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Brugada-Like Electrocardiographic Patterns Induced by Hyperkalemia

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Key Words: *Brugada syndrome; Brugada electrocardiographic pattern; electrocardiographic hyperkalemia signs.*

Summary. *Brugada syndrome was described in 1992 as a new clinical and electrocardiographic syndrome involving susceptibility to ventricular arrhythmias and sudden cardiac death in patients with no obvious structural heart disease.*

Brugada syndrome is characterized by a hereditary anomaly in the sodium ion channel (mutation of the SCN5A gene) identified by a wide QRS associated with the ST-segment elevation and the T-wave inversion in the right precordial leads. The Brugada-like electrocardiographic pattern can be caused by sodium channel-blocking drugs and electrolyte disorders. Hyperkalemia may produce multiple ECG abnormalities, including the ST-segment elevation and pseudomyocardial infarction with a resolution of these abnormalities after the correction of hyperkalemia.

This article describes 8 cases of pseudoanteroseptal myocardial infarction in acute renal insufficiency with hyperkalemia. The ST-segment elevation related to hyperkalemia is resolved by the reduced serum potassium level.

Clinicians should recognize that hyperkalemia is one of the etiologies of the Brugada-like electrocardiographic pattern.

Introduction

Brugada syndrome (BS) is a specific clinical and electrocardiographic, often familial, pathology, characterized by typical electrocardiographic changes and frequent sudden cardiac death in the absence of a structural heart disease or a coronary vascular pathology (1, 2). Three brothers Brugada, Spanish cardiologists, were first to describe the essence of this cardioelectrical pathology in 1992 (1, 3). Therefore, this syndrome was named after their name.

The Brugada-like electrocardiographic patterns (not BS) can appear in other situations. It is necessary to remember possible ST-segment elevations in leads V_1 through V_3 due to various conditions. They can be as follows: 1) atypical right bundle branch block; 2) acute, especially right, ventricular myocardial infarction; 3) acute pericarditis (myopericarditis); 4) hemopericarditis; 5) pulmonary embolism; 6) dissection of aortic aneurysm; 7) disorders of the central and autonomic nervous systems; 8) Duchenne's muscular dystrophy; 9) Friedreich's ataxia; 10) left ventricular hypertrophy; 11) arrhythmogenic right ventricular dysplasia; 12) mechanical compression of the right ventricular outflow tract; 13) electrical defibrillation; 14) early repolarization syndrome; and 15) hypothermia (1–3).

It is not difficult to correctly evaluate ST-segment changes in these conditions. Each of the pathologies has distinctive complaints and clinical signs, and is not related to BS, which is a genetic arrhythmogenic sodium channelopathy. ST-segment changes in the right precordial leads in the ECG in case of the Brugada syndrome are shown in Fig. 1. It is more difficult and more important to assess the signs of types 1, 2, and 3 Brugada-like ECG and partial right bundle branch block caused by electrolyte shifts, medications or their overdose, and intoxication. In these cases, it is not always easy to decide whether an electrolyte imbalance and drugs caused Brugada-like ECG images or showed previously unregistered BS ECG. These conditions could be hyperkalemia, hypercalcemia, cocaine intoxication, alcohol intoxication, and treatment and/or intoxication by different medications, such as class IA and IC antiarrhythmic agents, calcium channel blockers, psychotropic drugs, etc. (1–11).

Case Reports

We present the ECGs of 8 patients with Brugada ECG type 1 images caused by hyperkalemia. All the patients developed hyperkalemia in the oli-

