# A 39 Year-Old Male With Massive Hemoptysis

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**Citation**

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**Abstract**

Hemoptysis remains a distressing symptom and, at times, a challenging diagnostic problem. A careful history and detailed physical examination is the first step in identifying the cause of hemoptysis. The chest radiography may yield important information about the underlying cause of hemoptysis. However, in the majority of cases, additional testing is required, which most commonly consists of computarised tomography of thorax and fiberoptic bronchoscopy. Occasionally, the initial evaluation suggests an uncommon cause of hemoptysis that requires one or more specific confirmatory tests such as echocardiography. Here, we describe a patient with mitral stenosis who had massive hemoptysis and required valve replacement.

## INTRODUCTION

Hemoptysis remains a distressing symptom and, at times, a challenging diagnostic problem. A careful history and detailed physical examination is the first step in identifying the cause of hemoptysis. In some patients, such as those who have pulmonary embolism (PE), bronchogenic carcinoma, traumatic or iatrogenic lung injury, left ventricular faiulre (LVF), mitral stenosis (MS), and drug toxicity, the history and physical examination may provide the only clues to the diagnosis.

The chest radiography (CXR) may yield important information about the underlying cause of hemoptysis. However, in the majority of cases, additional testing is required, which most commonly consists of computarised tomography of thorax (CT) and fiberoptic bronchoscopy (FOB). CT should be very useful in patients with unexplained hemoptysis ([1]). FOB is most effective, however, in the diagnosis of bronchogenic carcinoma and other malignancies ([1]).

Occasionally, the initial evaluation suggests an uncommon cause of hemoptysis that requires one or more specific confirmatory tests such as echocardiography (echo) revealing the presence of LVF or MS. We describe a patient with MS who had massive hemoptysis and required valve replacement.

## CASE REPORT

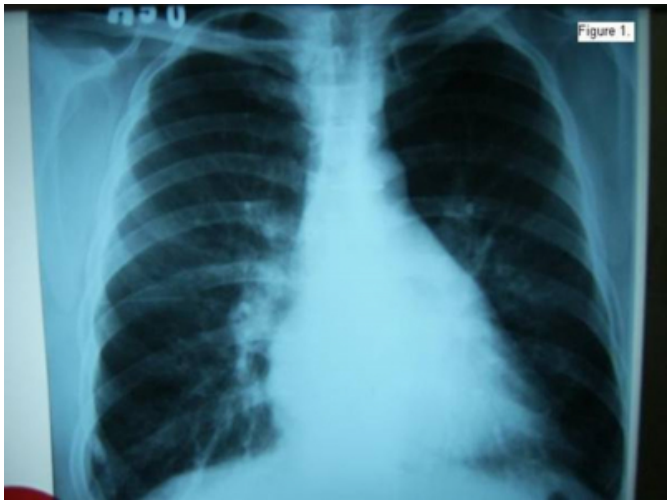
A 39 year -old -man admitted to emergency room with massive hemoptysis, severe dyspnea, sharp back and chest pain. He had had one episode of hemoptysis, coughing up about one glass of bright red blood one week ago and then the amount of expectorated blood decreased with a cough-supressant medication. He had a 15- pack-year smoking history. He had undergone a mechanical aortic valve replacement surgery 16 years ago, and had been taking oral anticoagulant therapy. Patient had no pneumonia, drug toxicity or iatrogenic or traumatic lung injury.

His physical examination was as follows: body temperature 36.6 oC, arterial blood pressure 92/68 mmHg, heart rate 76 beats/min, respiratory rate 32 breaths/min. His auscultation revealed scattered bilateral basal crackles, and a metalic heart sound was heard. He had no peripheral oedema or jugular vein distention.

Arterial blood gas analysis in room air was as the following pH=7.39, $PCO_2$=36 mmHg, $PO_2$=56 mmHg, $SaO_2$=88% and $HCO_3$=24 mEq/L. Erythrocyte sedimentation rate 44 mm/h. His hemogram, prothrombin time, INR ([1],[12]) and activated partial thromboplastin time were within normal limits. CXR revealed blunted right costophrenic recessus and enlarged right pulmonary artery (22mm)(Figure 1).

