Acute massive mitral regurgitation from prosthetic valve dysfunction

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Two cases of prosthetic valve dysfunction resulting in acute massive mitral regurgitation are reported; emergency operation was successful in both cases. Survival following complete dislodgement of the occluder of a disc valve, as occurred in one case, does not appear to have been reported before. The difficulty in diagnosis of sudden cardiac decompensation in patients with prosthetic valves is stressed, as is the need for urgent operation.

Prosthetic valve replacement is still associated with late complications with sufficient frequency to make it the operation of necessity rather than of choice. Thromboembolism and infection are the most commonly encountered problems and mechanical defects are reassuringly uncommon.

Acute massive mitral regurgitation from prosthetic valve dysfunction is particularly rare and usually rapidly fatal (Low and Lefemine, 1967; Samaan, 1969; Roberts and Morrow, 1968; Vasko and Leighton, 1968; Kalke et al., 1969; Ibarra-Perez et al., 1970; Wilson, 1970; DuPiest et al., 1973; Messmer, Rothlin, and Senning, 1973; Keen, 1974). This paper reports two such cases, in each of which an emergency operation had a successful outcome.

Case reports

Case 1

The patient, a 47-year-old woman, had undergone mitral valve replacement in 1964 at another London hospital. A Type I University of Cape Town mitral valve prosthesis had been inserted.

In 1968 she developed a persisting fever and was found to have splenomegaly, anaemia, finger clubbing, and haematuria. Investigations showed raised titres for Q fever antibodies, strongly suggesting active endocarditis. She was treated with tetracycline and made a good symptomatic recovery.

In September 1969 she noticed acute, but transient (10 minutes) loss of vision of the left eye, followed by severe dyspnoea and palpitation. She was admitted to another hospital, by which time she was shocked and cyanosed with a sinus tachycardia of 140/minute, a blood pressure of 60/30 mmHg (8/4 kPa), and gross rise in the venous pressure. Tachycardia made auscultation difficult, but a soft systolic murmur could be heard over the whole praecordium. There were bilateral crepitations, and a chest x-ray film confirmed gross pulmonary oedema. An electrocardiogram showed sinus tachycardia but no other significant abnormality. She was treated with furosemide, digoxin, and morphine with no improvement. Endotracheal intubation and intermittent positive pressure ventilation were employed and the patient was transferred to The Middlesex Hospital.

On admission the clinical picture was of tachycardia and acute severe left and right ventricular failure. In view of her previous history and in the absence of evidence of another cause, prosthetic valve dysfunction was suspected and emergency exploration of the valve was carried out. At operation all heart chambers were found to be of normal size; left atrial and pulmonary arterial pressures were raised and the left atrium showed systolic expansile pulsation. The University of Cape Town prosthesis had fractured circumferentially between the rigid Ivalon seating of the valve and the spongy Ivalon sewing ring (Fig. 1). About a quarter of the circumference was still attached, corresponding to the medial half of the mural cusp of the mitral valve; the prosthesis was otherwise lying free in the atrium. The suture-bearing cuff of the valve appeared to be
Fractured circumferentially throughout three-quarters of its circumference. The peripheral flange was left in the patient.

perfectly incorporated in the mitral annulus. There was no evidence of infection on the valve, which was perfectly clean, or in the left atrium, which contained no clot. At operation, separation of the prosthesis was completed, leaving the firmly-embedded sewing ring, and a No. 3M Model 6120 Starr-Edwards ball valve prosthesis was inserted.

The postoperative period was eventful, the patient developing tamponade, which was successfully relieved, and later, respiratory distress leading to cardiac arrest, which was successfully reversed after 30 minutes of cardiac massage. The patient remained ventilator-dependent until the sixteenth postoperative day, during which period a tracheostomy was performed; subsequent progress was satisfactory.

The response to animal inoculation with tissue obtained from the excised prosthesis showed the presence of Rickettsia burneti, but the antibody response obtained indicated non-viable organisms. A subsequent (November 1969) fall in antibody level in the patient’s serum suggested an absence or suppression of R. burneti as an antigenic stimulus, but it was decided to continue suppression of this organism indefinitely, currently using demeclocycline.

