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Brugada: a diagnosis or a sign?

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Key words: Brugada syndrome; hypercalcemia; phenocopy.

Abstract

A 59-year-old man presented with asthenia and dyspnoea. Admission ECG showed a type 1 Brugada pattern in leads V1–V2. The patient had no arrhythmic history and normal cardiac imaging. Laboratory tests revealed severe malignant hypercalcemia with renal dysfunction. After correction of calcium levels, the Brugada-like ECG changes completely resolved, and sodium-channel blocker testing was negative. This case illustrates malignant hypercalcemia as a reversible cause of Brugada phenocopy and underscores the importance of excluding metabolic disturbances before diagnosing Brugada syndrome and initiating risk stratification or device-based therapies.

Case report

A 59-year-old male patient was admitted to the emergency department of our institution for asthenia, exertional dyspnoea and general feeling of malaise. His vital parameters were normal: heart rate 90 beats per minute, blood pressure 130/80 mmHg, respiratory rate 15 breaths per minute, temperature 36.9 °C. The patient was a smoker and reported no prior pathology.

An ECG was recorded and transmitted to the on-call cardiologist. The ECG showed sinus rhythm, normal PR (155 msec) and a coved ST-segment elevation of V1 and V2 followed by a negative T wave. The pattern was suggestive of Brugada pattern (BrP) type I (Figure 1A).

At the cardiologist's visit, the patient denied history of syncope, nocturnal agonical respiration or family history of Brugada Syndrome (BrS) and nothing remarkable emerged from the objective examination and echocardiogram, which showed normal ventricular size and ejection fraction.

The surprise came from laboratory tests, which showed creatinine 2.7 mg/dl (0.5-1.2) and a calcium level of 18.30 mg/dl (8.2-10.4).

The patient underwent a chest and abdominal CT scan that detects the presence of renal masses (Figure 1C).

Hypercalcaemia was treated with fluids and diuretics. During hospitalization, the patient underwent continuous ECG telemetry monitoring for approximately 48 hours while the Brugada-type ECG pattern was present. No ventricular arrhythmias or other significant arrhythmic events were recorded. When calcium and creatinine were restored, J-waves on the ECG disappeared (Figure 1B). In addition, a bone scintigraphy was performed, which showed metastatic bone lesions (Figure 1D). The flecainide test was negative.

The patient underwent a nephrectomy and is still undergoing immunotherapy.

Discussion

The 2022 ESC guidelines state that "type 1 Brugada ECG pattern is characterized by J point elevation of >2 mV with coved ST elevation and T wave inversion in at least one right precordial ECG lead, V1 or V2, positioned in the second, third or fourth intercostal spaces. It is mandatory to exclude other conditions that may explain the type 1 pattern, so-called phenocopies".[1]

The term Brugada phenocopy is proposed to describe conditions that induce Brugada-like ECG manifestations in patients without true BrS.[2] The criteria for defining a Brugada phenocopy could be summarized as follows: i) the ECG pattern has a Brugada type 1 or type 2 morphology;[3] ii) the patient has an underlying condition that is identifiable; iii) the ECG pattern resolves after resolution of the underlying condition; iv) there is a low clinical pretest probability of true BrS determined by lack of symptoms, medical history, and family history; v) the results of provocative testing with flecainide, procainamide, ajmaline, or other sodium channel blockers are negative;[4] and vi) the results of genetic testing are negative (not a mandatory criterion, because the SCN5A mutation is identified in only 20% to 30% of probands affected by true BrS).[5,6]

Causes of BrS type 1 phenocopia can be:[2] i) metabolic conditions (hyperkalemia, hyponatremia, hypokalemia, hypothermia, hypercalcemia); ii) mechanical compression (pectus excavatum, mediastinal masses and haemopericardium); iii) ischemia; iv) myocardial and pericardial disease

The study by Sonoda *et al.* showed that 5 out of 89 patients with hypercalcemia exhibited a Brugada Pattern (BrP). The most common causes of hypercalcemia include malignancy, hyperparathyroidism, and renal dysfunction.[7]

Hypercalcemia is known to induce significant electrocardiographic changes, including shortening of the QT interval, prolongation of the PR interval and QRS duration, and an increased prevalence of J-point elevation.[8,9] These findings reflect the effects of elevated extracellular calcium on

cardiomyocyte ionic currents and are generally reversible after normalization of serum calcium levels.

J-point elevation associated with hypercalcemia may present with different morphologies, including early repolarization patterns, a scooped ST-segment appearance, and, in a minority of cases, Brugada-type ECG patterns. Sonoda reported Brugada-like findings in approximately 6% of patients with hypercalcemia, irrespective of its underlying etiology, suggesting a direct effect of calcium imbalance rather than an unmasking of a primary genetic channelopathy in most cases.

