

Pseudo Brugada Pattern Due to Hyperkalemia

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Abstract

Brugada syndrome is characterized by an absence of structural heart disease, unique Electrocardiographic (ECG) changes, and a high risk of ventricular fibrillation and sudden death. We describe a case of hyperkalemia presenting a Brugada pattern.

Keywords: Brugada syndrome; Hyperkalemia; Electrocardiography

Introduction

Brugada syndrome is characterised by a hereditary anomaly in the sodium ion channel (mutation of the SCN5A gene) identified by a wide QRS associated with elevation of the ST segment (J-point elevation), T-wave inversion, in the right precordial leads. Ventricular rhythm troubles and sudden death are associated in the absence of any underlying cardiopathy [1]. In this case report we describe a patient with hyperkalemia who manifested the Brugada pattern exclusively without any other ECG features of hyperkalemia.

Case

A 26-year-old man with a history of type 1 diabetes mellitus presented to the emergency department with nausea, vomiting and epigastric pain of 4 hours duration. Diabetic ketoacidosis was diagnosed based on a glucose level of 620 mg/dL, pH of 7.2 and a positive urine dipstick for ketones. Serum potassium measured 7.7 mmol/L. Initial electrocardiography revealed right bundle branch block and tall, peaked T waves, ST segment elevation in leads V1–V3 (Figure 1). Left ventricular motion was seen as a normal in echocardiography. Six hours after the patient received intravenous fluid, calcium gluconate, bicarbonate when the electrocardiogram was repeated, the ST-segment elevation and right bundle branch block, tall, peaked T waves disappeared completely (Figure 2). Creatine kinase, creatine kinase-MB and troponin I values were normal. Subsequently, a flecainide test did not reproduce ST-segment elevation. At the time of discharge, the patient was in good condition with normal electrocardiography.

Discussion

Brugada syndrome is characterised by a hereditary anomaly in the sodium ion channel (mutation of the SCN5A gene) identified by a wide QRS associated with elevation of the ST segment (J-point elevation), T-wave inversion, in the right precordial leads. Ventricular rhythm troubles and sudden death are associated in the absence of any underlying cardiopathy [1]. Sodium channel-blocking drugs (antiarrhythmics and tricyclic antidepressants); lithium, cocaine, hypothermia and electrolyte imbalances, such as hyperkalemia, can trigger Brugada pattern. Since the description of the Brugada syndrome in 1992, 15 cases with the specific type I Brugada pattern associated with hyperkalemia were reported from the literature by Littmann et al. [2] who added nine more cases of their own. Patients with Brugada pattern associated with hyperkalemia have a poor prognosis, but differentiation from Brugada syndrome is necessary in surviving patients because of long-term morbidity/mortality implications [2-5]. Littmann et al. [2] described the hyperkalemic Brugada pattern and noted significant differences in the ECG manifestations of these patients from the genetically determined Brugada syndrome. These differences

include wide complex rhythm or wide complex tachycardia without visible P waves, and abnormal axis deviation. Typically, in patients manifesting hyperkalemic Brugada sign, coved ST-segment elevation was usually superimposed on other ECG signs of hyperkalemia, such as peaked Twaves, QRS widening or flat P waves. This case series and literature review revealed that the hyperkalemic Brugada sign usually develops at serum potassium levels of 5.8– 9.4mEq/l (mean 7.8mEq/l) and in critically ill patients. A Brugada ECG pattern, especially when accompanied by widened QRS complex, axis shifts, and loss of P waves, may be an indication of severe and possibly life-threatening hyperkalemia [2]. The electrophysiologic mechanisms underlying ECGs of patients with the hyperkalemic Brugada pattern are still uncertain. It is probable that severe extracellular hyperkalemia, which results in inactivation of the cardiac sodium channel, may also cause a transient Brugada pattern in susceptible patients [2].

Conclusion

This case illustrates an increasingly seen phenomenon of Brugada ECG pattern precipitated by hyperkalemia.

