Reversible Cocaine-Induced Brugada Pattern in a Middle-Aged Man with Chest Pain

Saira Imran1,2, Maxwell Eyram Afari1,2, Ameya Hodarkar1,2, Fady Y. Marmoush1,2, Alice Y. Kim1,3

1. Department of Medicine, Memorial Hospital of Rhode Island, Pawtucket-RI.
2. Alpert Medical School of Brown University.
3. Harvard Medical School

Abstract

Brugada syndrome is an autosomal dominant disorder, characterized by electrocardiographic manifestations of a pseudo right bundle branch block and a distinctive ST segment elevation in the precordial leads, with a predisposition to ventricular arrhythmias and sudden cardiac arrest. Many medications and toxins have been reported to induce a Brugada pattern on electrocardiogram. We present the case of a middle-aged man who developed Brugada Type II electrocardiogram after cocaine ingestion.

Key words: brugada pattern, electrocardiogram, cocaine, chest pain

Citation: Imran S., Afari M.E., Hodarkar A., Marmoush F.Y., and Kim A.Y. Reversible Cocaine-Induced Brugada Pattern in a Middle-Aged Man with Chest Pain. International Cardiovascular Forum Journal. 2015;4:77-78. DOI: 10.17987/icfj.v4i0.141

Case Report

A 46-year-old man with a medical history of major depression and polysubstance abuse presented to the emergency room with moderate sub-sternal chest pain. He admitted to cocaine ingestion prior to coming to the ED. The review of systems was negative for palpitations, dizziness, and lower extremity edema, shortness of breath orthopnea, paroxysmal nocturnal dyspnea or syncope. His mother had a myocardial infarction at the age of 45 years but he denied any sudden cardiac death or syncope in the family. His home medications were gabapentin and oxycodone as needed for chronic back pain.

On presentation, he was afebrile, normotensive (137 x 96 mmHg), tachycardic (heart rate: 130 beats per minute and regular) and tachypneic (respiratory rate: 20 beats per minute) with oxygen saturation of 100 % on ambient air. His cardiopulmonary exam as well as the rest of the systemic examination was normal. An electrocardiogram revealed sinus rhythm, and ST segment elevation in leads V1-V2 with a distinct Brugada saddle-back pattern (Figure 1). Chest radiograph was unrevealing. Urine toxicology confirmed cocaine usage. Laboratory work up including serial cardiac enzymes, chemistry and a complete blood count were normal. An echocardiogram showed preserved ejection fraction, with no regional wall motion abnormalities.

The chest discomfort was relieved by sublingual nitroglycerin and oral benzodiazepine. Repeat EKG after a couple of hours showed the resolution of the Brugada II pattern (Figure 2). Patient enrolled into an inpatient substance abuse program and has since then not used cocaine. A follow up electrocardiogram, 6 months later confirmed sinus rhythm with normal QTc interval.

Discussion

Brugada syndrome is an autosomal dominant disorder with a predisposition to ventricular arrhythmias and sudden cardiac arrest. Brugada electrocardiogram pattern refers to distinctive segment elevation in the precordial leads without the associated clinical criteria for the syndrome (syncope, cardiac arrest, family history of sudden death <45 years and polymorphic ventricular tachycardia).

Two main electrocardiogram patterns have been described. In

![Figure 1: Electrocardiogram on presentation. This demonstrates segment elevation with the “saddle-back” (see arrow). Also evident is an incomplete right bundle branch configuration and heart rate of 87](image1.png)

![Figure 2: Follow up Electrocardiogram, 2 hours after the first electrocardiogram. This shows normal sinus rhythm, heart rate of 56, normal QRS and QTc and PR intervals. The “saddle” ST elevation has resolved](image2.png)
Figure 3: Electrocardiogram shows type I Brugada (Coved type) showing J point elevation and ST-segment elevation of ≥2 mm at its peak which descends with an upward convexity to an inverted T wave. A pseudo right bundle branch block (rSR’1) is also observed.

Figure 4: Electrocardiogram shows Type II Brugada (saddle-back type) which shows a gradually descending ST-segment of ≥2 mm, which gives rise to a gradually descending ST-segment elevation (remaining ≥1 mm above the baseline) followed by a positive T wave. A pseudo right bundle branch block (rSR’1) is noted.

Type I Brugada (coved type), the elevated ST segment (≥2mm) descends with an upward convexity to an inverted T wave (Figure 3). In type II Brugada pattern (saddle) the elevated ST segment descends towards the baseline and then suddenly rise to an upright or biphasic T wave (Figure 4). Both could have a pseudo right bundle branch block.

The Brugada syndrome is postulated to be due to the genetic mutation of the genes SCN5A and SCN10A. These genes are responsible for encoding the alpha subunits of the myocardial sodium channels. The defective sodium channel reduces the fast inward flow of sodium while at the same time causing a transient increase in the outward current affecting the early portion of the plateau phase of the action potential. The differential effect on the action potential between the endocardium and epicardium is responsible for the ST segment elevation on electrocardiogram.

Many medications and toxins have been reported to induce a Brugada pattern.1,2 Cocaine is a sympathomimetic stimulant which blocks the re uptake of norepinephrine, serotonin and dopamine. Its sympathomimetic effect potentiates tachyarrhythmia like supraventricular tachycardia, ventricular tachycardia and fibrillation. It acts by a) increasing sympathetic activity on myocardium and can increase automaticity,3 b)Just like several class IA and 1C anti-arrhythmic drugs, it can block the sodium channels and cause prolonged QT and a Brugada EKG pattern4-6 and c) it increases calcium inflow into the myocardial cells thus causing early after depolarization which is a mechanism for ventricular arrhythmias.

Management of cocaine-induced brugada entails supportive care. Benzodiazepines attenuate the sympathetic effect while nitroglycerin is helpful to counter vasospasm. The use of beta blockers should be avoided in cocaine use due to the concern with the unopposed alpha receptors effect which may exacerbate vasoconstriction. Some reports have suggested a role for bicarbonate in the treatment of cocaine induced arrhythmias.7 Alkalization of the extracellular pool increases the unionized fraction of cocaine and dissociation of cocaine from the sodium channels.

The prognosis of cocaine-induced Brugada pattern is unknown. It has been postulated that patients with Brugada pattern unmasked by toxins or medications are at high risk for ventricular arrhythmias.1 In a case series, 38% of the patients had cardiac arrest. It is suspected but has not been proven that patients with Brugada pattern could theoretically be having a genetic predisposition with an underlying sodium channel dysfunction. Since our patient did not undergo a drug challenge test (with flecainide), an underlying genetic defect cannot be ruled out.

Conclusion
Cocaine can induce Brugada pattern on electrocardiogram. Recognizing Brugada pattern is important to avoid misdiagnosis, especially when chest pain is the leading presentation, which could further lead to unnecessary medical interventions. It is unclear if drug-induced Brugada pattern has similar life threatening consequences as Brugada syndrome and this need to be investigated.

Statement of ethical publishing
The authors state that they abide by the statement of ethical publishing of the International Cardiovascular Forum Journal8.

Acknowledgement:
We thank Mobeen Ur Rehman, MD for sketching the Figures (3 and 4) to accurately depict Brugada pattern on electrocardiogram.

Consent:
Patient’s consent has been obtained prior to this submission.

Conflict of interest:
The authors declare that there is no conflict of interest.

Address for correspondence:
Maxwell Eyram Afari, Department of Medicine
Memorial Hospital of Rhode Island
Alpert Medical School of Brown University
111 Brewster Street, Pawtucket, RI 02860
E-mail: maxieafari@yahoo.co.uk

References

