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An atypical aortic valve non-bacterial thrombotic endocarditis in the course of multiple myeloma

Non-bacterial thrombotic endocarditis (NBTE) is characterized by small, sterile and loosely attached vegetations upon valve leaflets that develop usually in instances of debilitating disease such as metastatic cancer, renal failure, chronic sepsis, etc.[1]. Although clinically significant embolization may be frequently encountered[2], valvular dysfunction is rare in the course of NBTE.

A 40-year-old man with Ig G-lamda-type multiple myeloma known since 1990 and chronic renal failure on peritoneal dialysis since January 1994 was admitted to our emergency room complaining of sudden-onset chest pain and shortness of breath. A 4/6 systolic ejection murmur was auscultated along the left sternal border. An electrocardiogram suggested the presence of posterolateral ischaemia. Transthoracic two-dimensional echocardiography showed an immobile big rounded mass attached with a wide base to the aortic and ventricular surfaces of the aortic valve, creating an aortic valve stenosis with a mean gradient of 50 mmHg (Fig. 1).

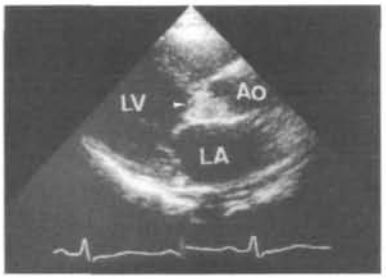

Figure 1 **Transthoracic echocardiography revealing the presence of a large aortic valve mass creating aortic valve stenosis (arrow).**

It was supposed that posterolateral ischaemia was due either to the partial obstruction of the right coronary artery ostium by this mass or to the embolization of a part of it in the right coronary artery. We opted not to perform coronary arteriography because of the risk of cerebral and/or peripheral embolization during catheterization of the ostia. The patient had not been febrile since the multiple myeloma had been diagnosed. Successive haemocultures showed no evidence of bacteria or fungus. At operation, the tumour was found to be a grey 2 × 1-cm soft and crumbly mass with a cauliflower appearance infiltrating the aortic valve leaflets and leaving a valve opening orifice of 6 mm of diameter. The mass was removed and the aortic valve was replaced with an aortic cryopreserved homograft. The patient developed acute hepatic insufficiency on postoperative day 3 and died in hepatic coma 2 days later. Histopathological examination of the tumour revealed a bland fibrinous material with some plasmocytoid cells among the lymphocytes and histiocytes at the insertion site with the valve. No bacteria or fungus were present in serial histological sections and cultures of the mass were negative. Immunocytochemical determination of the clonal constitution of the plasmocytoid cells did not confirm the eventuality of a plasmocytoma. In conclusion, based on these findings the diagnosis was consistent with NBTE.

Valve dysfunction due to NBTE is rare unless the affected valve has previous damage such as old rheumatic valvulitis or degenerative alterations. Our case shows that small vegetations in NBTE, usually single along the closure line of the leaflet, can increase in size and extensiveness by continuous fibrin accumulation and create valve dysfunction. Infective endocarditis should be considered in the differential diagnosis of NBTE and particularly in the setting of immunodeficiency due either to multiple myeloma or to its treatment. Clinical symptoms, positive blood cultures or presence of bacteria or fungus in the valve specimen are required for confirmation. However, some vegetations in infective endocarditis can undergo progressive sterilization, fibrosis and organization. This 'healed' form of infective endocarditis manifests as nodular excrescences on the valve leaflets and may mimic vegetations of NBTE. In these instances, pathological features of the valve specimen can serve to distinguish the 'healed' form of infective endocarditis from NBTE which is characterized by the presence of fibrin core and lack of organization and significant acute inflammatory reaction. In our case, the lack of the consistent signs of infective endocarditis, prophylactic use of antibiotics and potential complications of infective endocarditis in other organs at any time before the diagnosis of the mass during the course of the multiple myeloma precluded the eventuality of an episode of acute or subacute endocarditis.

This case demonstrates that an aortic valve mass formed in the setting of multiple myeloma with no clinical evidence of infective endocarditis or definitive consensus regarding the pathogenesis of the mass, should be suspected NBTE. Surgical intervention is mandatory in states of embolism or severe valvular dysfunction although it is impossible to definitely say what the fate of the patient could have been without surgery and to predict the prognosis of the multiple myeloma in this stage.