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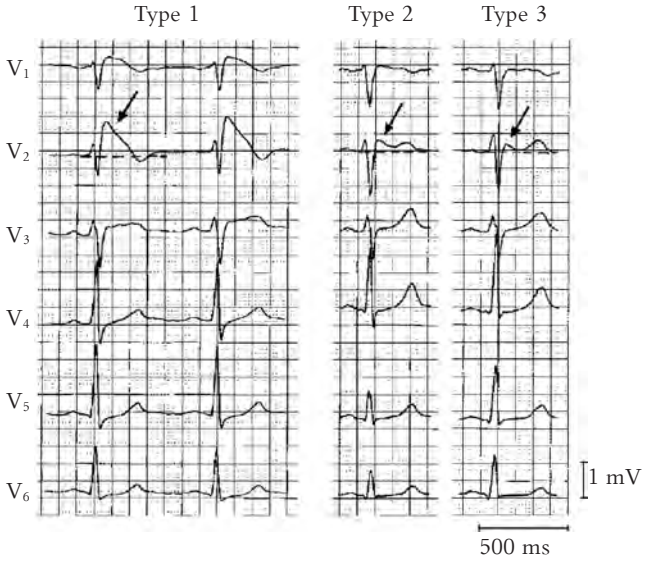


Fig. 1. ST-segment changes in the right precordial leads in the ECG in case of Brugada syndrome

goanuric period of acute renal failure. Kidney damage was caused by myoglobinemia due to muscle damage (deep burns, muscle crush syndrome, and acute compartment syndrome). In all the cases, the ECG changes were transient and disappeared when K^+ plasma levels returned to the reference values. None of the patients had a family history of BS, cardiac arrhythmias, or syncope. Therefore, the ECG changes were considered as the Brugada ECG patterns and not as BS.

Fig. 2 shows the ECGs of a 52-year-old man. The patient's death was caused by pneumonia in both sides. The autopsy showed that the coronary arteries were without lesions.

Fig. 3 shows the ECGs of a 63-year-old man.

Fig. 4 shows the ECGs of a 44-year-old patient.

Fig. 5 shows the ECGs of a 38-year-old woman.

The ECGs of other patients with hyperkalemia are shown in Figs. 6–9: Fig. 6 shows the ECG of a 40-year-old woman; Fig. 7, a 70-year-old woman; Fig. 8, a 51-year-old man; and Fig. 9, a 50-year-old man. The Brugada-like ECG images disappeared in all the ECGs of the patients after the correction of hyperkalemia. In Fig. 9, the ECG is particularly interesting because the specific ST-segment and T-wave changes are registered in the inferior leads (II, III, and aVF leads). The literature sources (3, 6) indicate that changes are possible in these leads, but we were able to detect only one such an example of the ECG.

Discussion

Electrocardiographic BS changes show a complete or partial right bundle branch block (3), but some real right bundle branch block may not be such. The ST-segment and T-wave changes in the right precordial leads (V_1 through V_3) are the most important recorded characteristics (Table).

In the same BS cases, a normal electrocardiogram can be registered or at different times all the mentioned types of ECG may be registered (1, 4). Sometimes, these specific ST-segment changes can be monitored in the inferior limb leads (6).

