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

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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. 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 potential triggers of the acquired Brugada-like electrocardiographic pattern.

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.

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

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