

RHYTHM DISORDERS AND ELECTROPHYSIOLOGY

CLINICAL CASE

Brugada Phenocopy in a Child With Parvovirus B19 Myocarditis



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ABSTRACT

BACKGROUND Sodium channelopathies predispose to life-threatening arrhythmias. Brugada phenocopies (BrPs) are nongenetic conditions that mimic Brugada syndrome and may have a reversible cause.

CASE PRESENTATION A 16-month-old child presented with a 12-day ventricular tachycardia storm and a spontaneous type 1 Brugada electrocardiogram pattern during sinus rhythm. The patient initially presented with no fever and normal ventricular function on admission that deteriorated severely within 6 days. Blood viral polymerase chain reaction was positive for parvovirus B19, and endomyocardial biopsy confirmed myocarditis. Targeted treatment led to full recovery.

DISCUSSION This is the first reported case to our knowledge of BrP caused by viral myocarditis. It highlights the diagnostic challenges and the importance of correct diagnosis and treatment.

TAKE-HOME MESSAGES In pediatric refractory ventricular tachycardia, even with a Brugada pattern (possible BrP), myocarditis should be considered, as early diagnosis and treatment can be lifesaving. Blood viral polymerase chain reaction is a highly sensitive diagnostic tool for diagnosis of viral myocarditis in children. (JACC Case Rep. 2025;30:105283)

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HISTORY OF PRESENTATION

A 16-month-old boy was admitted to a pediatric intensive care unit in a regional hospital following an episode of sudden loss of consciousness and tonic-clonic seizure shortly after lunch. He recovered spontaneously within 5 minutes. On admission, he was hemodynamically stable (heart rate of 170 beats/min), and a 12-lead electrocardiogram (ECG) revealed

TAKE-HOME MESSAGES

- In pediatric refractory VT, even with a Brugada pattern (possible BrP), myocarditis should be considered, as early diagnosis and treatment can be lifesaving.
- Blood viral PCR is a highly sensitive diagnostic tool for diagnosis of viral myocarditis in children.

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ABBREVIATIONS AND ACRONYMS

BrP = Brugada phenocopy
BrS = Brugada syndrome
CMR = cardiac magnetic resonance imaging
ECG = electrocardiogram
PCR = polymerase chain reaction
VT = ventricular tachycardia

a ventricular tachycardia (VT) with a right bundle branch morphology and superior axis VT (Figure 1). He reverted to sinus rhythm after amiodarone infusion, revealing a spontaneous type 1 Brugada pattern (Figure 2). An echocardiogram showed a structurally normal heart with preserved function. Blood tests showed initial troponin T of 579 ng/mL and N-terminal pro-B-type natriuretic peptide of 20,000 pg/mL, with all

electrolytes in normal range. His temperature was 36.5 °C.

Because of suspicion of Brugada syndrome (BrS), amiodarone was replaced by isoprenaline and quinidine. Persistent VT storm prompted transfer to our center.

PAST MEDICAL HISTORY

The patient had no known medical conditions, no recent viral infections, and no family history of sudden death or arrhythmias. ECGs of first-degree family members were normal.

DIFFERENTIAL DIAGNOSIS

First, on arrival, we obtained an echocardiogram that ruled out structural heart disease, coronary anomalies, intracardiac tumors, and hypertrophic or dilated cardiomyopathy. He presented with a left ventricular ejection fraction of 65%, so a severe myocarditis was considered less likely to be the cause of his arrhythmias. Second, we excluded any drug intake, any electrolyte imbalance, or any metabolic disease with a blood sample. Other channelopathies were also ruled out as ECG showed a type 1 Brugada ECG pattern. Overall, the spontaneous Brugada pattern supported the initial diagnosis of BrS.

INVESTIGATIONS

Subsequent 12-lead ECGs and continuous telemetry revealed alternance of sinus rhythm with type 1 Brugada pattern (Figure 2), different types of monomorphic VT (Figure 3), and polymorphic VT (Figure 4). During the first week, results of work-up for metabolic, neurologic, and toxic causes were normal. Electroencephalogram was normal. On day 10, a blood viral polymerase chain reaction (PCR) test was positive for parvovirus B19. For that reason, myocarditis was highly suspected despite the Brugada pattern and the atypical presentation. The following day, an endomyocardial biopsy confirmed inflammation with lymphocyte infiltration and necrosis, consistent with myocarditis, and PCR-positive parvovirus B19 was detected in the myocardium. Cardiac magnetic resonance (CMR) was deferred owing to clinical instability.