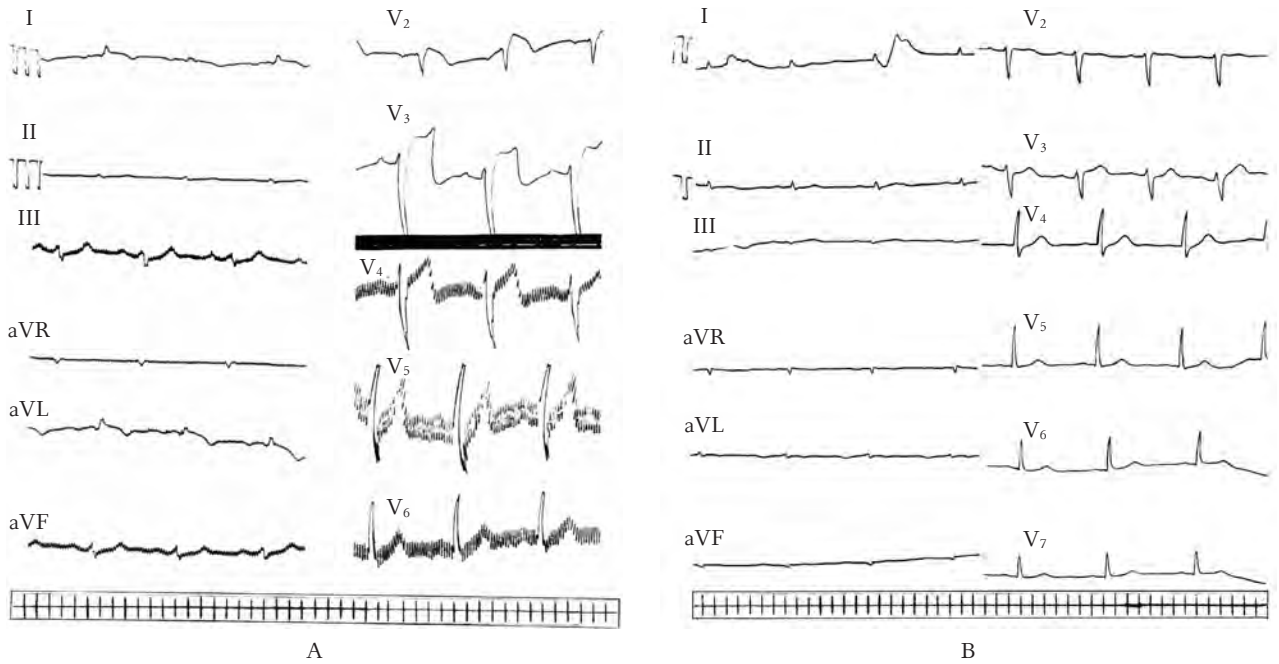


Fig. 2. The ECG of a 52-year-old man
A, K^+ 7.4 mmol/L; B, after hemodialysis, K^+ 3.9 mmol/L.

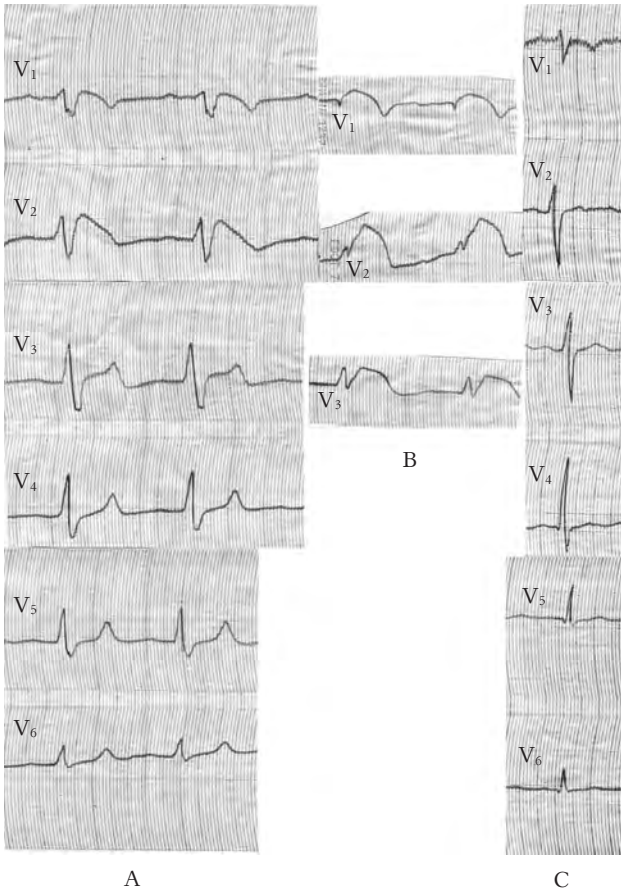


Fig. 3. The ECG of a 63-year-old man
A, K^+ 6.9 mmol/L; B, K^+ 7.2 mmol/L; and C, after hemodialysis, K^+ 4.2 mmol/L.

The BS diagnostic criteria include the type 1 ST-segment elevation greater than 2 mm and in more than one of the right precordial leads (V_1 through V_3), which are recorded spontaneously or during the tests with sodium blockers, and any one of the below mentioned: 1) documented ventricular arrhythmias, i.e., ventricular fibrillation, transient polymorphic ventricular tachycardia, and ventricular arrhythmias causing programmed electrical stimulation; 2) a family history (sudden death in the family up to the age of 45 years and type 1 ECG signs in family members since approximately 50% of the patients with BS have a family history); and 3) symptoms associated with arrhythmias (syncope and nocturnal agonal respirations) (1–3, 5, 6).

