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Case Reports

Hemoptysis Complicating Mitral Stenosis

Case report with attention to differential diagnosis and a review of the literature

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The differential diagnosis of hemoptysis in patients with mitral stenosis includes many primary pulmonary problems besides those peculiar to mitral stenosis. We describe the case of a 40-year-old woman with a 40-pack-year smoking history whose presenting symptom of hemoptysis was wrongly ascribed to pulmonary embolus and infarction, based on a presumably positive angiogram, and to chronic bronchitis, based on endoscopic findings at fiberoptic bronchoscopy, as provided by the referring physician. The features differentiating between cardiac and primary pulmonary disease are described, and the pathophysiology of hemoptysis in mitral stenosis is reviewed. In a case like the one we describe, hemoptysis is an important manifestation of severe valvular stenosis, and surgical intervention, i.e., mitral commissurotomy or mitral valve replacement, should be considered.

Though hemoptysis may result from many pulmonary and systemic diseases with pulmonary manifestations, severe hemoptysis usually results from intrinsic pulmonary disorders. An exception to this rule is pulmonary hemorrhage accompanying mitral stenosis. We report a case of mitral stenosis complicated by severe hemoptysis in which the significance of the association with mitral stenosis was overlooked and primary pulmonary disease was wrongly implicated initially as an etiologic factor.

Case Report

A 40-year-old white woman was transferred to Henry Ford Hospital for evaluation of hemoptysis. She had been well until one year before she was hospitalized, when a productive morning cough, dyspnea on exertion, and occasional paroxysmal nocturnal dyspnea developed. The symptoms progressed to the point that the patient was unable to care for her home without becoming dyspneic. Aside from a 40-pack-year smoking history, the remainder of the past medical history and review of systems was unremarkable.

Two months before she was admitted, the patient had been evaluated at a local hospital for hemoptysis. At that time, she had coughed approximately 100 cc of bright red blood. The chest x-ray examination was normal. A pulmonary angiogram reportedly showed a filling defect in the right lower lobe, and the patient was anticoagulated. Persistent hemoptysis sufficient to decrease the hemoglobin from 11 to 8 gm.% continued over the next several days. Fiberoptic bronchoscopy revealed changes of severe hemorrhagic bronchitis felt compatible with the patient's smoking history, and intensive bronchodilator therapy, including corticosteroids, was initiated.

When hemoptysis continued, anticoagulation was stopped because the risks of asphyxiation were felt to outweigh the risks of persistent or recurrent embolization. Auscultatory findings at the time of the first admission were compatible with mitral stenosis and confirmed by echocardiography. Both the referring physician and his consulting cardiologist felt that the association of hemoptysis and mitral stenosis was not significant because the positive pulmonary angiogram and the endoscopic features seemed to implicate primary pulmonary disease as the etiology. Hemoptysis gradually subsided, and the patient was discharged on bronchodilator therapy, but her dyspnea on exertion persisted. There was no recur-

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rence of hemoptysis until one day before the second admission when she coughed up approximately 90 cc of bright red blood. However, there were no other symptoms to suggest cardiopulmonary decompensation. After her condition had been stabilized with intravenous fluids, she was transferred to Henry Ford Hospital.

On arrival, the blood pressure was 92/52, the pulse 120/min, and the respiratory rate 24/min. There was minimal neck vein distention without hepatojugular reflux and no pedal edema. Bibasilar crackles were present. The point of maximal impulse was not displaced, and a right ventricular heave was present. The first heart sound was increased, as was the pulmonic second sound. An opening snap was heard close to the pulmonic second sound. There was I/VI systolic murmur best appreciated at the apex. The remainder of the physical examination was unremarkable.

After fluid administration, the blood pressure rose to 110/60, but the patient continued to cough up bright red blood. The chest roentgenogram showed some hilar prominence, but a normal cardiac silhouette and clear lung fluids (Fig. 1). The electrocardiogram showed a regular sinus rhythm, left atrial enlargement, and an incomplete right bundle branch block. The hemoglobin was 10 gm.%.

