Case Report

Toxic Myocarditis with Brugada Phenocopy in a Case of Aluminum Phosphide Poisoning

Digvijay D Nalawade, MD, DM, FACC, FSCAI[1], Ajitkumar Jadhav, MD, DNB[1*], Pratik S Wadhokar, DM[1], Anishkumar Khan, DM[1], Aryan Gupta, MBBS[2], Susheel Kumar Malani, MD, DM, FACC, FSCAI, FCSI[1]

[1]*Department of Cardiology, Dr. D Y Patil Medical College Hospital & Research Centre, Pimpri-Chinchwad, India.*
[2]*Department of Microbiology, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Dr. D. Y. Patil Vidyapeeth University, Pune, Maharashtra, India.*

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Abstract

Brugada phenocopy is considered when a Brugada-type ECG pattern is present but with a low likelihood of true Brugada syndrome, as indicated by negative family history, genetic testing, or provocative testing with drugs, or ECG normalization after the removal of precipitants. Brugada phenocopy has been reported due to various causes such as fever and electrolyte imbalance. We describe a 22-year-old man who presented with aluminum phosphide poisoning, resulting in severe metabolic acidosis, myocarditis, and profound myocardial depression. He developed transient Brugada-like ECG changes and multiorgan dysfunction, requiring intensive management, including mechanical ventilation and inotropes. Brugada phenocopy is a rare manifestation of aluminum phosphide-associated toxic myocarditis. After a week of treatment, there was a significant improvement in cardiac function and overall clinical status.

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Introduction

Brugada syndrome, characterized by ST-segment elevation in the right precordial leads and a predisposition to ventricular arrhythmias, poses a diagnostic challenge when ECG changes resembling its pattern are induced by non-genetic factors, known as Brugada phenocopy. Aluminum phosphide, widely used as a rodenticide and in suicides in India, releases phosphine gas causing severe cardiovascular effects such as refractory hypotension and myocarditis, with mortality rates ranging from 37% to 100%.[1] In rare instances, aluminum phosphide poisoning can unmask a Brugada pattern ECG. Our case illustrates transient ECG changes consistent with Brugada phenocopy post-poisoning, resolving within 24 hours despite normal electrolyte levels, likely due to toxic myocarditis.

[*]**Corresponding Author:** ***Ajitkumar Jadhav,*** *Assistant Professor of Cardiology, Department of Cardiology, OPD No. 2, First Floor, Hitech Building, Dr D. Y. Patil Medical College Hospital & Research Centre, Pimpri, Pune. 411018. Tel: +91 9028899494. E-mail: ajit92698@gmail.com.*

Case Report

A 22-year-old man presented to the emergency department 4 hours after suicidal ingestion of a rodenticide containing aluminum phosphide. The patient reported 4 to 5 episodes of vomiting and had no significant past medical history. On general examination, the patient was afebrile with a pulse rate of 120 beats per minute, a respiratory rate of 25 breaths per minute, blood pressure of 90/60 mm Hg, and oxygen saturation of 96% on room air. Bilateral crepitations were detected upon systemic examination. Initial arterial blood gas analysis revealed severe metabolic acidosis. Routine blood examination showed an elevated white blood cell count.

The patient received gastric lavage and was started on supportive therapy, including noninvasive ventilation and inotropes. Within a few hours of admission, he experienced an episode of atrial fibrillation with a rapid ventricular response, which subsequently reverted to sinus tachycardia. Two-dimensional echocardiography demonstrated normal left ventricular (LV) size and thickness with severely depressed LV ejection fraction of 15% to 20%, global LV

