MITRAL VALVOTOMY FOR DEXTRO-CARDIA WITH MITRAL STENOSIS

A CASE REPORT

BY

P. KOSHY, M.D.

Associate Professor of Medicine

T. THOMAS, M.B., B.S., F.A.C.S.

Acting Thoracic Surgeon

V. BENJAMIN, M.B., B.S.

Tutor, Department of Medicine

AND

N. GOPINATH, M.B., B.S., T.D.D.

Clinical Assistant, Department of Thoracic Surgery

*From the Departments of Medicine and Thoracic Surgery,
Christian Medical College and Hospital, Vellore,
South India*

Rheumatic valvular disease in dextrocardia with situs inversus is said to be rare. Bleyer and Saphir (1953) have reviewed the literature on five hitherto published cases, and reported a sixth. According to them, Owen is credited with the first case report of mitral stenosis in dextrocardia with situs inversus, but since he could not definitely establish the aetiological factor for the mitral stenosis the authors did not include it in the review. Of the six cases reported, two were cases of aortic valvular affection, three of mitral stenosis with insufficiency, and one a case in which both mitral and aortic valves were involved.

The purpose of this paper is to report a seventh case of rheumatic heart disease in a case of dextrocardia with situs inversus, producing pure mitral stenosis, and to record the successful performance of mitral valvotomy on such a case. So far as we are aware, this is the first case of surgical correction of mitral stenosis in dextrocardia to be reported.

Case Report

An Indian labourer aged 19 was admitted to the medical wards of the Christian Medical College Hospital, Vellore, on November 18, 1953. His only complaint was progressively increasing breathlessness with effort, of three years' duration. Until 1950 he could cycle five miles at a stretch, besides carrying on his job of unloading bags of tobacco. But from that time he has felt increasing dyspnoea. In 1952 he had to rest after walking half a mile. Three months prior to admission he was so incapacitated that he voluntarily left his employment. He had no history suggestive of definite rheumatic fever or of nocturnal cough or dyspnoea. He had blood-stained sputum once, two years previously. The family history had nothing significant.

Physical examination showed him to be physically somewhat underdeveloped, moderately anaemic, with no cyanosis and without any clubbing of fingers or toes. His temperature was 98.6° F. (37° C.), pulse 86 a minute, regular, and of normal tension and volume. His respiratory rate was 20 a minute and his blood pressure 110/70. There was no visible venous pulsation in the neck or obvious deformity of the chest. The apical impulse was visible and palpable in the fifth right intercostal space, in the mid-clavicular line. A well-marked presystolic thrill could be felt over the systolic impulse. Auscultation revealed, close to the apex on the right side, a loud first sound, a normal second sound, and a slow rumbling crescendo presystolic murmur in the same area. There was reduplication of the second sound in the pulmonary area, but the reduplication disappeared on deep inspiration—indicating that it was probably due to the opening snap (Mounsey, 1953). The

opening snap of the mitral valve was definitely well heard in this patient, just internal to the right nipple. Examination of the lungs revealed no abnormal physical signs. The liver and spleen were not palpable. There was no peripheral oedema. The central nervous system was normal.

His exercise tolerance was as follows: he was comfortable while walking on level ground for a distance of about 50 yards, but became uncomfortable if he tried to do more, and became acutely distressed on running. He was not able to do any work comfortably. He was classified as Grade 2 on the basis of the criteria laid down by Baker *et al.* (1952). Fluoroscopy revealed no obvious cardiac enlargement, but the pulmonary artery was somewhat prominent, and the left atrium was moderately enlarged, as observed by barium swallow. There was moderate enlargement of the right ventricle, but no enlargement of the left ventricle. Systolic expansion of the left atrium and calcification of the mitral valve were not visualized on fluoroscopy.

The electrocardiogram showed the standard Lead I to be a mirror image of Lead I in a normal individual, with inversion of all the complexes, which is characteristic of dextrocardia. aVR showed an RS pattern and an upward T wave, which suggests that it is facing the left ventricle in a vertical heart. aVL resembles an aVR tracing of a normally placed heart, showing a downward P and T, and this is pathognomonic of dextrocardia (with situs inversus) (Goldberger, 1950). Chest leads were taken on the right side of the chest as would normally be done on the left chest, and were designated VR₁, VR₂, VR₃, and VR₄. The R wave in VR₁ is suggestive of right ventricular hypertrophy.