BS is inherited in an autosomal dominant way. The *SCN5A* gene and its mutations are responsible for this disease (1, 12). This gene encodes cardiac sodium channels and is responsible for the cardiac action potential phase 0 (1, 5, 12), and BS is thoroughly an electrical heart disease, a channelopathy. The normal action potential of ventricular cardiomyocytes changes, and ion current variations occur in the endocardium and the epicardium. The inhibition of inward sodium (I_{Na}) currents (a disorder caused by the SCN5A mutation) causes the imbalance of input and output currents at the end of cell action potential phase I. This creates a transmural voltage gradient, a significant right ventricular transmural and epicardiac repolarization dispersion, and conditions for reentry arrhythmias (1, 3, 4, 13).

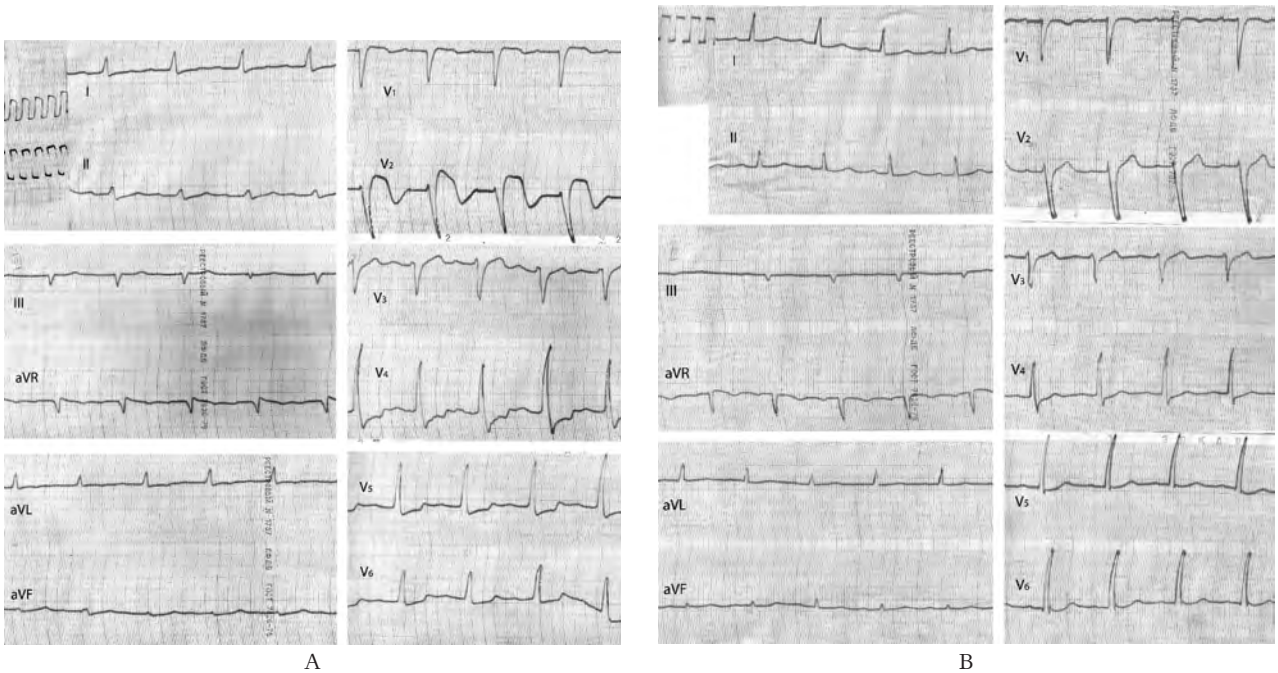
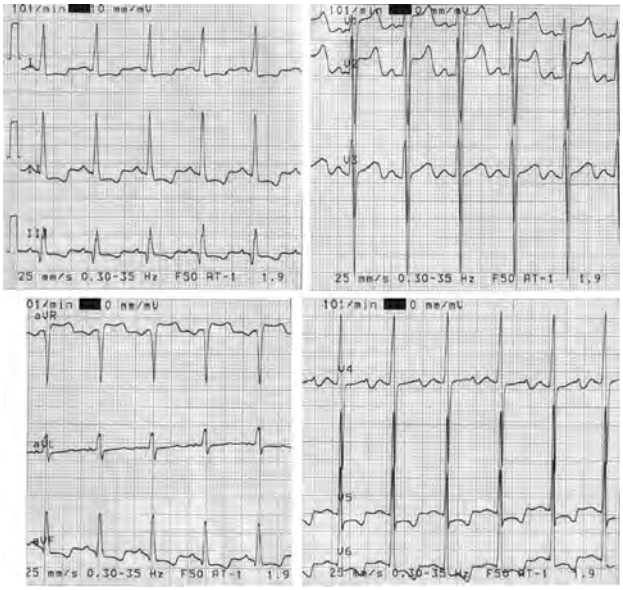
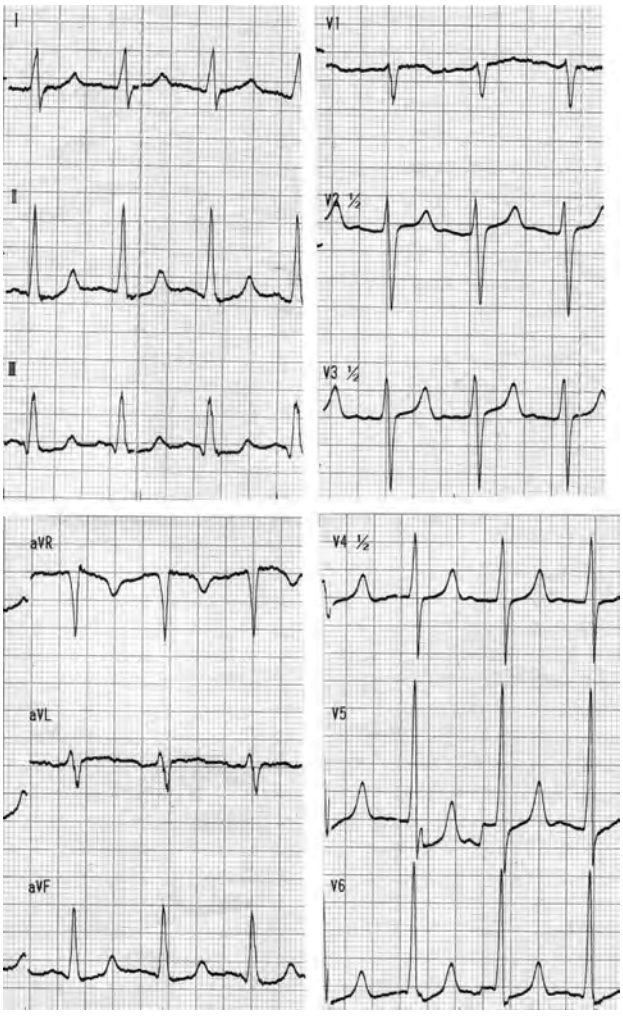


