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CASE REPORT

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Hypophosphatemia causing ST elevation in a critically ill noncardiac surgery postoperative patient

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Abstract

The patient is a 73-year-old female with peripheral vascular disease, coronary artery disease, and systemic lupus erythematosus, who underwent mesenteric artery bypass surgery. She suffered from a pneumonia after surgery, causing acute hypoxic respiratory failure and septic shock. Due to shock, she developed acute renal failure. She was intubated, ventilated, and received continuous veno-venous hemodialysis for renal failure. ST elevation was first observed on telemetry and subsequently confirmed on electrocardiogram. Marked ST elevation is present in the anterior leads with reciprocal ST depression in the inferior leads. A prolonged QT interval is also present. What is the most likely diagnosis?

KEYWORDS
electrocardiogram, hypophosphatemia, ST segment elevation

1 | CASE

The patient is a 73-year-old female with peripheral vascular disease, coronary artery disease, and systemic lupus erythematosus (SLE), who underwent mesenteric artery bypass surgery.

She suffered from a pneumonia after surgery, causing acute hypoxic respiratory failure and septic shock. Due to shock, she developed acute renal failure. She was intubated, ventilated, and received continuous veno-venous hemodialysis for renal failure.

ST elevation was first observed on telemetry and subsequently confirmed on electrocardiogram (ECG) (Figure 1). Marked ST elevation is present in the anterior leads with reciprocal ST depression in the inferior leads. A prolonged QT interval is also present.

What is the most likely diagnosis?

A. Acute pericarditis

B. Acute anterior ST elevation myocardial infarction (MI)

C. Electrolyte abnormality

It is crucial to rule out acute anterior ST elevation MI in this patient with vascular risk factors. However, urgent transthoracic echocardiogram (TTE) revealed normal biventricular function without segmental wall motion abnormalities. Thus, MI was unlikely. The patient is at risk for pericarditis from uremia and SLE, but the ST elevation was not concave up. It was convex up and there was no PR depression. Electrolytes were checked, which revealed normal potassium, but a severely reduced phosphate level of 1.1 mg/dl (normal 2.4–4.5). The patient's abnormal ECG was due to hypophosphatemia. Phosphate was repleted and the patient's ST abnormalities resolved (Figure 2).

ST elevation in MI is often accompanied with reciprocal change between leads III and aVL (Wang et al., 2003). Typically, the ST segment has a plateau, a shoulder, or is upsloping. In acute pericarditis, ST segment elevation occurs diffusely in the precordial and limb leads (Wang et al., 2003). Additionally, the PR segment is depressed, secondary to inflammation of the sub-epicardial layer of the atria. The ST elevation seldom exceeds 5 mm. In hyperkalemia, the ST elevation is often down-sloping. Additionally, it can present with other abnormalities, namely tall tented T waves, a wide QRS, and low amplitude or no P waves (Wang et al., 2003).

Hypophosphatemia has been demonstrated to be associated with arrhythmias. In a study of septic patients with hypophosphatemia,

