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Case Report

A tricky electrocardiogram: cocaine induced Brugada type 2 phenocopy

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Abstract: Background: The pharmacological effects of cocaine have been associated with different types of cardiac dysrhythmias and with Brugada pattern on the ECG, but currently only type 1 pattern has been described. We report a case of a transient Brugada type 2 pattern in a young cocaine abuser. Method: We report the clinical presentation of a 32-year-old male with a history of cocaine abuse. Result: The treatment and the resolution of the acute phase have been described; moreover we discuss the pathophysiology of the Brugada phenocopy in this specificclinical setting. Conclusion: The clinical impact of our case report underscores the necessity of prompt physician awareness of any ECG abnormality besides myocardial infarction in patients with cocaine abuse, such as any Brugada pattern, which could lead to ominous ventricular arrhythmias.

Keywords: ECG, ECG alterations, cocaine, Brugada

Introduction

Brugada syndrome is most frequently reported in young individuals in the absence of gross heart disease. It is a genetically determined disease caused by mutations in the sodium channels leading to high electrical vulnerability, reentrant ventricular arrhythmias and sudden cardiac death.

Cocaine abuse has been associated with different types of cardiac dysrhythmias and with a Brugada pattern on the ECG, but to date only type 1 pattern has been described. We report a case of a transient Brugada type 2 pattern in a young cocaine abuser.

Case report

A 32 years-old Caucasian male with a history of cocaine abuse was transferred to our emergency department complaining with psychomotor agitation followed by generalized seizures and collapse. Despite prompt administration of naloxone and intravenous diazepam, the patient was still critical presenting with cardiogenic shock. (ABP 80/40 mmHg). His body temperature was normal (36.8°C). Eventually, he was intubated and mechanically ventilated. Metabolic acidosis with elevated lactate values were documented at arterial blood gas test (**Table 1**).

His past medical history was unremarkable: no medication has been recently prescribed and his family history was negative for seizures, syncope, cardiac disease or any other major disease. Electrocardiogram (ECG) monitoring showed paroxysmal supraventricular tachycardia, followed by spontaneous conversion to sinus rhythm with V1-V3 leads mimicking Brugada type 2 pattern (**Figure 1**). Transthoracic echocardiography was unremarkable.

High sensitivity cardiac troponin I and serum electrolytes were within normal limits whereas toxicology studies were positive for cocaine metabolites and cannabinoids.

Sodium bicarbonate and fluid load were administered to correct metabolic acidosis, leading to pH normalization, clinical recovery and to normalization of the ECG morphology (**Figure 2**).

At discharge from the Emergency Department, the patient was stated on a follow-up program

Table 1. Arterial blood gas test values

	At Arrival O_2 100% in Vmask	Fluid & HCO_3^- Spontaneous Breath	After Restoration
pH	6.822	7.098	7.414
pCO_2	70.4 mmHg	35.7 mmHg	41.2 mmHg
pO_2	382.6 mmHg	79.0 mmHg	72.5 mmHg
Na^+	147.1 mmol/L	142.9 mmol/L	141.6 mmol/L
K^+	4.08 mmol/L	4.22 mmol/L	4.08 mmol/L
Lac	29.4 mmol/L	18.3 mmol/L	3.8 mmol/L
Gap	28.1 mmol/L	26.8 mmol/L	12.1 mmol/L
BE-ecf	-22.7 mmol/L	-18.7 mmol/L	1.8 mmol/L
HCO_3^-	11.6 mmol/L	11.1 mmol/L	26.6 mmol/L

and was referred to our Electrophysiology Department. A Holter examination performed after one month showed no malignant arrhythmias. Most notably, the flecainide test with 2 mg/kg over 10 minutes was negative, suggesting a different pathophysiological mechanism of the Brugada Pattern in this patient. In consideration of a negative flecainide test and of a negative family history for sudden cardiac death no further cardiac examinations were performed and the patient was referred to a detoxification program.

