# Brugada-Like Electrocardiographic Changes Induced by Hypokalemia

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## Introduction

Since its description in 1992, Brugada Syndrome has become the second cause of death among young adults in some countries[1]. The Brugada syndrome is an autosomal dominant disease with incomplete penetrance, which may cause syncope and sudden cardiac death in young individuals with a normal heart. It is characterized by an electrocardiographic (ECG) pattern of complete or incomplete right bundle branch block (RBBB) and ST-segment elevation in leads V1-V3. The characteristic ECG findings are seen in some patients not affected by the syndrome[2]. Several nongenetic factors have been mentioned in the literature as possible inductors of the Brugada ECG pattern[3]. It may be induced in some patients during febrile states, electrolytes abnormalities, cocaine use and drugs that have a sodium channel-blocking effect, such as antiarrhythmics, anesthetics, and tricyclic antidepressants, among others[4]. We describe a case of a 21-year-old man who presented with severe hypokalemia and dynamic electrocardiographic changes compatible with Brugada pattern[5].

## Case Report

A 21 year-old male, white, single, with diagnosis of hypokalemic periodic paralysis (a genetic disorder associated with potassium loss), presented to the emergency room with sudden-onset tetraparesis after several hours of sleep and two episodes of vomiting. There were no respiratory or swallowing difficulties. He had no family or personal history of sudden death nor had experienced any episodes of syncope. He was taking spironolactone and denied the use of other drugs or alcohol. On physical exam his pulse was regular, and blood pressure was 112/61 mmHg in both arms. The skin was cool and sweaty. No jugular venous distension, goiter or lymphadenopathies were observed. Heart examination showed regular rhythm, no murmurs and pulse of 95 bpm. Lung and abdomen assessment was unremarkable. There were no deformities or edema of the extremities and distal pulses were present and equal bilaterally. Neurologic examination showed flaccid paralysis of all extremities, which involved the proximal and distal muscles and included the hips and shoulders. Deep tendon reflexes were slightly diminished to 2 out of 4. Cranial nerve function was intact. Routine chemistry, liver enzymes and complete blood count were normal, except for a potassium level of 1.5 (3.5–5.5 mmol/). The analysis of thyroid function was normal.

The 12-lead ECG on admission showed sinus rhythm and incomplete RBBB with ST-segment elevation in leads V1–V2, with a coved-type ST segment elevation consistent with type 1 Brugada pattern (Figure 1). A chest radiograph showed a normal-sized heart. Six hours after initiation of intravenous potassium replacement, the patient's neurologic symptoms had completed resolved. Repeat ECG showed normal sinus rhythm and electrocardiographic abnormalities disappeared (Figure 2). Echocardiography was unremarkable.

## Discussion

Several nongenetic factors have been mentioned in the literature as possible inductors of the Brugada ECG pattern. The basis of the Brugada ECG pattern and Brugada syndrome remains a topic of debate, with arguments claiming both repolarization and depolarization abnormalities as being responsible[6,7]. In both events, decrease of inward sodium (Na+) current is generally accepted as being essential[8]. The list of drugs or conditions that can unmask or induce a Brugada ECG pattern is growing[9]. Factors that can unmask or modulate the Brugada ECG pattern are beta-blockers, tricyclic or tetracyclic antidepressants, lithium, local anesthetics, fever, hyperkalemia, hypercalcemia, hyponatremia, alcohol and cocaine toxicity[4,8,10,11]. The clinical meaning and the risk of arrhythmias that induce Brugada ECG pattern are unknown. Recent researches describe the risk of cardiac events in patients with a Brugada ECG pattern, without the Brugada syndrome, during acute medical situations[4]. Juntilla et al[4] collected data on 47 patients; 26 patients with the Brugada ECG pattern due to drugs or medications; 16 with this ECG pattern developed during a febrile episode and 5 related to electrolyte imbalances[4]. Of the 47 subjects with an acute Brugada ECG pattern, 24 (51%) had malignant arrhythmias, with 18 patients developed sudden cardiac

## Keywords

Brugada syndrome; bundle-branch clock; hypokalemic.

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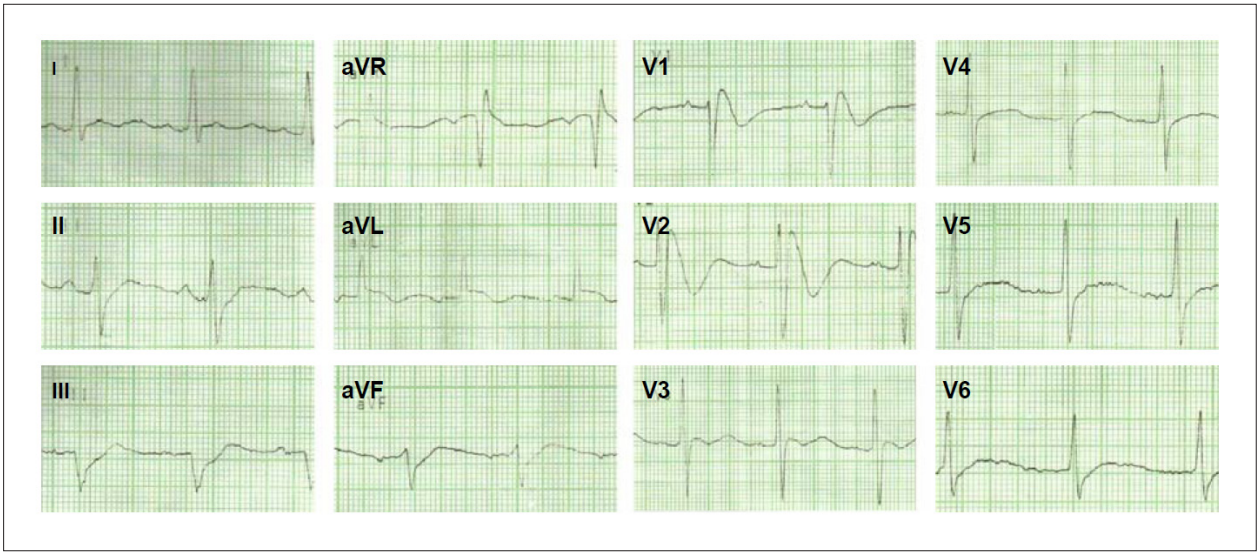


