# Cocaine And Hyperkalemia Unmasked The Electrocardiogram (EKG) Pattern Of Brugada Syndrome

A Hamo, F Al-Khitan, H Guirgis, P Wehner

**Citation**

A Hamo, F Al-Khitan, H Guirgis, P Wehner. *Cocaine And Hyperkalemia Unmasked The Electrocardiogram (EKG) Pattern Of Brugada Syndrome*. The Internet Journal of Cardiology. 2012 Volume 10 Number 2.

**Abstract**

A very few case reports have indicated that hyperkalemia can induce a Brugada pattern in the electrocardiogram (EKG). On the other hand, very rare case reports have indicated that cocaine has precipitated life-threatening arrhythmias associated with development of Brugada syndrome. We present a 26-year old patient with hyperkalemia secondary to muscle damage and renal insufficiency after a reported large intake of cocaine. The electrocardiogram showed a Brugada pattern. These EKG changes disappeared directly after normalization of serum potassium. We concluded that cocaine and the hyperkalemia were probably the culprit causes of the Brugada-pattern EKG. Unfortunately, the provided data in this case appear to be incomplete; the patient was found unresponsive at home, and we do not exactly whether or not he developed a cardiac arrest secondary to a malignant arrhythmia before the arrival of the Emergency medical services. This case highlights the importance of recognizing cocaine and hyperkalemia as potentialtriggers of the acquired Brugada-like electrocardiographic pattern.

## INTRODUCTION

Multiple clinical conditions may exacerbate or unmask the electrocardiogram (EKG) pattern of Brugada syndrome. Examples are hyperkalemia, hypokalemia, hypercalcemia, alcohol consumption, cocaine intoxication, a febrile state, and the use of sodium-channel blockers.

Very few case reports have indicated that hyperkalemia can induce a Brugada pattern in the electrocardiogram. On the other hand, rare case reports have indicated that cocaine has precipitated life-threatening arrhythmias associated with development of Brugada syndrome.

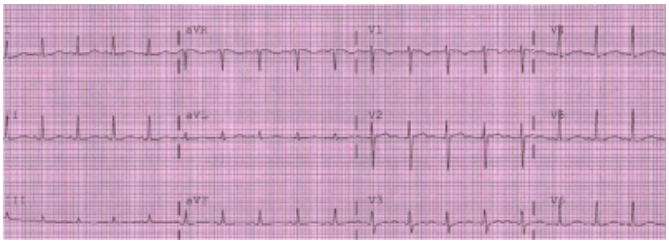
We present a young healthy patient with hyperkalemia secondary to muscle damage and renal insufficiency after a reported large intake of cocaine. The electrocardiogram showed a Brugada pattern. These EKG changes disappeared directly after normalization of serum potassium.

## CASE REPORT

A 26 year-old man with history of illicit drug use experienced a syncopal episode after using cocaine. His initial EKG showed incomplete right bundle branch block (RBBB) and ST-segment elevation in the right precordial leads (V1-V2) (Figure 1). Medical and family histories were unremarkable. Urine drug screen was positive for cocaine and marijuana. Chemistry panel revealed rhabdomyolysis, acute renal failure, and severe hyperkalemia of (7.5 meq/L ), which was treated emergently with administration of saline, calcium gluconate, insulin, in addition to sodium bicarbonate.

**Figure 2**

Figure 2: 12-Lead EKG demonstrated a resolution of ST-segment elevation shortly after treatment of hyperkalemia.



Eventually the Serial EKGs demonstrated a resolution of ST-segment elevation shortly after treatment (Figure 2).

{image:2}

## DISCUSSION

The case described here is most likely consistent with Brugada syndrome precipitated by cocaine and hyperkalemia, each has been individually reported as a precipitating factor. Unfortunately, the provided data in this

case appear to be incomplete; the patient was found unresponsive at home, and we do not know exactly whether or not he developed a cardiac arrest secondary to a malignant arrhythmia before the arrival of the emergency medical services.

Now it is known that cocaine, hyperkalemia, and the usage of certain medications such as sodium channel blocking agents may increase the risk of developing symptomatic Brugada syndrome. Whether the risk increases when hyperkalemia is combined with cocaine or any of these other agents has not been reported yet, but given the number of patients receiving such combinations, definitely deserves further investigative studies.