The mitral configuration in a dextrocardiac was well seen in a skiagram. It was also noticed that the barium was flowing into a stomach which was placed in the right hypochondrium, thus confirming the diagnosis of situs inversus.

An angiocardigram was done with the patient in the right posterior oblique position. The study of films at 8 and 11 seconds after injection showed an enlarged well-defined left atrium remaining opacified during that period. The small left ventricle remained poorly filled. Cardiac catheterization studies were not done, as the patient was not co-operative.

A diagnosis of mitral stenosis (grade II) in a dextrocardiac, with moderate incapacity of a progressive nature, was made, and as no obvious contraindications to operation were present he was submitted to a mitral valvotomy.

The operation was carried out on December 9 by one of us (T. T.), with endotracheal anaesthesia administered by Dr. K. Blott. The patient was placed on his left side and the thoracic cage was exposed by a right postero-lateral incision. The fifth rib was resected and the pleura was entered through the fifth rib bed. On opening the pericardium it was found that the heart was smaller than normal. The left auricle seemed to be somewhat dilated and there was no adequate ventricular filling. The auricular appendage was nearly 3.5 cm. long, and narrow, and its base was only about 2 cm. broad. It was felt that only the little finger could possibly enter the small auricular appendage. It was accordingly introduced through an opening, controlled both by a purse-string suture and by a Brock clamp. A tight mitral opening, not more than 0.5 cm. in diameter, was felt. A satisfactory commissurotomy was achieved. The finger was withdrawn and the auricular opening repaired. Histological examination of the auricular appendage revealed typical Aschoff bodies, thus establishing the aetiology of acquired mitral stenosis.

A post-operative follow-up study, about four months after operation, revealed that the patient's general condition was better. Whereas he could walk only 50 yards in comfort before operation, he now could walk about a mile. On physical examination the findings were more or less the same, the presystolic murmur being present as before. But there was a significant diminution in the intensity of the opening snap. The pulmonary second sound was reduced

in its intensity. His blood pressure was 108/70. The lungs were clear. He could now be classified as grade I.

Comment

The diagnosis of dextrocardia with mitral stenosis was well established from physical findings, electrocardiographic studies, and angiocardiology. The aetiology of mitral stenosis was only presumptive before operation, but was established beyond doubt from histological study of the atrial wall.

Dextrocardia of the mirror type with situs inversus is probably not as rare as is commonly believed; but unless the condition is associated with other congenital cardiac anomalies or cardiac disease no symptoms are produced. These symptoms, when they do appear, are those of the associated abnormality and not due to the dextrocardia *per se*. The signs in the heart are, of course, found on the right chest instead of on the left.

It is claimed by some workers that the condition is common in white persons. It is worth noting, however, that the case reported prior to this was in a negro (Bleyer and Saphir, 1953), and our case is in an Indian.

Summary

A case of pure mitral stenosis due to rheumatic infection in a true dextrocardiac (with situs inversus) is described.

The successful performance of valvotomy in such a case is reported.

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IDIOPATHIC PULMONARY HAEMOSIDEROSIS IN A YOUNG ADULT

BY

N. A. J. HAMER, M.B., M.R.C.P.

Captain, R.A.M.C.

(From the British Military Hospital, Singapore)

Idiopathic pulmonary haemosiderosis is being recognized with increasing frequency in recent years. Most of the cases described are in children, and the mortality rate is high. Considered in retrospect, the clinical picture is remarkably uniform, but early diagnosis may be difficult. Several cases of milder forms of the condition in young adults have been reported. In these the outlook is generally good and long remissions may occur. A further adult case is presented in this article.

Case Report

A National Service man aged 19 was first admitted to hospital in October, 1948, when aged 13, with a three-weeks history of intermittent frontal headache, pallor, and weakness. He described about six similar episodes since an attack of chicken-pox a few months previously. There was some dyspnoea on exertion. For four weeks he had been coughing up moderate amounts of brownish-red sputum. Oral iron therapy had had no apparent effect on the pallor. He had four sisters and nine brothers, all well except a brother with pulmonary tuberculosis. There had been no previous illness of significance.