MANAGEMENT

Initially, both isoprenaline and quinidine were used given the patient's type 1 ECG pattern and VT. Interestingly, both medications resulted in less ventricular arrhythmias compared with when the patient was given amiodarone or esmolol, which clearly

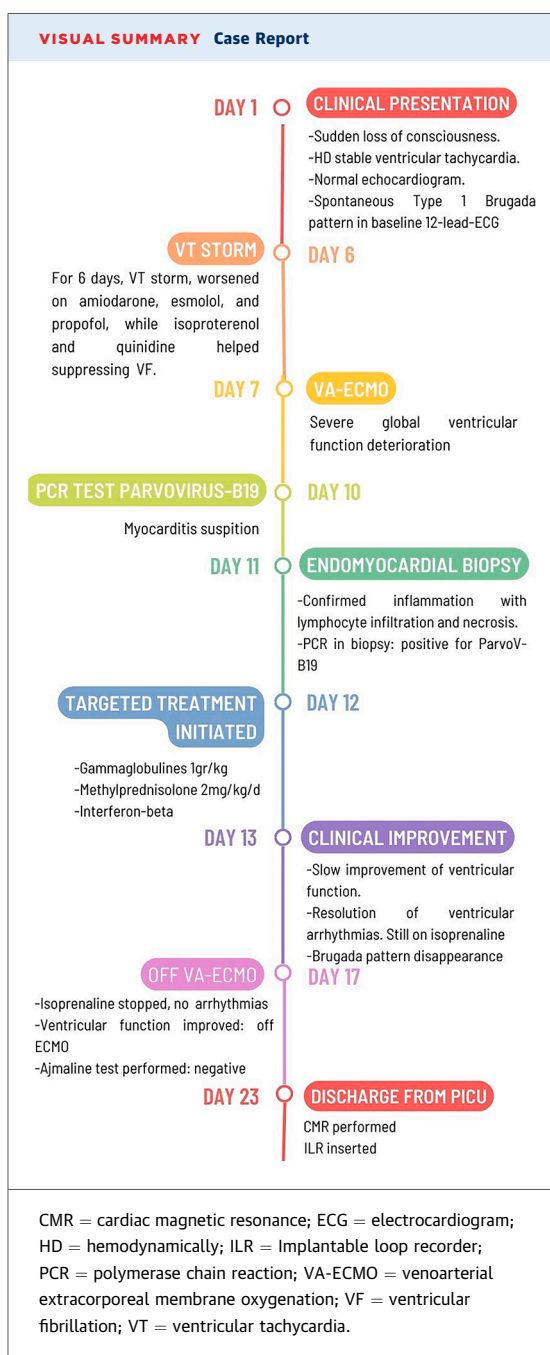
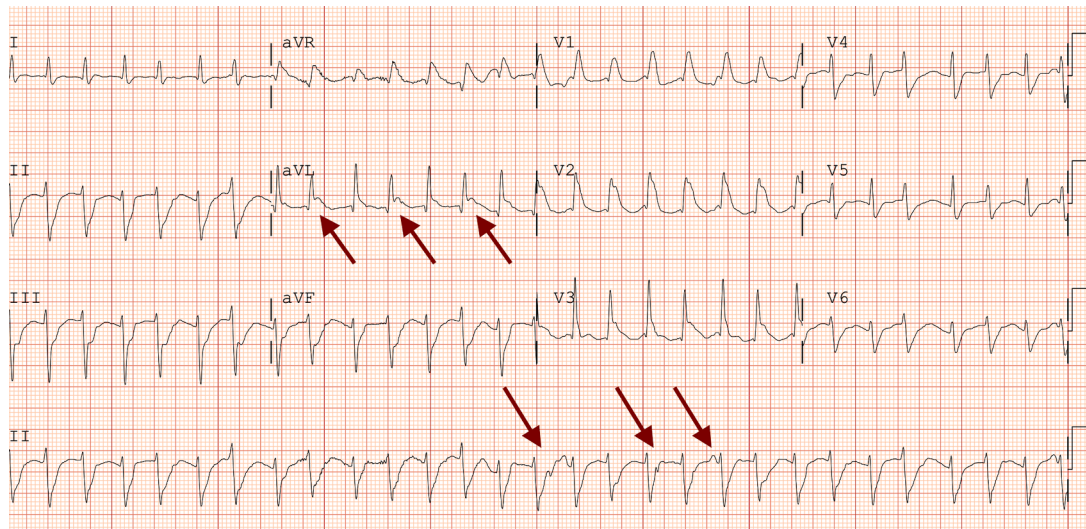


