CASE REPORT

All that looks like ''Brugada'' is not ''Brugada'': Case series of Brugada phenocopy caused by hyponatremia

Yashwant Agrawal [a,*], Sourabh Aggarwal [a], Jagadeesh K. Kalavakunta [b], Vishal Gupta [b]

[a] Department of Internal Medicine/Pediatrics and Internal Medicine, Western Michigan University, Homer Stryker School of Medicine, Kalamazoo, MI
[b] Department of Cardiology. Borgess Medical Center/Michigan State University, Kalamazoo, MI

[a,b] USA

Brugada syndrome (BS), a life-threatening channelopathy associated with reduced inward sodium current due to dysfunctional sodium channels, is characterized by ST-segment elevation with downsloping ''coved type'' (type 1) or ''saddle back'' (type 2) pattern in V1–V3 precordial chest leads (1, 2). Brugada phenocopy, a term describing conditions inducing Brugada-like pattern of electrocardiogram (EKG) manifestations in patients without true BS, is an emerging condition (3). We describe a case series of Brugada phenocopy with hyponatremia.

Keywords: Brugada syndrome, Electrocardiogram, Hyponatremia

Introduction

Brugada syndrome (BS), a life-threatening channelopathy associated with reduced inward sodium current due to dysfunctional sodium channels, is characterized by ST-segment elevation with downsloping ''coved type'' (type 1) or ''saddle back'' (type 2) pattern in V1–V3 precordial chest leads [1,2]. Brugada phenocopy, a term describing conditions inducing Brugada-like patterns of electrocardiogram (EKG) manifestations in patients without true BS, is an emerging condition [3]. We describe a case series of Brugada phenocopy with hyponatremia.

Case reports

Case 1

A 63-year-old Caucasian woman with a history of diabetes mellitus, hypertension, and schizoaffective disorder on haloperidol, presented to the emergency room with confusion and altered mental status. She was drinking up to 12 L of water and four to five 355-mL cans of beer every day. Physical examination including vitals was unremarkable except for confusion and disorganized thought process. Initial labs were significant for hyponatremia (Na^+ 112 mmol/L). Detailed family history was not significant for any cardiovascular disease including BS. EKG showed prolonged QTc (547 milliseconds)

Disclosure: Authors have nothing to disclose with regard to commercial support.

Received 30 January 2016; revised 5 February 2016; accepted 9 February 2016.
Available online 15 February 2016

* Corresponding author at: 1521 Shaffer Road, Kalamazoo, MI 49048, USA.
E-mail address: yashwantagrwal.agrawal@gmail.com (Y. Agrawal).

P.O. Box 2925 Riyadh – 11461KSA
Tel: +966 1 2520088 ext 40151
Fax: +966 1 2520718
Email: sha@sha.org.sa
URL: www.sha.org.sa

Peer review under responsibility of King Saud University.
URL: www.ksu.edu.sa
http://dx.doi.org/10.1016/j.jsha.2016.02.003

Production and hosting by Elsevier

and ''coved type'' ST elevations and deep T-wave inversions in leads V1–V3 (Fig. 1). However, no such changes were noticed on previous EKGs. Cardiac markers were within normal limits. Electrophysiological studies with programmed electrical stimulation to induce ventricular arrhythmias and left heart catheterization were unremarkable. A drug challenge test was not performed. Her haloperidol was held and water restriction initiated. Her sodium level improved gradually with serial EKGs showing resolution of ST elevations and QTc interval returning to normal (Fig. 2).

