An unusual Case of Diffuse ST Elevation Mimicking Acute Myocardial Infarction: a Challenge of Emergent Percutaneous Coronary Intervention?

CASE REPORT

Ching Tsai Hsu^{1,2}, Ching Han Liu³, Chin Sheng Lin¹,
Wei Shiang Lin¹, Po Jen Hsiao^{4,5,6}, Jun Ting Liou¹

Abstract

Coronary artery disease combined sepsis associated myocardial ischemia resulting in diffuse ST elevation on electrocardiogram has rarely been reported. We reported a rare case of diffuse ST elevation precipitated by septic shock and preexisting severe atherosclerosis heart disease. In clinical scenario, it is imperative for physicians to be aware of non-ischemic ST elevation etiologies and avoid inappropriate activation of the percutaneous coronary intervention (PCI) protocol, while not missing ST elevation myocardial infarction.

Keywords

Diffuse ST Elevation; Myocardial Infarction; Sepsis; Bacterial Myocarditis.

Introduction

Patients with sepsis can have elevated serum Troponin I values and several characteristic electrocardiogram (ECG) changes. However, sepsis rarely causes diffuse ST elevation on ECG. Furthermore, diffuse ST elevation on ECG is extremely rare individually in patients with acute myocardial infarction or septic shock [1-3]. We presented an unusual case of diffuse ST elevation precipitated by urinary tract infection complicated septic shock and preexisting severe atherosclerosis heart disease.

Case report

An 80-year-old man presented to our emergency department (ED) with new-onset of shortness of breath, no obvious chest pain was found. He

- 1 Division of Cardiology, Department of Internal Medicine, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, R.O.C.
- 2 Division of Cardiology, Department of Internal Medicine, Taoyuan Armed Forces General Hospital, Taoyuan, Taiwan, R.O.C.
- 3 Division of Cardiology, Department of Medicine, Kaohsiung Armed Forces General Hospital, Kaohsiung, Taiwan, R.O.C.
- 4 Division of Nephrology, Department of Internal Medicine, Taoyuan Armed Forces General Hospital, Taoyuan, Taiwan, R.O.C.
- 5 Division of Nephrology, Department of Internal Medicine, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, R.O.C.
- 6 Department of Life Sciences, National Central University, Taoyuan City, Taiwan, R.O.C.

Contact information:

Jun-ting Liou.

Address: Division of Cardiology, Tri-Service General Hospital, National Defense Medical Center, NO.325, Sec.2, Chenggong Rd., Neihu District, Taipei, Taiwan.

✉ ljtmail@gmail.com

Po-Jen Hsiao.

Address: Division of Nephrology, Department of Medicine, Tri-Service General Hospital, Number 325, Section 2, Cheng-Kung Road, Neihu 114, Taipei, Taiwan.

✉ a2005a660820@yahoo.com.tw

had history of type 2 diabetes mellitus and hypertensive cardiovascular disease. On initial assessment, his blood pressure was 77/48 mmHg with a heart rate of 60 beat per minute, respirations of 22 per minute, and temperature of 39.5°C. Initial laboratory investigations revealed the following values: white blood count (WBC), 17530 cells/mm³; neutrophils, 84.9%; serum creatinine 1.4 mg/dL; blood urea nitrogen, 73 mg/dL; creatine phosphokinase, 57 U/L; troponin I, 0.233 ng/mL; brain natriuretic peptide, 439 pg/mL and C-reactive protein, 29.67 mg/dL. Serum electrolytes and liver function tests were within normal limits. Urinalysis was significant for 3+ blood, 2+ protein, 3+ bacteria and >100 WBC/high-power fielder. Plain chest radiographs revealed a normal cardiac size and clear lungs. The ECG demonstrated junctional rhythms and diffuse ST elevation (**Figure 1A**). Transthoracic echocardiogram showed normal left ventricular systolic function and no pericardial effusion. We integrated all the laboratory data, ECG finding, and considered atypical manifestation of myocardial infarction in elderly. The patient then underwent emergent percutaneous coronary intervention (PCI) and it revealed no evidence of total occlusion or thrombus formation. Significant stenosis on the proximal part of left anterior descending coronary artery (LAD), left circumflex coronary artery (LCX) and right coronary artery (RCA) was noted (**Figure 1B & 1C**).

During hospitalization, blood and urine culture, taken on ED, revealed growth of staphylococcus aureus resistant to penicillin. Subsequent to culture result, intravenous antibiotics were prescribed. 7 days later after admission, the ST elevation normalized without evolution of Q wave or decreased R wave amplitude on ECG (**Figure 2**). The patient had an uneventful post PCI course and was discharged with no adverse events.

Figure 1: It demonstrated significant stenosis in the proximal part of LAD, LCX and RCA, respectively.

Figure 1A: ECG at ED showed diffuse ST elevation. Figure 1B & 1C: Coronary angiography showed no evidence of total occlusion or thrombus formation.

Figure 2: ECG at the 7th hospital day demonstrated normalization without prior diffuse ST elevation and without evolution of Q wave.

Discussion

Sepsis-associated ECG findings in patients with or without ischemic heart disease include significant attenuation of QRS complexes, prolonged QT interval, widening of QRS complex, bundle branch blocks and Osborn waves. These ECG changes returned to

normal following recovery from sepsis, showing reversible reductions of QRS amplitudes [1-3]. ST elevation is considered to reflect acute transmural myocardial ischemia and/or damage of myocardium due to thrombotic occlusion of an epicardial coronary artery. However, deviations of the ST segment due to non-ischemia etiologies are often seen. There were many non-ischemic ST elevation etiologies should be take into consideration. These include early repolarization pattern, hypertrophy of left ventricle, conduction defects (left bundle branch block or intraventricular conduction delay), pre-existing syndrome, acute pericarditis, myocarditis, pulmonary embolism, electrolytes imbalance (hyperkalemia or hypercalcemia), hypothermia and Takotsubo syndrome [2-4]. Diffuse ST elevation has been reported more in patients with acute pericarditis, which is characterized by pericarditic chest pain, pericardial effusion, pericardial friction rub, and ECG changes (such as new widespread ST elevation and PR depression) [4]. Previous case reports recommended Takotsubo syndrome and hydrogen sulfide intoxication could also cause diffuse ST elevation [5, 6].

In our patient, diffuse ST elevation on ECG may be precipitated by both septic shock and preexisting ischemic heart disease. Several possibilities and associated mechanisms have been proposed including direct bacterial invasion, bacterial toxins, and immune responses [7]. The differential diagnosis of acute bacterial myocarditis should be considered in this case because staphylococcus aureus was positive in both blood and urine cultures. Further histopathologic evidence may help confirm the diagnosis. This case highlights to be aware of non-ischemic ST elevation etiologies, while not missing ST elevation myocardial infarction.

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Conflict of interest

None of the authors have any conflicts of interest to declare.

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Abbreviations

ECG: electrocardiogram; ED: emergency department, PCI: percutaneous coronary intervention; LAD: left anterior descending coronary artery; LCX: left circumflex coronary artery; RCA: right coronary artery

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