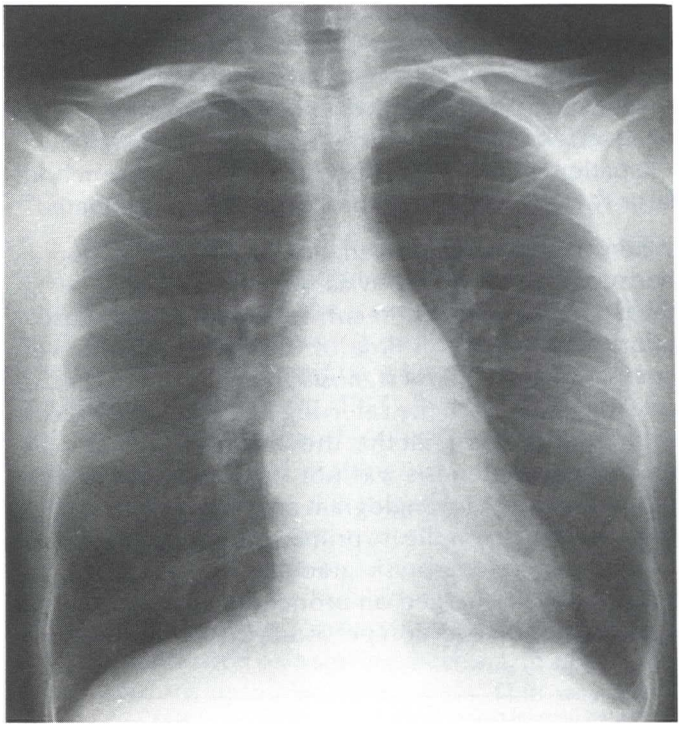

Fig. 1

Chest roentgenogram on admission revealing only slight hilar prominence bilaterally.

Although the degree of hemoptysis was considered somewhat excessive for pulmonary embolus and infarction, the history of a recent diagnostic angiogram for such, which had been inadequately treated, forced consideration of this diagnosis. Pulmonary angiography done shortly after admission revealed no evidence of embolus, and a subsequent review of the outside angiogram did not support the initial diagnosis of embolism either.

Treatment with bronchodilators and diuretics was started. Pulmonary function testing revealed a mild obstructive ventilatory defect ($FEV_1/FVC\%$ was 68%) with normal lung volumes and diffusion constant. Echocardiography revealed classical mitral stenosis, moderate left atrial (45 mm) and right ventricular (32 mm) dilatation, and findings supportive of pulmonary hypertension. With diuresis, hemoptysis ceased.

Because of the abnormalities appreciated at the referring institution, fiberoptic bronchoscopy was performed and revealed a prominent bronchial venous pattern. The interposed mucosa was not erythematous but only edematous. No focal bleeding sites could be identified.

Cardiac catheterization done on the third hospital day revealed a tightly stenotic mitral valve with a valve area of 0.7 cm^2 and a mean gradient of 17 mm Hg across the valve. Minimal mitral regurgitation was present. The right ventricular pressure was 65/3 mm Hg and the pulmonary artery pressure was 65/30 mm Hg, with a mean of 43 mm Hg. The cardiac index was normal, as were the coronary arteries.

Based on these findings, surgical intervention was indicated. At thoracotomy, significant deformity of the mitral valve was noted with extensive fibrosis extending into the papillary muscles. Commissurotomy was not considered technically feasible, and mitral valve replacement with a porcine valve was completed uneventfully.

Aside from a transient left pleural effusion, which cleared with diuresis, the patient's postoperative course was uneventful. Two months after discharge, she was asymptomatic even with exertion, and there had been no recurrent hemoptysis. While diuretics had been previously discontinued, theophylline was maintained because repeat spirometry continued to show mild airflow obstruction, though the $FEV_1/FVC\%$ had increased to 75% of predicted.

Discussion

The differential diagnosis of hemoptysis in patients with mitral stenosis obviously includes many primary pulmonary problems that may present with this symptom in addition to those peculiar to mitral stenosis. In the pres-

ent case, hemoptysis was wrongly ascribed to pulmonary embolus and infarction, based on a presumably positive angiogram, and to chronic bronchitis based on endoscopic findings at fiberoptic bronchoscopy by the referring physicians.

Despite this corroborating evidence, the degree of hemoptysis was unusual for either condition. While hemoptysis may occur with pulmonary infarction or pulmonary embolism with impaired bronchial venous flow, it would not be expected to be as extensive as it was in the present case. Similarly, while some parenchymal involvement on chest roentgenogram would have been expected with this degree of symptomatology, it was absent. Unfortunately, repeat pulmonary angiography was deemed necessary in this case despite the evidence implicating mitral stenosis as the cause of hemoptysis, because of the report of angiographically documented pulmonary embolic disease which had been inadequately treated. The degree of hemoptysis was also unusually brisk for bronchitis, particularly given the absence of factors suggesting an infective exacerbation and the slow response to intensive therapy at the referring hospital. Most cases of massive hemoptysis caused by primary pulmonary disease are due to active or inactive tuberculosis, lung abscess, carcinoma, or bronchiectasis; these diagnoses can usually be established by the chest roentgenogram and clinical history (1). These considerations alone increased the certainty that mitral stenosis resulted in the hemoptysis that was seen.

Wood, in his classic description of the features of mitral stenosis (2), distinguished five causes of hemoptysis that may complicate the course of the disease: pulmonary infarction, pulmonary edema, winter bronchitis, congestive hemoptysis, and pulmonary apoplexy. Pulmonary infarction occurred in 8.5% of 300 cases and was thought to result secondary to deep venous thrombosis in the legs, which, in turn resulted from low cardiac output and protracted bed rest. Infarction was considered to be more likely in patients with high pulmonary vascular resistance. Pink frothy sputum as a manifestation of pulmonary edema occurred in 6.5% of the cases, usually developing early in the course of the disease before increased pulmonary resistance developed. As pulmonary vascular pressures rose, alterations in cardiac output produced less dramatic alterations in pulmonary pressures so that hydrostatic edema formation was no longer favored.