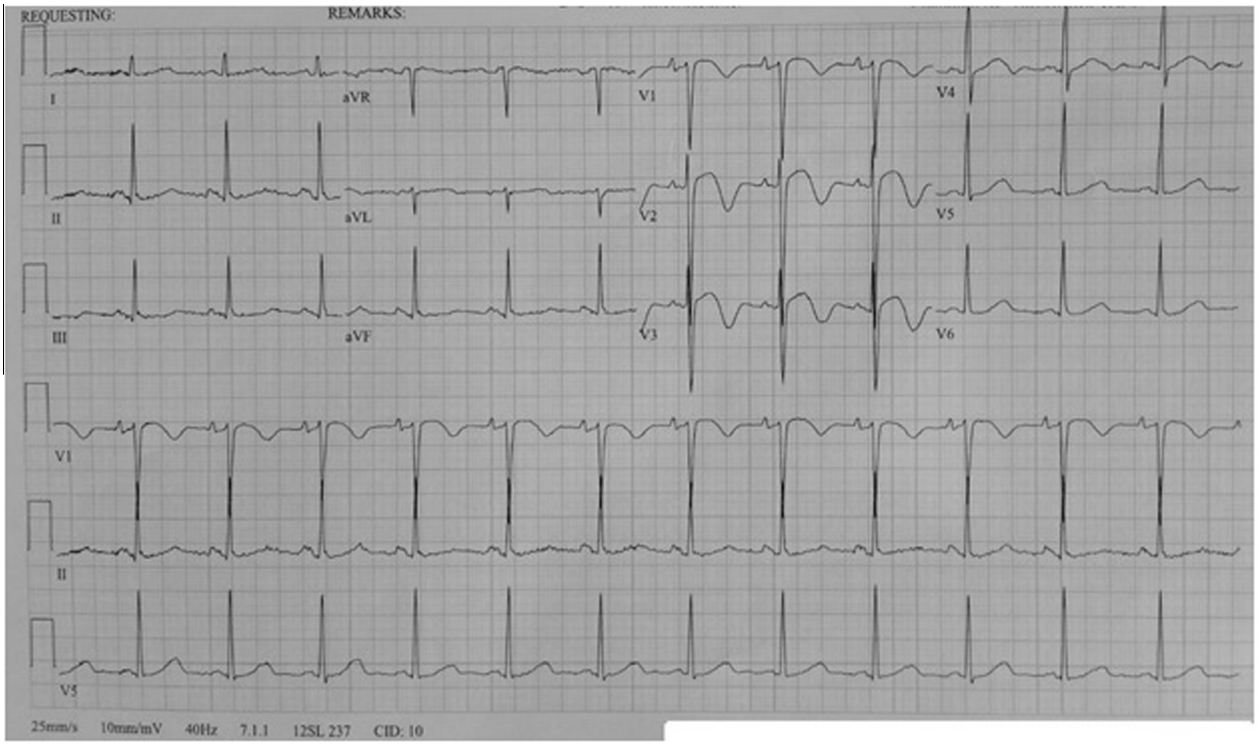

Figure 1. Brugada type 1 (coved type) pattern in leads V1–V3. Sodium 112 mmol/L, QTc 547 milliseconds.