She has since been followed up as an out-patient and was last seen in April 1975. She is able to lead a very active life and is employed full-time: she takes no medication except warfarin and demeclocycline. Her new mitral prosthesis continues to function well and the chest x-ray film shows only minimal cardiomegaly.

**Case 2**

The patient was a 32-year-old male Yugoslav student studying in England. In 1969 he had undergone mitral valve replacement in the U.S.A., a No. 30 Wada-Cutter prosthesis being inserted. The patient progressed satisfactorily and remained symptomatically well without cardiac or anticoagulant drugs, though when he was first seen in this country in October 1973 a systolic murmur was heard. By December 1973, the murmur had increased and the patient was referred for a surgical opinion. Before any further investigation could take place, however, he developed acute dyspnoea accompanied by sudden chest pain, palpitation, and the production of profuse amounts of pink, frothy sputum, and was admitted as an emergency to The Middlesex Hospital.

The patient was cyanosed, cold, sweaty, and grossly dyspnoeic. The pulse rate was 150/minute, jugular venous pressure was not raised, and the blood pressure was 80/0 mmHg (10·7/0 kPa). The gross tachycardia made auscultation difficult, but it was thought that there was a third heart sound and a systolic murmur; there were no thrills. Coarse crepitations could be heard throughout the lungs.

Initial treatment with intravenous frusenide, digoxin, diamorphine, and hydrocortisone was given, but with little beneficial effect. An electrocardiogram showed atrial tachycardia, and chest x-ray examination showed gross pulmonary oedema; the prosthetic valve ring could be seen. DC countershock was unsuccessful in restoring normal rhythm, as were carotid sinus and eyeball pressure. The clinical picture of gross pulmonary oedema of sudden onset suggested serious dysfunction of the prosthesis and no other explanation of his abrupt deterioration seemed likely. It was agreed that immediate surgical exploration was needed. Positive pressure ventilation was initiated, and the trachea and main bronchi were sucked out at frequent intervals with good effect before the patient was transferred to the theatre.

At operation, gross mitral regurgitation was
obvious from the systolic pulsation of the left atrium, though both left atrium and left ventricle were of normal size. When the left atrium was opened, the disc was found to be absent from the prosthesis. The left ventricle was explored, and the entire thoracic aorta was palpated via the left pleura, but the disc could not be found. The ring of the Wada prosthesis was excised, and a No. 3M Model 6120 Starr-Edwards prosthesis was inserted. The mid-line incision was extended and a hand was passed into the abdomen, but the disc was not palpable in the abdominal aorta or iliac arteries.

Postoperatively progress was initially slow, but basically uneventful. The new prosthesis functioned efficiently, and the pulmonary oedema cleared over the next few days. On the third postoperative day thoracic and abdominal aortography was carried out and a possible filling defect was seen at the aortic bifurcation; echocardiography also suggested that the disc might be present at this site. Laparotomy was performed on the 4th day and the disc was eventually found, not at the aortic bifurcation, but at the level of the renal vessels, behind the pancreas. It was removed through an incision in the lower descending aorta with some difficulty as it proved to be considerably larger than expected and had sharp protuberances.

The Teflon disc showed signs of wear at the pivot sites where it had been bearing on the metal retaining struts of the prosthesis (Fig. 2). Notching had occurred at these points, allowing the disc to open more than its design intended until eventually it had slipped out of position entirely.

Twenty-seven days after his admission the patient was well enough to be discharged home, and at this time was taking digoxin, frusemide, slow K, and warfarin. Later, all medication was stopped, with the exception of the anticoagulant. The patient has since been seen on three occasions as an out-patient, most recently in July 1974. His progress has been excellent; there are no cardiac murmurs, and chest x-ray examination shows further improvement. He had returned to full-time employment and has since emigrated to Australia.

Discussion

Both cases illustrate a design problem of prosthetic valves where mobile elements join or impinge on rigid elements of the structure. However, in reporting seven years’ experience with four models of the University of Cape Town prosthesis in 122 patients, Schrire and Barnard (1970) did not meet the complication described in the present paper.

