

Massive haemoptysis as the presenting symptom in mitral stenosis

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Mitral stenosis is a well known cause of haemoptysis, but a severe, sudden pulmonary haemorrhage is a rare presenting symptom in a hitherto symptom free person.

Case report

A 25 year old man had no prior symptoms and had been engaged in sport until a week before admission to hospital, when he had a "mild cold" with cough and one episode of chills. On the day of admission, after coitus, he had suddenly started to cough and expectorated large amounts of blood. On admission to hospital he was severely dyspnoeic and during three hours he expectorated about 1000 ml of bright red blood without clots. The blood pressure was 120/70 mm Hg, pulse rate 120/min, and respiratory rate 50/min. The rectal temperature was 39.5°C. The apex beat was normal in location but accentuated. No cardiac murmurs were found. Crackles and wheezes were heard over the lower halves of both lungs. The rest of the physical examination revealed no abnormality. The haemoglobin was 10.4 g/dl with 18×10^9 leucocytes/l. Arterial blood analysis showed a mild respiratory alkalosis (pH 7.44, oxygen tension 86 mm Hg (11.5 kPa), carbon dioxide tension 29 mm Hg (3.9 kPa)). Sputum obtained after the haemoptysis had ceased showed Gram positive cocci and leucocytes on microscopic examination. Sputum cultures yielded mixed normal flora. Blood cultures examined later were negative. The electrocardiogram showed sinus tachycardia with incomplete right bundle branch block and a QRS axis of +110°. The P waves were 3 mm high and peaked in lead II, and had a negative late phase in lead V₁. A chest radiograph showed diffuse lung shadowing and an enlarged heart with blurred borders.

The patient was thought at first to have a fulminant pulmonary infection and treatment with high dose broad spectrum antibiotics was started. In the next hours respiratory distress increased, and progressive hypoxaemia and hypercapnia necessitated intubation and assisted ventilation. Further auscultation of the heart revealed an accentuated first sound and a possible opening snap. Diuretics and digoxin were given. Haemodynamic measurements made by Swan-Ganz catheter showed raised pressures in the right ventricle (40/7 mm Hg), pulmonary artery (42/22 mm Hg), and pulmonary capillary wedge (22 mm Hg).

When the heart rate slowed, the typical auscultatory findings of mitral stenosis were heard in addition to a proto-

diastolic high pitched murmur at the left sternal border. An echocardiogram showed a calcified mitral valve with an area of 1.1 cm^2 , enlargement of the left atrium, and thickening of the aortic valve leaflets.

In the following hours the patient improved, the pulmonary bleeding ceased, and the temperature returned to normal. Twenty four hours later the chest radiograph still showed some pulmonary congestion with the typical cardiac configuration of mitral stenosis. The electrocardiogram now showed a normal QRS axis and P waves of normal size. Two further minor episodes of dyspnoea and haemoptysis were controlled with additional diuretic treatment.

Cardiac catheterisation confirmed the diagnosis of severe mitral stenosis with mild aortic regurgitation. Right sided intracardiac pressures were similar to those found previously: right ventricle 40/5 mm Hg, pulmonary artery 45/26 mm Hg, pulmonary capillary wedge 28 mm Hg. Measurements on the left side were: left ventricle 110/4 mm Hg, mitral valve gradient 21 mm Hg, and ejection fraction 72%.

One month after open mitral commissurotomy, the patient was doing well.

Discussion

Wood¹ defined several types of haemoptysis in mitral stenosis. He found sudden massive haemoptysis ("pulmonary apoplexy") in 18.3% of his patients. A bleeding of lesser degree, blood stained sputum accompanying congestion ("congestive haemoptysis"), was found in 16.5% of his series. He pointed out that in 12.7% massive haemoptysis was an early symptom, occurring before the appearance of effort dyspnoea.

Massive haemoptysis was attributed by Woodward and Dexter² to bleeding from pulmonary capillaries and bronchial arterioles. Shunting of blood from congested pulmonary veins to bronchial veins, which tend to bleed easily, would explain more readily a profuse haemorrhage, as suggested by Fergusson *et al.*³

The pleurohilar veins, which connect the pulmonary and azygos venous beds, is another source of bleeding suggested by Gilroy *et al.*⁴ Wood¹ argues that massive haemoptysis occurs when pulmonary venous pressure rises suddenly, and the development of high pulmonary resistance prevents bleeding in advanced disease. He considered massive haemoptysis to be self limited and of minor prognostic importance. Diamond and Genovese,⁵ however, reached different conclusions. Comparing two groups of patients with mitral stenosis and haemoptysis, they found that five out of seven non-operated patients died acutely, in contrast to only two deaths among seven operated patients. The

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authors concluded that massive haemoptysis carries a grave prognosis and should be treated by early commissurotomy or valve replacement. It seems that, in contrast to the relatively high prevalence of pulmonary apoplexy in Wood's series,¹ this condition is not frequent and has been rarely reported since. Similar cases of pulmonary bleeding during intercourse in patients with mitral stenosis were noted by Thompson.⁶

Our patient showed how this rare presentation can be a pitfall in rapid diagnosis. Physical effort and fever were the precipitating factors leading to extreme tachycardia, pulmonary congestion, and haemoptysis. The right-sided intracardiac pressures were not strikingly high, and the clinical picture seems to have been the result of the rapid increase of pressure in previously normal pulmonary vessels.

Massive aspiration of blood was probably an important factor, which caused asphyxiation and aggravated the hypoxaemia caused by pulmonary oedema. Pulmonary bleeding is known to cause diffuse radiographic shadowing that tends to disappear rapidly.

The electrocardiographic pattern of "acute cor pulmonale" is well known in pulmonary embolism. Although

the same mechanism may play a role in acute right ventricular strain of other causes, we are not aware of a similar description in mitral stenosis.

Mitral stenosis should be considered in the differential diagnosis of severe pulmonary haemorrhage, and in doubtful cases echocardiography and right heart catheterisation may be helpful.

References

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