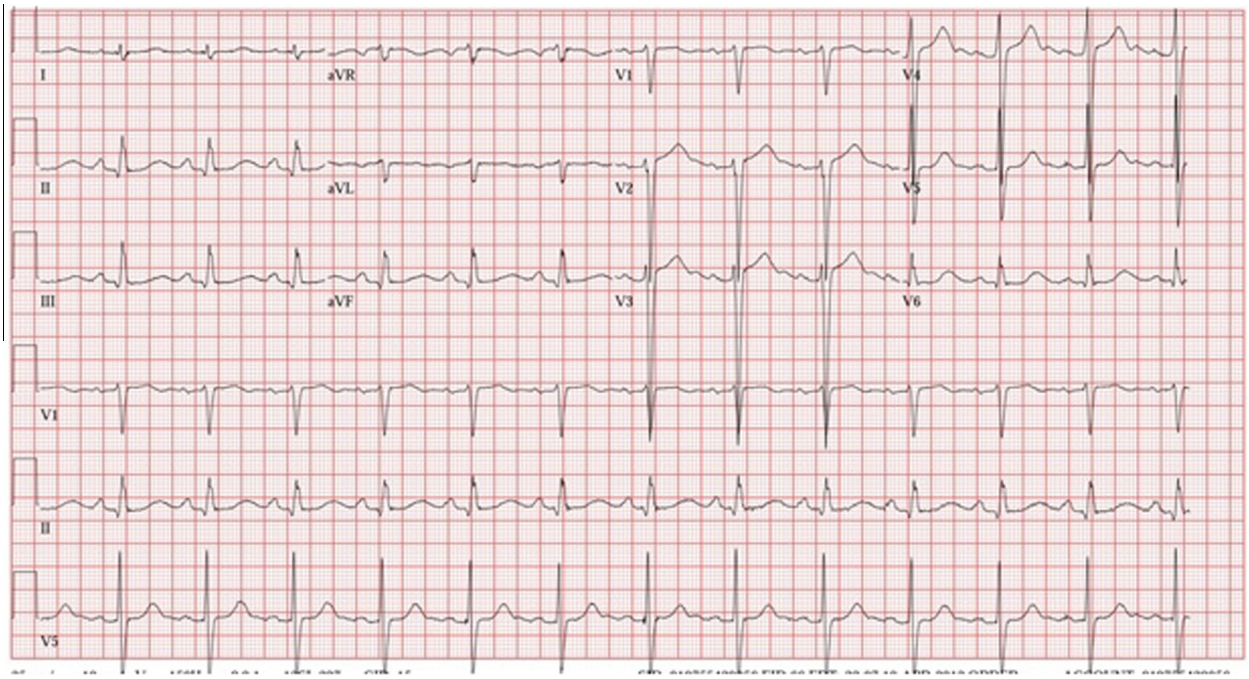

Figure 2. Resolution of Brugada type pattern. Sodium 130 mmol/L, QTc 526 milliseconds.

Case 2

A 54-year-old white man, with a history of hypertension, presented to the emergency room complaining of lethargy, vomiting, anorexia, and decreased fluid intake for 7 days. He denied any cardiovascular symptoms. Physical examination was unremarkable except for signs of dehydration. Initial labs revealed significant hyponatremia (Na^+ 106 mmol/L) with EKG showing prolonged QTc (526 milliseconds) and a ''saddle back'' type ST elevation in leads V2–V3 (Fig. 3). Detailed family history did not reveal BS. Telemetry did not show any evidence of arrhythmia. Electrophysiological studies which included programmed

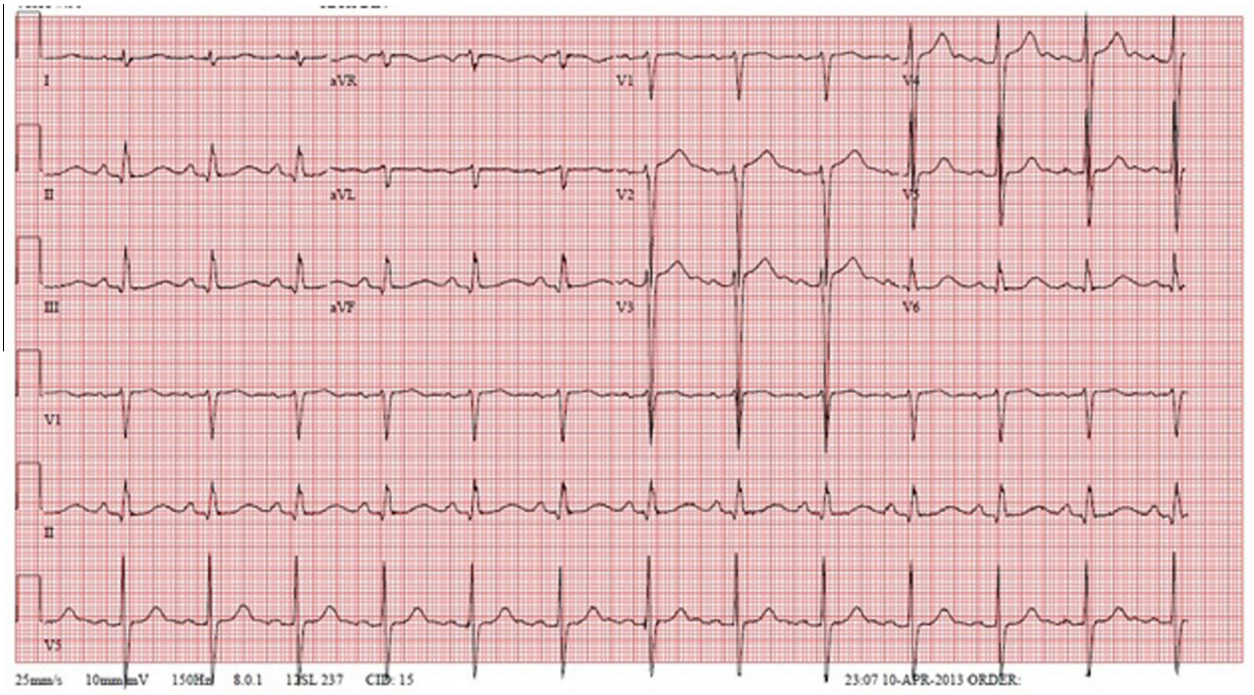

Figure 3. Brugada type 2 (saddle back) patterns in leads V2–V3. Sodium 106 mmol/L, QTc 526 milliseconds.

