchoscopic view of the trachea demonstrating engorged veins in the trachea and proximal right and left bronchial trees.

bilateral calcified pulmonary nodules most pronounced in the right upper lobe, and a dilated left atrium. No pulmonary embolism or arteriovenous malformations (AVM) were seen.

A transthoracic echocardiogram (TTE) revealed a left ventricular ejection fraction of 35–45%, mild left ventricular hypertrophy, and severe mitral valve stenosis with moderate mitral valve regurgitation.

Sputum AFB smear, blood cultures, HIV, ANA, ANCA and anti-GBM antibodies were obtained and all negative. Rheumatoid factor was elevated. Bronchoscopy (Fig. 3) revealed engorged veins in the trachea and bilateral proximal bronchial trees.

Results of the clinical work-up made infection, malignancy, TB, vasculitis or AVM unlikely. The dilated left atrium and severe mitral stenosis seen on TTE was confirmed by transesophageal echocardiogram (TEE), which showed a mean gradient of 12 mmHg and Wilkins score of 11 (see Figs. 4–6). Left heart catheterization was performed and showed no evidence of coronary artery disease.

Given these findings, the patient's massive hemoptysis was attributed to severe mitral valve stenosis in the setting of rheumatic heart disease. Given her Wilkins score of 11, she was not a candidate for balloon valvuloplasty, and ultimately underwent successful mitral valve replacement with a pericardial bioprosthetic valve and resection of anterior and posterior papillary muscles.

3. Discussion

Hemoptysis carries a broad differential including airway diseases such as bronchiectasis, pulmonary parenchymal diseases of infectious or rheumatic cause, pulmonary AVM's, hematologic disorders, and numerous drugs and iatrogenic injuries [14]. Due to this plurality, a thorough history is critical to guide clinical decision making.

While hemoptysis is a well-known and documented sequela of mitral stenosis (MS), its occurrence in contemporary western medicine has become rare [2]. This is likely due to the dramatic decline in the prevalence of rheumatic heart disease in developed nations and the earlier detection, as well as subsequent management, of valvular disease preventing the sequela of more severe disease. Currently, rheumatic fever (RF) mostly affects children in developing countries, especially in areas of widespread poverty [15].

Common causes of severe mitral stenosis in developed countries include mitral annular calcification, radiation-associated valve disease, Fabry's disease, Whipple's disease, mucopolysaccharidosis, methysergide therapy, carcinoid valvular disease, endomyocardial fibrosis, and systemic autoimmune disease (such as systemic lupus erythematosus and rheumatoid arthritis) [16].

Other cardiac conditions may produce hemodynamic abnormalities similar to those of native valvular MS. These include atrial myxomas, large infected vegetations, ball valve thrombi, and degenerated stenotic bioprosthetic mitral valves [17].

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