mesenchymal tumours, cutaneous lentiginosis, hyperfunctional endocrine disorders, and tumors of the peripheral nerves.[3] Symptoms depend on location, size, and mobility of the tumor. Symptoms may be due to obstruction of the ventricle, with the onset of dyspnea, secondary to pulmonary edema, syncope, and even sudden death. Systemic embolization occurs in 25% to 40% of cases due to tumor fragmentation, with the brain being the most frequent destination. Limb embolisms, as in our case, in visceral, coronary, and renal arteries are rare. Complete embolization of the tumor has also been reported. Patients may present with symptoms including fever, skin rash, joint pain, and weight loss, as a result of the production of cytokines (interleukin-6) and growth factors by the myxoma.[4]

Transthoracic echocardiography is the method of choice for the diagnosis of cardiac myxomas. However, transesophageal ultrasound has greater sensitivity (almost 100%) than transthoracic ultrasound (90%).[5] The treatment of choice is surgical resection even in asymptomatic patients. It must be performed very soon after diagnosis due to the risk of embolism and sudden death. Surgical mortality varies from 2% to 5%, and the prognosis after resection is excellent. Recurrence of these tumors varies from 0.4% to 5% at 22 years of treatment. Therefore, follow-up with echocardiography is recommended at least every 5 years. Cardiac myxomas have greater recurrence in young males, multifocal cases, and familial forms.[6] In these patients, follow-up must therefore be more stringent.

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Severe Community-Acquired Methicillin-Resistant *Staphylococcus aureus* Endocarditis in a Child With Structurally Normal Heart: a Case Report

Endocarditis bacteriana grave por Staphylococcus aureus *resistente a meticilina adquirida en la comunidad en un niño con corazón estructuralmente normal*

To the Editor,

A 2-year-old male infant was admitted to the hospital with a history of rapid decline in general health and persistent hyperthermia (38.5 °C to 39 °C) over the previous 6 days. No history of congenital heart disease or previous hospitalization was reported by parents. General inspection revealed eutrophic child, easily irritable, and small pustular lesions over both legs. Rapid pulse (120 bpm), dry and pale mucosa, and bilateral subconjunctival hemorrhagic spots were noted. Large erythematic area over the right hypochondrium, diffuse petechial exanthema over the lower limbs, and left proximal forefinger interphalangeal joint swelling were also observed (Fig. 1).

Cardiac examination revealed hyperdynamic precordium and 3+/4+ systolic murmur was heard over the apex irradiating to the left axilla and posterior torso.

Laboratory findings on admission were white blood cell count 24 500/μL with 25% rod-like neutrophil, platelet 21 000/μL, hematocrit 24.7%, creatinine 0.4 mg/dL, GGT 54 IU/L, alkaline phosphatase 518 IU/L, sodium 130 mEq/L, and potassium 2.9 mEq/L. Bidimensional color Doppler echocardiogram with parasternal short axis and apical long axis views showed left chambers enlargement (left ventricular diameter 37 mm diastolic and 23 mm systolic). A filamentary vegetation was observed extending from the ventricular aspect of the anterior mitral valve leaflet to the left ventricular outflow tract adherent to the noncoronary aortic cusp (Figs. 2A and B; Movies 1 and 2). Severe mitral and mild aortic regurgitations were also present (Movie 3).

An acute infective endocarditis (IE) was thus considered and before starting an empiric antibiotic schedule (ceftriaxone, oxacillin, and vancomycin), two blood specimens were cultured. A surgical intervention was considered but disapproved by the cardiac team.

In 48 h, metabolic acidosis, respiratory distress with sign of spontaneous bleeding from lung parenchyma, and anisocoria evolved. New echocardiogram showed that sub-aortic filamentary vegetation was no longer present and right ventricle was dilated. A new image adhered to the septal tricuspid valve leaflet was observed (Figs. 2C and D; Movies 4 and 5). The child evolved with bilateral mydriasis, progressing to death.

Staphylococcus aureus (*S. aureus*) was isolated from all blood samples, and was sensitive to vancomycin, clindamycin, and sulfamethoxazole-trimethoprim and resistant to both oxacillin and penicillin.

Current report describes a case of aggressive IE caused by *S. aureus* involving 3 cardiac valves in an otherwise healthy male infant. In children, IE is commonly associated with congenital or rheumatic heart disease.[1] In healthy children, bacteremia secondary to skin infection or due to central catheters invasion, particularly in neonates, is associated with IE, and *S. aureus* has been the most frequent infective agent isolated from blood cultures.[1,2] Community-acquired methicillin-resistant *S. aureus* (CA-MRSA) is a rapidly growing worldwide problem.[3] Reassuringly, the prevalence of CA-MRSA colonization in a community's young children remains very low[4]. Milstone et al. reported a 61% CA-MRSA prevalence among all MRSA-colonized children in a pediatric intensive care unit.[5] Jaggi et al. reported that invasive CA-MRSA represented 12.7% of all MRSA strain isolated in pediatric ICU, but none related to IE.[6] Children with CA-MRSA infections

