

Case Report

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Acute Inferior Pseudo Infarction Pattern in a Patient with Hyperosmolar Hyperglycemic State



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Abstract

ST elevation myocardial infarction (STEMI) is a medical emergency that requires timely revascularization for better outcomes. However, other causes of ST elevation (STE) should always be considered in the differential diagnosis.

Of these causes, diabetic ketoacidosis has been reported in the past, even in normokalemic patients, however, to our knowledge, hyperosmolar hyperglycemic state (HHS) has never been reported to be associated with ST elevations. Here, we present a case of HHS in a patient who had inferolateral STE on EKG with normal coronary angiogram

Keywords: ST elevation myocardial infarction; Hyperosmolar hyperglycemic state

Introduction

ST elevation myocardial infarction (STEMI) is a medical emergency, and the earlier the revascularization is achieved via thrombolysis or percutaneous coronary intervention, the better the prognosis. However, other causes of ST elevation (STE) should always be considered in the differential diagnosis, and electrocardiographic clues to differentiate between them have to be sought in order not to delay treatment in time sensitive situations, and at the same time to avoid unnecessary studies or procedures with their added risks [1]. Here, we present a case of hyperosmolar hyperglycemic state (HHS) in a patient who had inferolateral STE on EKG with normal coronary angiogram.

Case History

A 50-year-old Hispanic male, with past medical history significant for hypertension and liver transplantation for alcoholic liver cirrhosis, currently on Tacrolimus, presented to the Emergency Department (ED) complaining from left-sided chest pain for 1 hour, associated with nausea, vomiting and generalized weakness. Electrocardiogram was done, which showed STE in inferior leads with reciprocal changes in the lateral leads. 15-lead Electrocardiogram showing ST elevation in leads II, III and aVF, with reciprocal changes in lead aVL. Immediately patient was transferred to the catheterization laboratory, and left

heart catheterization was performed, which did not show any obstruction.

In the ED physical examination revealed blood pressure of 115/71mmHg, heart rate of 87 beats/min, normal heart sounds without any murmur or additional sounds. Lungs were clear to auscultation and there was no peripheral edema. However, on examining the extremities, patient was noticed to have abscess in his right hand.

Diagnostic work up was significant for blood glucose of 928, serum osmolality of 335, hemoglobin A1c of 9.7%, acute kidney injury with creatinine of 1.94, metabolic acidosis with a wide anion gap of 16, lactic acidosis and uremia. Serum acetone was negative repeatedly. The initial serum Potassium level was 4.8.

Patient was treated for (HHS), with Intravenous fluids and insulin infusion with resolution of his symptoms and improvement in his BUN/creatinine levels. Repeat EKG showed resolution of the STE, without any Q waves or new T wave inversions. 15-lead electrocardiogram showing resolution of the ST elevations in the inferior leads noted on EKG obtained on presentation to the ED. Serial Troponins showed an increase in the Troponin I from 0.01 on admission to 0.17 after 12 hours. Subsequently a transthoracic echocardiogram (TTE) was done which showed normal systolic function with no wall motion abnormalities.

Later, patient's right hand abscess was drained surgically, cultured and started on wide spectrum antibiotics. The patient was discharged home on oral antibiotics with proper follow up plan.

Discussion

STE can be due to STEMI, but other causes include normal STE, early repolarization pattern, normal variant STE, left bundle branch block (LBBB), pulmonary embolism, acute pericarditis, acute myocarditis, Brugada Syndrome, arrhythmogenic right ventricular cardiomyopathy, transthoracic cardioversion, Prinzmetal's angina and hyperkalemia. These can be differentiated in most of the cases by the pattern of the ST elevation [1]. ST-segment elevations may arise from metabolic acidosis or other metabolic abnormalities specific to DKA. Coronary vasospasm might also play a role [2].

Few cases about ST segment elevations in patients with diabetic ketoacidosis (DKA) with hyperkalemia were reported in the past [3,4], but there are also fewer case reports of these ST segment elevations in DKA patients who are normokalemic [4], and it is debatable whether the ST elevation is a primary repolarization abnormality or an artefact caused by merging of the terminal R' portion of the QRS with the T wave. It is also unclear whether the changes are due to acidosis or other metabolic abnormalities specific to diabetic ketoacidosis [4].

Our case is interesting because ST -segment elevations has never been reported in patients with HHS, to our knowledge. Although the mechanism of myocardial necrosis in our patient is unclear, it might have been a late consequence of severe acid-base and electrolyte disturbances that might have triggered coronary spasms leading to ischemic myocardial necrosis. The coincidence

of infectious myocarditis is less likely, but cannot be excluded [5].

Similarly, in our patient though the left heart catheterization did not show any significant coronary abnormality, but the fact that the patient presented with typical chest pain and he had a 10-fold increase in the Troponin I levels, similar mechanisms may play a role in HHS, especially if it is combined with acidosis due to other causes.

Conclusion

STE can be due to several causes besides STEMI. And though time is of utmost importance, other causes of ST elevation should be considered in the differential diagnosis, otherwise, patient may be subjected to unnecessary diagnostic and therapeutic procedures with their added risks. ST elevation due to DKA is a rare electrocardiographic manifestation that emergency providers should be familiar with, the role of HHS is yet to be clarified.

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