Red cell survival after aortic valve replacement with Björk-Shiley prosthesis in presence of sickle-cell trait

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SUMMARY A case of aortic valve replacement with a Björk-Shiley disc prosthesis in a patient with sickle-cell trait is presented. Six months after operation red cell survival was not significantly reduced. On theoretical and clinical grounds it is concluded that the presence of sick-cell trait does not contribute to late postoperative haemolysis after heart valve replacement.

There are few reports of valve replacement in patients with sickle-cell disorders. Though there is at least one opinion to the contrary (Craenen et al., 1972), it is considered that prosthetic valves should be avoided in these patients. It is thought that the haemolysis usually observed after the insertion of prostheses would be increased in the presence of red cells containing haemoglobin S (Yacoub et al., 1970; deLeval et al., 1974). A case is presented which challenges this contention.

Case report

A 53-year-old Jamaican woman presented with left ventricular failure associated with severe aortic regurgitation.

INVESTIGATIONS

Chest radiograph showed dilatation of the left ventricle and an aneurysm of the ascending aorta.

Electrocardiogram indicated sinus rhythm, left axis deviation, and severe left ventricular hypertrophy with repolarisation abnormalities on the left praecordial leads. At cardiac catheterisation the left ventricular end-diastolic pressure was 16 mmHg rising to 30 mmHg after exercise and the aortic pressure was 180/60 mmHg.

Cineangioigraphy confirmed that there was aneurysmal dilatation of the ascending aorta and gross aortic regurgitation.

Haemoglobin was 13.5 g/dl; RBC 4·93 x 10¹²/l; PCV 0·41; MCV 83 fl; MCH 27·4 pg. Blood film was normal. Haemoglobin electrophoresis showed the presence of haemoglobin A + S with a haemoglobin S concentration of 34 per cent, establishing the diagnosis of sickle-cell trait.

OPERATION

After initial medical treatment, operation was carried out on 8 October 1975. The aortic valve was replaced with a Björk-Shiley prosthesis, size 25, and the ascending aorta with a 30 mm crimped, woven ‘dacron’ graft.

Histological examination of the aorta showed calcified atheroma.

Normothermic cardiopulmonary bypass was maintained for 190 minutes using a Modulung-teflo oxygenator (Travenol). Bilateral coronary artery perfusion was maintained after an initial ischaemic interval of 6 minutes. There was a total of 19 minutes of non-perfusion of the right coronary artery.

The following precautions were taken to minimise the dangers of sickling in either the systemic or coronary circulations during operation and the postoperative period (Yacoub et al., 1970; deLeval et al., 1974). The systemic circulation was maintained using a high flow (2·4 l/min per m²) perfusion technique while on cardiopulmonary bypass, with an arterial Po₂ in excess of 110 mmHg. Local hypoxia and acidosis in the coronary circulation were avoided by minimising the time of total myocardial ischaemia. After the period of bypass the arterial oxygen tension was maintained above 93 mmHg, with inspired oxygen fractions not exceeding 0·40. Any respiratory or metabolic acidosis was corrected by adjustment of the minute volume or by the administration of a small dose of sodium bicarbonate.

The concentration of haemoglobin S was reduced by exchanging 2 litres of the patient’s blood for fresh donor blood and Hartman’s solution and by the use of a haemodilution bypass technique.
In the immediate postoperative period the concentration of haemoglobin S was 6 per cent of the total haemoglobin. Plasma viscosity was reduced by haemodilution resulting in a packed cell volume of 0·20 during operation. The temperature was maintained at 37°C.

POSTOPERATIVE COURSE

The early postoperative course was complicated by the occurrence of ilio-femoral venous thrombosis after cannulation of the iliac vessels at surgery and by the development of systemic hypertension. She was discharged on bendrofluprazide, supplementary potassium, propranolol, and warfarin.

Six months after operation she was clinically well with no clinical anaemia. Haemoglobin was 13·4 g/dl; reticulocyte count 1 per cent, and blood film was normal, with no evidence of mechanical fragmentation. Red cell survival was measured using $^{51}$Cr. The $T_{50}$ $^{51}$Cr was 24 days (normal 25 to 33 days).

Discussion

Mild haemolysis is usually detectable after prosthetic valve replacement though this would not normally be clinically serious. The Björk-Shiley prosthesis compares favourably with other prosthetic devices in causing minimal haemolysis, as indicated by serum lactate dehydrogenase activity (Björk et al., 1974; Slater et al., 1974). Ahmad and his colleagues (1976) have measured red cell survival in patients with Björk-Shiley valves in the mitral position and they have found minimal red cell destruction. Haemolysis arising because of a mitral prosthesis is unlikely to be less than with an aortic prosthesis (Crezells et al., 1972). The amount of the haemolysis in this case is similar to Ahmad's finding and would be accounted for by the presence of the valve and any further explanation involving sickle-cell trait would be unnecessary.

$^{51}$Cr-labelled red cell survival in sickle-cell trait is normal (Weinstein et al., 1954). This accords with the presence of a normal mechanical fragility of red cells containing Hb A and S, under physiological conditions. In their classic study on the biophysics of sickle-cell disease, Harris and his coworkers (Harris et al., 1956; Griggs and Harris, 1956) showed that changes in the mechanical fragility of red cells containing Hb S were directly related to the degree of sickling. In sickle-cell trait sickling only occurs at oxygen pressures lower than 30 mmHg. Within the physiological range of blood oxygenation and acid-base balance, the cell containing Hb A and S has normal cell morphology and is mechanically as robust as the normal red cell.

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Conclusion

Theoretical consideration and the experience of this patient indicate that sickle-cell trait does not contribute to haemolysis late after heart valve replacement. Therefore, there seems to be no disadvantage in using a prosthetic device as compared with a biological valve, such as a free homograft or a mounted heterograft. The hazards of acid-base imbalance, inadequate oxygenation, and blood stasis, both in the systemic and coronary circulation during the operation and the postoperative period, are emphasised.

References


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