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Case Report

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Brugada Phenocopy Induced by Electrocution: About a Case

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Abstract

Brugada Syndrome (BrS) is an inherited channelopathy secondary to a sodium channel defect (SCN5A), predisposing to ventricular arrhythmia and sudden death. The BrS electrocardiogram is often dynamic and can change from a type 1 to a type 2 pattern, passing through periods of normalisation. The term Brugada phenocopy was proposed by Pérez-Riera et al to describe the electrocardiographic aspect of Brugada observed in other situations, such as acute myocarditis, muscular dystrophy, or secondary to other agents such as electrocution, the taking of drugs such as psychotropic drugs, prolonged exposure to cold or carbon monoxide intoxication. Electrocution can cause serious cardiovascular damage, in particular malignant ventricular arrhythmias. We report the case of a 59-year-old patient who consulted the emergency ward for a cardiac work-up following electrocution. An ECG was performed, which revealed a transient Brugada type 1.

In this case, we discuss the diagnostic procedures for this channelopathy, which carries a risk of ventricular rhythm disorders and sudden death, and the therapeutic management.

Keywords: Brugada Syndrome, Canalopathy, hereditary, Ventricular rhythm disorders, Sudden death, Brugada phenocopy, Electrocution, Other situations, Brugada type 1, Type 2.

Introduction

Brugada syndrome (BrS) is a congenitally inherited cardiac channelopathy characterized by type 1 and type 2 electrocardiogram patterns in leads V1-V3 that predisposes individuals to malignant ventricular arrhythmias and sudden cardiac death [1]. Brugada phenocopies (BrP) are clinical entities that have ECG patterns identical to congenital BrS but are elicited by various other factors, such as myocardial ischemia, metabolic abnormalities, mechanical mediastinal compression and poor ECG filters, cardiomyopathy [2,3,5]. Baranchuk and colleagues characterized this condition in 2012, and since then the phenomenon has been increasingly reported [4].

We report the case of Mr. O. K., 59 years old, painter, who consults in the emergency room of cardiology for the management of repolarization disorders resembling acute coronary syndrome, following an ECG made in the balance sheet of an electrocution. The patient was accidentally electrocuted by 240 V. He was thrown to the ground with a brief loss of consciousness for a few seconds followed by spontaneous recovery. He has no history of heart disease and does not report chest pain, palpitations or sudden or premature death in his family.

Examination at admission: Cardiovascular examination is without abnormality; BP=120/80 mm Hg; Fc=66 bpm; no evidence of HF, SpO2: 95%; no neurological deficit; abdomen supple; no skin burns.

Surface ECG performed immediately: Brugada type 1: Sus shift of the segment st in V1V2V3 domed, with negative T in the same leads. QTc normal.

Echocardiographic evaluation was normal. Routine laboratory tests, including blood urea, serum creatinine, serum electrolytes and cardiac biomarkers, are normal. The patient was observed in the intensive care unit for 24 hours. The ECG returned to normal after 24 hours. In view of the absence of a family history of sudden death and similar cases in siblings and the notion of a trigger event that could lead to the ECG changes mentioned above, he was not subjected to further investigations, such as induction with Class I antiarrhythmic drugs or electrophysiological studies.

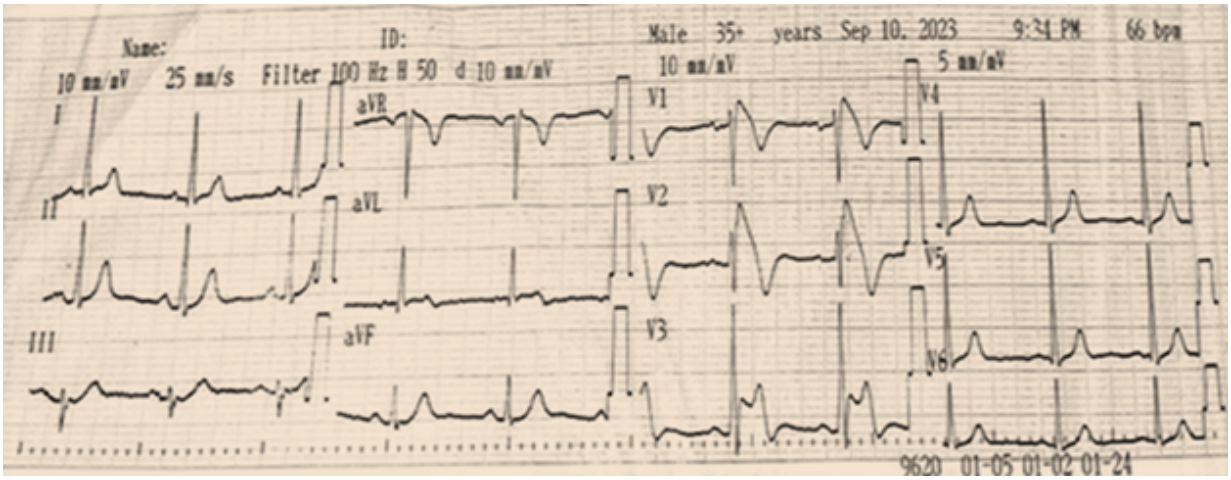


Figure 1: ECG at hospitalization.

