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Brugada-like pattern and myocarditis in a child with multisystem inflammatory syndrome: overlap or differential diagnosis?

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A 5-year-old Caucasian boy was admitted to the hospital because of fever, tachypnoea, chest discomfort, hypotension, and skin rash. Twelve-lead electrocardiogram (ECG) recorded at admission is shown in *Figure 1*. Transthoracic echocardiogram revealed normal left ventricular diameters, septal hypokinesia, systolic dysfunction (ejection fraction 45%), and mild pericardial effusion.

Blood test revealed moderate elevation of high sensitive Troponin (highest value 434 ng/L, n.v. <34), marked elevation of brain natriuretic peptide (721 ng/L, n.v. <100), and inflammatory marker (C-reactive protein 12 mg/dL, n.v. <1). Serology for SARS-CoV-2 was positive (IgG), while molecular PCR assay on nasopharyngeal swab was negative. The troponin curve displayed multiple spikes with a long plateau phase.

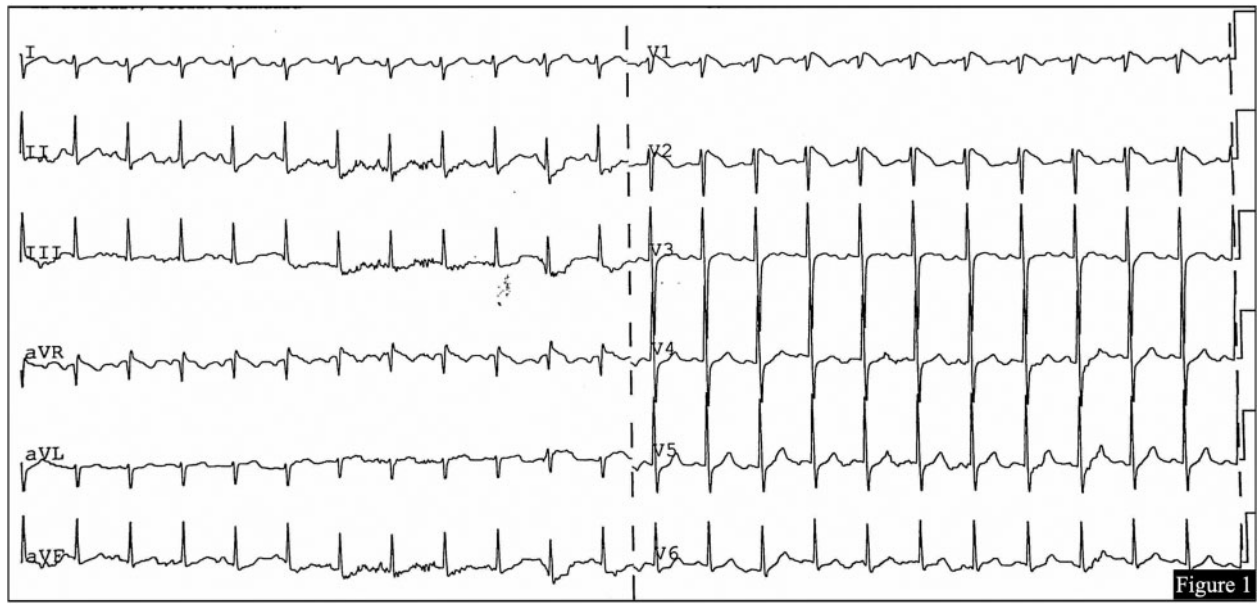

Figure 1 First electrocardiogram recorded at admission showing coved ST-elevation in V1 and V2.

