Acute Myocarditis Versus Ventricular Noncompaction Cardiomyopathy in Infancy: Role of Magnetic Resonance

Miocarditis aguda frente a miocardiopatía no compactada en el lactante: utilidad de la resonancia magnética

To the Editor,

Acute myocarditis is an inflammatory process affecting the myocardium that mainly occurs secondary to a viral infection. The definitive diagnosis is established by endomyocardial biopsy, but because this method is invasive, it is performed only in selected patients. A presumptive diagnosis can be reached by taking a clinical history and performing noninvasive complementary tests. Among these, cardiac magnetic resonance (CMR) imaging plays an important role, and experience with this technique is increasing in children. CMR can detect the tissue changes inherent to myocarditis, such as edema, hyperemia, and necrosis. To establish the diagnosis, at least 2 of the 3 CMR criteria must be fulfilled. The sensitivity of the test varies according to the clinical pattern: sensitivity is very high in the forms presenting with precordial pain, but is lower in patients with dilatation and ventricular dysfunction. In these patients, the differential diagnosis should be made with other conditions such as dilated cardiomyopathy or noncompaction cardiomyopathy. This latter disease can present with a pattern of heart failure at any age, and the diagnosis is established on the basis of echocardiographic and CMR criteria. We present a series of 3 patients admitted to our center between April 2015 and September 2016 with clinical signs and symptoms of cardiogenic shock. The patients’ characteristics are described in Table. All required inotropic support and mechanical ventilation. After they had been stabilized, the patients were extubated, and standard heart failure therapy was initiated. The first 2 patients had experienced an infectious process before symptom onset. At admittance they underwent electrocardiography, blood analysis to determine myocardial injury markers, and polymerase chain reaction (PCR) testing for cardiotropic viruses in blood and respiratory secretions. CMR was performed during the first 3 days following admittance, with the patients under sedation and breathing spontaneously. Only 1 patient tested PCR-positive in a blood sample, with detection of parvovirus B19 (Table). All 3 patients had left ventricular dilatation and hypertrabeulation, and they met the diagnostic criteria of noncompaction cardiomyopathy both on CMR and echocardiography. There was a good correlation between the ejection fraction determined by CMR and the estimated value obtained with the Teichholz formula on echocardiography (Table). A hyperintense signal on CMR T2 sequences and increased early gadolinium uptake were seen in all patients. Of note, both the hyperintensity and signal increase in the early phase following gadolinium administration were localized at the trabeculated region, but did not reach the compacted myocardium (Figure). In the second and third patient, the right ventricle also showed considerable trabeculation. In the first patient, who tested PCR-positive for parvovirus B19 in blood and had a poor clinical course, endomyocardial biopsy was carried out. The results were normal, and diagnosis of acute myocarditis was ruled out. Thus, the diagnostic orientation in the 3 patients was noncompaction cardiomyopathy with severe ventricular dysfunction.

At the time of writing, the 3 patients have been stable, show moderate-severe ventricular dysfunction, and are receiving heart failure treatment.

In conclusion, we wish to convey the usefulness of CMR for the etiological diagnosis of ventricular dysfunction in pediatric patients. This technique should be among the first to be used in these patients, as it can avoid the need for invasive examinations that are not without risk, such as endomyocardial biopsy. This procedure should be carried out in selected patients, particularly in the pediatric population. In infants with noncompaction cardiomyopathy and severe left ventricular dysfunction, the differential diagnosis with acute myocarditis can be challenging. Trabeculation appears hyperintense on T2 images and slow flow due to severe

César Caro Martínez,a,* José M. Andreu Cayuelas,b Ginés Elvira Ruiz,c Helena Albellén Iglesias,c Arcadio García Alberola,b,d and Sergio Manzano Fernándezb,d

aServicio de Cardiología, Hospital Vega Baja, Orihuela, Alicante, Spain
bServicio de Cardiología, Hospital Clínico Universitario Virgen de la Arrixaca, Instituto Murciano de Investigación Biosanitaria, El Palmar, Murcia, Spain
cServicio de Medicina Interna, Hospital Clínico Universitario Virgen de la Arrixaca, El Palmar, Murcia, Spain

dDepartamento de Medicina Interna, Facultad de Medicina, Universidad de Murcia, Murcia, Spain

* Corresponding author: E-mail address: ccaro1980@gmail.com (C. Caro Martínez).

Available online 7 June 2017

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http://dx.doi.org/10.1016/j.rec.2017.03.032

1885-5857
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ventricular dysfunction may lead to gadolinium retention in the intertrabecular spaces. The absence of the characteristic myocarditis pattern of edema and patchy subepicardial hyperemia, and the lack of late contrast uptake can guide the diagnosis toward ventricular dysfunction secondary to noncompaction cardiomyopathy.

Ferran Gran,⁎,⁎⁎ Maria Martínez-Villar,⁎ Anna Sabaté-Rotés,⁎ Amparo Castellote,⁎ Ferran Rosés-Noguer,⁎ and Dimpna C. Albert⁎

⁎Unidad de Cardiología Pediátrica, Hospital Universitario Materno-Infantil Vall d’Hebron, Universidad Autónoma de Barcelona, Barcelona, Spain
⁎⁎Servicio de Pediatría, Hospital Vega Baja, Orihuela, Alicante, Spain
⁎⁎⁎Servicio de Radiología, Hospital Universitario Materno-Infantil Vall d’Hebron, Universidad Autónoma de Barcelona, Barcelona, Spain

⁎ Corresponding author:
E-mail address: fgranipina@gmail.com (F. Gran).

Available online 18 May 2017

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http://dx.doi.org/10.1016/j.rec.2017.04.027
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