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CASE REPORT

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Complete detachment of an aortic valve prosthesis 10 years after implantation

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Abstract A 47-year-old male patient died unexpectedly 10 years after replacement of the aortic valve with a Carbomedics heart valve prosthesis required for post-endocarditic valve stenosis. The man was in regular medical attendance by his general practitioner and in hospital. Clinical data and examinations did not suggest the reoccurrence of endocarditis. Three months before his death a haemolytic anaemia of unknown genesis was diagnosed. One afternoon, while lifting a heavy object, the man suffered acute chest pain and collapsed. Resuscitation failed and the patient died in hospital. Autopsy revealed the completely detached valve prosthesis within the ascending aorta. Histological examination confirmed a chronic endocarditis at the site of the valve implantation.

Keywords Aortic valve · Embolisation · Complete detachment · Endocarditis · Late complication

Introduction

The majority of sudden deaths are cardiac deaths, most of them based on internal diseases such as myocardial ischaemia or infarction [1, 2], but also conduction system abnormalities can cause such sudden deaths [3]. In rare cases also external mechanisms such as myocardial contusion following from blunt trauma can cause sudden death [4]. Prosthetic heart valves are still associated with severe or even fatal complications, but problems of structural valve failure in older generations of prosthetic valves now seem to be solved. Mechanical failure, the best known example of which is the (outlet) strut fracture in Björk-Shiley prostheses [5, 6, 7], is also a thing of the past. Nevertheless, life-threatening complications can still occur. A rare late complication of an implanted modern mechanical bileaflet prosthesis is presented in this report.

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Case report

Clinical history

A 47-year-old male patient who suffered from post-endocarditic aortic valve stenosis received a size 23 Carbomedics heart valve prosthesis in March 1993. The postoperative course was uneventful, the patient was in regular medical attendance of his general practitioner and in hospital. In 1999, an increased level of inflammatory parameters (e.g. white blood count, C-reactive protein) was documented in the medical records for the first time and the man received antibiotics for 8 weeks. In 2000, the man had a transient ischaemic attack with a temporary aphasia and a dysfunction of the fine motoric activities of the right side of the body. To rule out valvular dysfunction several echocardiograms were performed, but these showed no pathological findings. In 2001, the patient required a permanent pacemaker implantation for symptomatic complete heart block. In December 2002, 3 months before his death, a haemolytic anaemia was diagnosed. A possible mechanical haemolysis associated with the prosthesis was ruled out as "the previous haemograms had always shown regular values". Further echocardiographic investigations (transoesophageal and transthoracic) did not show any prosthetic valve dysfunction or endocarditic vegetation and clinically, no aortic regurgitant murmur was detected. Routinely performed bacterial cultures were negative, extensive medical investigations showed no pathological findings. In January 2003, a biopsy of the iliac crest was done to find out the cause of the haemolysis, and further examinations were planned to rule out an oncological disease.

One afternoon in February 2003, the patient suffered acute chest pain for the first time, when he lifted a heavy object. The general practitioner carried out an electrocardiogram which revealed a regular sinus pattern. Auscultation of the heart showed normal heart and prosthetic sounds. The condition of the man deteriorated quickly during the next minutes: attempts at resuscitation failed and he was finally declared dead in hospital. A clinical autopsy was performed to investigate the cause of death.

Autopsy results

At autopsy, performed 20 h postmortem, the internal organs (heart, kidneys, liver) showed signs of acute congestion; both pleura spaces contained each about 700 ml of clear thin fluid. The heart weighed 710 g with both ventricles showing marked hypertrophy. Myocardial infarctions of different durations were noted in the left ventricular posterior wall, whereas the coronary arteries showed only minor signs of arteriosclerosis. The aortic valve was completely detached and was found free within the ascending aorta. Some soft tissue fragments from the aortic root, where the valve had been sutured, were still attached to the ring of the valve. These particles were almost circular and had regularly formed margins. (Fig. 1). At the site of valve implantation, remnants of sutures were detected and the whole area had an irregular surface. Calcified material was attached on several places and a foraminated recess was found (Fig. 2).