The greatest experience with the Wada-Cutter valve has been reported from Houston, Texas, where 150 patients had valves replaced by this prosthesis during 1969 (Hallman et al., 1970). No cases of dysfunction of the prosthesis in the mitral position were reported after a follow-up period of approximately one year. In 1972, Wada reported that he had used this valve in 106 patients, including 48 in the mitral position, and in only one case was replacement of a defective prosthesis necessary (Wada, Komatsu, and Kamata, 1972).

Complete dislodgement of a Wada-Cutter disc valve occluder has been reported previously on one occasion. In 1973, DuPriest and his colleagues reported the sudden death of a 48-year-old man whose mitral valve had been replaced three years earlier. Gross pulmonary oedema and congestion were noted at necropsy, and the disc was found at the apex of the left ventricle. The disc had a notch at one pivot point and was thinned at the other pivot point. At an angle of 50°, the disc slipped from the four projections which ordinarily retain it. As in our own case the disc had become dislodged because of wearing of the Teflon; DuPriest and his colleagues forecast that further cases would occur, and suggested that as durable alternatives were available continued clinical use of the Wada-Cutter valve should be reconsidered.

Björk (1970) also reported variance of Wada-Cutter valves (in the aortic position) with grooving of the Teflon disc where it strikes the metal shoulder of the valve ring; this had led to the death of two patients within one year, though in neither case was this the result of dislodgement of the disc. Pulse duplicator studies showed that the Teflon disc of the Wada prosthesis was more susceptible to wear than
Discs of other materials used in the construction of prostheses.

Disc dislodgement has occurred from a Björk-Shiley mitral prosthesis (Messmer et al., 1973), and a case of dislodgement of a deformed silicone rubber ball from a Cutter-Smelloff prosthesis has been reported by Keen (1974). The case of disc dislodgement reported in the present paper had a successful outcome, and in this respect appears to be unique. If the poppet had been a ball rather than a disc, it is probable that total occlusion of the aorta would have occurred leading to a sudden increase in peripheral resistance, left ventricular embarrassment, and sudden death as in Keen’s case. The present case also emphasizes the difficulty in locating a disc embolus; all peripheral pulses were present, and neither radiography nor echocardiography located it accurately.

Several authors have stressed the difficulty in making a definite diagnosis in prosthetic valve dysfunction, particularly in the early postoperative period. Auscultation, phonocardiography, echocardiography, and fluoroscopy may be inconclusive; left heart catheterization with cineangiography appears to be the only reliable means of confirming the diagnosis (Thomas, 1971), but may be precluded by the urgency of the situation. Absence of the opening click is an ominous sign of malfunction of ball or disc valve prostheses, whether this sign is the result of insufficient peripendicular force to move the occluder from thrombus or tissue ingrowth, swelling of the poppet itself, or complete absence of the ball or disc. Leachman and Cokkino (1969) have reported 6 cases of dehiscence of a disc valve in which the characteristic opening click of the prosthesis was absent, and concluded that if the absence of the click can definitely be documented, such a patient should be considered for corrective surgical therapy at the earliest possible time. Auscultation, however, may be exceedingly difficult in such cases, because of extreme tachycardia, the obviously small cardiac output, and the loud pulmonary added sounds. With free reflux, a systolic murmur is not present, but a third sound may be heard. Phonocardiography has been used in conditions of malfunction of mitral valve prostheses and may prove helpful in confirming auscultatory signs (Craigie, Hutchin, and Sutton, 1970; Wise, Webb-Peploe, and Oakley, 1971).

The two cases reported here illustrate the difficulty in diagnosis. It is our view, however, that a patient with a prosthetic valve, who experiences sudden cardiac decompensation that cannot be readily attributed to a pathological cause such as myocardial infarction, or speedily controlled medically, should be re-explored urgently for prosthetic dysfunction. Operation should not be delayed to permit sophisticated investigation which may confirm the diagnosis but vitiate treatment.

References


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