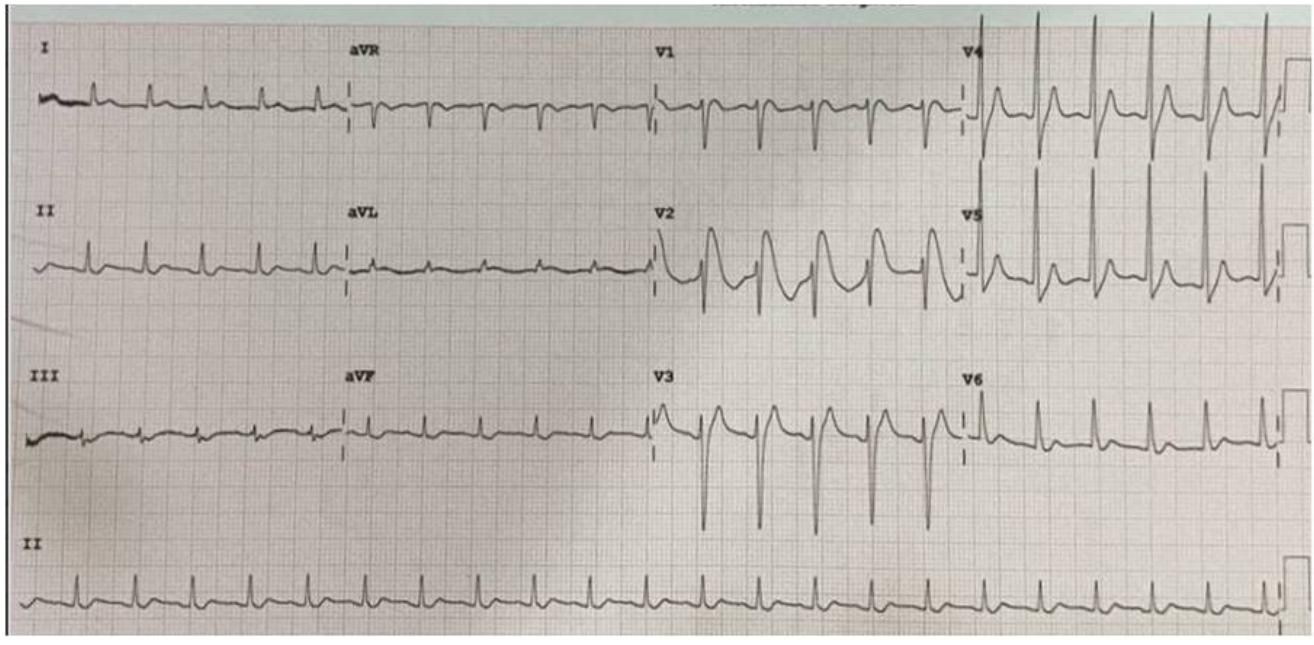

Figure 1. The ECG shows ST-segment coving in leads V_1 to V_3 with a right bundle branch block, suggestive of a Brugada-like pattern.

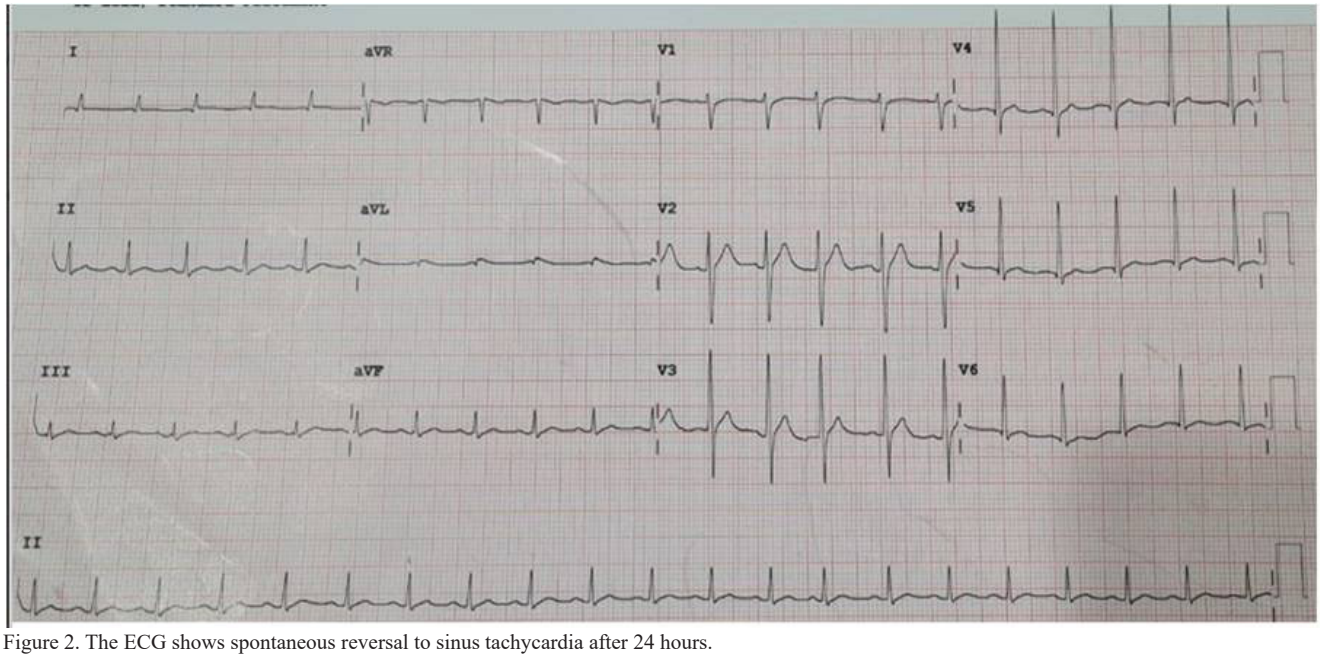

Figure 2. The ECG shows spontaneous reversal to sinus tachycardia after 24 hours.

hypokinesia. Markedly elevated cardiac enzymes (creatinine phosphokinase MB: 392 U/L and troponin I: 7325 pg/mL) were suggestive of myocarditis.

On day 2, the ECG showed ST-segment coving in leads V_1 to V_3 with a right bundle branch block, indicative of a Brugada-like pattern (Figure 1). Retrospective history taking revealed no history of syncope, palpitations, or a family history of sudden cardiac death. Serum electrolytes, including potassium and magnesium, were within normal limits. This ECG abnormality spontaneously reverted to sinus tachycardia the following day (Figure 2).

