'Toxic' ST elevation

Amal Paul,¹ Anandaroop Lahiri²

¹Department of Cardiology,
Christain Medical College and
Hospital, Vellore, Tamil Nadu,
India

²Department of Cardiac
Electrophysiology and Pacing,
Christain Medical College and
Hospital, Vellore, Tamil Nadu,
India

Correspondence to
Dr Anandaroop Lahiri,
anandaroop_lahiri@yahoo.com

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DESCRIPTION

A young man was admitted to the emergency department after consuming zinc phosphide, with severe drug refractory hypotension. ECG (refer [figure 1](#)) revealed ST segment elevation involving anterior, lateral and inferior leads. Serum levels of potassium, calcium, magnesium and phosphorous were normal; serum troponin-T

Figure 1 ST segment elevation involving anterior, lateral and inferior leads.

Learning points

- Toxins are an important cause of ST segment elevation that need to be suspected in the emergency department. Phosphide overdose is a significant cause of cardiotoxicity, mediated by phosphine.
- ST segment elevation occurring in multiple territories is unlikely to be due to an acute coronary syndrome. Such situations demand the consideration of other causes of ST segment elevation.

was 1126 ng/nL (upper limit of normal 14 ng/nL), with associated metabolic acidosis. Echocardiogram revealed severe left ventricular systolic dysfunction. Severe myocardial depression with histopathological evidence of myocyte vacuolation and myocytolysis has been occasionally described with aluminium phosphide toxicity, primarily mediated by phosphine. The absence of an antidote makes the mortality high, once refractory shock ensues. Our patient succumbed to this condition. Zinc phosphide toxicity mediated by phosphine can have similar multisystem involvement, though dramatic electrocardiographic changes are uncommon.¹² However, as evidenced by our report, ST segment elevation involving multiple leads on the ECG, though rare, maybe a hazardous manifestation of zinc phosphide poisoning leading to a fulminant course.

Contributors AP: involved in the management of the patient and collection of data with regard to the patient. AL: involved in the preparation of the manuscript and editing of the image.

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