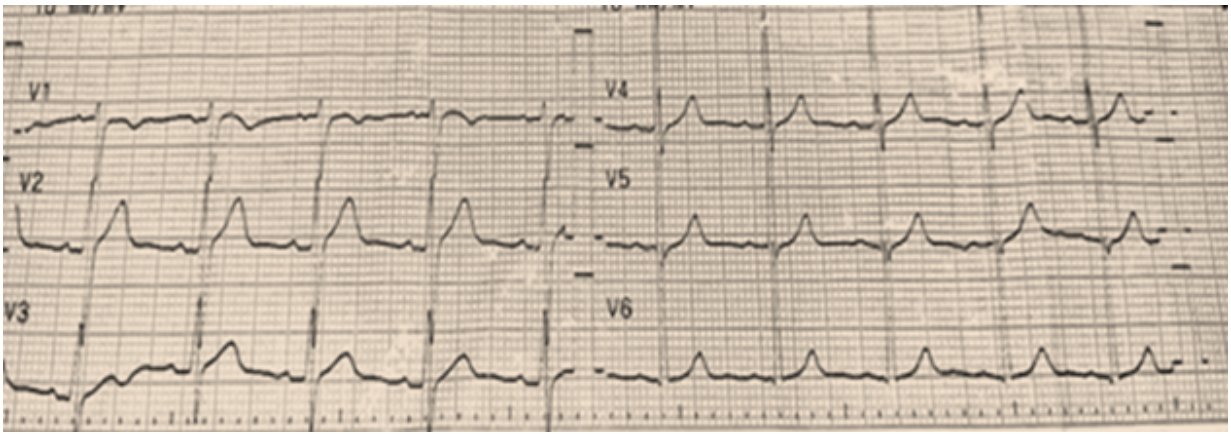
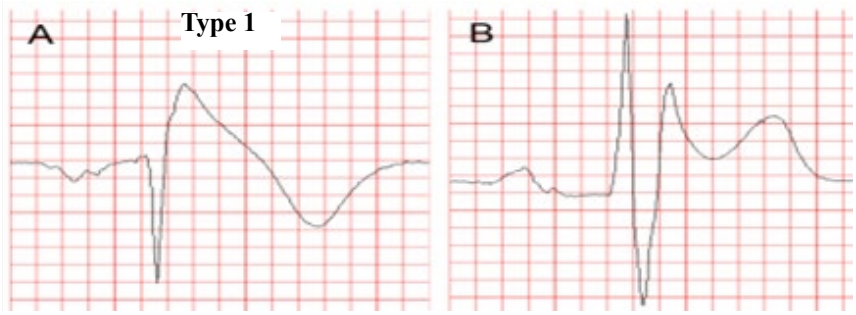


Figure 2: ECG 24h after hospitalization.

Discussion

BrP are clinical entities that are etiologically distinct from true congenital BrS. BrP are defined by ECG patterns that are identical to BrS but are elicited by various clinical circumstances The term phenocopy was chosen because it was previously used to describe an environmental condition that imitates one produced by a gene; therefore, it served as a reasonable and succinct description for all acquired Brugada-like ECG manifestations [4, (14,18]. Since the initial reports, type 1 BrP have been reported in the context of an acute inferior ST-segment elevation myocardial infarction with right ventricular involvement [2,6]; concurrent hyperkalemia, hyponatremia and acidosis [7,8], acute pulmonary embolism [9,10] and hypokalemia [11,12,16 ,18]. Similarly, type 2 BrP have been reported immediately post-electrocution accidental injury [12,13]. True congenital BrS is characterized by two ECG patterns in leads V1-V3: The typical type 1 "coved" or the type 2 "saddleback" patterns (figure 3). The type 1 pattern has a high take-off ST segment elevation that is ≥ 2 mm followed by a down-sloping concave or rectilinear ST-segment with a negative symetric T-wave (Figure (1). The type 2 pattern is defined as a high take-off (r') that is ≥ 2 mm from the isoelectric baseline, followed by ST-segment elevation that is convex with respect to the isoelectric baseline with elevation ≥ 0.05 mV, with variable T-wave in lead V1 and positive or flat T-wave in lead V2 (Figure 3) [19].



Figue 3: Brugada type ECG patterns.

Conclusion

Electrocution causes a transient Brugada-type electrocardiographic aspect. These patients may be at high risk of developing life-threatening ventricular arrhythmias and sudden death. A monitoring is strongly recommended within 24 to 48 hours.

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