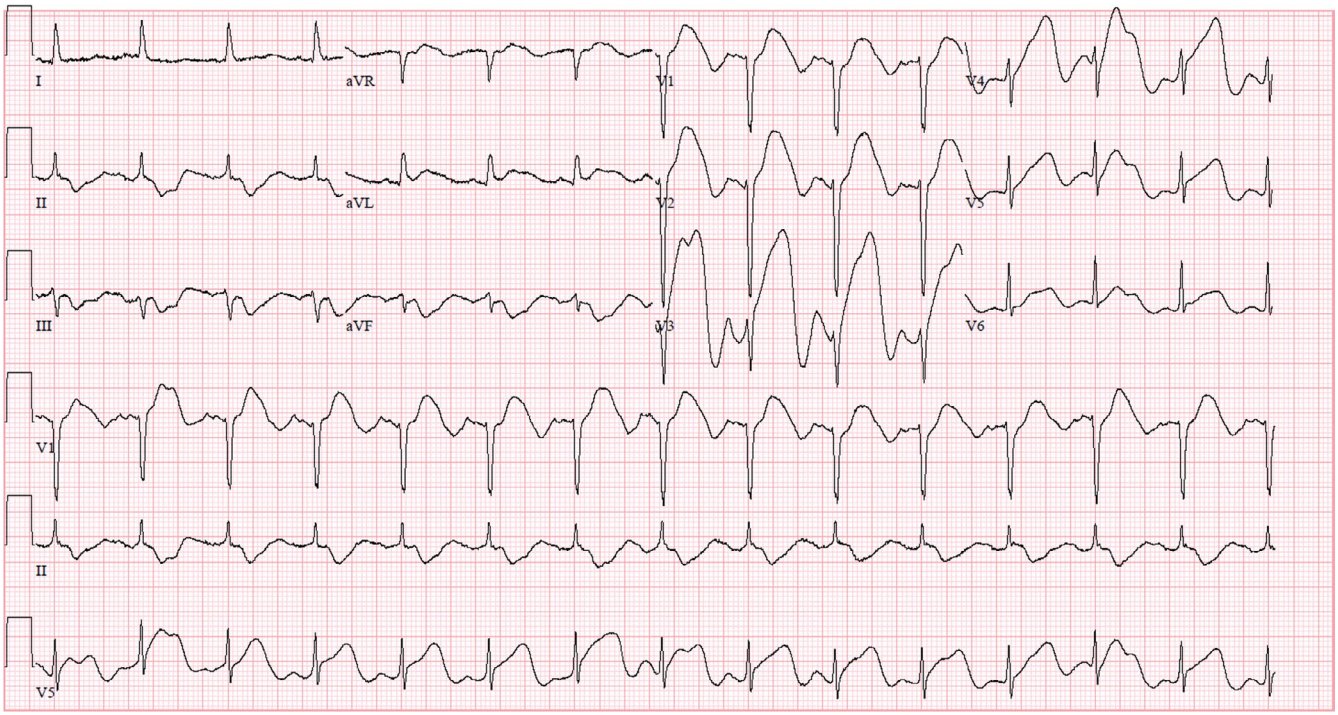

FIGURE 1 Electrocardiogram

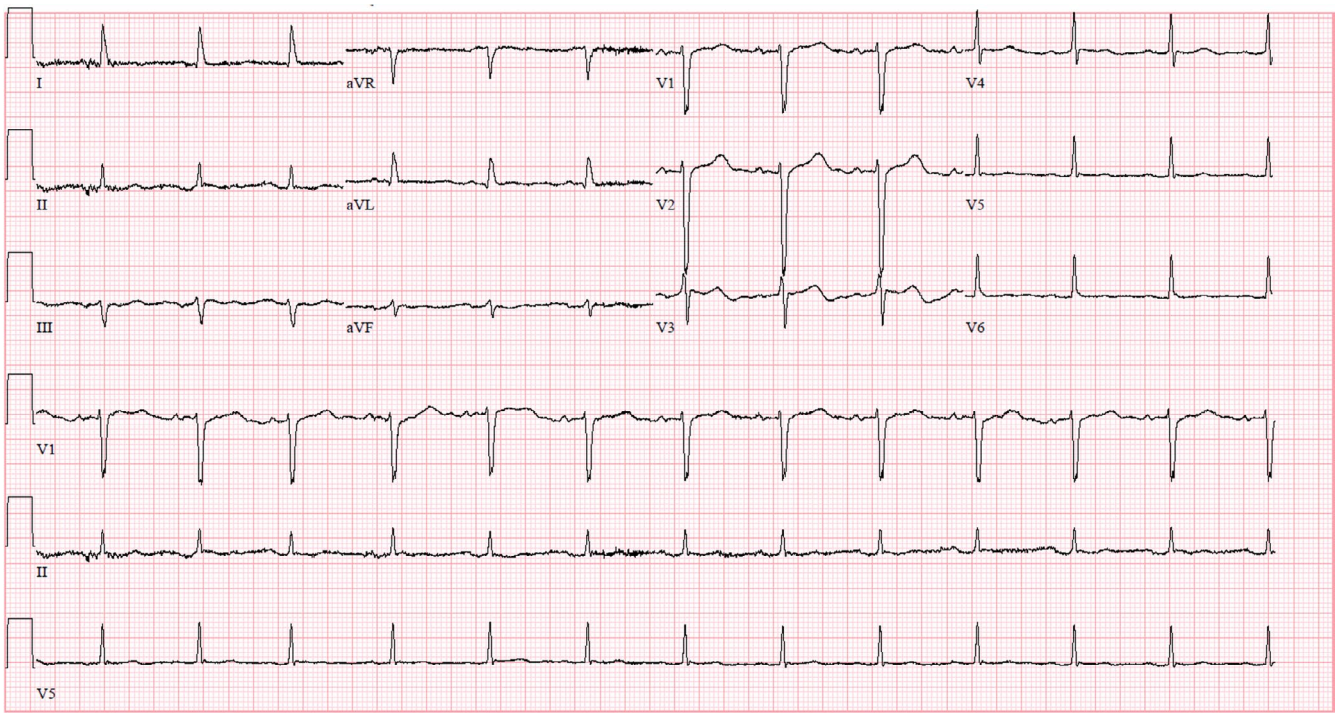

FIGURE 2 Electrocardiogram after phosphate repletion

phosphate replacement was found to reduce the incidence of arrhythmias. (Schwartz et al., 2014) In acute MI, hypophosphatemia has been found to be an independent predictor of ventricular tachycardia (Ognibene et al., 1994).

There are few reports that describe the ECG changes associated with hypophosphatemia. One case report demonstrated an ECG pattern of ST segment elevation in leads V1 to V3, consistent with a type 1 Brugada pattern (Meloche et al., 2016). The patient

was found to have profound hypophosphatemia. Upon correction of it, the type 1 Brugada pattern resolved. The patient eventually underwent provocative testing with procainamide with a negative response, excluding true Brugada syndrome.

Experimental studies on the role of hypophosphatemia in arrhythmias is limited. A number of potential mechanisms have been postulated, including depletion of myocardial ATP leading to abnormalities in potassium and phosphorus transport (Ognibene et al., 1994). In the patient with Brugada phenocopy from hypophosphatemia, the authors hypothesize that alterations of the inward sodium current and the transient outward potassium current in cardiac myocytes lead to a voltage gradient in the right ventricle, producing the Brugada pattern (Meloche et al., 2016).

CONFLICT OF INTEREST

The authors declare that they have no competing interests. The results presented in this paper have not been published previously in whole or part, except in abstract form.

AUTHORS' CONTRIBUTIONS

JJ manuscript. GM manuscript and supervision.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The study did not require review by our institutional review board. This article does not contain any studies with animals performed by any of the authors.

CONSENT FOR PUBLICATION

Not applicable.

CODE AVAILABILITY

Not applicable.

DATA AVAILABILITY STATEMENT

Data are safely kept in a password-protected security system at Thomas Jefferson University Hospital. The data sets used and/or analyzed during the current study are de-identified and available from the corresponding author on reasonable request.

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