Winter bronchitis was found in roughly one quarter of cases and was attributed to chronic pulmonary venous congestion. There was, however, no correlation between the incidence of winter bronchitis and the occurrence of symptoms of pulmonary venous congestion, left atrial pressure or size, or oxygen saturation. Nevertheless, mitral commissurotomy ameliorated bronchitis in most cases.

Congestive hemoptysis was present in 16.5% of cases. Though not clearly defined by Wood, this syndrome probably developed in a similar manner as pulmonary edema (i.e., pulmonary venous hypertension), but to a lesser degree. Dyspnea was characterized as severe, while the hemoptysis was limited to blood staining or streaking.

Pulmonary apoplexy, on the other hand, was defined as sudden, profuse hemoptysis often antedating the onset of dyspnea. Present in 18.3% of cases, it was the first manifestation of mitral stenosis in 42.5% of cases in which it occurred. Apoplexy developed within one year of effort-related dyspnea in another 12.8%. It was recurrent in 58%, often precipitated by pregnancy, severe exertion, or episodes of acute rheumatic fever. It frequently developed in patients with mixed stenosis and regurgitation.

Wood ascribed apoplexy to sudden increases in pulmonary venous pressure before pulmonary hypertension was present. The development of pulmonary hypertension was felt to protect against profuse hemorrhage by damping the degree of pressure rise within the pulmonary vascular tree. Wood stated that when this type of hemoptysis occurred in the presence of pulmonary hypertension, it always occurred at least three years before the measurement of increased pressure, suggesting its development at a time when pressures were lower.

Though often an early symptom of mitral stenosis, the degree of stenosis is usually severe before apoplexy develops. Earlier explanations attributed the hemoptysis to pulmonary arterial and venous hypertension and congestion with vascular rupture (3). Others felt sudden dilatation of pulmonary capillaries led to diapedesis of erythrocytes into alveoli (4). In 1944, however, Ferguson, et al (3) demonstrated varices of the bronchial veins in cases of mitral stenosis and felt that these were responsible for the hemoptysis seen. This view was also supported by the autopsy studies of Gilroy in 1952 (5). Bronchial veins dilate in response to the volume load experienced within the pulmonary circulation and thus serve to decompress the pulmonary vascular tree. These authors also felt that the "stiff lung" or "Lungenstarre" seen at surgery for mitral stenosis was due to hydrostatically-induced interstitial edema from bronchial venous engorgement.

Wood considered pulmonary apoplexy to be a benign, self-limited condition, with hemorrhage serving to decom-

press the bronchial venous system (1). Other authors have pointed out that deaths (usually due to asphyxia) may result from pulmonary hemorrhage, and several such cases were actually described before Wood's report appeared (4,6,7).

Reports of mitral commissurotomy done for severe, life-threatening recurrent hemoptysis first appeared in the late 1950s (8-10). These reports uniformly documented a cessation of hemoptysis following the procedure. Similar results have also been seen after mitral valve replacement (11,12). Braunwald has pointed out that successful mitral valve replacement will result in a consistent decrease in pulmonary vascular resistance, despite an increase in pulmonary blood flow (13). Greater preoperative resistances usually predicted a greater postoperative benefit. After closed commissurotomy, pulmonary resistance, on the other hand, may either decline, increase, or remain unchanged. Braunwald postulated that the apparent differences between the two techniques can be attributed to the completeness of the relief of the stenosis, with valve replacement completely relieving stenosis. Similarly, with more complete commissurotomy, the more likely will be the fall in pulmonary vascular resistance. Diamond and Genovese (12) reviewed 14 cases of hemoptysis complicating mitral stenosis and noted no deaths when aggressive operative intervention had taken place. Because the pattern of pulmonary hemorrhage is unpredictable, they recommend early intervention in all cases. Based on the apparent pathophysiology, both anatomic and hemodynamic, this seems justifiable.

As regards pulmonary function in mitral stenosis, reductions in forced expiratory flow rates, diffusing capacity, and compliance have all been described (14). These changes can be attributed to increased parenchymal stiffness due to engorgement of intropulmonic lymphatics and blood vessels, both pulmonary and bronchial, and to interstitial edema. In the present case, bronchoscopic findings felt compatible with chronic bronchitis (seen by the referring physician at the referring institution) in reality reflected these pathologic changes with bronchial venous hypertrophy and an edematous interposed mucosa, rather than an inflammed mucosa, being evident. The persistence of changes in spirometry after surgery suggests either that some permanent alterations in the pulmonary vasculature or interstitium had developed or that primary pulmonary disease was present. In the present case, the latter explanation is favored in light of the patient's long smoking history.

Thus, in patients with mitral stenosis, hemoptysis should be considered an indication that the degree of stenosis is advanced. Despite apparent abnormalities in pulmonary function testing, one should be reluctant to ascribe hemoptysis to pulmonary disease, particularly in the presence of a relatively normal chest roentgenogram. In such situations, emergent valve replacement or commissurotomy should be strongly considered.

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