which have a recognized anti-inflammatory and antithrombotic action).

Furthermore, South Asians in our series presented a significantly lower prevalence of hypertension, smoking, and cocaine abuse than the white Spanish population, with similar values of low-density lipoprotein cholesterol, but a lower prevalence of previous treatments with statins. Studies performed in Britain have reported contradictory results regarding the presence of these risk factors in South Asian patients, and this has been related to the heterogeneity of the subgroups analyzed, which makes extrapolation to our setting difficult. For its part, the risk profile observed among the young Spaniards in our series is in keeping with previous findings, in which early infarction has been associated with high rates of smoking and hypercholesterolemia, and glucose metabolism disorders have a smaller role. Our study highlights the specific features of cardiovascular risk in South Asians in our setting and the differences with respect to the young Spanish population.

There were no significant differences between the 2 groups in the clinical presentation of AMI (with or without ST elevation). Nonetheless, among patients with ST elevation AMI, there was a significantly higher percentage of anterior AMI in South Asian patients. In addition, coronary angiography showed more severe disease in these patients, and a larger number were considered to have nonrevascularizable lesions. AMI without coronary lesions was more common in the white Spanish population. These observations indicate that coronary disease in South Asians is related more to accelerated atherosclerosis than to predominantly thrombotic or vasospastic processes.

Despite the differences described, the 2 groups presented a similar in-hospital clinical course with low complication rates and mortality, which was likely due to the young age of the patients, low prevalence of comorbidities, absence of significant differences in the treatment received, and the improved in-hospital prognosis of AMI observed in our setting.

This series is the first in Spain to address ischemic heart disease in South Asian immigrant patients. Knowledge of the differential characteristics of this ethnic group can facilitate the development of primary and secondary prevention strategies adapted to their risk profile.

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Constrictive Infectious Pericarditis Caused by Propionibacterium acnes

Pericarditis constrictiva por Propionibacterium acnes

To the Editor,

Propionibacterium acnes is a slow-growing Gram-positive anaerobic bacillus, which is part of the bacterial flora of the skin and is also present in mucous membranes. Isolated cases of pericarditis caused by P. acnes have been reported but, even though this microorganism is a frequent cause of the disease, its characteristics have not been described. We report the characteristics of 5 patients with constrictive infectious pericarditis caused by P. acnes—initially manifesting as constriction in 3 and as effusive-constrictive syndrome in 2—characterized by a torpid clinical course, minimal signs of infection, much inflammatory activity and the need for surgery and prolonged treatment with antibiotics, anti-inflammatory drugs, and corticosteroids.

The 5 patients were attended between 2006 and 2011. All underwent transthoracic echocardiography and 3 underwent magnetic resonance imaging studies. For diagnostic purposes, the microbiology results were combined with the echocardiogram results in 4 patients and with the magnetic resonance imaging results in 1. Pathologic analysis was performed in 4 patients.

The mean age was 44.4 years; 4 were men. Possible predisposing factors are shown in the Table. The predominant symptoms were asthenia, chest pain and symptoms of heart failure with elevated venous pressure, dyspnea and edema of the lower limbs; 3 patients had pulsus paradoxus. One patient had fever and another had chylous ascites with tension. The mean diagnostic delay was 30 weeks. One patient had hypogammaglobulinemia, adenopathy and pulmonary infiltration caused by lymphangectasia, which disappeared once pericardial constriction had been resolved. All electrocardiograms showed low voltage in precordial leads, with ST-segment depression and T wave inversion in 3 of them.

All chest X-rays showed cardiomegaly. Echocardiographic and magnetic resonance imaging studies are described in the Table. One patient had a calcified mass, with pericardial effusion and lateral myocardial hypokinesia. The Figure shows the surgical specimen, illustrating growth of P. acnes, together with an magnetic resonance imaging scan.

