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*Case Report*

# ST segment elevation in a patient with myocardial infarction after primary PCI due to cardiogenic shock, hyperkalemia, and acidosis

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**Abstract:** With the adoption of revascularization as the standard treatment method for acute myocardial infarction (AMI) in recent decades, obvious benefits include significantly reduced short-term mortality. Cardiogenic shock is the main life-threatening complication and occurs in 10% of patients after AMI. Myocardial necrosis, damaged microvasculature, stunned myocardium, and hormonal/endocrine responses contribute to the occurrence and development of cardiogenic shock. Ischemia-hypoxia in the myocardial tissue and organ can result in organ dysfunction and metabolic abnormalities, including acidosis, anoxia, and hyperkalemia. This leads to a series of diseases and complications, e.g. worsening of cardiac function, acute renal failure, respiratory failure, and even multiple organ failure. In the current case, ECG after successful primary PCI showed changes strongly reflecting STEMI. However, no thrombus or stenosis was found in the stent upon cardiac catheterization. This case highlights the importance of early recognition and aggressive management of AMI patients who develop cardiogenic shock accompanied with acidosis, hyperkalemia, and acute renal dysfunction.

**Keywords:** AMI, cardiogenic shock, acidosis, hyperkalemia, acute renal dysfunction

## Introduction

Cardiogenic shock is the main life-threatening complication of acute myocardial infarction (AMI) and occurs in 10% of patients [1-3]. Ischemia-hypoxia in the myocardial tissue and organ can result in organ dysfunction and metabolic abnormalities, including acidosis, anoxia, and hyperkalemia. This leads to a series of diseases and complications, such as worsening of cardiac function, acute renal failure, respiratory failure, and even multiple organ failure.

## Case description

A 53-year-old male patient suddenly developed retrosternal pain without obvious cause for 8 hours. After admission, 18-lead electrocardiogram (ECG) showed arched ST segment elevation in leads V1-V4 (**Figure 1A**). CK-MB was 24 U/L, for cTNI at 0.63 ng/m, BNP at 1850 pg/mL, blood glucose at 21.17 mmol/L, serum potassium at 5.18 mmol/L, serum creatinine at 100.5 μmol/L, and eGFR at 75 ml/min. The patient had a history of type 2 diabetes for 7 years (treated with phenformin and gliquidone) and smoking for 30 years. Coronary angiography showed 99% proximal stenosis in the LAD and 90% localized stenosis in the D1. Stents were implanted in LAD and D1 lesions. Seventeen hours after primary PCI, the patient developed palpitation, aggravated chest distress, and shortness of breath, accompanied with profuse sweating and tachypnea. ECG showed that arched ST segment elevation in leads V1-V4 was more significant than before PCI (**Figure 1B**). According to ECG changes and symptoms, stent thrombosis could not be excluded. CAG was performed again, and the results showed no thrombus and stenosis in the LAD and D1 stents. During contrast radiography, the patient developed significant chest distress and shortness of breath, and invasive arterial pressure decreased to 66-74/40-50 mmHg. He was considered a high-risk patient with combined hemodynamic instability and