**Figure 1**

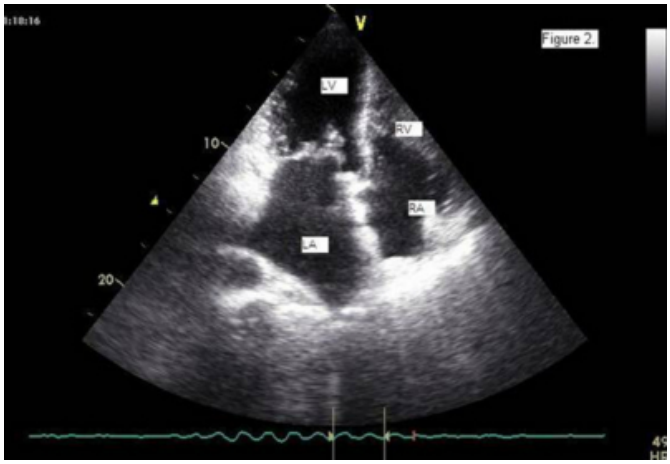
Figure 1: Posteroanterior chest radiography of the patient with massive hemoptysis. Enlarged right pulmonary artery is seen.




ECG revealed that the patient was in sinus rhythm. Helical contrast-enhanced CT revealed no a parenchymal nodule, cavity or infiltrate, endobrochial mass, bronchiectasis and vascular abnormality such as emboli in the pulmonary arteries. Since he had prosthetic aortic valve due to rheumatic fever, it could be associated with MS. So, a careful and detailed cardiac auscultation revealed a loud first heart sound, a rumbling, low-pitched diastolic murmur localized to the apical area. Patient had no apparent clue of the LVF such as S3 gallop, disseminated fine crackles in the lung areas and peripheral oedema. Also, he had no cardiomegaly on CXR. Complete echo revealed a severe MS (mitral valvul area: 1.0 cm2 and a mean mitral valve gradient of 18.8 mmHg by Doppler) with thickened and calcified mitral leaflets (Figure 2). Grade 1 mitral regurgitation and grade 2-3 tricuspid regurgitation were established. Mean pulmonary artery pressure (PAP) was 50.2 mmHg by Doppler Echo. He had left atrial enlargement (51 mm) with normal functions of left ventricular ejection fraction and mechanical aortic valve.

**Figure 2**

Figure 2: Thickened and calcified mitral leaflets by two-dimensional echocardiography in the patient with severe mitral stenosis.




Hemoptysis resolved within a few days, and did not reoccur. The etiology of massive hemoptysis in our patient was a complication of severe MS. So, a mitral valve replacement after the cardiac catheterization and coronary angiography was planned.

## DISCUSSION

Hemoptysis in MS may occur frequently, but massive pulmonary hemorrhage is uncommon ([2]). No definition of massive hemoptysis is generally accepted, although the most commonly used criteria require between 200 mL and 600 mL of blood over 24 hours ([3]). In about 60% of patients, CXR is definitely abnormal or ‘localizing'- it demonstrates a mass, cavity, infiltrate, atelectasis, or other finding that is likely to be directly related to the cause of hemoptysis. In the presence of a normal or nonlocalizing (abnormal but nonspecific findings) CXR, CT detects an unsuspected cause of hemoptysis, most commonly bronchiectasis or a parenchymal nodule or cavity, in approximately 50% of patients. When performed after a nondiagnostic FOB, CT still identifies a potential bleeding site in about one third of these patients (1). In our case, since the CT was normal, we did not consider to perform FOB immediately.

We confirmed a severe MS by echo as a cause of massive hemoptysis, however, the patient had normal mechanical aortic valve functions, and no LVF. Additionally, oral anticoagulant therapy of the patient was not effective with a normal INR value. In patients with severe MS, pulmonary vein-bronchial vein shunts occur. Their rupture may cause hemoptysis. Wood has differentiated between several kinds of hemoptysis complicating MS ([4]): 1-Sudden hemorrhage

(previously called ‘pulmonary apoplexy'). Although the hemorrhage is often profuse, it is only rarely life-threatening ([5]). It results from the rupture of thin-walled, dilated bronchial veins. 2-Blood-stained sputum associated with attacks of paroxysmal nocturnal dyspnea. 3-Pink, frothy sputum characteristic of acute pulmonary edema with rupture of alveolar capillaries. 4-Pulmonary infarction, a late complication of MS associated with heart failure. 5-Blood-stained sputum complicating chronic bronchitis. The edematous bronchial mucosa in patients with chronic MS increases the likelihood of chronic bronchitis.

CT often identifies an unsuspected cause of hemoptysis, even in patients whose CXR is normal or nonlocalizing, and may provide a road map for bronchoscopy, as well as other important information in patients who already have a presumptive diagnosis. If CT suggests a disorder that is amenable to bronchoscopic diagnosis, such as neoplasm, infection, bronchiolithiasis, or interstitial lung diasease, FOB is performed next in the diagnostic evaluation. On the other hand, a detailed history and a careful physical examination of the patients may give us several clues for hemoptysis complicating MS. So, a complete echocardiographic assessment of patients confirms the diagnosis of MS identifying an unsuspected cause of hemoptysis.

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