Clinical Case

Reverse Takotsubo cardiomyopathy in a pediatric patient: atypical presentation of reversible ventricular dysfunction.

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ABSTRACT

Takotsubo syndrome (TTS) is a transient cardiomyopathy that mimics acute myocardial infarction in the absence of obstructive coronary artery disease. Its "reverse" form, characterized by basal systolic dysfunction with apical preservation, is rare in pediatric patients but may result in significant morbidity and even mortality. We report the case of a 9-year-old boy with reverse TTS who was admitted due to severe hypertension, bradycardia, and acute neurological deterioration with decreased alertness. Echocardiographic and magnetic resonance imaging tests confirmed the basal pattern of myocardial dysfunction, with complete recovery within 20 days. This case underscores the need to consider reverse TTS in the differential diagnosis of acute pediatric heart failure, particularly in the setting of possible adrenergic crises, and highlights the importance of early diagnosis and multidisciplinary follow-up.

INTRODUCTION

Takotsubo syndrome (TTS), also called stress cardiomyopathy, is a transient myocardial dysfunction resembling acute coronary syndrome, but with no evidence of obstructive CAD, and is characterized by transient systolic dysfunction of the left ventricle with anomaly of parietal motion.[1] Initially described in 1990 in Japan, its pathophysiology is still not fully understood, although the participation of a sudden increase in catecholamines is acknowledged as a central factor in its development, and can be associated to multiple emotional and physical triggers.[2]

Although TTS is more frequent in postmenopausal women, in the last two decades it has been reported in men, teenagers and children.[1,3] In the pediatric population, it is a rare entity but with a potential severe hemodynamic compromise and risk of associated mortality. In comparison to adults, pediatric patients present atypical pattern of ventricular involvement more frequently, as well as a higher proportion of affected males.[1,3]

The classical form represents 80% of cases and presents apical region hypokinesis.[2] The “inverse” or basal subtype of TTS represents a rare morphological pattern, characterized by hypokinesis of the basal and midventricular segments with apical preservation, and has been mainly linked to emotional, physical, neurological or endocrine stressors.[4,5] Adrenergic crises, as those induced by external stress or pheochromocytoma, are one of the main triggers of this subtype.[6,7]

Recent evidence suggests that the inverse subtype could be related to a differential distribution of β-adrenergic receptors in the myocardium, with the basal segments being more sensitive to sympathetic stimulation in young people.[4,8] Moreover, it has been proposed that transient myocardial inflammation would also contribute to the ventricular dysfunction observed in the acute phase of TTS.[9]

This report describes a case of TTS in a pediatric patient, emphasizing his clinical presentation, diagnostic evaluation and favorable evolution, with the aim of contributing to the scarce existing literature about this population.

CASE PRESENTATION

Male, 9-year-old patient, with history of erosive lichen planus, in treatment with weekly methotrexate. As relevant family history, myofibrillar myopathy stands out, secondary to amines deposits in grandfather and aunt, and presence of spontaneous bilateral carotid dissection in his mother, with good course.

The patient presented clinical symptoms characterized by nausea, sweating and fever episode. Within 24 hours, in a scenario of a family argument and a fight during an online videogame play, he evolved with intense headache and two episodes of syncope. Testing in the ER yielded the following upon admission: Glasgow score 10/15, blood pressure 185/145 mmHg, 40 bpm bradycardia, glycemia of 150 mg/dl and ECG with sinus bradycardia and negative T waves in V1, V2, V3. Two doses of atropine were administered with no clinical response, and due to acute neurological deterioration (Glasgow <8), orotracheal intubation was carried out; he was admitted in the pediatric ICU, and invasive monitoring of blood pressure was started. Sedoanalgesia was applied with fentanyl, as well as inotropic treatment.

