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Brugada phenocopy induced by aluminum phosphide intoxication: a case report

Fenocopia de Brugada inducida por intoxicación con fosfuro de aluminio: reporte de un caso

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Palabras clave:
Brugada, fenocopia, aluminio, fosfuro, intoxicación.

ABSTRACT

Introduction: Brugada phenocopy is an electrocardiographic phenomenon that presents a type 1 or 2 Brugada pattern triggered by various underlying clinical conditions. **Case report:** a 23-year-old male patient was admitted due to the ingestion of aluminum phosphide. At admission, an electrocardiogram showed a Brugada type 1 pattern with a transthoracic echocardiogram showing a left ventricular ejection fraction of 34% and apical dyskinesia. A second electrocardiogram was performed 24 hours later with sinus tachycardia and no evidence of the Brugada pattern. After 72 hours of clinical improvement, the apical contractility was reported as normal. **Conclusion:** this acute stress-induced cardiomyopathy by Aluminum phosphide intoxication met the criteria to be regarded as a type 1 class B Brugada phenocopy.

RESUMEN

Introducción: *la fenocopia de Brugada es un fenómeno electrocardiográfico que presenta un patrón de Brugada tipo 1 o tipo 2 desencadenado por diversas condiciones clínicas subyacentes.* ***Caso clínico:*** *paciente masculino de 23 años que ingresó por ingesta de fosfuro de aluminio. Al ingreso, el electrocardiograma mostró patrón de Brugada tipo 1 con un ecocardiograma transtorácico que mostró una fracción de eyección del ventrículo izquierdo del 34% y discinesia apical. Se realizó un segundo electrocardiograma 24 horas después con taquicardia sinusal y sin evidencia del patrón de Brugada. Luego de 72 horas de mejoría clínica se reportó la contractilidad apical como normal.* ***Conclusión:*** *esta miocardiopatía aguda inducida por estrés por intoxicación con fosfuro de aluminio cumplió con los criterios para ser considerada como fenocopia de Brugada tipo 1 clase B.*

INTRODUCTION

Brugada phenocopy is an electrocardiographic phenomenon characterized by presenting a type 1 or 2 Brugada pattern triggered by various underlying clinical conditions that resemble the Brugada phenotype in an individual not carrying the genetic mutation.[1] This case highlights the fact that the search for Brugada phenocopies triggering factors remains open, and the list continues to grow, confirming the importance of timely recognition of Brugada phenocopies and their clinical approach.

CASE PRESENTATION

A 23-year-old male patient was admitted due to the intentional ingestion of a tablet with 1.7 grams of aluminum phosphide and 1.3 grams of excipients. An electrocardiogram (ECG) was performed *(Figure 1)*. The patient was admitted with neurologic deficit and shock; cardiovascular support, mechanical ventilation, and vasopressors were initiated.

Cardiac biomarkers (troponin I, brain natriuretic peptide) were elevated beyond the 99th percentile values at admission; no

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electrolyte imbalance was present when the ECG was taken; a transthoracic echocardiogram was performed, documenting a 34% biplane left ventricular ejection fraction (LVEF) with apical dyskinesia. A second ECG was performed 24 hours later, showing sinus tachycardia without evidence of the Brugada pattern *(Figure 2)*. After 72 hours of admission, vasopressors were discontinued, and the patient was weaned from mechanical ventilation. A second transthoracic echocardiogram showed improvement in systolic function (LVEF 48% by biplane) without alterations in apical contractility. In a 24-hour Holter, the Brugada pattern was not documented again. Due to the patient's age and contractility improvement, the patient was not considered in need of coronary angiography. Acute stress-induced *(tako-tsubo)* cardiomyopathy was presumed. The patient continued to show clinical improvement until discharged.

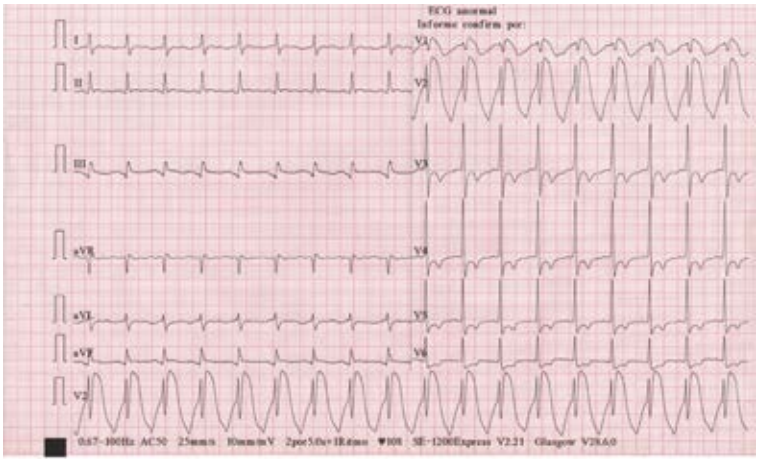

Figure 1: Electrocardiogram was taken at the presentation in the emergency room.