Discussion

The Brugada syndrome is a dominant genetic pathophysiology involving loss-of-function sodium channel mutations that may lead to life-threatening ventricular tachyarrhythmias and sudden cardiac death [1]; the syndrome is characterized by the presence of specific patterns on the ECG in absence of structural cardiac abnormalities [2-4].

Recently, the term "Brugada phenocopy" has been used to describe these cases, provided they fulfill the following criteria: there is an identifiable trigger, it resolves after removal of the trigger (if possible), and the results of a provocative test with sodium channel blockers are negative [5].

In patients with Brugada phenocopy the typical ECG pattern can be unmasked by exposure to drugs (class IC antiarrhytmic drugs, calcium channel blockers, betablockers, antianginal and psychotropic drugs), pathological conditions (such as metabolic disorders, ischemia, mechanical compression, myocardial and pericardial disease) or others agents (cocaine, alcohol, opioids) [6]; acute patients presenting with a Brugada-type ECG are at considerably higher risk of sudden cardiac death and should be considered as a medical emergency [7]; the ECG abnormalities may disappear once underlying condition is treated or resolved.

The direct and indirect (i.e., antivagal) effects of cocaine on the electrophysiological properties of the heart are well-known: more specifically, it has a sodium-channel blocking effect and may increase intracellular calcium concentration, leading to malignant ventricular arrhythmias. Moreover, cocaine intoxication may favor electrolyte imbalance and increase the electrical susceptibility of the myocardium to arrhythmias.

Table 2 depicts all previous reports in current literature; we observed that all patients were young male with age ranging between 18 and 49 years old (mean 34 ± 9 years), and presented to the ER specifically with a Brugada type 1 phenocopy [2-4, 6, 8-13].

The clinical setting of our patient was the one of a cocaine intoxication associated with a type 2 Brugada pattern on the ECG. To our knowledge, this is the first description of the association of the ECG abnormality in patients with cocaine abuse. The potentially lethal clinical setting was promptly recognized by ER physicians: favoring rapid treatment of acidosis with fluid resuscitation and bicarbonate administration. When considering the electrophysiological properties of the heart, bicarbonate may exhibit a specific sodium-channel blocking action [14]. Moreover, the increase in extracellular pH and the creation of an alkaline milieu may increase the unionized fraction of cocaine, resulting in a dissociation of the drug from the sodium channels. The only favorable feature of our clinical case was related to the absence of any electrolyte imbalance which could have worsened myocardial susceptibility towards arrhythmias.

The clinical relevance of our case report lies in the prompt awareness of any ECG abnormality in patients with cocaine abuse besides myocardial infarction, such as any Brugada pattern, which could lead to appropriate treatment in order to prevent malignant ventricular arrhythmias.