**Figure 1 -** *Twelve-lead electrocardiogram of the patient shows incomplete right bundle branch block with a coved-type ST-Segment Elevation in leads V1-V2.*

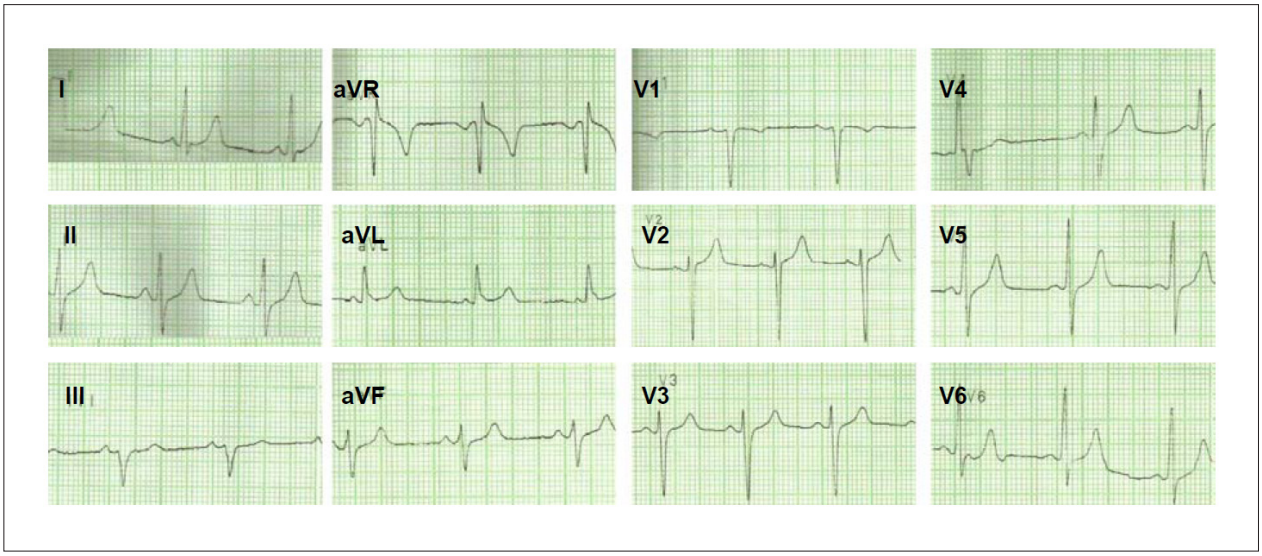


**Figure 2 -** *Twelve-lead electrocardiogram of the patient shows disappearance of both ST-segment elevation and incomplete right bundle-branch block (RBBB) after potassium replacement.*

death, 3 had ventricular tachycardia episodes, and 3 had syncope. Tsai et al[12] described the Brugada ECG pattern during a hypokalemic periodic paralysis in a man with thyrotoxicosis[12].

In patients with Brugada syndrome there also have been reports of induction of ventricular tachycardia and ventricular fibrillation associated with transient hypokalemia[13,14]. Hypokalemia induces QT prolongation and several ventricular arrhythmias because of an elevation of the resting membrane potential, prolongation of action potential duration, and an increase in the automaticity in cardiac myocytes[13]. The loss of the action potential dome due to transient outward of potassium current (Ito)-mediated phase 1 in right ventricular epicardium generates a transmural voltage gradient that underlies ST-segment elevation, similar to that observed in Brugada syndrome[14].

The most common electrocardiographic alterations induced by hypokalemia include increased U-wave amplitude, T-wave depression and rectification and increased duration of ST-segment, with consequent increase in QT interval[15]. These findings are also identified on the admission ECG, which exhibited the Brugada pattern (Figure 1), and could be useful for the differential diagnosis. Hypokalemia is usually well tolerated in the absence of

structural heart disease, especially in the presence of normal QT on the ECG[14].

The Brugada ECG pattern may be a transient phenomenon in individuals who do not have genetically determined disease and should not be considered benign. There is some evidence proposing that this transient ECG pattern is a risk factor for the development of life-threatening cardiac arrhythmias and should be aggressively treated.

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## Author contributions

Conception and design of the research: Gazzoni GF, Bergoli LCC, Soares JLF, Kalil C, Bartholomay E; Acquisition of data: Borges AP, Bergoli LCC, Soares JLF; Analysis and interpretation of the data: Gazzoni GF, Borges AP, Bergoli LCC, Soares JLF, Bartholomay E; Writing of the manuscript: Gazzoni GF, Borges AP, Bergoli LCC, Kalil C, Bartholomay E; Critical revision of the manuscript for intellectual content: Gazzoni GF, Borges AP, Bergoli LCC, Soares JLF, Kalil C, Bartholomay E.

### Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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### Study Association

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