References

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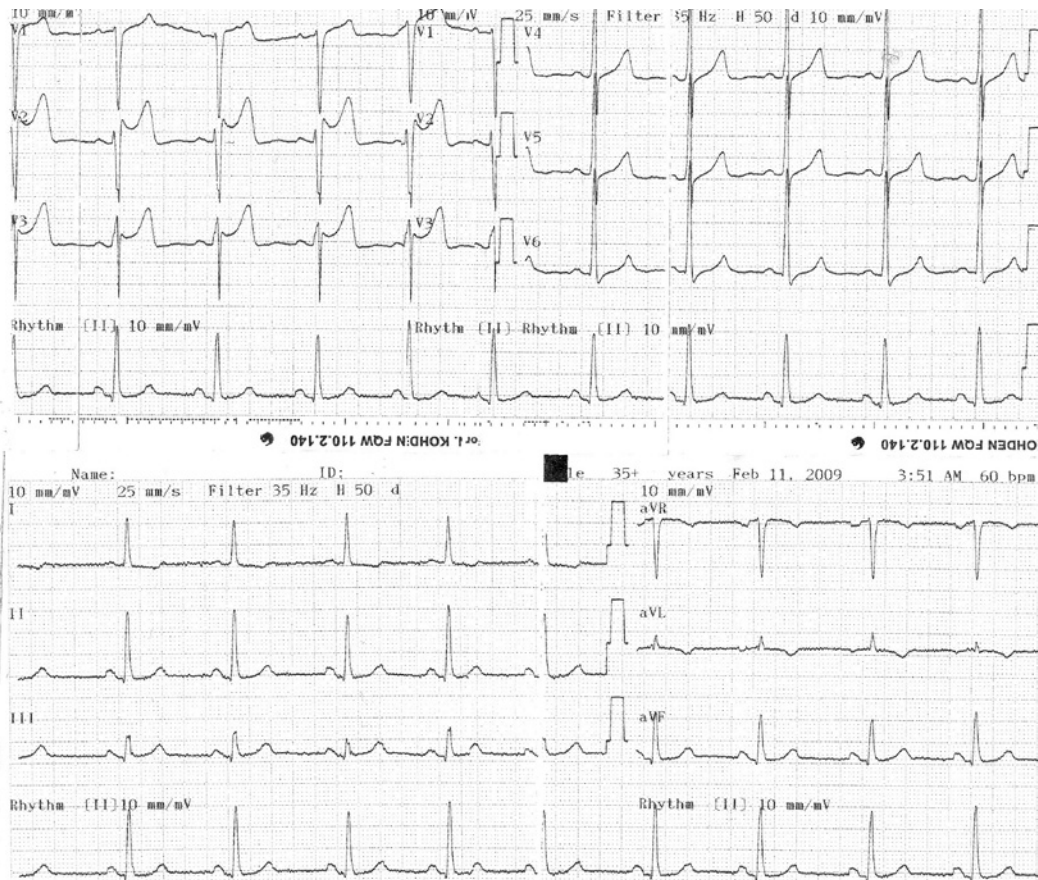


Figure 1: 12-lead ECG of the patient when his serum potassium level was more than 7 mEq/l demonstrated covered ST-segment elevation in V1 –V3, consistent with a Brugada pattern.

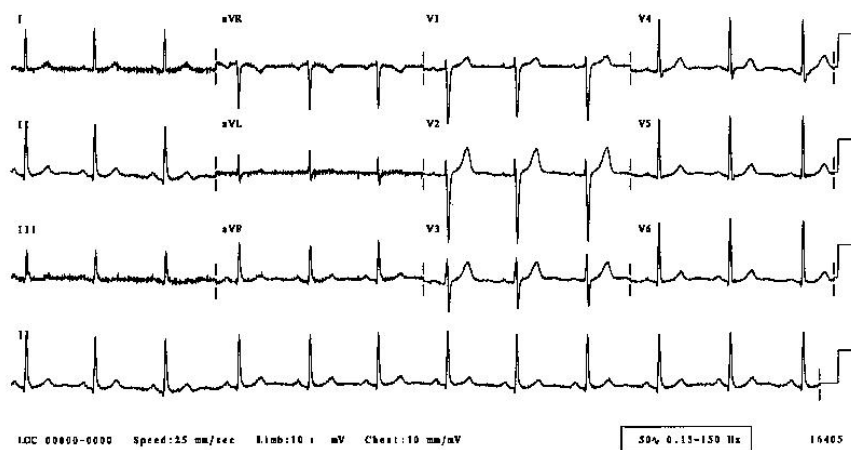


Figure 2: Normal electrocardiography after medical treatment.