Fig. 4. The ECG of a 44-year-old patient
A, K^+ 6.9 mmol/L; B, after hemodialysis, K^+ 4.0 mmol/L.



A



B

Fig. 5. The ECG of a 38-year-old woman
A, K^+ 5.6 mmol/L; B, after hemodialysis, K^+ 4.2 mmol/L.

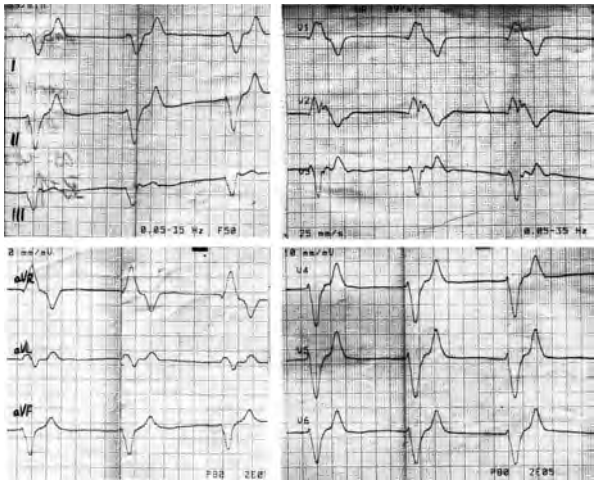


Fig. 6. The ECG of a 40-year-old woman (K^+ 6.7 mmol/L)