## CONCLUSION

Although Brugada syndrome is relatively uncommon, its association with sudden cardiac death mandates its prompt recognition and treatment. On occasion, diagnosis of Brugada syndrome is made difficult by temporary normalization of the EKG. This case highlights the importance of recognizing cocaine and hyperkalemia, as potential triggers of the acquired Brugada-like electrocardiographic pattern.

### References

1. Brugada P, Brugada J. Right bundle branch block, persistent ST segment elevation and sudden cardiac death: a distinct clinical and electrocardiographical syndrome. A multicenter report. J Am Coll Cardiol 1992;20:1391–6.
2. Brugada J, Brugada R, Antzelevitch C, Towbin J, Nademanee K, Brugada P. Long-term follow-up of individuals with the electrocardiographic pattern of right bundle-branch block and ST-segment elevation in precordial leads V1 to V3. Circulation 2002;105:73–8.
3. Bebarta VS, Summers S. Brugada electrocardiographic pattern induced by cocaine toxicity. Ann Emerg Med. 2007; 49: 827–829.
4. Brugada R, Brugada P, Brugada J. Electrocardiogram interpretation and class I blocker challenge in Brugada syndrome. J Electrocardiol Suppl. 2006; 39: S115–S118.
5. Belhassen B, Viskin S, Fish R, Glick A, Setbon I, Eldar M. Effects of electrophysiologic-guided therapy with Class IA antiarrhythmic drugs on the long-term outcome of patients with idiopathic ventricular fibrillation with or without the Brugada syndrome. J Cardiovasc Electrophysiol 1999;10: 1301–12.
6. Wilde AA, Antzelevitch C, Borggrefe M, Brugada J, Brugada R, Brugada P, Corrado D, Hauer RN, Kass RS, Nademanee K, Priori SG, Towbin JA; Study Group on the Molecular Basis of Arrhythmias of the European Society of Cardiology. Proposed diagnostic criteria for the Brugada syndrome: consensus report. Circulation. 2002; 106: 2514–2519.
7. Porres JM, Brugada J, Urbistondo V, García F, Reviejo K, Marco P. Fever unmasking the Brugada syndrome. Pacing Clin Electrophysiol. 2002; 25: 1646–1648.
8. Vernooy K, Sicouri S, Dumaine R, Hong K, Oliva A, Burashnikov E, Timmermans C, Delhaas T, Crijns HJ, Antzelevitch C, Rodriguez LM, Brugada R. Genetic and biophysical
9. basis for bupivacaine-induced ST segment elevation and VT/VF: anesthesia unmasked Brugada syndrome. Heart Rhythm. 2006; 3: 1074–1078.
10. Dumaine R, Towbin JA, Brugada P, Vatta M, Nesterenko DV, Nesterenko VV, Brugada J, Brugada R, Antzelevitch C. Ionic mechanisms responsible for the electrocardiographic phenotype of the Brugada syndrome are temperature dependent. Circ Res. 1999; 85: 803–809.
11. Kurita T, Shimizu W, Inagaki M, et al. The electrophysiologic mechanism of ST-segment elevation in Brugada syndrome. J Am Coll Cardiol 2002;40:330–4.
12. Kovacic JC, Kuchar DL. Brugada pattern electrocardiographic changes associated with profound electrolyte disturbance. Pacing Clin Electrophysiol 2004;27:1020–3.
13. Hermida JS, Jandaud S, Lemoine JL, et al. Prevalence of drug-induced electrocardiographic pattern of the Brugada syndrome in a healthy population. Am J Cardiol 2004;94:230–3.
14. Antzelevitch C, Brugada P, Borggrefe M, et al. Brugada syndrome: report of the second consensus conference: endorsed by the Heart Rhythm Society and the European Heart Rhythm Association [published erratum appears in Circulation 2005;112:e74]. Circulation 2005;111:659–70.
15. Bezzina C, Veldkamp MW, van Den Berg MP, et al. A single Na(+) channel mutation causing both long-QT and Brugada syndromes. Circ Res 1999;85:1206–13.

## Author Information

**Abdrhman Hamo, MD, M.MED**
Department of Cardiovascular Services and Internal Medicine, Joan C. Edwards School of Medicine, Marshall University

**Farah Al-Khitan, MD**
Department of Cardiovascular Services and Internal Medicine, Joan C. Edwards School of Medicine, Marshall University

**Hany Guirgis, MD**
Department of Cardiovascular Services and Internal Medicine, Joan C. Edwards School of Medicine, Marshall University

**Paulette Wehner, MD**
Department of Cardiovascular Services and Internal Medicine, Joan C. Edwards School of Medicine, Marshall University