CASE REPORT

Hyperkalemia-Induced Brugada Pattern with Electrical Alternans

Renguang Liu, Qinghua Chang, M.D.

From the Cardiovascular Institute of the First Affiliated Hospital of Liaoning Medical College in Jinzhou, Liaoning Province, China

The Brugada syndrome is a genetic disease (mutation of the *SCN5A* gene) that predisposes to syncope and life-threatening sudden cardiac death. The case highlights the importance of recognizing hyperkalemia, as potential triggers of the acquired Brugada sign with electrical alternans.

Ann Noninvasive Electrocardiol 2013;18(1):95–98

Brugada syndrome; hyperkalemia; electrocardiogram; electrical alternans; Intracerebral hemorrhage

The Brugada syndrome is a genetic disease (characteristically with a gene mutation affecting sodium channel function) that predisposes to syncope and sudden cardiac death (malignant arrhythmias). The syndrome is usually characterized by J-point elevation (formed J wave), coved-type ST-segment elevation, and T-wave inversion in the right precordial leads (type I). This ECG characteristics have already been highlighted. It is reported that more than 20 kinds of diseases except Brugada syndrome have recently emerged as triggers of the Brugada sign. We report a rare case of dynamic Brugada electrocardiographic pattern with electrical alternans incited by the effect of hyperkalemia.

CASE REPORT

A 49-year-old man was admitted to our hospital because of monolateral lower extremity paralysis and transient unconsciousness for 2 hours. His medical history included hypertension and chronic renal insufficiency. On admission, his blood pressure was 160/90 mmHg and computed tomography (CT) signs provided right basilar artery bleeding. Laboratory investigations were noteworthy for

serum potassium of 8.83 mmol/l, urea of 50.01 mmol/l, crea of 1412 mmol/l, and HCO_3^- of 8.9 mmol/l. The clinical diagnoses included hypertension, intracerebral hemorrhage, and chronic renal insufficiency. His ECG recording (Fig. 1) demonstrated regular QRS widening without visible P waves. The QRS complex duration was 160 milliseconds (prolonged 80 milliseconds compared with normal QRS complex duration in Fig. 3) and was in qR pattern in lead V_1 and qRs pattern in lead V_2 . There was a prominent J wave followed by a coved type ST-segment elevation in lead V_1 and a saddle-back type ST-segment elevation in lead V_2 resembling the Brugada syndrome. The long V_2 recording presented QRS-J-T electrical alternans (refer to Fig. 1, the 2nd, 4th, 6th beats, prominent decrease in S waves and T waves, elevation in J waves). ECG characteristics demonstrated hyperkalemia-induced sinoventricular conduction, intraventricular block, and Brugada-type electrocardiogram with electrical alternans. As the improvement of acidosis and hyperkalemia, there was a progressive normalization of the ECG (Fig. 2). When serum potassium was 7.75 mmol/l, QRS duration decreased to 100 milliseconds and qRs pattern transformed into rS pattern in lead V_2 . J waves

Address for correspondence. Prof. Renguang Liu, Cardiovascular Institute, The First Affiliated Hospital of Liaoning Medical College, Renmin Street, Jinzhou 121001, Liaoning Province, China. Fax: 0086-0416-4197105; E-mail: liurenguangleaoshi@126.com

Conflict of interest: All authors declare that we have no conflict of interest.

Figure 1. The ECG recorded with a serum potassium of 8.83 mmol/l shows regular QRS widening without visible P waves. The QRS complex is in qR pattern in lead V₁ and qRs pattern in lead V₂. There is a prominent J wave followed by a coved type ST-segment elevation in lead V₁ and a saddle-back type ST-segment elevation in lead V₂.

and ST-segment elevation stood still, but both of them were diminished. When serum potassium decreased to 6.16 mmol/l, an ECG tracing (Fig. 3) showed that there was a sinus rhythm without visible J waves, the QRS complex duration was 80 milliseconds and the QRS complex was in rS pattern in lead V_{1,2} but T waves were still tall and upright. When the potassium concentration was returned to 4.2 mmol/l, a new electrocardiographic study was carried out, in which peaked T wave reverted to normal.

DISCUSSION

Although hyperkalemia can display diverse manifestations on electrocardiography, a presentation mimicking a Brugada pattern is very unusual.