Although J-point elevation, including Brugada and early repolarization patterns, has been associated with an increased risk of ventricular tachyarrhythmias and sudden cardiac death,[10] sases of ventricular arrhythmias induced by hypercalcemia are uncommon.[7,11] This apparent discrepancy may be explained by the stabilizing effect of elevated extracellular calcium on the cardiac cell membrane, which reduces myocardial excitability despite the presence of potentially arrhythmogenic electrophysiological changes.[12]

The pathophysiological mechanisms underlying hypercalcemia-induced J-point elevation are not fully understood. A shortened QT interval likely plays a contributory role, and transmural heterogeneity of early repolarization has been proposed as a key mechanism. In particular, reduced inward current through the Na^+/Ca^{2+} exchanger, heterogeneously expressed across the ventricular myocardium, may be more pronounced in the epicardium than in the endocardium, leading to accentuation of the action potential notch and the appearance of J-point elevation and Brugada-like ECG patterns.[13]

In this context, the Brugada-type ECG pattern observed in our patient should be interpreted as a secondary and reversible phenomenon related to malignant hypercalcemia rather than as a manifestation of BrS. This case highlights the importance of recognizing metabolic and electrolyte disturbances as reversible causes of Brugada-like ECG patterns, in order to avoid misdiagnosis and inappropriate risk stratification or therapeutic interventions.

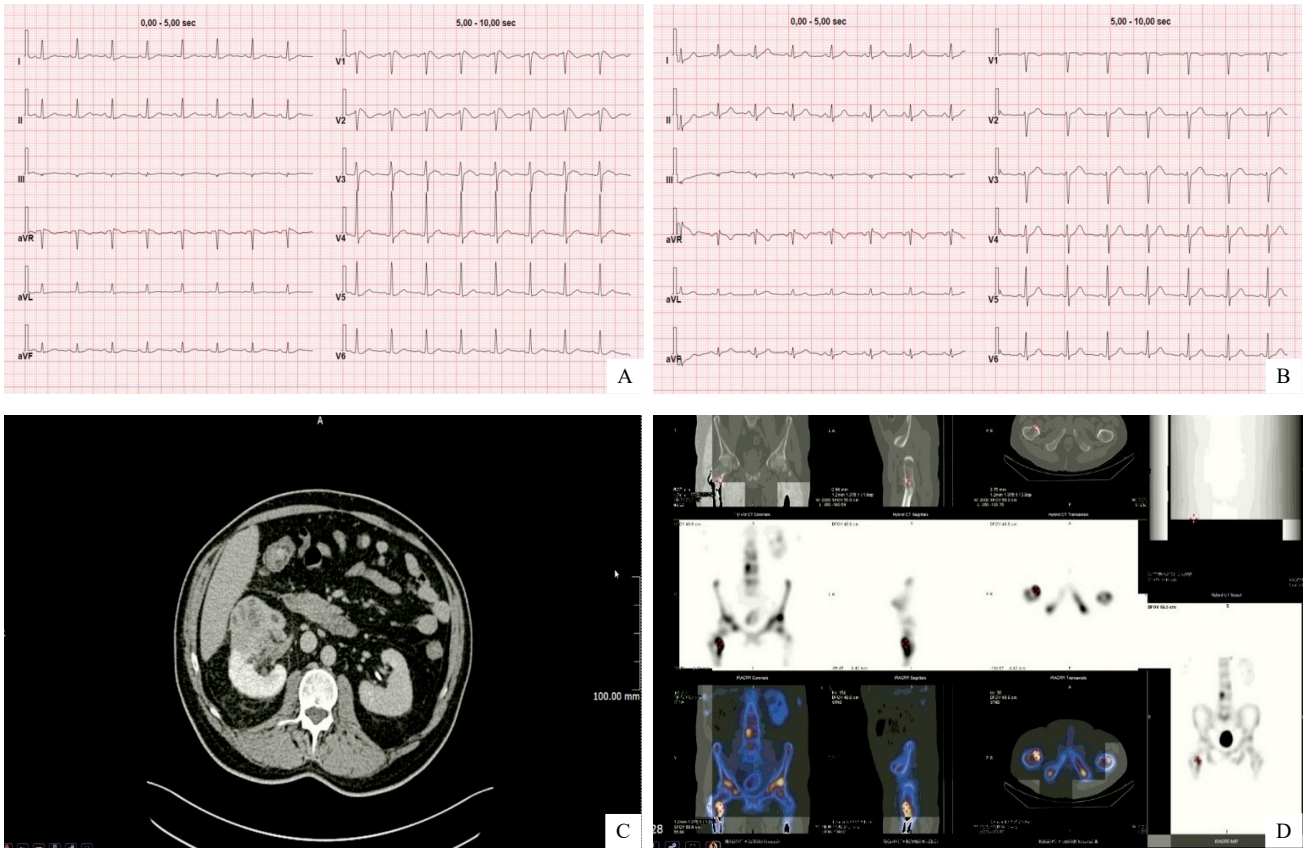

Figure 1. A) The ECG shows sinus rhythm and a J poin elevation and negative T waves in V1 and V2 like a type 1 Brugada pattern; B) Disappearance of the type 1 Brugada pattern after normalization of calcium levels; C) Presence of renal masses in the TC scan; D) Bone metastases on scintigraphy.

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