Brugada-Like Electrocardiographic Pattern Due to Hyperkalemia

S. Kurisu et al: *Clin Cardiol* 2009;32:E23

To the editor:

Dr. Satoshi Kurisu and his colleagues¹ describe an interesting case of a patient who developed a Brugada pattern electrocardiogram (ECG) due to hyperkalemia.¹ It is amazing to observe the changes reverting to normal as hyperkalemia resolves.

Brugada syndrome is characterized by wide QRS associated with an elevation of the ST-segment (J-point elevation) and T-wave inversion in leads V₁ to V₃. It is due to a hereditary anomaly in the sodium ion channel (mutation of the *SCN5A* gene) and may manifest as syncope and sudden death. However, Brugada pattern ECG or Brugada sign is reported to occur due to anatomic compression of the right ventricle, electrolyte abnormalities like hyperkalemia,^{1,2} hyperglycemia, hyponatremia, and acidosis. An isolated case of Brugada pattern due to electrocution³ has also been reported. The Brugada-like ECG pattern may be found approximately in 0.10% of healthy persons.⁴

Most cases with hyperkalemia-induced Brugada sign were reported in a series of critically ill males and the hyperkalemic Brugada pattern differed in many ways from the ECG of patients with the genetic Brugada syndrome.⁵

These patients had diminished or absent P waves, wide complex rhythms with marked QRS widening, and an abnormal QRS axis.

The patient described in this case report had nearly absent P waves, widened QRS interval, and tall tented T waves along with the Brugada sign. These associated hyperkalemic ECG changes help differentiate this Brugada pattern from the genetic Brugada syndrome. Prompt recognition of these subtle ECG changes may help initiation of early treatment of hyperkalemia.

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Author reply:

I thank Dr. Kumar for his thoughtful comments and appreciate his great interest in our report.

We have previously reported clinical profiles of 40 consecutive patients with symptomatic hyperkalemia (serum potassium >6.0 mmol/L). In this series, diminished or absent P waves and QRS widening were frequently found. However, Brugada sign was found in only 1 patient (2.5%), suggesting that the development of the Brugada sign during hyperkalemia is the exception rather than the rule.

Most patients with hyperkalemia induced Brugada sign are critically ill and there has been no report demonstrating

electrophysiological study or sodium channel blocker challenge after its resolution.

Further studies are necessary to clarify whether patients with Brugada sign during hyperkalemia have a hereditary anomaly in the sodium channel.

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