Severe Hemoptysis During Pregnancy Treated by Mitral Commissurotomy


Pulmonary bleeding in mitral stenosis is not commonly regarded as an indication for operation. A case is reported in which mitral commissurotomy was performed during pregnancy primarily because of recurrent profuse hemoptysis. Some relevant points are briefly discussed.

PULMONARY apoplexy is a common symptom in mitral stenosis. According to Wool^1 such hemorrhages tend to be self-limiting and are never fatal. In his experience they are not in themselves an indication for mitral commissurotomy. Moreover, during pregnancy, operation is not often warranted and most of the reported cases were submitted to operation because of pulmonary edema.

CASE REPORT

The patient was a 19-year-old married woman of average build and nutrition. Five years previously she had left-sided chest pain and breathlessness on exertion. There was no history of acute rheumatism. Clinically, presystolic and middiastolic murmurs at the mitral area supported a diagnosis of mitral stenosis. In addition there was a systolic murmur at the aortic area and a short diastolic murmur at the left sternal border.

Breathlessness on exertion persisted with little change in severity during the ensuing 5 years. From her history, exercise tolerance was grade 2. Hemoptysis became a recurrent symptom some 3 years before the present illness. It was never severe and apart from these episodes she had neither cough nor sputum. Two years before admission, mitral commissurotomy had been considered elsewhere but was withheld, largely because of insufficient disability. The cardiac shadow at that time showed a mitral configuration (fig. 1).

Six days before admission on May 3, 1956, she started to cough up blood while walking but not exerting herself unduly. She thought that about 1 pint in all was lost. During the next few days 4 further hemoptyses of lesser severity took place.

On admission the patient was weak, pale, cyanosed, and dyspneic. She was about 4 months pregnant. The pulse was of small volume and regular at 120 per minute. The blood pressure was 95/55 mm. Hg. There was no peripheral venous engorgement, no edema, and no palpable hepatic enlargement. The respiratory rate was 36 per minute. Rhonchi were present over both lungs but no fine crepitations were heard. A portable radiograph (fig. 2) showed increased vascularity of the left lung and collapse of most of the right lung. Much of her dyspnea was thought to be due to the pulmonary collapse.

As a result of the mediastinal shift a distinct cardiac impulse could be felt 8 cm. to the right of the midline. Nevertheless the predominantly right ventricular quality of the precardial systolic impulse could be appreciated. The apex beat was in the fifth left interspace, 8 cm. from the midsternal line. At the mitral area the first sound was loud, raised in pitch, and was preceded by a presystolic murmur. A well-marked opening snap was followed by a long diastolic murmur. There was a systolic thrill and murmur at the aortic area and a short early diastolic murmur down the left sternal border. It was considered that stenosis of the mitral valve was the main lesion and the cause of the hemoptysis. The electrocardiogram showed P waves consistent with this diagnosis and there was no evidence of right or left ventricular hypertrophy.

During the next 2 days the hemoglobin level fell from 11.2 to 9.1 Gm. per cent. Recurrent bleeding over a further 3 days brought the level down to 7.0 Gm. per cent. By this time the patient was exceedingly ill although the blood pressure was 90/55 mm. Hg. During the next 24 hours 1080 ml. of citrated blood were given slowly, raising the hemoglobin level to 8 Gm. per cent. Massive hemoptysis, however, recurred on the following day with a loss of 1200 ml. of bright red blood in the course of a few hours. Her condition was again critical. Although restoration of blood volume and hemoglobin was needed, the awareness of the role of congestion of the pulmonary circuit in producing further bleeding and pulmonary edema indicated extreme caution in further blood transfusion. Over the next 8 days slight hemopt-
sis continued while she received the packed cells from 1620 ml. of blood. At the end of this period she was still critically ill and 2 further hemoptyses of about 300 ml. each took place.

At this point, conservative policy was abandoned. There seemed little likelihood that bleeding would cease and there were still more than 4 months of pregnancy to run. It was decided to perform mitral commissurotomy when her condition should have improved sufficiently to permit operation. By this time the collapsed right lung had expanded fully (fig. 3). Over the next 11 days the packed cells from 2160 ml. of blood were transfused, causing the hemoglobin level to rise to 12 Gm. per cent. The general condition improved, pulse rate fell to 100 per minute, but the blood pressure did not rise above 90/60 mm. Hg. At the end of this time a further hemoptysis of about 300 ml. occurred and as soon as bleeding ceased, operation was carried out.

The patient had also received continuous medication with phenobarbital and penicillin. With the exception of a few days, morphine sulfate, 10 mg., was given every 8 hours. Mersalyl, 1 ml.,
was given thrice weekly, in the interval between the second and third major bouts of bleeding. Although attended by moderate increases in the urinary output, the drug did not prevent the third and fourth episodes of hemoptysis. There was no evidence of abnormal breakdown of transfused blood cells.

