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 metilicina 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 erythematous
area over the right hypochondrium, diffuse petechial exanthema
over the lower limbs, and left proximal forehead interphalangeal
ging 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 second-
ary 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

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tended to be younger African-American descendents and admitted to the hospital 1 week after initial infection.\textsuperscript{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\textit{S. aureus} susceptible to a wide range of antibiotics, including sulfamethoxazole-trimethoprim and clindamycin.\textsuperscript{3} Over 95\% of CA-MRSA strains carry toxins-encoding genes (eg, Pantone-Valentine), predisposing to severe soft-tissue infections and necrotizing pneumonia.\textsuperscript{3}

\begin{figure}[h]
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\includegraphics[width=\textwidth]{figure1.png}
\caption{A, left forefinger, proximal interphalangeal joint swelling. B, erythematous 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.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure2.png}
\caption{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).}
\end{figure}
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. 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 counterpulsion. 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.