Brugada-like electrocardiography pattern induced by severe hyponatraemia

Ashenafi Tamene, Srinivasan Sattiraju, Kyuhyun Wang, and David G. Benditt*

Cardiovascular Division, Department of Medicine, Cardiac Arrhythmia Center, University of Minnesota Medical School, MMC 508, 420 Delaware Street SE, Minneapolis, MN 55455, USA

* Corresponding author. Tel: +1 612 625 4401; fax: +1 612 624 4937, Email: bendi001@umn.edu

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The characteristic electrocardiographic (ECG) findings of Brugada syndrome, specifically the down sloping coved elevation of the ST-segment ending in an inverted T-wave in the right precordial leads, have been linked to reduced inward sodium current due to dysfunctional sodium channels. Additionally, however, many sodium channel blocking agents are known to either unmask or induce this ECG pattern. Severe hyponatraemia may be expected to have the same effect by reducing inward sodium current. Here, we report a case in which the presenting ECG exhibited a ‘Brugada-like’ pattern during severe isolated hyponatraemia, with subsequent normalization as hyponatraemia improved.

Case report
A 63-year-old male presented with profound generalized body weakness with inability to perform routine activities of daily living. Dietary history revealed daily consumption of ~8 L of diet cola. He had no prior history of syncope or arrhythmias, but had known hypertension, type 2 diabetes, dyslipidaemia, and bipolar disorder. Medications included metoprolol, hydrochlorothiazide, lisinopril, valproic acid, and oral hypoglycaemics. His family history was without known arrhythmic disorders or sudden cardiac death. Physical examination was remarkable only for drowsy mental status. Initial metabolic studies revealed severe hyponatraemia (101 mmol/L) and normal serum potassium (K⁺, 3.6 mmol/L). Electrocardiography showed inverted P-waves in inferior leads (II, III, aVF) suggesting a low atrial rhythm at 45 b.p.m. Although J-point elevation of 0.2 mV was noted in only one precordial lead (V2), the down-sloping coved ST-segment elevations in anterior precordial leads with associated T-wave inversions in V1 and V2 suggested a Brugada-like ECG pattern (Figure 1). Acute myocardial infarction was excluded by negative cardiac biomarkers. The patient improved clinically with correction of serum sodium (Na⁺). Hyponatraemia was attributed to poor dietary salt intake, psychogenic polydipsia, and hydrochlorothiazide diuretic. Neurological manifestations that have been associated with severe hyponatraemia such as seizure, confusion, and coma were not observed. His antihypertensive medications were held but valproic acid for bipolar disorder was continued. ~36 h after admission his serum Na⁺ was 119 mmol/L. Follow-up ECG showed normalization of ST-elevations and disappearance of the Brugada ECG pattern (Figure 2). A pharmacological provocation test for Brugada pattern was recommended but the patient declined.

Discussion
We report a case in which isolated severe hyponatraemia (101 mmol/L) was accompanied by an ECG pattern in which the ST and T-wave changes resembled a Brugada pattern (albeit not type-1), and which subsequently returned toward normal when the serum Na⁺ level improved.

The basis of the Brugada ECG pattern and Brugada syndrome remains a topic of debate, with arguments having been made for both repolarization and depolarization abnormalities being responsible. In any event, diminution of inward sodium (Na⁺) current is generally accepted as being essential. The reversible nature of the Brugada-like ECG finding in our patient after normalization of hyponatraemia, suggests that a diminished transmembrane Na⁺ concentration gradient was the basic cause in this instance.

To our knowledge, only two previous similar observations have been published, but both also exhibited concomitant abnormalities of serum potassium (K⁺). Mok et al. reported a Brugada-like ECG pattern during combined severe hyponatraemia (serum Na⁺, 111 mmol/L) and hypokalaemia (serum K⁺, 1.7 mmol/L) in a 64-year-old male who complained of weakness and dizziness. The Brugada-like ECG pattern persisted despite normalizing serum K⁺, but disappeared when serum Na⁺ was corrected; the authors attributed the finding to reduced inward Na⁺ current due to diminished ionic gradient. In the second case, a Brugada-like ECG pattern was reported in the setting of diabetic ketoacidosis (glucose of 1232 mg/dL) with severe hyponatraemia (serum Na⁺, 105 mmol/L), but concomitant severe hyperkalaemia (serum K⁺, 7 mmol/L) likely contributed to the abnormal ECG.

Brugada syndrome has been linked to mutations in the cardiac sodium channel gene. However, ST-segment changes are sometimes concealed, and are only unmasked by sodium channel blockers such as flecainide, ajmaline, or procainamide. The list of drugs or conditions that can unmask or induce a Brugada ECG pattern is growing. Potentially, based on the observation reported here, severe...
Hyponatraemia can be added to the list of circumstances in which a Brugada-pattern ECG may be mimicked; however, whether an arrhythmic risk is associated with this scenario is unknown at this time.

In summary, severe hyponatraemia appeared to be responsible for inducing ECG changes that, while not a type-1 Brugada, does resemble the Brugada pattern. Consequently, marked reduction of serum Na\(^+\) should be included in the growing list of drugs or conditions known to induce ECG findings that resemble the Brugada pattern. Whether induction of the Brugada pattern in this manner is associated with increased susceptibility to ventricular tachyarrhythmias is currently uncertain.

Conflict of interest: none declared.

References

**Figure 1** Twelve lead electrocardiogram obtained upon admission (serum sodium concentration of 101 mmol/L) showing ST-segment changes that resemble a Brugada ECG pattern. A downward coving of the ST-segment with T-wave inversion is present in the anterior precordial leads. The QRS exhibits late rightward forces of uncertain origin.

**Figure 2** Twelve lead electrocardiogram showing normalization of ST-segment elevations at serum sodium concentration of 119 mmol/L. When compared with the admission ECG, the anterior precordial leads now exhibit a more normal ST-segment and T-wave pattern. A prolonged PR interval is also noted.

