## Scientific letters

Transient Trifascicular Block Secondary to Tricuspid Valve Endocarditis

Bloqueo trifascicular paroxístico secundario a endocarditis infecciosa sobre válvula tricúspide

To the Editor,

A 76-year-old woman presented with fever that began 2 days previously, along with general malaise and dyspnea. She had a history of hypertension and dyslipidemia, and was undergoing dialysis for chronic end-stage renal failure via a permanent internal jugular vein catheter. Laboratory studies revealed leukocytosis with left shift, along with white blood cells and bacteria in urinary sediment. She was admitted to hospital with a diagnosis of febrile syndrome probably due to a urinary tract infection, and antibiotic treatment was initiated with levofloxacin. Four days after admission, she had a long episode of nausea

and sweating. Her blood pressure was raised and physical examination revealed bradycardia with a left parasternal systolic murmur. An electocardiogram (Fig. 1A) revealed a widened QRS interval with an escape rhythm of 30 bpm due to third-degree atrioventricular block (AVB). A decision was therefore taken to immediately implant a temporary pacemaker via the jugular vein. A few hours later the patient recovered her normal rhythm but with first-degree AVB and complete left bundle-branch block (Fig. 1B). Due to the febrile syndrome and a continued murmur, a transthoracic echocardiogram was carried out. Two large vegetations were observed on the tricuspid valve; the most mobile measured 10×16 mm and was anchored to the free edge of the valve (Fig. 2), while a second, smaller growth was observed at the insertion of the septal leaflet. A transesophageal echocardiogram undertaken to complete the study confirmed the earlier findings and ruled out involvement of the left valves or other complications. Three separate blood cultures revealed growth of methicillin-sensitive *Staphylococcus epidermidis* and a urine culture was negative. Infectious endocarditis due to *S epidermidis* was diagnosed, probably secondary to catheter infection. On this basis, treatment was initiated with gentamicin and cloxacillin, and the dialysis catheter was surgically removed. Electrocardiographic monitoring over the next 72 h revealed progressive narrowing of the QRS interval until it reached 86 ms, with persistence of first-degree AVB (Fig. 1C). After a week of treatment with antibiotics, blood cultures were negative and no new conduction abnormalities were observed in the electrocardiogram. Therefore, the temporary pacemaker was removed. The catheter tip culture was positive for *S epidermidis*. After 5 days of combined antibiotic therapy, gentamicin was suspended and treatment continued with cloxacillin alone. The patient's condition followed an unfavorable course with worsening renal failure and development of systemic congestion, resulting in death 23 days later.