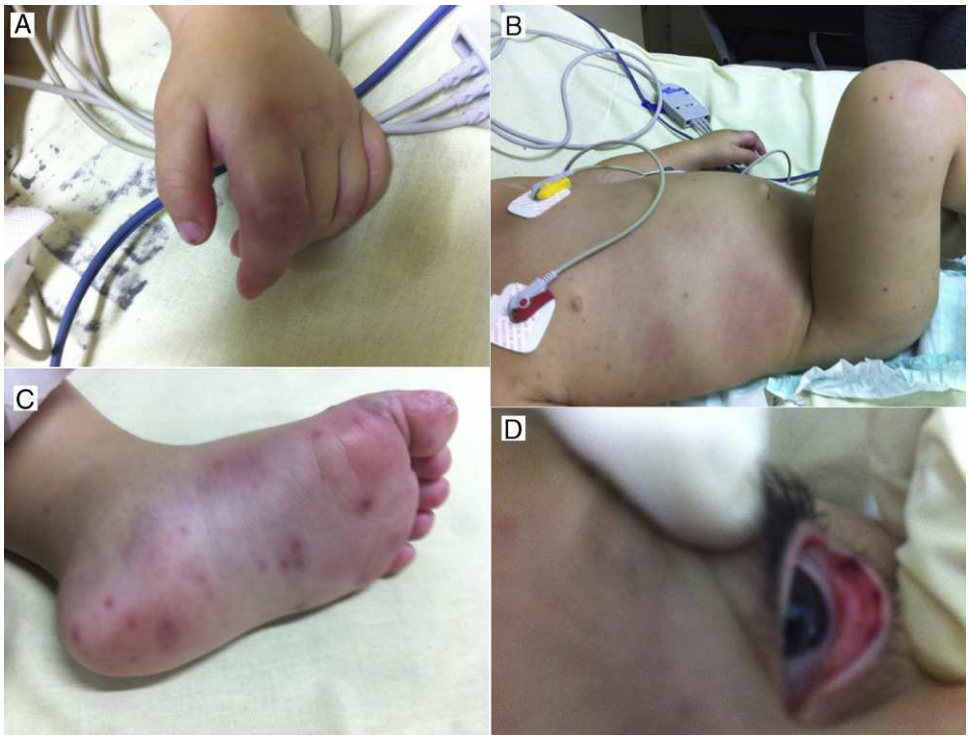

Figure 1. A, left forefinger, proximal interphalangeal joint swelling. B, erythematic spot over the right hypochondrium and diffuse petechial exanthema over the right lower limbs. C, petechial exanthema over the right foot. D, subconjunctival hemorrhage spots.

tended to be younger African-American descendents and admitted to the hospital 1 week after initial infection.[5,6] Markers of CA-MRSA are no hospitalization, no dialysis or surgery, no exposure to interventional procedures, and neither MRSA colonization nor confinement in care or performing centers in the previous year, and isolated *S. aureus* susceptible to a wide range of antibiotics, including sulfamethoxazole-trimethoprim and clindamycin.[3] Over 95% of CA-MRSA strains carry toxins-encoding genes (eg, Pantone-Valentine), predisposing to severe soft-tissue infections and necrotizing pneumonia.[3]