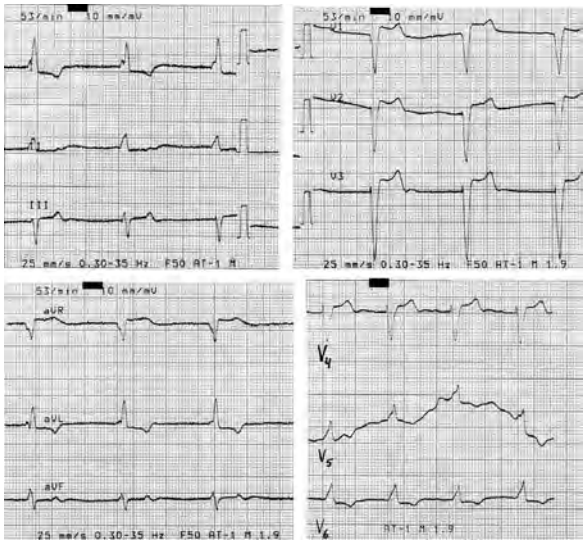


Fig. 7. The ECG of a 70-year-old woman (K^+ 6.0 mmol/L)

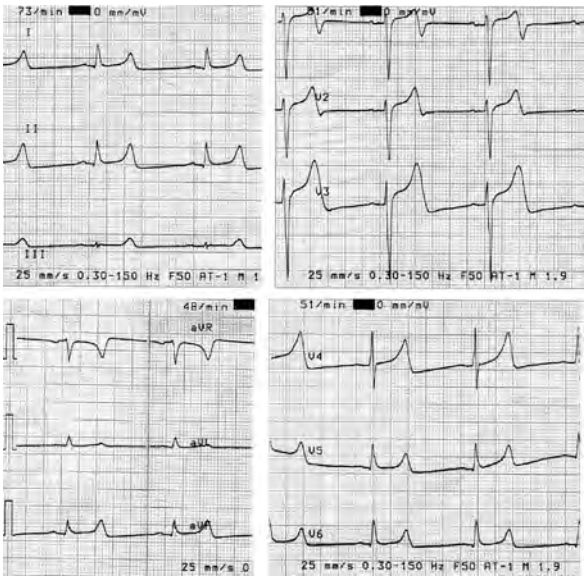


Fig. 8. The ECG of a 51-year-old man (K^+ 6.9 mmol/L)