Medical treatment, microbiology and histology results are outlined in the Table. P. acnes grew in the samples between days 10 and 12. The results of blood cultures (3 patients) were negative. P. acnes was penicillin-sensitive in all patients. The 5 patients
received intravenous beta-lactam antibiotics, followed by oral antibiotics for a mean of 5.6 months. Four received corticosteroids and 3 received nonsteroidal antiinflammatory drugs (NSAIDs) plus colchicine. Surgical treatment and outcome are described in the Table.

Pericarditis caused by *P. acnes* is mainly found in men and is associated with cardiac surgery and immunsuppression.²,₃ Iseki et al.¹ describe a patient with comorbidity and a calcified mass containing caseous material, as found in 1 of our patients (Figure). Fever has only been described in 1 other case.¹ Chest pain has also been described, as have signs of right heart failure and cardiac taponade.¹,³ Late growth has been described in other infections caused by *P. acnes*⁴ and, in the context of compatible symptoms, it should not

### Table

Characteristics of the 5 Patients With Constrictive Infectious Pericarditis Caused by *Propionibacterium acnes*.

<table>
<thead>
<tr>
<th></th>
<th>Man, 55 years</th>
<th>Man, 26 years</th>
<th>Man, 31 years</th>
<th>Man, 72 years</th>
<th>Woman, 38 years</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Predisposing factor</strong></td>
<td>Respiratory infection</td>
<td>Tonsillitis, dental caries</td>
<td>Dental infection</td>
<td>Not reported</td>
<td>2 week-long respiratory infection</td>
</tr>
<tr>
<td><strong>Symptoms and diagnostic delay</strong></td>
<td>Pleuritic pain, dyspnea, heart failure, 2 months</td>
<td>Chest pain, dyspnea, palpitations, syncope, 2 weeks</td>
<td>Dyspnea, chest pain, right heart failure, fever, 9 months</td>
<td>Asthenia, progressive dyspnea, right heart failure, 20 months</td>
<td>Chest pain, asthenia, dry cough, dyspnea, right heart failure, 1 month</td>
</tr>
<tr>
<td><strong>Echocardiogram</strong></td>
<td>Constrictive infectious pericarditis, mild pericardial effusion, bilateral pleural effusion</td>
<td>Constrictive infectious pericarditis with cardiac taponade, moderate-to-severe effusion of up to 21 mm</td>
<td>Increased intrapericardial space, solid/fluid material causing constriction</td>
<td>Pericardial thickening with mild effusion, effusive-constrictive syndrome</td>
<td></td>
</tr>
<tr>
<td><strong>Magnetic Resonance Imaging</strong></td>
<td>Substantial pericardial effusion, hypointense nodular areas</td>
<td>Pericardial thickening, dilated inferior vena cava</td>
<td>Pericardial thickening with associated effusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Pathology</strong></td>
<td>Pericardial thickening, whitish colored nodule</td>
<td>Pericardial thickening</td>
<td>First surgical intervention: pericardial thickening; second intervention: adhesions, calcified nodules with caseous appearance</td>
<td>Pericardial thickening</td>
<td>Pericardial fluid with hematic material</td>
</tr>
<tr>
<td><strong>Macroscopic</strong></td>
<td>Chronic inflammation, reactive fibrosis</td>
<td>Acute inflammation, fibrillin deposit, mesothelial hyperplasia</td>
<td>Marked fibrosis and foci of lymphocytosis</td>
<td>Intense pericardial fibrosis</td>
<td>Not performed</td>
</tr>
<tr>
<td><strong>Microscopic</strong></td>
<td><em>P. acnes</em> + <em>Staphylococcus epidermidis</em> in pericardial tissue</td>
<td><em>P. acnes</em> in pericardial fluid and pericardial tissue</td>
<td><em>S. epidermidis</em> in fluid in first surgical intervention; <em>P. acnes</em> in pericardial tissue in second intervention</td>
<td><em>P. acnes</em> in pericardial fluid and ascitic fluid</td>
<td><em>P. acnes</em> in pericardial fluid</td>
</tr>
<tr>
<td><strong>Biologic samples</strong></td>
<td><strong>Surgical treatment</strong></td>
<td>Subtotal pericardectomy with pericardial patching in right cavities</td>
<td>Pleuropericardial pericardial window-type surgical drainage</td>
<td>First subtotal pericardiectomy, second patched epicardectomy and residual pericardiectomy</td>
<td>Total pericardiectomy with decortication</td>
</tr>
<tr>
<td><strong>Medical treatment and outcome</strong></td>
<td>1 month ceftriaxone + amoxicillin-clavulanic acid 1 month + amoxicillin and corticosteroids 6 months more</td>
<td>1 relapse, resolved in 1 year</td>
<td>Amoxicillin-clavulanic acid + amoxicillin 6 months + doxycycline 2 months, corticosteroids 3 months</td>
<td>1 relapse requiring ASA + colchicine, resolved in 21 months</td>
<td>Post-second surgery, pencillin G sodium, amoxicillin 10 months + moxifloxacin 8 months, corticosteroids + colchicine + NSAID 10 months</td>
</tr>
</tbody>
</table>