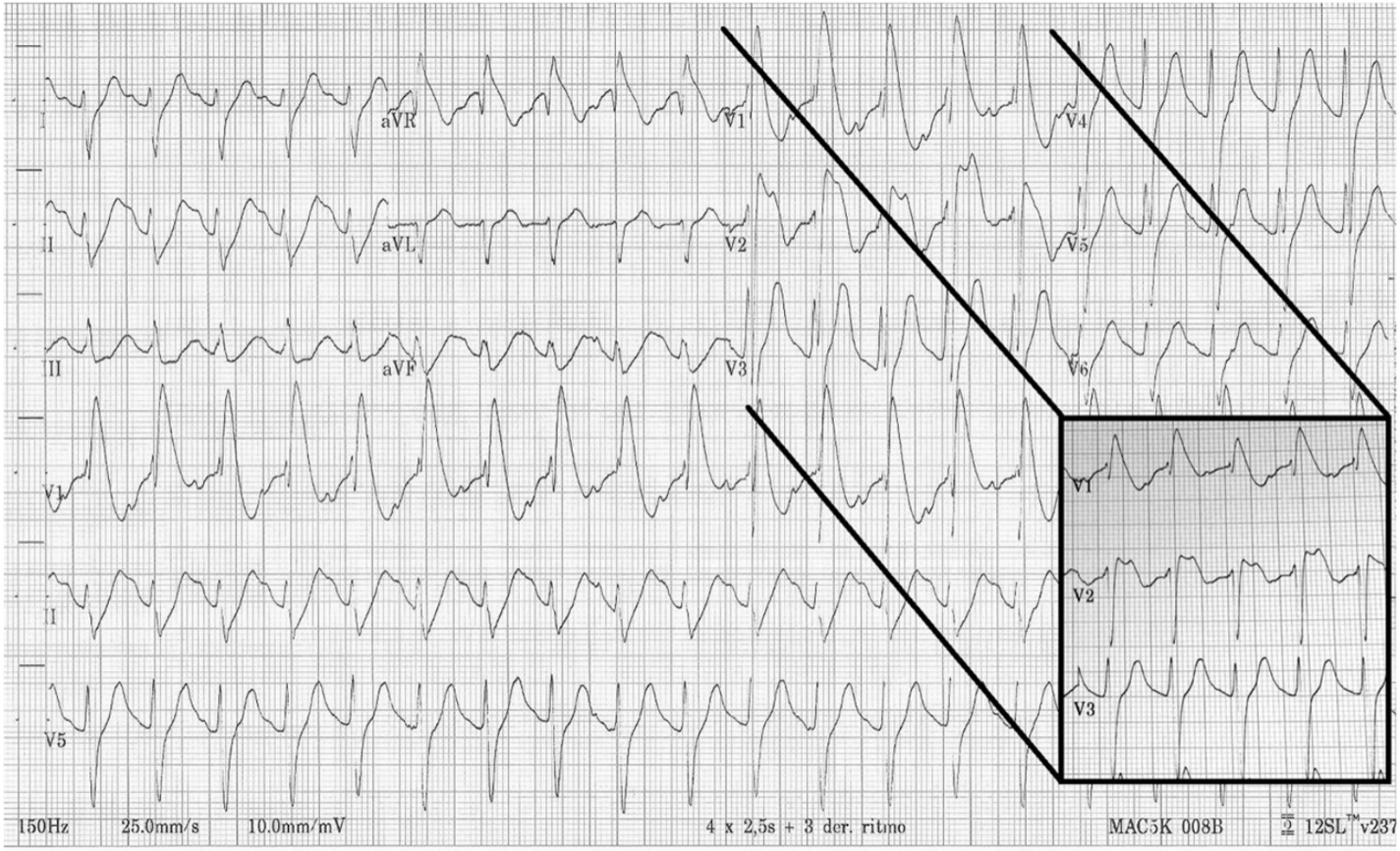

Figure 1. ECG: sinus rhythm with V1-V3 leads mimicking Brugada type 2 pattern.