FIGURE 1 Electrocardiogram on Admission



Broad complex tachycardia at a heart rate of 170 beats/min, with right bundle branch block morphology and late precordial transition, superior axis, and ventriculoatrial dissociation. Red arrows show dissociated P waves. Electrocardiogram speed 25 mm/s, voltage 10 mm/mV.

worsened his episodes of ventricular arrhythmias. Amiodarone was stopped eventually owing to toxicity with QRS broadening and third-degree atrioventricular block (Figure 5). By day 7, global ventricular function deterioration required venoarterial extracorporeal membrane oxygenation and urgent septostomy for left atrial decompression. Once parvovirus B19 myocarditis was confirmed by biopsy (Figure 6), targeted treatment according to our protocol¹ was initiated immediately on day 12 including gamma globulins, methylprednisolone, and interferon beta.

OUTCOME

Within 24 hours, ventricular function started improving, and ventricular arrhythmias resolved. Interestingly, the Brugada pattern also disappeared. The patient was weaned from extracorporeal membrane oxygenation 5 days later, and isoprenaline and quinidine could be discontinued with no further ventricular arrhythmias.

Once the patient recovered, we performed an ajmaline challenge, which was negative. Genetic testing showed a heterozygous SCN4A variant of

uncertain significance (p.Ala927Val). CMR performed 5 weeks later showed no abnormalities or gadolinium enhancement, with fully recovered biventricular function.

The patient remained well until discharge with no further evidence of Brugada pattern in his repeated 12-lead ECG, and we could not identify any further tachyarrhythmias. A loop recorder was implanted for continuous monitoring. He was discharged after 5 weeks in the hospital. After 1 year, the patient remains well, with no further cardiac symptoms or arrhythmic events recorded.

DISCUSSION

BrS is a genetic sodium channelopathy characterized by a type 1 ECG pattern. It is associated with life-threatening arrhythmias and might be triggered by fever or some drugs. Diagnosis is based mainly on the ECG pattern, clinical symptoms, and loss-of-function mutations in the SCN5A gene, which encodes the alpha subunit of the cardiac sodium channel.² In BrS, effective control of VT during the acute arrhythmic phase has been reported with the infusion of isoprenaline, leading to the suppression of electrical

FIGURE 2 Baseline 12-Lead Electrocardiogram in Sinus Rhythm Showing a Right Bundle Branch Block Pattern With Coved ST-Segment Elevation >2 mm Followed by a Negative T-Wave in the Right Precordial Leads, Suggesting a Spontaneous Type 1 Brugada Pattern



V₅ to V₆ correspond to high precordial leads placed at third intercostal space. Electrocardiogram speed 25 mm/s, voltage 10 mm/mV.