Over the following days, the patient's condition deteriorated, with multiorgan involvement, including liver and renal dysfunction. Inotrope requirements increased, and the patient was transitioned to invasive mechanical ventilation. After a week of multidisciplinary care and treatment, the patient's condition improved, and he was successfully weaned off the ventilator. Follow-up 2D echocardiography revealed improved LV ejection fraction (40%–45%). Additionally, cardiac enzyme levels showed a reduction.

Discussion

Aluminum phosphide is a widely used rodenticide and a common suicidal agent in India.[1] Mortality rates due to aluminum phosphide poisoning are very high, ranging from 37% to 100%.[1] The release of phosphine gas, a protoplasmic poison, inhibits cellular ATP synthesis by blocking cytochrome oxidase and induces cellular hypoxia, predominantly affecting the cardiovascular system and causing refractory hypotension, myocarditis, heart failure, and other complications.[1] Various ECG abnormalities have been reported, such as atrial fibrillation, supraventricular or ventricular premature contractions, ST-T changes, atrioventricular dissociation, right or left bundle branch block, prolonged PR interval, and sinus tachycardia.[2] Only a few case reports have documented the unmasking of Brugada pattern ECG associated with this poisoning.

Brugada syndrome, first described by Brugada and Brugada in 1992, is an inherited channelopathy characterized by ST-segment elevation in the right precordial leads V_1 to V_3 (in the absence of acute coronary syndrome), right bundle branch block, susceptibility to ventricular tachycardia, and structurally normal heart.[3] These ECG changes may occur spontaneously or be unmasked by sodium channel blockers.[4] Clinical manifestations include male predominance, syncope, or cardiac arrest due to sudden arrhythmia, with rapid polymorphic ventricular tachycardia being the most typical presentation that can degenerate into ventricular fibrillation, leading to sudden death.[3] The syndrome follows an autosomal dominant inheritance pattern, with genetic mutations involving the SCN5A gene in 20% to 25% of cases and GPD1-L in 1% of cases.[3] Three types of ECG patterns have been identified: type I (coved-type ST-segment elevation in precordial leads V_1, V_2, and V_3), type II (saddle-back type), and type III (right precordial saddle-back type ST-segment with mirror deviation from the isoelectric segment).[3]

Brugada phenocopy is a clinical entity etiologically distinct from true congenital Brugada syndrome, resulting from metabolic, ischemic (right coronary artery), compressive (mediastinal mass), myocardial/pericardial (myocarditis/pericarditis), and miscellaneous (fever and ECG filters) causes.[4,5] According to Anselm et al,[5] the diagnostic distinction between Brugada phenocopy and true congenital Brugada syndrome depends on 3 factors: the resolution of the Brugada pattern upon the removal of the inciting cause, negative provocative testing with sodium channel blockers, and low clinical pretest probability of true Brugada syndrome determined by the absence of symptoms, relevant medical history, and family history.

In our case, provocative and genetic tests were not conducted; however, ECG changes were considered Brugada phenocopy due to the negative medical and family history and spontaneous reversal within 24 hours. While the exact cellular mechanisms underlying Brugada phenocopy remain unknown, transient alterations of sodium channels, not reproducible with a sodium channel provocative test, or malfunction of other ion channels may be involved.[5]

Previous reports have suggested that these changes could result from associated hypomagnesemia or the direct toxic effects of aluminum phosphide on the myocardium.1-3 In our case, electrolyte levels were normal; nonetheless, LV dysfunction, elevated cardiac biomarkers, and clinical presentation indicated probable toxic myocarditis, which may have caused the Brugada phenocopy.

Conclusion

Understanding the rare association between Brugada phenocopy and aluminum phosphide poisoning is crucial due to the potential for life-threatening arrhythmias. Further investigations are warranted to rule out true Brugada syndrome.

References

1. Guru S, Kumar R, Behera A, Patra S, Kumar P Jr. Aluminium Phosphide-Induced Expression of Covertly Present Brugada Pattern in Electrocardiogram: A Rare Case Report. Cureus 202020;12:e10552.
2. Nayyar S, Nair M. Brugada pattern in toxic myocarditis due to severe aluminum phosphide poisoning. Pacing Clin Electrophysiol 2009;32:e16-17.
3. Singh Mahajan D, Singh JB, Chaudhry RK. Brugada type ECG pattern in a case of aluminium phosphide poisoning. jemds

2012;1:932-935.
4. Dharanipradab M, Viswanathan S, Kumar GR, Krishnamurthy V, Stanley DD. Yellow phosphorus-induced Brugada phenocopy. J Electrocardiol 2018;51:129-131.
5. Anselm DD, Evans JM, Baranchuk A. Brugada phenocopy: A new electrocardiogram phenomenon. World J Cardiol 2014;6:81-86.