Severe conduction disorders are a rare manifestation of infectious endocarditis. According to data from patient registries, their occurrence is between 1% and 15% of cases. They are associated with worse prognosis due to an increased risk of embolus and with higher mortality. Conduction abnormalities are caused by spread of the infection from the valves to
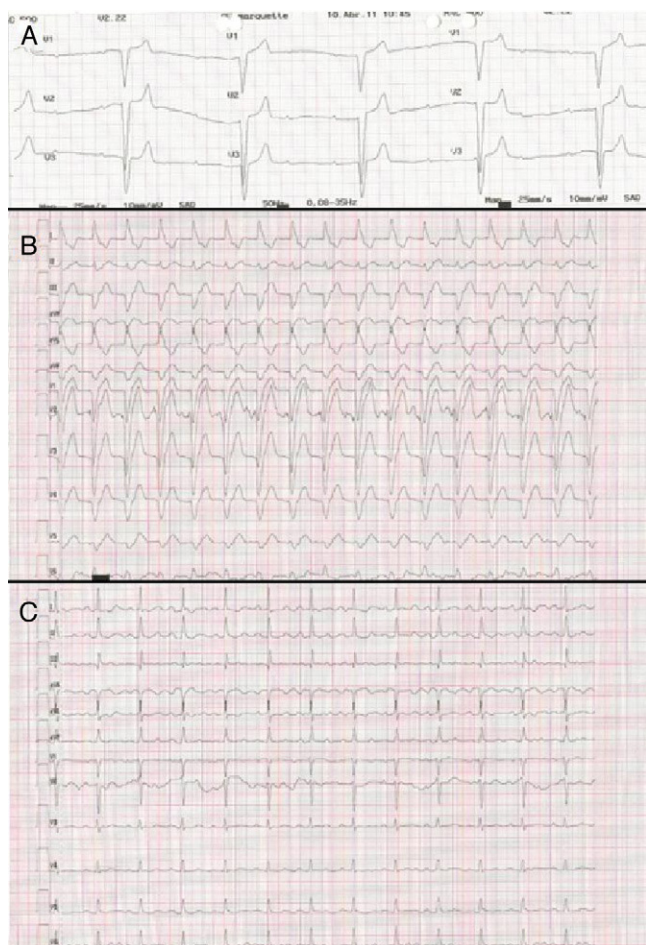
Figure 1. A, Third-degree atrioventricular block with widened QRS intervals and escape rhythm. B, A few hours after implantation of a temporary pacemaker, first-degree atrioventricular block is visible alongside complete left bundle-branch block. C, Three days later, the electrocardiogram reveals first-degree atrioventricular block with narrowed QRS intervals.
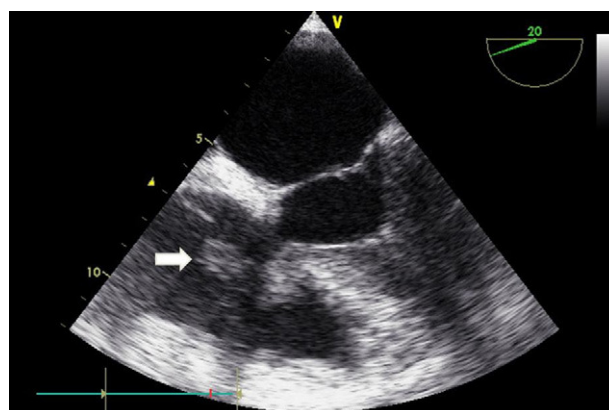
Figure 2. Transesophageal echocardiogram revealing a large vegetation on the free edge of the tricuspid valve (white arrow) and a smaller vegetation on the septal leaflet.

the conduction pathways and are generally associated with perivalvular complications. Third-degree AVB is most often associated with disease affecting the left valves (aortic, 36%; mitral, 33%) and cases of tricuspid endocarditis are rare (2%).[1] This is because of the anatomical relationship with the atrioventricular node, which is situated at the centre of Koch's triangle, close to the noncoronary aortic leaflet and the anterior mitral leaflet; the tricuspid septal leaflet, on the other hand, is located at the base of Koch's triangle. In a study of patients with endocarditis and complete AVB, pathology workup revealed the presence of an infection, usually accompanied by abscesses and fistulas, that affected the conduction pathways; in cases involving paroxysmal AVB, inflammation was observed at this level, which would explain the reversibility of the event.[2] Electrocardiographic monitoring is important in patients with endocarditis, as the appearance of conduction abnormalities can alert us to the appearance of perivalvular complications; similarly, once a conduction block has been detected, its disappearance in less than a week indicates a good response to antibiotic treatment.[3]

Right-sided endocarditis is an underdiagnosed condition, probably due to its atypical clinical presentation,[4] although vegetations can be readily observed even by transthoracic echocardiography.[5] The case described here is exceptional in that it involved isolated tricuspid endocarditis appearing with complete AVB and left bundle-branch block that was reversed upon treatment with antibiotics. Cases of conduction abnormalities in association with tricuspid valve endocarditis have rarely been described in the literature, although there appears to be a tendency for the block to be reversed with medical treatment, without the need to implant a permanent pacemaker or resort to surgery.[6]

Noelia Martínez-Urueña,* Carolina Hernández, Iria C. Duro, María G. Sandín, Eduardo Zatarain, and Alberto San Román

Instituto de Ciencias del Corazón (ICICOR), Hospital Clínico, Valladolid, Spain

* Corresponding author:
E-mail address: noemu83@hotmail.com (N. Martínez-Urueña).