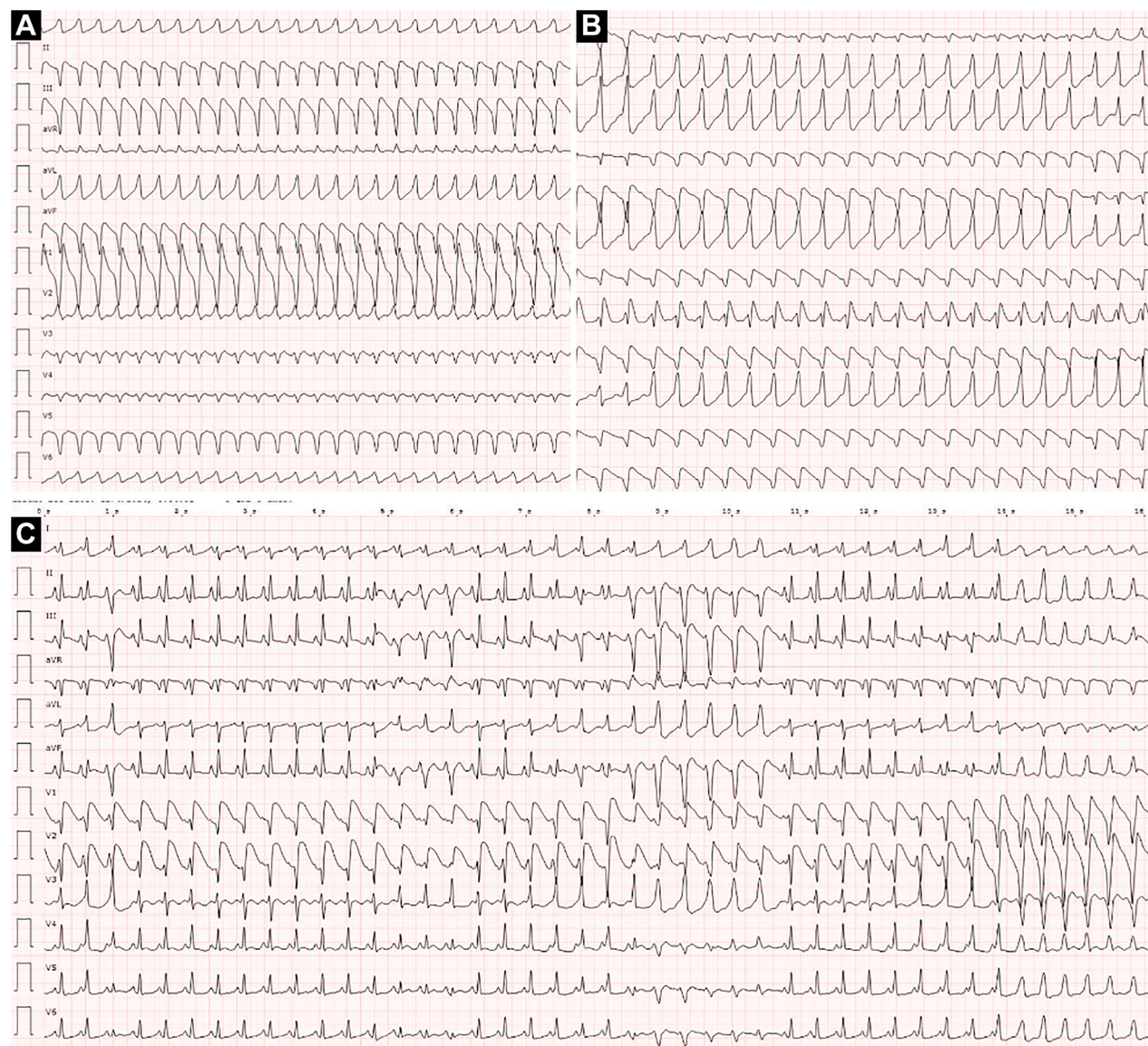
storm as well as normalized ST-segment elevation. In the more chronic phase, quinidine has been described as protective from inducible ventricular arrhythmias and even recommended as baseline treatment in high-risk patients who are not candidates for implantable cardioverter-defibrillator.³

However, Brugada phenocopies (BrPs) must be considered. BrPs are conditions that can mimic the type 1 Brugada pattern without an underlying genetic channelopathy. In 2014, Anselm et al⁴ introduced the term Brugada phenocopy and started an international registry. In 2016, Antzelevitch et al⁵ described myocarditis as one possible cause among others.

Inflammation in the right ventricular outflow tract epicardium may simulate the ECG pattern and the arrhythmogenic characteristics of BrS. So far, there have been 5 reported cases of myocardial involvement and BrP: 2 cases of hematologic diseases, 1 case of amyloidosis, and 1 case of Chagas disease, all due to cell infiltration, and 1 case of aluminum poisoning myocarditis.⁶ In 2015, Gottschalk et al⁷ proposed systematic diagnostic criteria to differentiate true BrS from BrP to avoid erroneous diagnosis of BrS (Table 1).