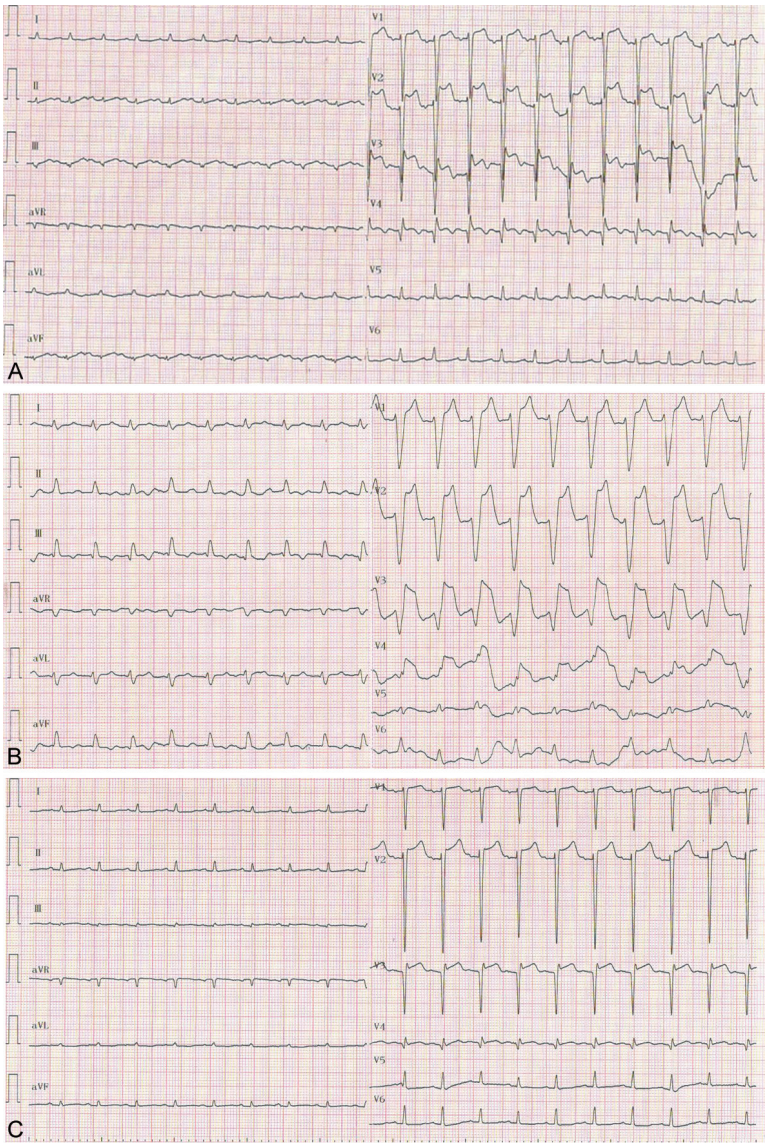


**Figure 1.** A. Before PCI, a sinus rhythm of 131 times/minute was obtained, with a PR interval of 184 ms, QRS duration of 106 ms, and arched ST segment elevation in leads V1-V4. B. Seventeen hours after PCI, a sinus rhythm of 111 times/minute was obtained, with a PR interval of 268 ms, QRS duration of 166 ms, and significantly worsened arched ST segment elevation in leads V1-V4 compared with the condition at admission. C. Four hours after CRRT, sinus rhythm returned to normal, with a heart rate of 114 beats/minute and a PR interval shortened to 206 ms; QRS duration was 88 ms, and the ST segment in leads V1-V4 fell back to baseline.

cardiogenic shock, and an IABP was implanted. Furthermore, the patient developed upper gastrointestinal bleeding twice, losing 500 ml of blood in total. Echocardiography showed a left ventricular ejection fraction (EF) of 45% with hypokinesis of the anterior wall. BNP was 3500 pg/mL, for urea at 15.5 mmol/L, creatinine at 243 μmol/L, eGFR at 24.7 ml/min, no urine ketone body, potassium at 7.92 mmol/L, and GLU at 33 mmol/L. Blood gas analysis indicated pH 7.21 and 6.1 mmol/L lactic acid. The patient also showed sustained ventricular tachycardia, and was restored to sinus rhythm after 200 J electrical cardioversion. Continuous renal replacement therapy (CRRT) was performed, and a ventilator was used for assisted respiration. Temporary pacemaker treatment was performed to prevent cardiac arrest caused by hyperkalemia, and insulin pump therapy was continuously performed to control blood glucose. Upon successful treatment, ECG was performed 4 hours after CRRT transfer, which showed normal sinus rhythm, with a heart rate of 114 beats/minute. The PR interval and QRS duration were shortened more than before treatment, and the ST segment in leads V1-V4 fell back to baseline (**Figure 1C**). After the patient's symptoms and various test parameters improved, he was discharged. Re-examination before discharge showed BNP at 825 pg/mL, hemoglobin at 112 g/L, creatinine at 96 μmol/L, eGFR at 76 ml/min/1.72 $m^2$, and potassium at 4.16 mmol/L.

## Discussion

With revascularization adopted as the standard treatment method for acute myocardial infarction (AMI) in recent decades, obvious benefits include significantly reduced short-term mortality [1, 2]. Metabolic acidosis is especially present in patients with cardiogenic shock after AMI. Increased lactate levels identify patients with critical illness, cardiogenic shock and very poor prognosis [4, 5]. In this patient, 17 hours after successful primary PCI, ECG showed significant ST elevation without thrombus and stenosis in the LAD and D1 stents. ECG findings likely mim-

icked STEMI due to hyperkalemia along with acidosis and anoxia. Similar cases have been reported by multiple studies [6, 7]. Metformin is the standard treatment according to current guidelines for patients with established type 2 diabetes. Metformin-associated lactic acidosis (MALA) is a rare but deadly complication [8, 9]. In this case, lactic acid levels were significantly elevated, but it was difficult to achieve a diagnosis of MALA. Cardiogenic shock caused by extensive myocardial infarction resulted in ischemia/hypoxia of all organs and tissues. In the setting of renal compromise due to ischemia/hypoxia, accumulating metformin exacerbates metabolic acidosis further [8, 9].

It has been reported that the incidence of acidosis soon after reperfusion is associated with contrast-induced nephropathy in patients with first-time ST-segment elevation AMI [10]. In the current case, acidosis and acute renal insufficiency occurred shortly after reperfusion. In addition, hyperkalemia was present at the same time. Therefore, hemodialysis can constitute an effective therapeutic option in this patient. In the management, auxiliary breathing by mechanical ventilation was also very important for this patient after acute cardiogenic shock with acute respiratory failure.

The intra-aortic balloon pump (IABP) could enhance coronary perfusion in AMI patients with cardiogenic shock [11]. It has been reported that 3.7% of patients develop cardiogenic shock during acute hospitalization, with an overall in-hospital mortality rate of 41.4% [12]. To stabilize and rescue patients from the slippery slope of hemodynamic progression to hemometabolic cardiogenic shock, hemodynamic conditions should be assessed as soon as possible, with early use of acute mechanical circulatory support devices in AMI patients [13].

In summary, the current case was very complicated and seriously life-threatening. After AMI, significant complications emerged one after another and interacted with each other. Early recognition and aggressive management of patients developing cardiogenic shock is critical.

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## Disclosure of conflict of interest

None.

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