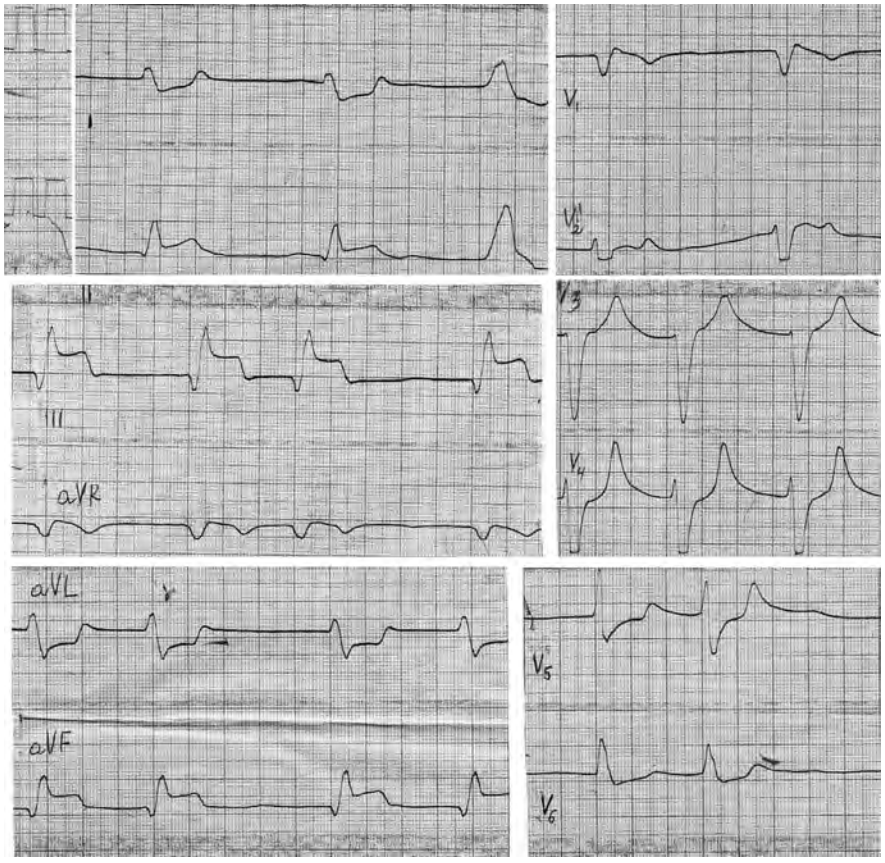


Fig. 9. The ECG of a 50-year-old man (K^+ 6.7 mmol/L)

Table. ST-Segment and T-Wave Abnormalities in Brugada Syndrome

Sign	Type 1	Type 2	Type 3
J wave amplitude	⩾2 mm*	⩾2 mm	⩾2 mm
T wave	Negative	Positive or biphasic	Positive
ST-T configuration	Coved type	Saddle-back type	Saddle-back type
Terminal part of ST segment**	Gradually descending	Elevated ⩾1 mm	Elevated <1 mm

*1 mm=0.1 mV.
**The last, second part of the half ST segment.

Hyperkalemia (the reference range of K^+ level in blood plasma is from 3.6 to 5 mmol/L) can be the cause of acute renal failure, metabolic acidosis, tissue catabolism, potassium salt overdose, etc. The most common signs of hyperkalemia are a tall, sharp T wave, QT-interval shortening, a decreasing conductivity of the atria or atrioventricular junction, and, later, of the ventricles. Rarely, hyperkalemia can cause an ST-segment dislocation to the top in the precordial leads. Previously, the changes, as mentioned above, were regarded as anterior myocardial pseudoinfarction. Even in 2010, these changes sometimes were still interpreted in such a way (14). However, in recent years, a strange pseudoinfarction ECG pattern caused by hyperkalemia was correctly associated with the Brugada ECG type 1 in scientific literature (10, 14). One patient (10) was investigated due to suspicion of BS (diagnosis was not confirmed), and the other one was not examined (14). In all the cases, if the plasma potassium level was within the reference range, these ECG changes disappeared (10, 14). Sometimes, during the examination of hyperkalemia-induced ECG changes, ST-segment shifts characteristic in the presented ECG are not considered.

How hyperkalemia may include the Brigada-like ECG patterns? Hyperkalemia reduces the resting membrane potential, i.e., a diastolic current of injury that inactivates sodium channels. This inactivation is heterogeneous in the heart muscle, but it is rather expressed in the anteroseptal area (15).

In the literature, where hyperkalemia-induced Brugada ECG patterns were described, the plasma K^+ level ranged from 6.0 to 8.8 mmol/L (10, 14,

15). The plasma K^+ level of our patients varied from 5.6 to 7.4 mmol/L. With the exception of 1 patient, the K^+ levels of all the patients were greater than 6.0 mmol/L.

Conclusions

Attention should be paid to the fact that without life-threatening Brugada syndrome, there are many reasons that can lead to the Brugada-like ECG pattern. Hyperkalemia, a electrolyte abnormality commonly observed in a clinical setting, is one of the possible reasons. An incorrect ECG evaluation, e.g., supposedly showing myocardial infarction, can cause diagnostic and treatment errors. An incorrect diagnosis of Brugada syndrome, but not a Brugada-like ECG pattern, can cause considerable confusion to the patient's prognosis and to the patient's family. In addition, it is important not to use drugs blocking cardiac sodium channels when these ECG changes are recorded as this could cause arrhythmias.

Statement of Conflict of Interest

The authors state no conflict of interest.

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