Our patient presented with a diagnostic type 1 Brugada pattern, but lacked known clinical criteria

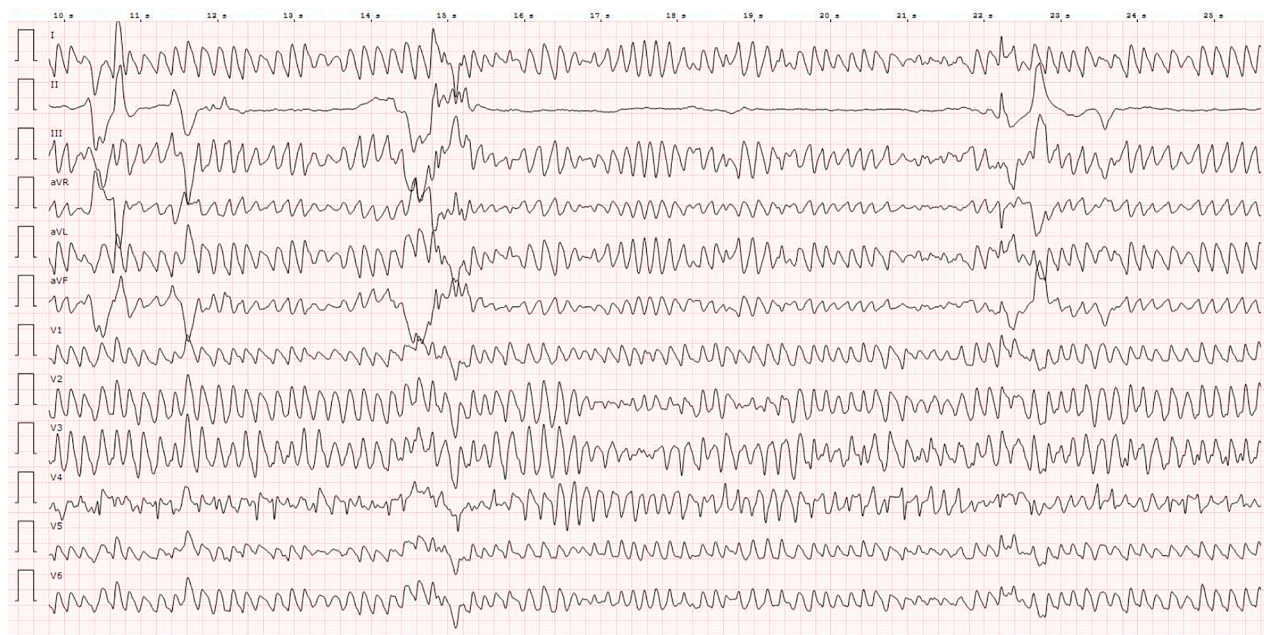
FIGURE 3 Different Ventricular Tachycardia Episodes Recorded by Electrocardiogram



(A) Ventricular tachycardia (VT) with superior axis and left bundle branch morphology. (B) VT with inferior axis and right bundle branch morphology. (C). Sinus rhythm with Brugada pattern and runs of nonsustained VT of different morphologies. V₅ to V₆ correspond to high precordial leads placed at third intercostal space in (A and B). Electrocardiogram speed 25 mm/s, voltage 10 mm/mV.

for BrS such as fever, previous syncope, and family history. The resolution of the electrical changes after myocarditis treatment initiation with negative ajmaline challenge and inconclusive genetics further supported the BrP diagnosis.

Myocarditis is an inflammatory disease of the myocardium that can lead to arrhythmias and is often underdiagnosed as it can present as sudden cardiac death or as the only symptom in the acute phase.⁸ The diagnosis of myocarditis is based on the clinical

FIGURE 4 Polymorphic Ventricular Tachycardia

Electrocardiogram speed 25 mm/s, voltage 10 mm/mV.

findings, blood biomarkers, ECG, echocardiography, and CMR using the Lake Louise Criteria.⁹ However, endomyocardial biopsy remains the gold standard, requiring an inflammatory cellular infiltrate with or without associated myocyte necrosis, following the Dallas Criteria.¹⁰ In children, a positive PCR viral blood test has high sensitivity in viral myocarditis (in adults its sensitivity is lower).¹¹ In our patient's case, the positive viral blood PCR test suggested a parvovirus B19 myocarditis, later confirmed with an endomyocardial biopsy and a positive PCR in this sample.