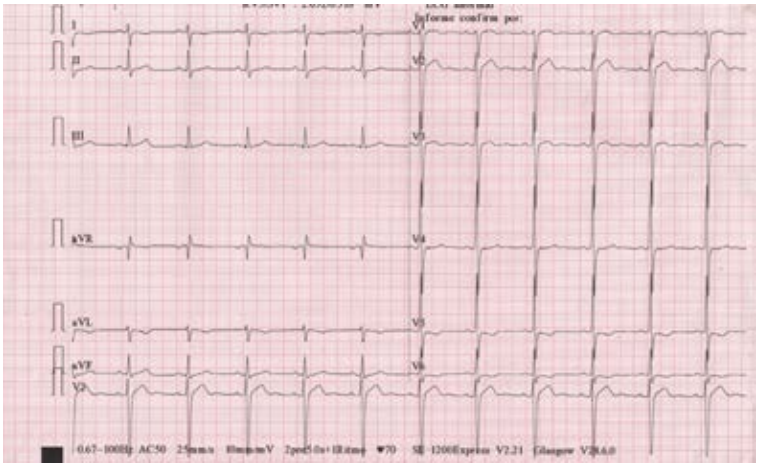

Figure 2: Electrocardiogram taken after patient's recovery.

DISCUSSION

Aluminum phosphide (Alp) is used to preserve grains worldwide. It is also one of the most dreaded poisons. The poisoning mechanism after ingestion of Alp is thought to be secondary to the release of phosphine gas by a chemical reaction of the phosphide with water and hydrochloric acid in the stomach.[2] Phosphine is a metabolic poison that noncompetitively blocks cytochrome C oxidase in rat liver preparation.[3] It also boosts the extra-mitochondrial release of oxygen free radicals, resulting in lipid peroxidation and protein denaturation of the cell membrane.[4]

A cardinal feature of Alp poisoning is early hypotension (within six hours) with a clear mental state. Features of shock include a thready pulse, cold extremities, sweating, and oliguria present in 70% of patients.[5] Myocardial injury is common with global hypokinesia of the left ventricle and interventricular septum with decreased LVEF on echocardiography, which occurs during the first 1 to 4 days in up to 50% cases.[6] Apical dyskinesia was not found in Alp poisoning in previous reports. In this case, it was attributed to *tako-tsubo* cardiomyopathy.

In Alp intoxication nonsurvivors, examination of myocardial tissue has been reported with focal areas of congestion, necrosis, and edema along with inflammatory cell infiltration; histopathological changes related to left ventricular dysfunction have been reported.

The ECG may show the following:

1. Widening of QRS complex.[4]
2. ST depression.
3. ST elevation.
4. T inversion in V2 and V3.
5. Arrhythmias and conduction disturbances include atrial fibrillation, ventricular tachycardia, wandering atrial pacemaker, complete heart block, bundle branch block, and sinus arrest.[7]

Asymmetrical T-wave inversion in precordial leads was attributed to the acute stress-induced cardiomyopathy found in the patient.

Previous cases of Brugada phenocopy induced by intoxication by Alp have been reported, none of them in conjunction with acute stress-induced cardiomyopathy.[8,9]

The diagnostic criteria for Brugada phenocopy have been established (The first four criteria are mandatory):

1. ECG pattern with type 1 or type 2 Brugada morphologic criteria.
2. Presence of an underlying condition that is identifiable and reversible.
3. Resolution of the ECG pattern upon elimination of the underlying condition.
4. Low pretest probability for Brugada syndrome determined by the lack of symptoms, clinical history, and family history.
5. A provocative negative test with a sodium channel blocker drug (e.g., ajmaline, flecainide, or procainamide).
6. A negative genetic test.[1]

CONCLUSIONS

This acute stress-induced cardiomyopathy by Aluminum phosphide intoxication met the criteria to be considered a type 1 class B Brugada phenocopy according to the Morphologic classification system from the international registry of Brugada phenocopies.

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