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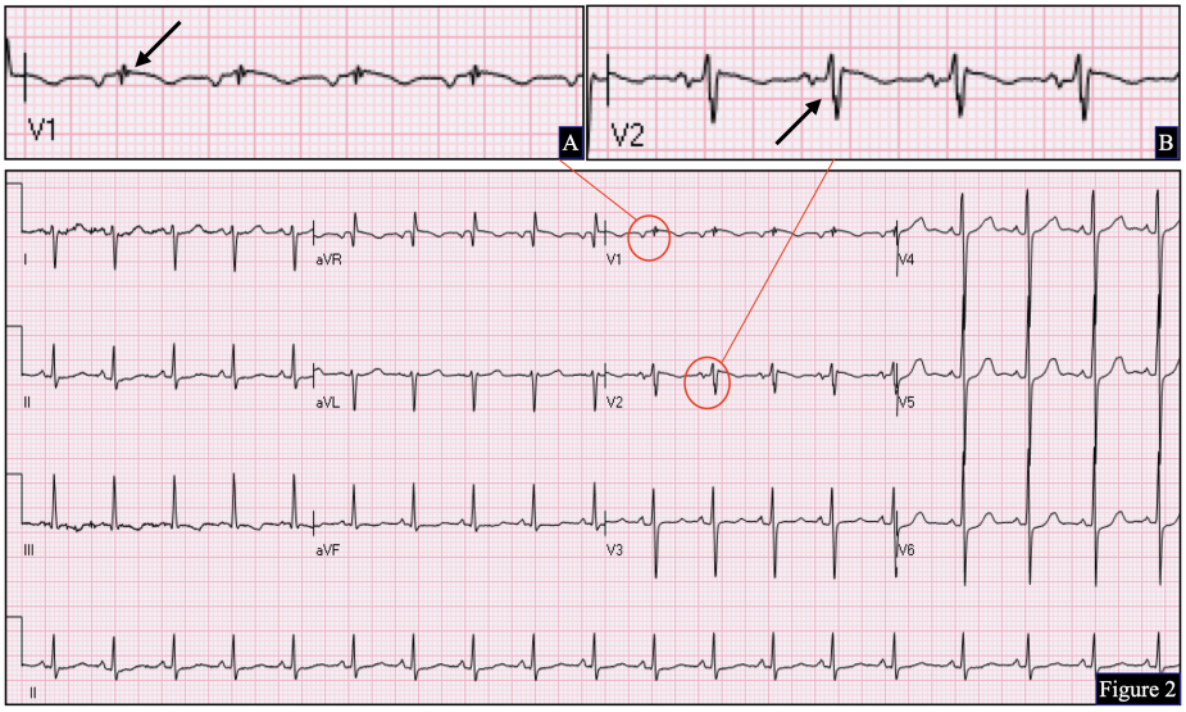

Figure 2 Electrocardiogram recorded the 3rd day of admission showing evolutive changes in V1 and V2 characterized by voltage decrease and QRS fragmentation. (*A*) Magnification insert showing detail of fragmented QRS in V1. (*B*) Magnification insert showing detail of fragmented QRS in V2.

The presented clinical scenario satisfies the diagnostic criteria of multisystem inflammatory syndrome in children (MIS-C). In*Figure 1*, 12-lead ECG shows sinus tachycardia (130 b.p.m.) and marked ST-elevation in V1–V2, and right axis deviation. The differential diagnosis of these localized repolarization abnormalities includes Type 1 (coved-type) Brugada pattern triggered by fever, acute focal myocarditis, and regional transmural ischaemia secondary to Kawasaki-like coronaritis in the setting of MIS-C.[1,2]

Fever is a known trigger able to unmask Brugada pattern, and the observed ST-elevation in V1–V2 is compatible with the classical type I Brugada (coved-type). However, clinical features, elevated markers of myocardial damage, and echocardiographic findings favour an acute structural process rather than pure electrical disease.

Electrocardiogram recorded the following days showed QRS fragmentation and voltage reduction in V1–V2 (*Figure 2*), consistent with myocardial oedema and fibrosis.[3]

The hypothesis of acute myocardial ischaemia was considered unlikely since chest pain features were indicative of pericardial irritation (e.g. increasing by cough and deep inspiration), and there were no echocardiographic signs of inflammatory coronary involvement. In addition, the troponin trend did not display a typical ischaemic pattern.

The patient was treated with intravenous immunoglobulin and steroids. Symptoms, including fever, remitted gradually in a few days, ventricular function, and ECG normalized in 1 week (Supplementary material online, *Figure S1*)

This clinical-electrocardiographical course supports the diagnosis of a Brugada phenocopy during acute inflammatory syndrome fulfilling the clinical criteria of MIS-C. Furthermore, the isolated ST-elevation in V1–V2 followed by transient QRS fragmentation and voltage reduction, together with increase of inflammatory and myocardial damage markers are best interpreted as evolutive myocardial inflammation, with preponderant septal involvement, and subsequent regression. To our knowledge, this is the first reported case of Brugada ECG pattern due to a myocardial inflammatory disease. We recognize that this hypothesis is somewhat speculative as we do not have histopathologic or cardiac magnetic resonance evidences of focal myocarditis. Indeed, we felt that these investigations were not justified in this case, owing to the patient age and favourable clinical course. Similarly, we did not deem appropriate to perform neither pharmacological challenge nor SCN5A testing.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

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