CONCLUSIONS

To our knowledge, there are no previous reports in the literature of viral myocarditis manifesting with BrP. Myocarditis should be considered in cases with

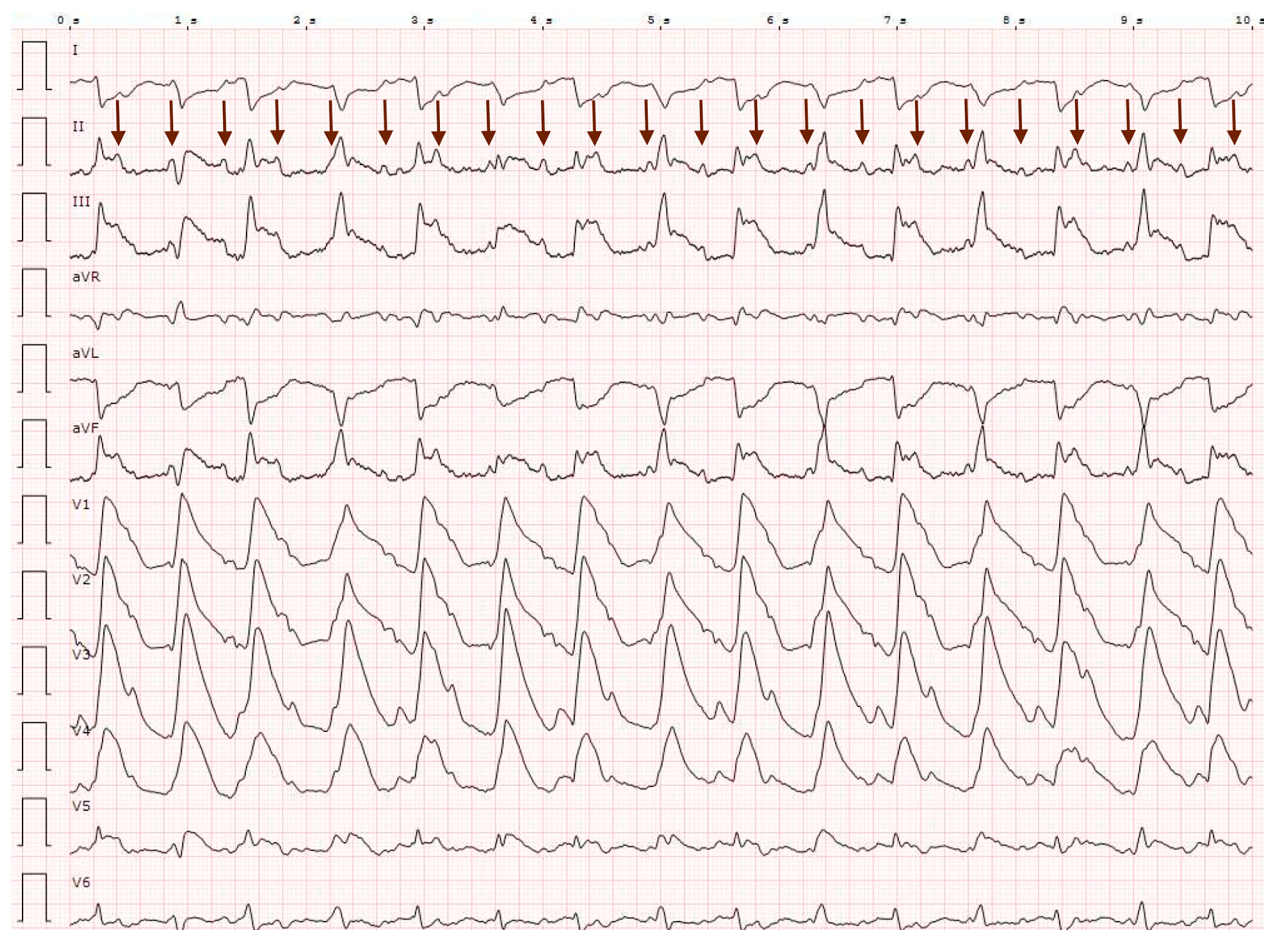
any refractory ventricular arrhythmias, even in the rare scenario in which they occur with a Brugada pattern, as this may be a BrP. In this case, prompt treatment can completely shift the progression toward rapid resolution of an otherwise lethal disease. In children, a positive blood viral PCR test is a sensitive tool when myocarditis is suspected.