The initial laboratory tests showed leukocytosis, with no other alterations. Blood, urine and cerebrospinal fluid cultures were negative, just as toxicological tests. He received empirical treatment with 3 doses of ceftriaxone. Infectious causes were

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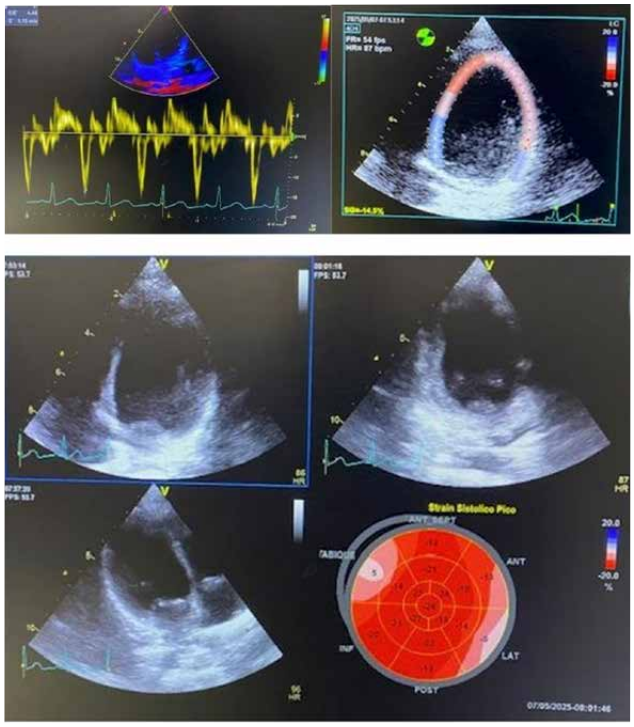

FIGURE 1.
Ecocardiograma control a las 24 hs

ruled out (negative viral serology), as well as endocrinological, neurological and nephrologic causes. CT and brain MRI were normal.

The initial echocardiogram showed a shortening fraction of 13% and ejection fraction of 28% in a context of global myocardial dysfunction, with troponin <2 ng/l and CPK MB of 22 U/l.

In control echocardiogram within 24 hours, he showed hypocontractility of basal sectors with preserved apical contractility (basal shortening fraction 18% and apical shortening fraction 29%), and normal origin of coronary arteries, with no heart valve diseases *(Figure 1)*. Cardiac MRI confirmed the pattern of basal hypocontractility with apical preservation, with no late enhancement or signs of myocarditis, findings compatible with inverse Takotsubo syndrome *(Figure 2)*.

The troponin curve showed a peak of 889 ng/l within 24 h after admission; ProBNP reached 2418 pg/ml within 36 h after admission; and normal electrocardiogram within 8 h after admission.

Twenty-four hours after admission, with clinical and hemodynamic improvement, scheduled extubation was achieved and sedoanalgesia was suspended. Inotropic support was suspended 48 h after admission, coinciding with progressive troponin decrease (31 ng/l) and ProBNP (465 pg/m). Echocardiogram before discharge showed recovery of ventricular function (baseline shortening fraction 21%, apical shortening fraction 26%).

The patient was discharged 6 days after admission, under treatment with enalapril to reduce postload, and outpatient follow-up through the Pediatric Cardiology Service. The patient presented normal catecholamines. Control 15 days after discharge presented echocardiogram with normalization of cardiac function (basal shortening fraction 36%, apical shortening fraction 43%) *(Figure 3)*.

DISCUSSION

Inverse Takotsubo cardiomyopathy represents an atypical form of presentation, with predominant basal compromise of the left ventricle and preserved apical contractility.[4] This pattern is more commonly observed in young patients, including teenagers and children, and has been linked to adrenergic crisis, acute neurostimulation or pheochromocytoma that could be triggered by emotional stress.[5,6,7]

In pediatrics, TTS is associated to more severity upon admission, with higher rates of cardiogenic shock, and lower ejection fractions in comparison to adults. A multicenter study showed that more than 50% of pediatric patients were males, contrasting with the predominantly female pattern in adults.[1] Furthermore, in pediatrics, physical triggers (neurological diseases, severe infections) were the main triggers in more than 90% of cases.[1,3] In this case, no clear stressor factor was identified, but it was associated to a context of family argument and virtual argument in a videogame.