ASA, acetylsalicylic acid; NSAID, nonsteroid antiinflammatory drugs; *P. acnes*, *Propionibacterium acnes*; *S. epidermidis*, *Staphylococcus epidermidis*.

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**Figure.** Cardiac magnetic resonance image of a patient with constrictive infectious pericarditis. A: cine echo image of short-axis plane gradient; pericardial effusion (**_*_), pericardial thickening (**_*) and 1.5 cm diameter hypointense nodule image (arrow), corresponding to mass full of material, in which *Propionibacterium acnes* was cultivated (B). LP, left posterior; MRN, magnetic resonance nuclear; RA, right anterior.
be considered contamination.\textsuperscript{3} The electrocardiographic and X-ray studies showed abnormalities similar to those described elsewhere.\textsuperscript{1,2} Echocardiograms have only previously been described in 2 patients with percutaneous pericarditis caused by \textit{P. acnes}; both had pericardial effusion.\textsuperscript{1,2} Mookadam et al.\textsuperscript{3} reported that 34 of 49 cases of pericarditis caused by \textit{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 pericardietomy.\textsuperscript{1} Three of our patients needed a wide pericardial resection and patch epicardiectomy. Inflammatory infiltration and fibrosis confirmed that despite minimal virulence, \textit{P. acnes} has an immunostimulatory effect on the mononuclear phagocyte system, which produces inflammatory mediators such as metalloproteases and tumor necrosis factor alpha.\textsuperscript{6} 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 \textit{P. acnes}.\textsuperscript{6}

The antibiotic treatment was prolonged since \textit{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 \textit{P. acnes} is similar to that of tuberculous pericarditis, with a tendency to constriction. We would include \textit{P. acnes} in the differential diagnosis of constrictive 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 \textit{P. acnes}.\textsuperscript{4}

### CoreValve® Aortic Bioprosthesis Implantation in a Patient With Situs Inversus Totalis With Dextrocardia

**Implante de bioprótesis aórtica CoreValve® en un paciente con situs inversus totalis con dextrocardia**

**To the Editor,**

Dextrocardia occurs in 1/12 000 pregnancies, of which approximately a third are associated with inversion (mirror imaging) of the other visceral organs (situs inversus totalis).\textsuperscript{1} In these cases, inversion of the normal anatomy can hamper the performance of fluoroscopy-guided interventional procedures.

We describe a 78-year-old man with situs inversus totalis and symptomatic severe aortic stenosis who was referred to our hospital for transcatheter implantation of an aortic valve. The patient had previously been considered ineligible for conventional aortic valve replacement due to high surgical risk (EuroSCORE logistic, 21%: porcelain aorta). The procedure was performed using a right femoral approach but was complex due to inversion of the cardiac anatomy. Classic ventriculography with 5 segments was performed using a 30° left oblique projection (the usual view is a 30° right oblique projection); a 10° caudal and a 10° right oblique projection were used to align the 3 Valsalva sinuses. Following aortic valvuloplasty with ventricular overdrive pacing, a 29-mm CoreValve\textsuperscript{®} self-expanding aortic valve prosthesis (Medtronic, Irvine, California, United States) was successfully implanted.

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