Fig. 1 Completely detached aortic valve prosthesis found in the ascending aorta. Note the remains of sutures and the nearly circular aortic particles. The two lightest patches are used as abutments when suturing the valve in the implantation site

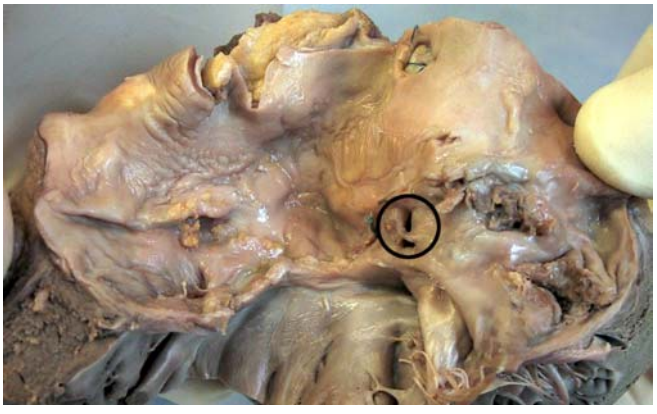

Fig. 2 Left ventricular outflow tract and ascending aorta showing the area from which the valve had been detached. Note the necrotic and fibrotic parts with calcified areas. The circle marks the aperture of the recess which was filled with thrombotic material

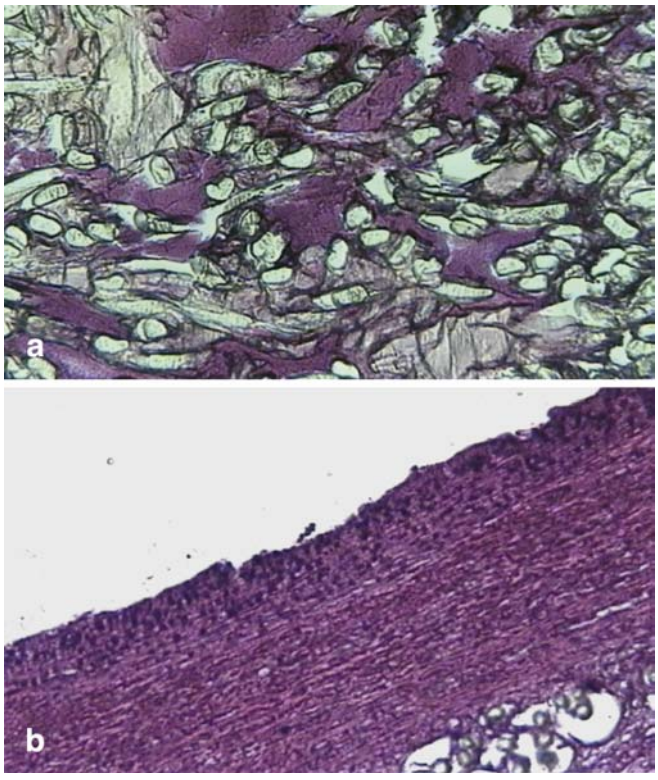

Fig. 3 Histological slides showing segments of the tissue pieces which were still attached to the valve. **a** Synthetic sutures and connective tissue (HE, magnification ×100), **b** parts of the aorta wall with massive infiltration of predominantly granulocytes in the outer margin (HE, magnification ×40)

Histological examination revealed that the tissue around the sutures showed reactive changes and the aortic valve sutures were partly covered with endothelium and connective tissue. In the entire area from which the valve had been detached there were marked signs of inflammation. Focal inflammation infiltrates consisting of abundant granulocytes, histiocytes and lymphocytes were found in the tissue pieces from the aortic wall, which were still attached to the valve (Fig. 3), and also in the area from which the valve had been detached (Fig. 4). No bacteria were seen histologically.

Additional findings were the presence of necrosis with capillary proliferation and mild perifocal reactive astrogliosis in parts of the cerebrum and cerebellum.

Discussion

Total detachment and embolisation of an implanted mechanical aortic valve is a very rare event. A review of the literature revealed only two fatal cases, both involving Starr-Edwards ball-valve prostheses. In 1966 Cohn et al. [8] reported three patients who had aortic valve replacement with a Starr-Edwards ball-valve prosthesis and developed acute bacterial endocarditis with typical clinical features several months after the operation. In each case the infection resulted in detachment of the prosthetic valve and massive aortic regurgitation, in one case the