At operation the pulmonary artery was large and tense, measuring 4 cm. in diameter. The mean pulmonary artery pressure was estimated at 65 mm. Hg. The left atrium was also enlarged and tense and the pressure therein was in excess of 35 mm. Hg. The mitral diaphragm was elastic and the stenosis extreme, measuring 0.5 cm. in its greater diameter. Both commissures were split by finger and knife to the atrioventricular ring. The final opening was 3 cm. across; no incompetence resulted. There was a systolic thrill over the base of the aorta but this was slight and not thought to indicate functionally important aortic valvular disease. The left atrial pressure fell to a normal level while that in the main pulmonary artery reached approximately 35 mm. Hg.

Postoperative progress was uneventful. There was no further hemoptysis apart from some dark red staining of the sputum on the first day. Immediately after the operation, the blood pressure was 120/55 mm. Hg. and the diastolic pressure rose progressively to 65 to 70 mm. Hg. The patient was kept in the hospital for 5½ weeks after operation. Antenatal care was also uneventful. She was admitted for 2 weeks of bed rest in the eighth month and again some 2 weeks before the expected time of delivery. At full term she was delivered of a healthy infant weighing 6 lb. 15 oz. Labor was normal, the first stage lasted 12 hours 45 minutes and the second stage 25 minutes.

Following delivery the patient's condition was excellent and she was allowed home after 9 days. When seen 5 months later she was well, without cardiac disability and there had been no further hemoptysis. The lung fields remained clear (fig. 4).

Histologic examination of the tip of the lingula of the left lung indicated that the pulmonary arteries showed minimal intimal thickening with some reduplication of the internal elastic lamina and medial hypertrophy with moderate sclerosis changes; the lumen-to-wall ratio was 2.9/1. Moderate pericapillary fibrosis existed while the pulmonary veins were the site of muscular hypertrophy.

**DISCUSSION**

Hemoptysis in mitral stenosis is often a feature of the earlier phases of the clinical course, at a time when breathlessness on exertion may not be great and when dyspnea at rest and congestive cardiac failure are absent. Such was the situation in this case. Some hold the view that this form of pulmonary bleeding is venous, coming from the bronchial veins or pulmonary-bronchial venous anastomoses. The frankly red blood in this case was in keeping with hemorrhage from the venous side of the pulmonary circulation. According to Wood, sudden profuse hemoptysis in mitral stenosis is due to sudden rise in pulmonary venous pressure and may be regarded as "a safety valve, and is inevitably self-limiting, ceasing when the pulmonary venous pressure has fallen sufficiently." In his experience pulmonary apoplexy in mitral stenosis is not a serious symptom and has never proved fatal. Thompson and Stewart found that the amount of blood coughed up had no prognostic significance, and this form of pulmonary hemorrhage was never a serious threat to life. With particular reference to their experience of mitral stenosis in pregnancy, Marquis and Mulcahy have not had to recommend operative intervention because of hemoptysis.

It was in recognition of the good immediate prognosis in such hemoptysis that the initial conservative management of this case was instituted and continued. The time came, however, when continuance of this regime seemed to carry a greater risk to life than surgical intervention. The gratifying outcome of this single case obviously does not prove the correctness of the decision made when considered out of context. On the other hand it was expected that as soon as the obstruction at the mitral valve had been relieved, the tendency to hemoptysis would diminish or disappear.

Several factors may have contributed to the severity of the bleeding. The main ones appear to have been pregnancy and the collapse of the right lung. While hemoptysis had occurred during the previous 3 years, it had never reached the frequency and profusion attained during the fourth and fifth months of pregnancy. At this time it is well known that increased blood volume and cardiac output are becoming established. These
in turn will tend to cause a rise in pressure in the pulmonary circulation in the presence of mitral stenosis.

Collapse of the right lung presumably resulted from aspiration of blood into the bronchial tree. It is likely that the source of the first hemorrhage was also on the right side. It is not known whether subsequent bleeding came from the same or from the opposite side or from both. It would seem possible, however, that the severity of the bleeding may in some way have been aggravated by the collapse of the lung, since the more massive hemorrhages occurred during this period and were less severe when the right lung had regained aeration and function. During the period of collapse blood would have been diverted away from the airless lung to augment the flow to the left lung bed, thereby increasing the pressure in the left pulmonary circulation. Again, within the collapsed lung itself, altered pressure relations, particularly between the air passages and the pulmonary veins may have contributed to the unusual severity of the hemorrhages.

Later in pregnancy deficiency of fibrinogen might conceivably be an aggravating cause of such pulmonary bleeding. In the present case there was no abnormality of blood clotting.

Summary
A case is presented in which mitral commissurotomy was performed in the fifth month of pregnancy because of recurrent profuse hemoptysis. After relief of tight mitral stenosis, hemoptysis ceased. Pregnancy continued to term without complication and a normal healthy child was delivered.

Some points relevant to hemoptysis in mitral stenosis complicating pregnancy are discussed.

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SUMMARIO IN INTERLINGUA
Es presente un caso in que commissurotomia mitral esseva executate in le quinte mense de pregnanția a causa de recurrente e profuse hemoptysis. Post le alleviamento del stricte stenosis mitral, le hemoptysis cessava. Le pregnanția continuava sin complication, e un infant normal naseva a termino.

Es discutite certe aspectos de hemoptysis in stenosis mitral como complication de pregnanțias.

REFERENCES
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