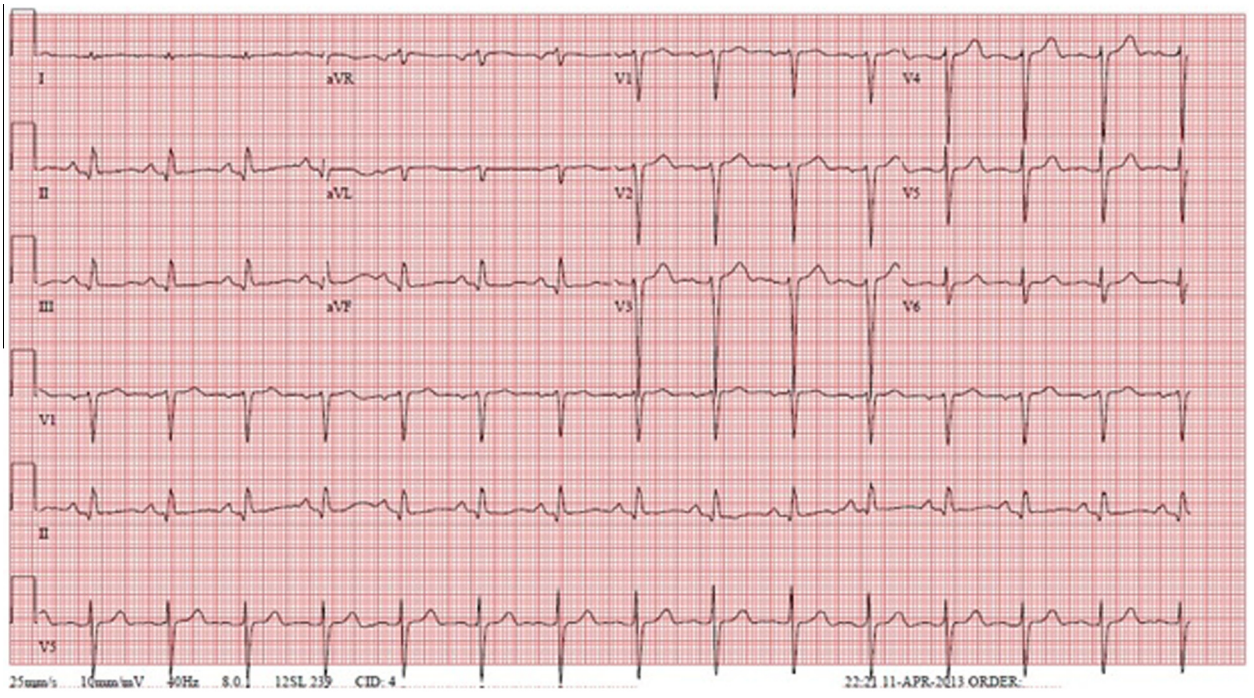

Figure 4. Resolution of Brugada type patterns. Sodium 128 mmol/L, QTc 462 milliseconds.

electrical stimulation to induce ventricular arrhythmias and left heart catheterization were unremarkable. A drug challenge test was not performed. He was fluid resuscitated with gradual return of sodium level towards normal, and serial EKGs showing resolution of EKG findings with improving sodium level (Fig. 4).

Discussion

Brugada phenocopy associated with hyponatremia has been very rarely described. There have been very few isolated case reports [4–7]. This, to the best of our knowledge, is the first case series of Brugada phenocopy with hyponatremia. Sodium channel blockers are used to unmask and/or induce EKG-manifestations of BS in susceptible patients. Electrophysiologically, hyponatremia works similarly by decreasing the electrochemical gradient and causing decreased inward current, leading to Brugada phenocopy. We believe that reduced transmembrane gradient was responsible for Brugada phenocopy in our patient which was reversible and resolved with improvement in sodium levels and potentially transmembrane gradient. BS can be differentiated from early repolarization syndromes (forme frustes) by more than 2 mm ST-elevation in the right precordial leads with greater than 110 milliseconds QRS duration which was not appreciated in both these patients [8]. Prognostic implications of these changes are unknown; however, both our patients were doing fine at the 12-month follow up at the cardiology office. Management of these patients is supportive with intensive observation. Clinicians should be aware of the association of Brugada phenocopy with hyponatremia and be vigilant for a diagnosis of true BS in cases where EKG findings fail to resolve with supportive management.

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