Prompt recognition of this electrocardiographic entity may expedite the initiation of appropriate diagnosis and treatment. Reviewing literatures¹⁻⁶ in recent 10 years, we can conclude that the hyperkalemia Brugada sign usually developed as follows: (1) Serum potassium levels were usually 5.8–9.4 mmol/l; (2) Patients were always in critically ill or administered medicine that could block sodium channel and prolong QT-interval, or committed cocaine challenge, alcohol intoxication, hyperglycemia, hyponatremia, thiamine deficiency, fever, and so on; (3) ECG characteristics of patients who exhibited the hyperkalemic Brugada sign manifest widened QRS complex, axis shifts, and especially loss of P waves. These clinical and electrocardiographic characteristics not only differentiate it from the genetic-type Brugada but also may be an

Figure 2. The ECG recorded with a serum potassium of 7.75 mmol/l reveals that QRS duration decreases to 100 milliseconds and qRs pattern transforms into rS pattern in lead V₂. J waves and ST-segment elevation stand still, but both of them are diminished.

Figure 3. The ECG recorded with a serum potassium of 6.16 mmol/l demonstrates that there is a sinus rhythm without visible J waves, the QRS complex duration is 80 milliseconds and the QRS complex is in rS pattern in lead V_{1,2} but T waves are still tall and upright.

indication of severe and possibly life-threatening hyperkalemia. Therefore immediate treatment of hyperkalemia rather than a generic treatment of wide complex rhythm or wide complex tachycardia may be life saving. Our patient suffered intracerebral hemorrhage and chronic renal insufficiency and the electrocardiogram revealed widened QRS complexes, abnormal QRS axis, and absence of P waves. The main clinical and electrocardiographic characteristics of our case were remarkably similar to those found in the literatures. The ECG pattern resembled Brugada syndrome, which completely resolved after positive correction of the abnormal electrolyte. This further confirmed our diagnosis.

The electrophysiologic mechanisms underlying these ECG abnormalities are displayed as follows: (1) Hyperkalemia-induced widened QRS complexes and absence of P waves: Hyperkalemia decreases the resting membrane potential, which leads to inactivation of sodium channels. The inactivation of sodium channels decreases the velocity and amplitude of depolarization in the phase 0 of the action potential, which slows down the capability of conduction to lose it. The negative conduction effects of hyperkalemia depend on the tissue involved. The atrial and ventricular myocardial are more sensitive than the autonomous conduction system. Therefore, severe hyperkalemia can cause regular QRS widening and sinoventricular conduction (absence of P waves) pattern in the electrocardiogram. Once the autonomous conduction system was involved, atrioventricular, the branch or the bundle branches block may emerge. (2) The electro-

physiologic mechanism underlying hyperkalemia-induced ECG abnormalities is uncertain. Conceivably, the negative effects of hyperkalemia may be heterogeneous throughout the ventricles. Being more pronounced in some regions, then the antero-septal wall appears to be more sensitive to these negative effects, depolarization in this region is delayed much more serious and leads to J-point and ST-segment elevation in the right precordial leads. If depolarization of considerable myocardial cells in the antero-septal wall were significantly delayed, initial r waves would vanish and q waves would appear in the right precordial leads (simulating acute antero-septal myocardial infarction in Fig. 1). Our case also displayed QRS-J-T electrical alternans. This phenomenon may be induced by regularly delayed depolarization.

REFERENCES

1. Tanawuttiwata T, Harindhanavudhi T, Bhan A, et al. Hyperkalemia-induced Brugada pattern: An unusual manifestation. *J Cardiovasc Med* 2010;11:285–287.
2. Ortega-Carnicer J, Benezet J, Ruiz-Lorenzo F, et al. Transient Brugada-type electrocardiographic abnormalities in renal failure reversed by dialysis. *Resuscitation* 2002;55:215–219.
3. Garnier F, Mathe A, Bruyeres R. Images in cardiology: Electrical aspects of Brugada: Hyperkalaemia and intoxication with Phenothiazines. *Heart* 2008;94:1578.
4. Irani F, Kasmani R, Kanjwal Y. Hyperkalemia and cocaine induced dynamic Brugada-type electrocardiogram. *Eur J Emerg Med* 2010;17:113–115.
5. Kovacic JC, Kuchar DL. Brugada pattern electrocardiographic changes associated with profound electrolyte disturbance. *Pacing Clin Electrophysiol* 2004;27:1020–1023.
6. Littmann L, Monroe MH, Taylor L 3rd, et al. The hyperkalemia Brugada sign. *J Electrocardiol* 2007;40:53–59.