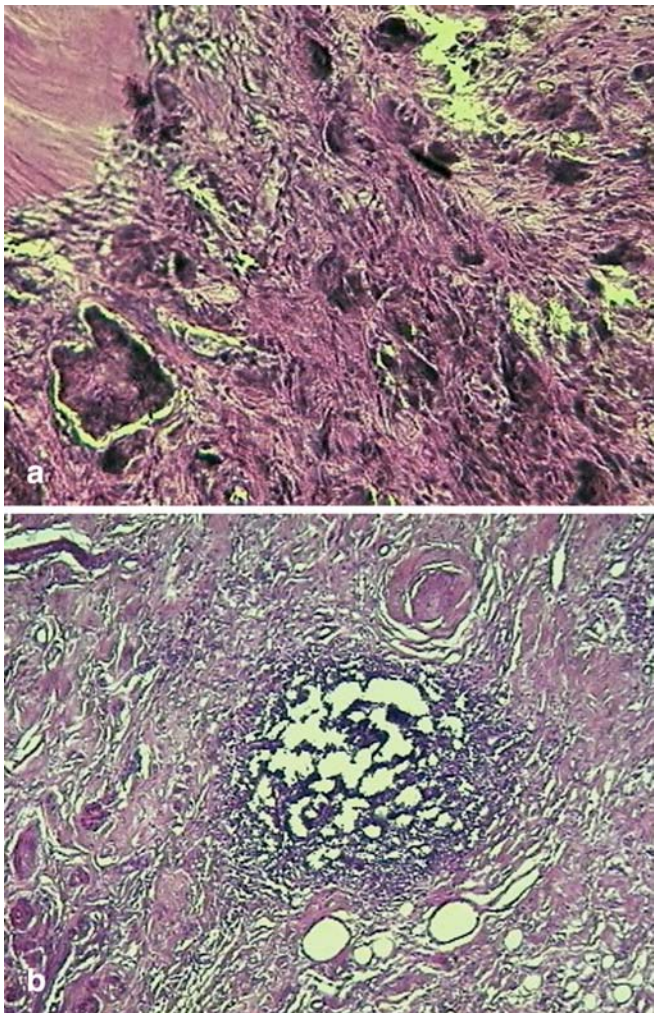

Fig. 4 Histological slides showing the site of the valve implantation. **a** Giant cells around the remnants of sutures (HE, magnification ×40), **b** parts from the aorta wall with massive infiltration of predominantly lymphocytes (HE, magnification ×40)

valve was totally detached and displaced into the aortic arch. Because of the extent of the infection, secondary operative interventions were regarded as inadvisable. Cohn et al. [8] suggested a more comprehensive antibiotic regimen for such patients.

The second case of a completely detached aortic Starr-Edward ball-valve prosthesis was described by Bruhlmann et al. [9] in 1982 which occurred 11 years after replacement. The reason for detachment in this case was not found and the prosthesis was translocated into the thoracic aorta. No clinical or laboratory data suggested the existence of an endocarditis, but permission for autopsy was denied in this case.

Additionally, a review of the literature resulted in three reports concerning survival after complete translocation of prosthetic aortic valves: Stoneburner et al. [10] described a case of successful management of dehiscence and embolisation of an aortic Starr-Edward ball-valve prosthesis to the aortic arch, 5 years after implantation in a patient with a history of drug abuse. Amsel and Walter [11] reported a case of survival after dislocation of a Björk-Shiley prosthesis into the left ventricular cavity, 6 months after insertion. Villani et al. [12] described a case of complete embolisation of an Omnicarbon mechanical prosthesis into the abdominal aorta. In all three survivors bacterial endocarditis was diagnosed and 2 patients showed postoperative residual problems [10, 11].

This case is the first report concerning a Carbomedics prosthesis, a modern bileaflet prosthesis, which was first implanted in 1987 [13]. In reports about short-term [14], medium-term [15, 16, 17] and long-term [13, 18, 19, 20] experiences, the Carbomedics prosthetic heart valve has proven to be a highly reliable device with no structural failures and a low incidence of valve-related complications which results in a low over-all mortality. Comparative studies of different mechanical aortic valves demonstrated no clinically relevant differences among the tested bileaflet valves [21, 22]. Most common complications associated with an implanted bileaflet prosthesis are valve thrombosis, embolism, anticoagulant-related bleeding, paravalvular leakage and prosthetic valve endocarditis.

In this case, the clinical data and investigations did not suggest the existence of an endocarditis, nonetheless the microscopic investigations of autopsy tissue revealed a chronic inflammation process at the site of valve implantation. As aortic regurgitation never appeared in clinical examinations or in echocardiography, it is assumed that the valve must have functioned properly most of the time. It appears to have come to an increasing partial dehiscence and eventually detachment of the prosthetic valve some weeks previously; this is also suggested by the morphology of the detached pieces from the aortic root. Paravalvular leakage could have accounted for the initially unexplained haemolytic anaemia. The necrotic areas found in parts of the cerebrum and cerebellum suggest the occurrence of embolisations, associated either with endocarditis or with inappropriate anticoagulation treatment. Myocardial infarction could have resulted from embolised endocarditic or thrombotic vegetation into the coronary arteries; another explanation for the myocardial scarring is a suspected temporary valve dehiscence with partial occlusion of the coronary ostia. However, the possibility that the valve prosthesis was ultimately detached by resuscitation measures cannot be completely ruled out, although this has not been described in the past and is therefore unlikely. The lack of clinical features suggesting valve malfunction or myocardial infarction resulted in the fatal outcome in our case.

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