be considered contamination. The electrocardiographic and X-ray studies showed abnormalities similar to those described elsewhere. Echocardiograms have only previously been described in 2 patients with infectious pericarditis caused by *P. acnes*; both had pericardial effusion. Mookadam et al. reported that 34 of 49 cases of pericarditis caused by *P. acnes* needed surgery. However, these authors did not specify the type of surgery performed. The only report specifying the type of surgery describes a partial pericardiotomy. Three of our patients needed a wide pericardial resection and patch epicardietomy. Inflammatory infiltration and fibrosis confirmed that despite minimal virulence, *P. acnes* has an immunostimulatory effect on the mononuclear phagocyte system, which produces inflammatory mediators such as metalloproteases and tumor necrosis factor alpha. This microorganism has been associated with inflammatory diseases such as sarcoidosis, which would explain the need for a combined NSAID, colchicine and corticosteroid regimen. Doxycycline was selected as the maintenance antimicrobial treatment of choice, due to its ability to inhibit the metalloproteases of *P. acnes*.

The antibiotic treatment was prolonged since *P. acnes* resists phagocytosis as an intracellular microorganism. Length of treatment has not been defined but we consider that a minimum 4 weeks are needed, which should be extended to several months in patients who relapse.

The pericardial response to infection caused by *P. acnes* is similar to that of tuberculous pericarditis, with a tendency to constriction. We would include *P. acnes* in the differential diagnosis of constricitive infectious pericarditis or idiopathic, viral and postsurgical effusive-constrictive syndrome, which has become increasingly frequent in recent years. The incubation time of surgical samples should be lengthened or polymerase chain reaction techniques be used to rule out infection caused by *P. acnes*.

The patient was stable and asymptomatic when returned to the coronary unit. However, 24 h later he experienced cardiac tamponade secondary to right ventricular free wall perforation by the temporary pacemaker lead and required surgery. Three days later the patient experienced high-grade atrioventricular block and consequently a permanent dual-chamber pacemaker was implanted. The patient was discharged 10 days later, with no further incidents (Figure).

Dextrocardia is a rare anomaly of the heart position. Most cases with situs solitus are associated with other cardiac or noncardiac malformations. However, patients with situs inversus totalis (as in our patient) rarely have other associated malformations and, therefore, it is not unusual them to reach older ages in which degenerative aortic stenosis is common.

Inversion of cardiovascular structures is an added procedural difficulty for percutaneous aortic valve implantation. The most important difficulties are related to stable positioning of the temporary pacemaker when crossing the aortic valve with the straight guidewire or attempting to align the 3 Valsalva sinuses to assess correct positioning of the prosthesis. In fact, our patient experienced late perforation by the pacemaker leads, possibly related to malpositioning. In cases such ours, in which the abnormal cardiac anatomy can affect the operator’s spatial orientation, we recommend careful catheter handling and conscientious selection of the angiographic projections (usually opposite to those seen in a patient with levocardia). To our knowledge, this is the first case of the implantation of a CoreValve aortic valve prosthesis in a patient with situs inversus totalis.
Valvular Heart Disease in Hyperprolactinemic Patients Treated With Low Doses of Cabergoline

Afección valvular cardiaca por dosis bajas de cabergolina en pacientes hiperprolactinémicos

To the Editor,

Between 1992 and 1997, a valvular heart disease similar to that produced by carcinoid tumors associated with fibrosis, leaflet retraction, and calcification was reported in patients treated with ergotamine derivatives (fenfluramine-phenetermine). The activation of 5-hydroxytryptamine (serotonin) 2B (5-HT2B) receptors, which are highly abundant in the valvular endocardium, through signaling pathways mediated by Src kinases, stimulates fibroblast mitogenesis, with increased extracellular matrix production. It also causes the histological changes typical of this restrictive valvular heart disease, referred to as fen-phen since it was first described. In recent years, the development of this type of valvular heart disease has also been reported in patients with Parkinson's disease treated with dopamine agonists and, more recently,

REFERENCES


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