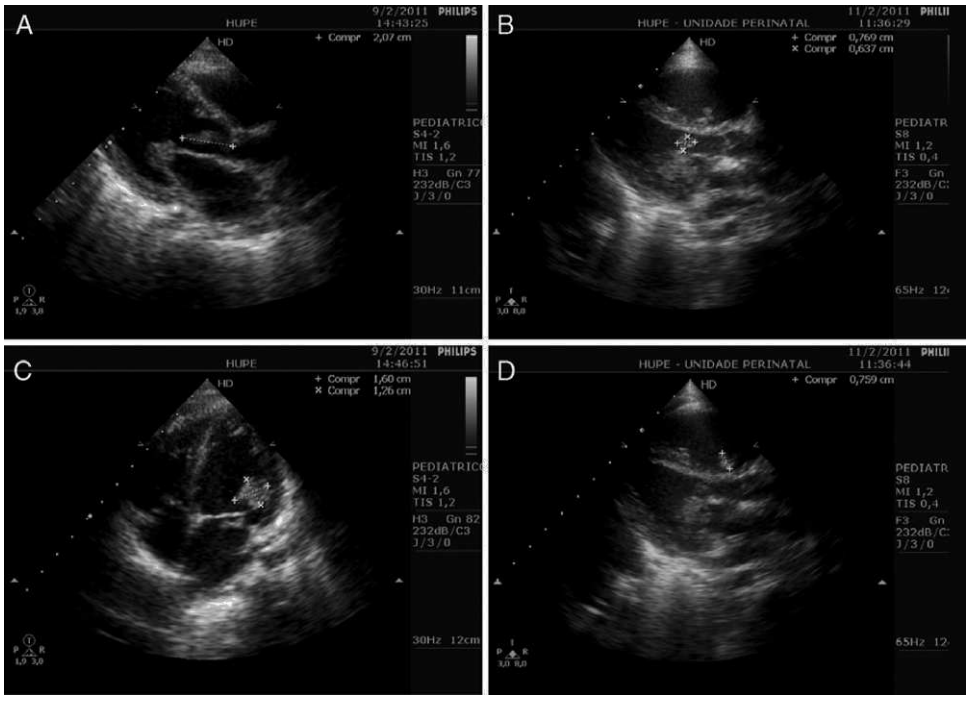

Figure 2. A, subaortic filament-shaped vegetation, extending from the anterior mitral valve leaflet to the noncoronary aortic cusp (marks). B, disc-shaped vegetation, attached to the anterior mitral valve leaflet (marks). C, large vegetation over the ventricular aspect of the anterior mitral valve leaflet and subvalve apparatus. Subaortic filamentary vegetation was no longer observed, and a tiny portion remained attached to the mitral valve (marks). D, disc-shaped vegetation, attached to the septal tricuspid valve leaflet (marks).

The history of no previous hospitalization, findings of diffuse petechial exanthema and abdominal spot, and isolation from blood cultures of MRSA sensitive to sulfamethoxazole-trimethoprim confirmed this rare case of CA-MRSA IE. The extensive vegetation affecting mitral, aortic, and tricuspid valves and the clinical signs of necrotizing pneumonia and severe systemic embolization are evidence of the virulent strain of the infective agent in this case.

No clear recommendation can be obtained from the current case, owing to the fatal outcome. Jaggi et al. successfully employed surgical interventions in CA-MRSA infections not associated with IE, with no deaths.[6] High diagnostic suspicion and early surgical intervention in selected cases may be beneficial.

SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.rec.2011.06.011.

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Ventricular Fibrillation, an Emergency in Electrophysiology

La fibrilación ventricular, una emergencia electrofisiológica

To the Editor,

We present the case of a 78-year-old woman who was admitted to our service with a diagnosis of anteroseptal myocardial infarction. Angiography revealed severe stenosis of left main coronary artery and a critical lesion in proximal left anterior descending coronary artery. Following successful percutaneous revascularization of these lesions, the patient, who was stable and asymptomatic, was transferred to the coronary care unit. A few hours later, she began to have repeated episodes of ventricular fibrillation (VF). She was treated with beta blockers and amiodarone and underwent deep sedation, orotracheal intubation and implantation of an intraaortic balloon pump for counterpulsation. Despite all these measures, it was not possible to control the electrical storm, and external defibrillation was necessary on more than 100 occasions during the first 12 h.

Although this complication occurred over the weekend, given the instability of the patient the decision was made to discuss the case with the electrophysiology team. Upon reviewing the results of the telemetric study, we observed that each episode of VF began with a monomorphic premature ventricular complex (PVC) (Fig. 1), with a QRS width of only 120 ms. Thus, suspecting that

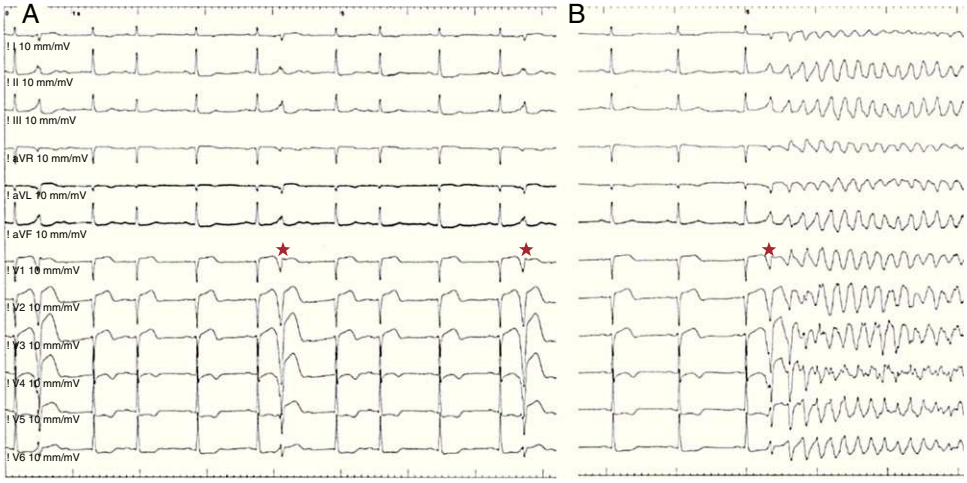

Figure 1. A, high-density monomorphic premature ventricular complex (asterisks). B, the premature ventricular complexes induce repeated ventricular fibrillation episodes.