REFERENCES

1. DiNubile MJ, Calderwood SB, Steinhaus DM, Karchmer AW. Cardiac conduction abnormalities complicating native valve active infective endocarditis. Am J Cardiol. 1986;58:1213–7.
2. Wang K, Gobel F, Gleason DF, Edwards JE. Complete heart block complicating bacterial endocarditis. Circulation. 1972;46:939–47.
3. Dinubile MJ. Heart block during bacterial endocarditis: a review of the literature and guidelines for surgical intervention. Am J Med Sci. 1984;287:30–2.
4. Revilla A, López J, Villacorta E, Gómez I, Sevilla T, Del Pozo MA, et al. Endocarditis derecha aislada en pacientes no adictos a drogas por vía parenteral. Rev Esp Cardiol. 2008;61:1253–9.
5. Shah PM. Valvulopatía tricuspídea y pulmonar: evaluación y tratamiento. Rev Esp Cardiol. 2010;63:1349–55.
6. Fordyce CB, Leather RA, Partlow E, Swiggum EA. Complete heart block associated with tricuspid valve endocarditis due to extended spectrum β-lactamase-producing *Escherichia coli*. Can J Cardiol. 2011;27:263.e17–20.

Percutaneous Closure of Mitral Paravalvular Leaks in Patients With Aortic Valve Prostheses

Cierre percutáneo de fugas perivalvulares mitrales en pacientes con prótesis metálica aórtica

To the Editor,

Paravalvular leaks (PVL) develop after implantation of prosthetic heart valves in 2% to 12% of the cases, and the standard treatment is repeat valve replacement.[1] Nevertheless, although the subject is controversial,[2] it has recently been shown that it is feasible to apply percutaneous closure in a growing number of patients.[3–5] The percutaneous closure of mitral PVL is more complex than that of aortic leaks since it is necessary to establish a circuit with a long guide wire along which the delivery sheath is advanced.[3–5] In patients with mitral PVL who also have a metallic aortic valve prosthesis, this device adds to the difficulty because the guide wire can interfere with the aortic prosthesis and complicate the procedure. For this reason, some surgeons and interventionalists opt for the transapical approach.[6]

In our center, percutaneous closure of a mitral PVL has been successfully performed in 2 patients who also had metallic aortic valve prostheses. The first case was that of a 79-year-old woman with heart failure in New York Heart Association functional class IV, who had undergone valve surgery 18 years earlier. She had a normally functioning 21-mm Björk prosthetic valve in aortic position and a 27-mm Björk prosthetic valve in mitral position, with posterior dehiscence that produced severe periprosthetic regurgitation. In addition, she had severe left ventricular dysfunction, chronic atrial fibrillation, and a history of stroke with sequelae, and had had placement of an implantable cardioverter defibrillator. The estimated surgical risk was 17% according to the EuroSCORE and 18% according to the Society of Thoracic Surgeons score. In this patient, percutaneous closure of the PVL was carried out successfully and without complications; it involved the implantation, in 2 separate procedures, of a 14/5-mm and an 8/3-mm Amplatzer Vascular Plus III occluder, respectively.

The second patient was a 74-year-old woman who, 26 years earlier, had undergone the implantation of a 21-mm Omnicarbon aortic valve prosthesis, which was functioning normally, and a 27-mm Omnicarbon mitral valve prosthesis with posterior dehiscence and moderate regurgitation. She had moderate heart failure (New York Heart Association functional class II) and severe hemolysis requiring frequent blood transfusions. She also had a history of stroke with sequelae and presented with atrial fibrillation. Her EuroSCORE was 13% and her Society of Thoracic Surgeons score, 7%. Percutaneous closure with a 10/3-mm Amplatzer Vascular Plug III occluder (AGA Medical) was carried out successfully and without complications.

In both patients, the procedure was performed under general anesthesia using 3-dimensional transesophageal echocardiography (Figure). Transseptal puncture was carried out using the standard technique (Müllins sheath and Brockenbrough needle). Once access to the left atrium had been gained, a 6-Fr angiography catheter was introduced to enable access to the left ventricle using a hydrophilic guide wire. After crossing the dehiscence, the guide wire was advanced through the aortic valve prosthesis to the descending aorta where, following retrograde mobilization of the loop system from the femoral artery, it was entrapped and externalized through the femoral artery. Once this circuit had been established, the delivery sheath was advanced, together with its dilator, through the interatrial septum, the PVL, and left ventricle.

The Vascular Plug III occluder was then advanced to left ventricle, where the ventricular portion was released and, after it had been withdrawn to the level of the annulus of the mitral valve prosthesis, was released in its entirety, and correctly placed in all 3 procedures.