

**CASE REPORT****OPEN ACCESS**

Pseudo Myocardial Infarction in Diabetic Ketoacidosis: A Case Report and Review of Literature

Nida Wali Khan & Uzma Anwar

Resident Physician-Medicine Dept. Khyber Teaching Hospital

We present a rare case of pseudo myocardial infarction in a patient with diabetic ketoacidosis (DKA) and discuss the diagnostic challenges, management, and implications for clinical practice. This case highlights the importance of considering alternative diagnoses in patients presenting with chest pain and elevated cardiac biomarkers, especially in the context of metabolic derangements such as DKA.

Keywords: Diabetes; Ketosis; Acidosis; Myocardial Infarction

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INTRODUCTION

Diabetic ketoacidosis (DKA) is a life-threatening complication of diabetes mellitus, characterized by hyperglycemia, ketonemia, and metabolic acidosis (1). While DKA has been associated with various cardiovascular complications, pseudo myocardial infarction is a rare and often overlooked diagnosis. Pseudo myocardial infarction refers to a condition in which a patient presents with symptoms and laboratory findings suggestive of myocardial infarction (MI), but without the

presence of acute coronary artery occlusion (2). The differentiation between true MI and pseudo myocardial infarction is critical for appropriate management and avoiding unnecessary interventions.

CASE REPORT

A 45-year-old male with a history of type 2 diabetes mellitus presented to the emergency department with severe chest pain, dyspnea, and altered mental status. The patient had a blood pressure of 140/90 mmHg, heart rate of 110 bpm, respiratory rate of 28 breaths/min, and oxygen saturation of 92% on room air. Initial laboratory investigations revealed hyperglycemia (glucose: 550 mg/dL), metabolic acidosis (pH: 7.1, bicarbonate: 8 mEq/L), and positive serum ketones. Cardiac biomarkers were significantly elevated, with a troponin I level of 2.5 ng/mL (reference range: <0.04 ng/mL) and a CK-MB level of 30 ng/mL (reference range: 0-5 ng/mL) (3).

An electrocardiogram (ECG) demonstrated sinus tachycardia, nonspecific ST-segment and T-wave changes, but no definitive evidence of ST-segment elevation myocardial infarction (STEMI) or non-STEMI. The patient was started on an insulin infusion, intravenous fluids, and potassium supplementation for the management of DKA (4). He was also given aspirin and sublingual nitroglycerin for presumed MI.

Over the next 24 hours, the patient's clinical condition and laboratory parameters improved. Serial ECGs showed resolution of the previously noted ST-segment and T-wave changes. Transthoracic echocardiography revealed normal left ventricular systolic function with no regional wall motion abnormalities (5). A coronary angiogram was performed, which demonstrated no significant coronary artery disease.

DISCUSSION

This case highlights the challenge of differentiating pseudo myocardial infarction from true MI in a patient with DKA. Pseudo myocardial infarction has been reported in various clinical settings, including myocarditis, pericarditis, and pulmonary embolism (6). In the context of DKA, several factors, such as electrolyte imbalances, acidosis, and the direct effects of ketone bodies on myocardial cells, may contribute to the development of pseudo myocardial infarction (7).

A high index of suspicion and a thorough diagnostic workup, including serial ECGs and echocardiography, are crucial for accurate diagnosis (8). The management of pseudo myocardial infarction in DKA primarily involves the correction of the underlying metabolic derangements, with close monitoring for resolution of ECG changes and improvement in cardiac biomarkers. In some cases, a coronary angiogram may be necessary to exclude significant coronary artery disease, as demonstrated in our patient.

Understanding the pathophysiology of pseudo myocardial infarction in DKA can help clinicians avoid unnecessary interventions and potential complications associated with aggressive antiplatelet or anticoagulant therapy in patients without true MI. Additionally, this case underscores the importance of promptly recognizing and treating DKA, as prompt management may lead to the resolution of pseudo myocardial infarction and improvement of the patient's clinical condition.

Further studies are needed to better understand the prevalence and risk factors for pseudo myocardial infarction in DKA, as well as the optimal diagnostic and management strategies for this unique patient population. Clinicians should remain vigilant for this rare but important

complication of DKA and consider alternative diagnoses in patients presenting with chest pain and elevated cardiac biomarkers in the setting of metabolic acidosis.

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