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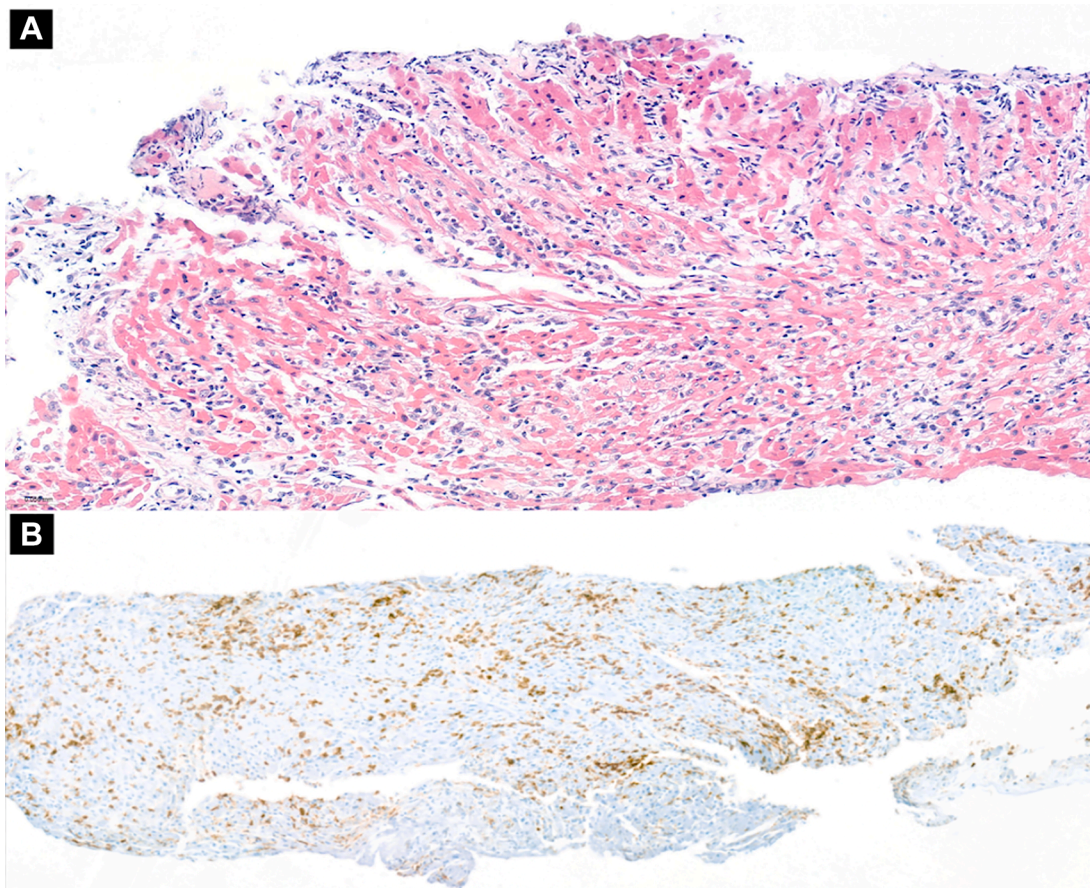
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FIGURE 5 Amiodarone Intoxication



QRS broadening and complete atrioventricular block with an accelerated escape rhythm. Red arrows point to P waves. V₁ with type 1-like Brugada pattern. Electrocardiogram speed 25 mm/s, voltage 10 mm/mV.

