Type I Brugada pattern associated with diabetic ketoacidosis in a patient with type II diabetes mellitus

A 42-year old male with known insulin-dependent diabetes mellitus and otherwise unremarkable medical history presented with nausea, vomiting, and atypical chest pain for 2 days. His initial vital signs were remarkable for sinus tachycardia (101 bpm) and tachypnea (20 breaths per minute). He did not have a personal or family history of arrhythmia-related symptoms, ventricular tachycardia or fibrillation (VT/VF), or premature sudden cardiac death (SCD). Electrocardiography (ECG) revealed ST elevations (V1–V2) with associated T wave inversions (Fig. 1a). A baseline ECG performed 6 months ago showed no abnormalities. Serial troponin T measurements were normal. His initial laboratories were notable for hyperglycemia, hyponatremia, hyperkalemia, ketonuria, and metabolic acidosis (Table 1). Upon further questioning, it was found that he had no history of referable cardiac symptoms. There was no family history for SCD, recurrent syncope, or arrhythmia. He was diagnosed with and treated for diabetic ketoacidosis. His clinical status stabilized with normalization of electrolytes and pH (Table 1). After the medical treatment, ECG (Fig. 1b) showed improvement, and he had a negative provocative testing with a sodium channel blocker (ajmaline). He had an uneventful discharged and, at 10 months after treatment, he is doing well.

Brugada syndrome (BrS) is a relatively rare genetic channelopathy; however, Brugada pattern is an ECG phenomenon, which is now known as Brugada phenocopy (BrP). The diagnostic distinction between BrP and true congenital BrS focuses on a few key features. First, patients with BrP have a reversible underlying condition, such as adrenal insufficiency, hypokalemia, or myocardial ischemia, that elicits or induces the Brugada ECG pattern. Once this underlying condition resolves, there is prompt normalization of the ECG. Second, patients with BrP have a low clinical pretest probability of true congenital BrS as opposed to a high clinical pretest probability in patients with BrS, who have a history of VF, polymorphic VT, nonvagal syncope, and a family history of sudden cardiac. Third, patients with BrP have a negative provocative challenge with a sodium channel blocker, whereas those with true congenital BrS have a positive provocative challenge. Therefore, ECG alone cannot be used as a diagnostic tool. Implantation of an implantable cardioverter defibrillation in an asymptomatic patient without a spontaneous type 1 Brugada electrocardiographic pattern has no benefit.

Table 1. Patient's laboratory results on admission versus before discharge

Tests	On admission	Before discharge
Venous pH	6.94	7.4
Potassium (mmol/L)	6.8	3.7
Bicarbonate (mmol/L)	8	21
Sodium (mmol/L)	122	135
Chloride (mmol/L)	75	105
Calcium (mmol/L)	8.2	7.9
Glucose (mg/dL)	900	225
Urea Nitrogen (mg/dL)	45	13
Creatinine (mg/dL)	1.44	0.72
Urine ketone	++++	-

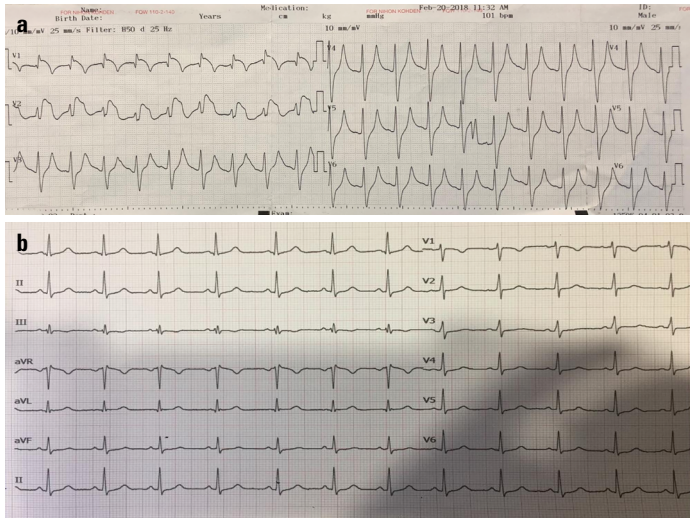

Figure 1. (a) Electrocardiography depicts coved-type ST-segment elevation in leads V1-V2 with associated T wave inversions during diabetic ketoacidosis. (b) Electrocardiography indicates resolution of the prior findings before the discharge

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