On examination he was quiet and intelligent, and gave a clear story. He was very pale and listless. There were

no signs of cardiac failure or jaundice, and no haemorrhagic manifestations. His tongue was normal. There were no enlarged lymph nodes and the spleen could not be felt, but a sharp rubbery liver edge was palpable 1 in. (2.5 cm.) below the costal margin. The pulse rate was 110 per minute and the blood pressure 120/80 mm. Hg. The heart and chest were normal, as was the central nervous system.

He was transfused immediately after admission to hospital. The haemoglobin level after transfusion was 9 g.%. This rose to 11 g.% after two weeks, and remained at this level until discharge one week later. No iron therapy was given at this time. The E.S.R. and the leucocyte count were normal. No urobilinogen was found in the urine.

The patient did not attend for observation, but was re-admitted with severe anaemia six months after leaving hospital. Dark-red sputum was produced during most of the time between the admissions to hospital. There were no new physical signs. The haemoglobin value was 4.1 g.%. The red cells showed anisocytosis and were poorly filled with haemoglobin. The icteric index was 8 units, and no urobilinogen was found in the urine. A red-cell fragility test showed haemolysis from 0.56% to 0.32% saline, almost the same as a control.

After a week, treatment with oral iron was started and the haemoglobin level rose rapidly, reaching 12.4 g.% after four weeks, when the patient was discharged. During this period the chest was x-rayed, but no abnormality was reported. He remained completely free of symptoms from July, 1949, until December, 1953, a period of 4½ years.

In December, 1953, the cough with reddish-brown sputum returned. This attack lasted six weeks. There were several minor recurrences during the next few months. He began his national service in October, 1953, and at the end of April, 1954, he arrived in Singapore; the haemoptyses appeared again a few days later. After three weeks he reported sick and a radiograph of the chest showed miliary mottling of the lung fields.

On admission to hospital his only symptom was a mild frontal headache on exertion. Apart from a slight sternal depression no abnormalities were found on examination. The mucosae were of good colour and there was no cyanosis or jaundice. The mouth and tongue were normal. There was no finger-clubbing. No lymph nodes were enlarged, and the spleen and liver were not palpable.

Investigations.—Several blobs of dark-brown sputum with a little mucus were produced each day. On microscopy numerous macrophages containing haemosiderin granules were seen, and a very few red blood cells. No acid-fast bacilli were found on direct examination or culture of six specimens. X-ray examination of the chest showed a normal cardiac shadow, confirmed on screening. There was diffuse mottling throughout the lung fields, maximal in the middle zones. There was no suggestion of a clear centre in any of the markings. Blood picture: Hb, 14.5 g.%; red cells, 5,000,000 per c.mm.; P.C.V., 45%; E.S.R., 36 mm. in the first hour (Wintrobe). Absolute values: M.C.V., 90 cubic μ ; M.C.H., 29 $\gamma\gamma$; M.C.H.C., 32%. Reticulocyte count, 2.5%; leucocyte count, normal; platelet count, 200,000 per c.mm.; bleeding-time, 2 minutes; clotting-time, 4 minutes; prothrombin time, 15 seconds (equal to control). Capillary fragility test, negative. The indirect Coombs test and trypsin cell test showed no antibodies. Cold agglutination was negative at 1 in 4. Red-cell fragility showed haemolysis from 0.52% to 0.28% saline. Blood W.R. and Kahn test were negative; serum bilirubin, 0.5 mg. per 100 ml. on two occasions; serum proteins, 7.5 g.% (albumin 5.5 g.%, globulin 2 g.%). An E.C.G. was normal. The urine was normal on repeated tests, and no urobilinogen was present. The Mantoux test was negative to 1 in 10,000, but positive to 1 in 1,000 O.T. X-ray films of the skull, hands, long bones, and abdomen were normal.

During three months' observation there has been little change in the clinical condition. Daily haemoptyses continue, but the haemoglobin has remained between 13.3 and