FIGURE 6 Hystopathologic Findings From Endomyocardial Biopsy



(A) Hematoxylin and eosin staining reveals cellular infiltrate with myocyte necrosis. (B) Immunohistochemical staining for CD3, revealing the presence of T lymphocytes.

TABLE 1 Comparison Between BrP and BrS		
	BrP	BrS
Brugada ECG pattern	Present	Present
Resolution of Brugada ECG pattern	Complete resolution when underlying condition is treated	ECG changes are dynamic; may reappear more or less frequently, especially with fever, electrolyte disturbances, or specific drugs that can induce it
Clinical history	No relevant personal or family history; presence of an identifiable current condition that might trigger Brugada ECG pattern	History of syncope, ventricular arrhythmias, or cardiac arrest; family history of BrS or sudden cardiac death
Provocative testing (ajmaline, flecainide, or procainamide)	Negative	Positive
Genetic testing	Negative	Positive in one-third of cases; negative in about two-thirds
BrP = Brugada phenocopy; BrS = Brugada syndrome; ECG = electrocardiogram.		

REFERENCES

1. Esmel-Vilomara R, Dolader P, Izquierdo-Blasco J, et al. Parvovirus B19 myocarditis in children: a diagnostic and therapeutic approach. *Eur J Pediatr*. 2022;181(5):2045–2053. <https://doi.org/10.1007/s00431-022-04406-x>
2. Priori SG, Wilde AA, Horie M, et al. HRS/EHRA/APHRS expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes: document endorsed by HRS, EHRA, and APHRS in May 2013 and by ACCF, AHA, PACES, and AEPC in June 2013. *Heart Rhythm*. 2013;10:1932–1963. <https://doi.org/10.1016/j.hrthm.2013.05.014>
3. Ohgo T, Okamura H, Noda T, et al. Acute and chronic management in patients with Brugada syndrome associated with electrical storm of ventricular fibrillation. *Heart Rhythm*. 2007;4(6):695–700. <https://doi.org/10.1016/j.hrthm.2007.02.014>
4. Anselm DD, Gottschalk BH, Baranchuk A. Brugada phenocopies: consideration of morphologic criteria and early findings from an international registry. *Can J Cardiol*. 2014;30:1511. <https://doi.org/10.1016/j.cjca.2014.09.023>
5. Antzelevitch C, Yan GX, Ackerman MJ, et al. J-Wave syndromes expert consensus conference report: emerging concepts and gaps in knowledge. *J Arrhythm*. 2016;32(5):315–339. <https://doi.org/10.1016/j.joa.2016.07.002>
6. Baranchuk A, Nguyen T, Ryu MH, et al. Brugada phenocopy: new terminology and proposed classification. *Ann Noninvasive Electrocardiol*. 2012;17(4):299–314. <https://doi.org/10.1111/j.1542-474X.2012.00525.x>
7. Gottschalk BH, Anselm DD, Brugada J, et al. Expert cardiologists cannot distinguish between Brugada phenocopy and Brugada syndrome electrocardiogram patterns. *Europace*. 2016;18(7):1095–1100. <https://doi.org/10.1093/europace/euv278>
8. Peretto G, Sala S, Rizzo S, et al. Arrhythmias in myocarditis: state of the art. *Heart Rhythm*. 2019;16(5):793–801. <https://doi.org/10.1016/j.hrthm.2018.11.024>
9. Friedrich MG, Sechtem U, Schulz-Menger J, et al. Cardiovascular magnetic resonance in myocarditis: a JACC White Paper. *J Am Coll Cardiol*. 2009;53:1475–1487.
10. Cooper LT. Myocarditis. *N Engl J Med*. 2009;360:1526–1538. <https://doi.org/10.1056/NEJMr0800028>
11. Simpson KE, Storch GA, Lee CK, et al. High frequency of detection by PCR of viral nucleic acid in the blood of infants presenting with clinical myocarditis. *Pediatr Cardiol*. 2016;37:399–404. <https://doi.org/10.1007/s00246-015-1290-6>

KEY WORDS Brugada phenocopy, electrical storm, parvovirus B19 myocarditis, type 1 Brugada ECG pattern, ventricular tachycardia