As to pathophysiology, it is proposed that a massive discharge of catecholamines generates transient myocardial dysfunction through direct toxicity on the myocytes, coronary vasospasm,

FIGURE 2.
Imágenes de resonancia cardíaca

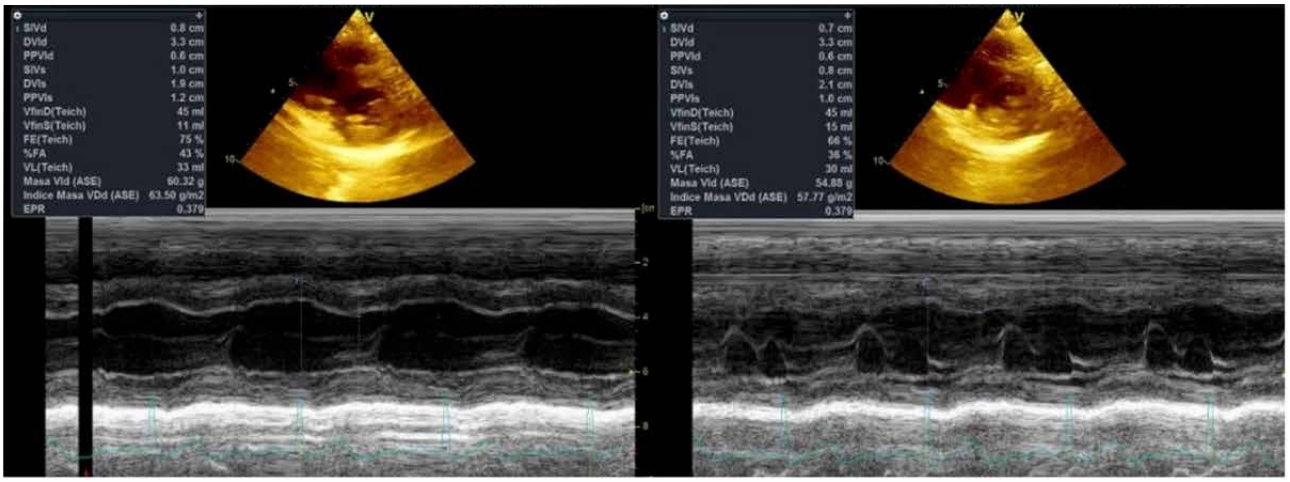

FIGURE 3.
Ecocardiograma a los 15 días.

microvascular dysfunction and adrenergic desensitization.[2,4,7] The adrenergic trafficking model explains how β2 overstimulation may induce a regional negative inotropic response, particularly in the segments with greater density of receptors, like the ventricular base in young individuals.[8]

Inverse TTS has been described in association with pheochromocytomas and catecholamine-secreting tumors, in which sympathetic hyperstimulation generates a characteristic distribution of ventricular dysfunction.[6,7,10] Yuan et al., reported a case of pheochromocytoma rupture with presentation like basal TTS, emphasizing the need to consider this pathology in differential diagnosis.[10]

On the other hand, the more recent studies have explored the role of systemic and myocardial inflammation. Scally et al showed that, by MRI and inflammatory markers, in the acute phase of TTS there is a significant inflammatory response, both local and systemic, that may contribute to ventricular dysfunction.[9]

The clinical presentation of inverse TTS may resemble other causes of acute heart failure, such as myocarditis, congenital heart diseases, or intoxication. The absence of coronary lesions, moderate elevation of biomarkers, echocardiography with regional patterns, and a rapid recovery of ventricular function guide the diagnosis of TTS.[2,4,11,12]

Management is essentially support treatment: therapy for heart failure and therapy with long-term beta blockers are recommended in selected scenarios.[4,11,13] Full recovery usually occurs within days or weeks, although in some cases recurrence has been documented, including the inverse subtype.[8,9] In this patient, ventricular function recovery took twenty days, and he remained medicated with enalapril for a month.

This case contributes to the growing literature on TTS in pediatrics, highlighting the significance of an early identification and multidisciplinary approach. In spite of its rarity, it should be included in the differential diagnosis of acute ventricular dysfunction in children, particularly in a scenario of adrenergic crises.

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