cardiographic pattern induced by a clearly identifiable condition that reverts after correction, low pretest probability of Brugada syndrome, and negative results in induction and genetic tests.¹ However, in this particular patient, we believe that the fact that inflammatory syndrome plus fever were considered a potential reversible condition when diagnosing Brugada phenocopy deserves special attention. The clinical conditions traditionally reported to exhibit Brugada pattern in phenocopies are related to: a) metabolic conditions: b) mechanical compression: c) ischemia and pulmonary embolism; d) myocardial and pericardial disease, and e) electrocardiogram-amplitude modulation.⁴ Fever is not usually listed among the conditions, as elevated body temperature outside the physiologic range is considered a genuine trigger for uncovering true Brugada patterns because the arrhythmogenic potential of cardiac sodium channels is increased at high temperatures.^{5,6} Additionally, from a clinical rather than pathophysiologic standpoint, numerous reports and case series have shown that patients with fever that induced a Brugada-like electrocardiogram have later experienced malignant arrhythmia.⁷

Induction and genetic tests were used in this patient to support the Brugada phenocopy diagnosis. However, the sensitivity of induction tests is only 70% to 80%, and this specific patient experienced slight QRS widening after flecainide administration, although with no clear ST-segment changes.⁸ Only a few Brugada syndrome cases are due to known gene mutations, and patients who have exhibited Brugada pattern in the context of fever could later experience malignant arrhythmia despite no genetic predisposition.^{7,9} Therefore, in the event of an unusual predisposing factor for the diagnosis of Brugada phenocopy, as seen on this occasion, the ability of these tests to define the condition is somewhat debatable, as negative results are not too useful. An alternative diagnostic approach could include serial tests with increasing doses of sodium channel blockers to improve the diagnostic accuracy of provocation tests if there is diagnostic uncertainty.

Accurate differentiation between Brugada syndrome and Brugada phenocopies has major prognostic implications for patients. The diagnostic process should include a careful assessment of all established criteria for both entities while considering that the current information available seems to indicate that triggering factors of Brugada-like electrocardiographic pattern seem to have varying molecular and pathophysiologic consequences as well as different implications for the clinical prognosis. This will ensure an appropriate approach to therapy and follow-up for each individual patient.¹⁰

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A.F. Miranda-Arboleda and J.M. Farina participated in the article text and subsequent corrections. A. Baranchuk had the idea for the article and reviewed the final version.

CONFLICTS OF INTEREST

None.

Andrés Felipe Miranda-Arboleda,^{a,b} Juan María Farina,^c and Adrian Baranchuk^{a,*}

^aDivision of Cardiology, Electrophysiology and Pacing, Queen's University, Kingston Health Sciences Centre, Kingston, Ontario, Canada

^bDepartamento de Cardiología, Hospital Pablo Tobón Uribe, Medellín, Antioquia, Colombia

^cDepartment of Cardiovascular and Thoracic Surgery, Mayo Clinic, Phoenix, Arizona, United States

* Corresponding author:

E-mail address: barancha@kgh.kari.net (A. Baranchuk).

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Fever and Brugada electrocardiographic pattern. Response

Fiebre y patrón electrocardiográfico de Brugada. Respuesta

To the Editor,

First of all, we would like to thank Miranda-Arboleda et al. for their interest in our article.¹ Their thoughtful comments have given us the opportunity to clarify certain clinical aspects of our case that could affect its interpretation.

Indeed, the case report describes electrocardiographic (ECG) findings consistent with a Brugada pattern in the context of fever and clinical findings compatible with COVID–19-associated multisystem inflammatory syndrome in children (MIS-C). The pattern, however, persisted for approximately 4 days after the fever had subsided. While fever is a precipitating factor for ECG abnormalities in Brugada syndrome, afebrile patients with this disorder often have normal findings.² The Brugada pattern disappeared on day 5 of admission, although the ECG did show repolarization abnormalities, possibly indicative of myocardial involvement.

MIS-C can cause inflammation and myocardial involvement³ with reduced ejection fraction and increased N-terminal pro-B-type natriuretic peptide (NT-proBNP) and troponin levels (observed in our patient). This myocardial involvement could be in keeping with conditions that have been linked to Brugada phenocopy. The improvements in cardiac function and clinical and biochemical parameters (reduction in NT-proBNP and troponin levels) in our patient all coincided with the disappearance of the Brugada pattern.

Because the boy had a Brugada type 1-like pattern that resolved on improvement of the acute inflammation, in addition to a negative flecainide challenge and genetic study and, above all, a very low pretest probability of Brugada syndrome (no compatible clinical manifestations or family history of the syndrome), we believe he met al.l the criteria for Brugada phenocopy. We also believe, however, that close long-term monitoring is warranted, and if the patient shows any additional symptoms, we will, as suggested, perform a provocation test with incremental doses of sodium channel blockers, assuming of course that the benefits outweigh any possible risks.

AUTHORS' CONTRIBUTIONS

Lead author: R. Santiago-Cortés. Revision and supervision: M. Clavero Adell, D. Palanca Arias, and A. Ayerza Casas.

CONFLICTS OF INTEREST

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Rebeca Santiago-Cortés,* Marcos Clavero-Adell, Daniel Palanca-Arias, and Ariadna Ayerza-Casas

Departamento de Cardiología Pediátrica, Hospital Universitario Miguel Servet, Zaragoza, España

* Corresponding author:

E-mail address: rebeca.sancor91@gmail.com (R. Santiago-Cortés).

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Clinical management in cardiology. Extended hospitalization

Gestión clínica en cardiología. La hospitalización extendida

To the Editor,

Care outcomes in cardiology inpatient units do not depend exclusively on their internal functioning. The main quality indicators of hospitalization units are determined by the influence of external factors that are dependent on other cardiology service units, other hospital services, the emergency department, or the organization of care for chronic cardiovascular disease.¹ From this perspective, it seems reasonable to suggest that it should be the hospitalization units themselves that, by extending their boundaries, promote and participate in the implementation of strategies to positively influence these factors. This approach is what we define as "extended hospitalization" (figure 1).

Within the service itself, cardiology hospitalization units should ensure the prompt performance of complementary tests by managing delays and establishing agreements and

prioritization criteria with the imaging, electrophysiology, and cardiac catheterization units. The cancellation of scheduled diagnostic and therapeutic procedures should be minimized, because, in addition to interfering with the effectiveness and efficiency of health care, it has relevant negative implications for the patient's perception of service quality and is a frequent cause of complaints. Promoting bedside echocardiography by physicians in the hospitalization unit avoids patient travel, improves satisfaction and the doctor-patient relationship,² and reduces the workload of the imaging units by reserving their participation for doubtful or more complex cases. In addition, it provides enormous educational value, because it allows the immediate establishment of correlations with exploratory findings. This aspect facilitates the improvement of these skills which, sadly, are currently in disuse. These advantages can be extended to the use of techniques such as signal-averaged electrocardiography or vectorcardiography, whose use, despite their proven clinical benefit, is unjustifiably marginal.

Beyond the boundaries of the cardiology department, the establishment of rapid and efficient cardiology care in the



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