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Coronary artery mycotic aneurysm discovered by transoesophageal echocardiography

In February 1991, an 18-year-old woman was admitted to hospital with a week-long fever. She was known to have had asymptomatic aortic regurgitation since childhood. At presentation, she had a grade 4 diastolic murmur and a new mitral regurgitation. A *Streptococcus mitis* grew from blood cultures and the diagnosis of acute bacterial endocarditis was made. The patient was treated with appropriate antibiotics and became apyreic after 2 days. The first transthoracic echocardiogram revealed worsened aortic insufficiency, mitral regurgitation as a result of anterior leaflet prolapse and vegetations on the anterior leaflet and below the aortic valve. Six weeks later, the regular weekly ECG showed an acute anterolateral myocardial infarction. There had been no accompanying thoracic pains. Transoesophageal echocardiography was performed and revealed the double valvular lesion but the vegetation located below the aortic valve had disappeared. Transoesophageal echocardiography also revealed a paracardiac mass, round and apparently empty measuring 1 cm × 1 cm, located in the anterior interventricular groove (Fig. 1). No pulsatile flow was detected by Doppler examination. Selective coronary angiography revealed an aneurysm of the left anterior descending artery at its midportion. The other coronary arteries were normal. The patient was scheduled for surgical correction. Mitro-aortic replacement comprised a St. Jude prosthesis and resection of the coronary artery aneurysm was completed by a left internal thoracic artery graft of the anterior interventricular artery. Histological examination of the aneurysm confirmed its mycotic origin; the artery wall was fibrosed and invaded by inflammatory cells. There was a fresh thrombosis in situ. The 3-year follow-up examination revealed a healthy and asymptomatic patient.

Coronary artery aneurysms are rarely described as a paracardiac mass visualized by transoesophageal echocardiography. Two cases have recently been reported in adults[1,2]. Both patients were asymptomatic and

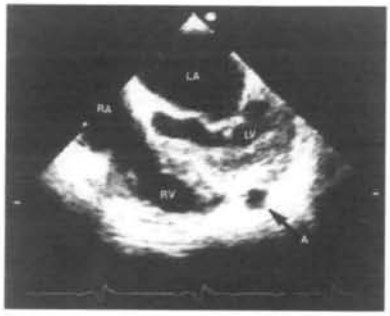

Figure 1 **Transoesophageal echocardiogram, apical four-chamber view. A=aneurysm of the left coronary artery; LA=left atrium; LV=left ventricle; RA=right atrium; RV=right ventricle.**

the aneurysms were revealed by transoesophageal echocardiography. In an autopsy review of 89 cases, Daoud *et al.*[3] found that 52% of all cases of coronary artery aneurysm were atherosclerotic, 17% congenital, 11% mycotic or embolic, 11% dissecting and 4% luetic. The mycotic origin of the coronary artery aneurysm described here may be affirmed by the context of fever, the disappearance of a vegetation and the histologic examination. The thrombosis of the aneurysm may have explained the myocardial infarction on the ECG and the absence of flow on Doppler during the transoesophageal echocardiogram. Two cases of coronary artery mycotic aneurysm have reported previously[4,5]. Both were diagnosed by coronary angiography, late after the bacterial endocarditis was treated and considered cured. Both patients died after a short course and the diagnosis of coronary artery mycotic aneurysm was confirmed by autopsy examination, with findings similar to our case.

To our knowledge, this is the first description of a coronary artery myocardial aneurysm revealed by transoesophageal echocardiography. This probably accelerated the therapeutic course and permitted, in our case, a favourable outcome in this life-threatening complication.

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Distinguishing between the warm-up phenomenon and training during repeated exercise testing

We read with interest the recent article entitled 'Mechanisms of the warm up phenomenon'[1]. In this study three exercise tests were performed on 15 patients with a 10 min interval between the first and second test and 2 h between the second and third. They found an increase in time to 1 mm ST depression and time to angina in both the second and third tests; however, the rate pressure product at 1.5 mm ST depression (ischaemic threshold) was increased only in the second test and was not significantly changed in the third compared with the baseline test. They concluded that the reduction in ischaemic threshold seen in the second test may be due to an improvement of myocardial perfusion (collateral flow) or to ischaemic preconditioning, whereas the changes the seen in the third test may be due to the training effect.

The training effect in repeated exercise testing has been recognized for some time. Burkart *et al.*[2] found, in 10 healthy subjects who performed two exercise tests with a 30 min rest period, a reduction in pulmonary artery pressure, aortic pressure, cardiac output and oxygen uptake in the second test compared to the first. Smokler *et al.*[3] performed a retrospective analysis to examine the reproducibility of exercise testing in 63 patients with angina who had performed more than one exercise test with varying time intervals between the tests. The study showed that the