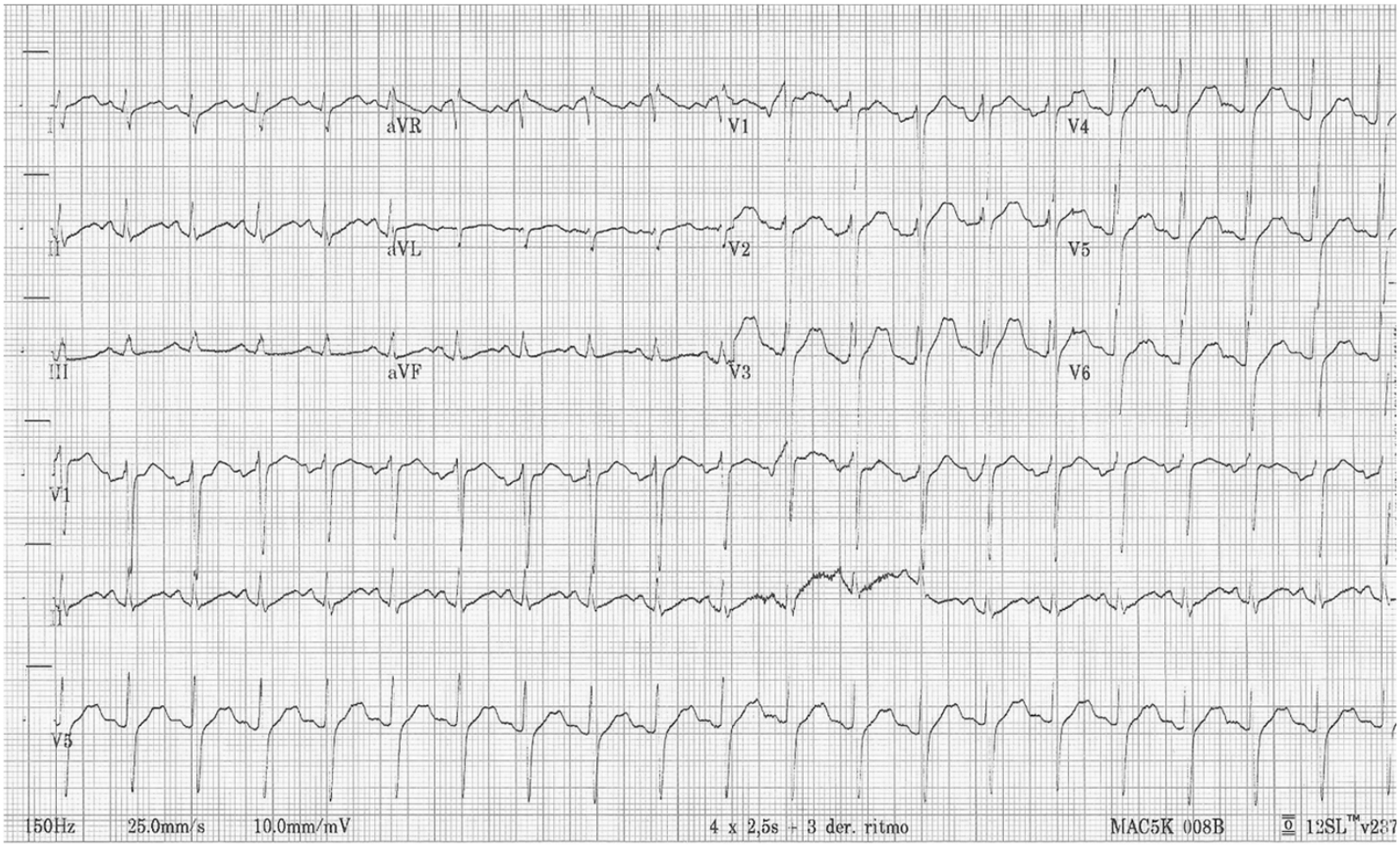

Figure 2. ECG after restoration.

Table 2. All previous reports in current literature

Case N°	Demographic Characteristic		Toxicology			Electrocardiographic Presentation		
	Gender	Age	Intake modality	Drugs co-assumption	Alcohol Intake	Brugada Type	QRS Wide	Axis Deviation
1	M	29	Crack	No	Yes	1	Yes (RBBB)	No
2	M	37	Ingestion	No	No	1	Yes (RBBB)	Yes
3	M	22	NR	No	No	1	Yes (RBBB)	No
4	M	18	Insufflation and Ingestion	No	No	1	No	No
5	NR	33	Ingestion	No	No	1	No	No
6	M	27	Ingestion	No	No	1	Yes	Yes
7	M	44	NR	No	Yes	1	No	No
8	M	48	NR	No	No	1	Yes	NR
9	M	41	NR	Yes, THC & Opiates	No	1	No	NR
10	M	39	Ingestion	No	No	1	Yes	NR
11	M	37	Ingestion	No	No	1	Yes	NR
12	M	49	NR	Yes, THC & Opiates	No	1	No	NR
13	M	29	NR	No	No	1	No	NR

Disclosure of conflict of interest

None.

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