Abstract Form

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Project Title:	STEMI Secondary to Hyperglycemic Hyperosmolar State

Research Category (please check one):

☐	**Original Research**	☒	**Clinical Vignette**	☐	**Quality Improvement**	☐	**Medical Education Innovation**

Abstract

Introduction

Diabetes mellitus is a metabolic disorder which results in the inability to control blood glucose levels leading to hyperglycemia, a state where patients may be asymptomatic. However typical features of hyperglycemia include polyuria, polydipsia, weight loss, and blurry vision. In severe cases patients may develop a condition known as hyperglycemic hyperosmolar state (HHS) where patients become dehydrated, tachycardic, and even hypotensive. The typical patient presents with a several-week history of decreased oral intake, weight loss, and mental confusion. This is usually precipitated by an event such as infection, stroke, or myocardial infarction, however sequelae of ST-elevation MI because of a hyperglycemic hyperosmolar state is rare. Here we present a case of a patient who was found to be in HHS further complicated by findings of STEMI.

Case Presentation

A 53-year-old African American female with uncontrolled diabetes, hypertension, hyperlipidemia, and alcohol abuse presented to our institution with a 2-week history of generalized weakness and fatigue accompanied by 15-pound weight loss, blurry vision, polyuria, polydipsia, and an intermittent right side chest pain rated as a sharp, 2/10 pain at the time. Review of systems was negative for headache, nausea, vomiting, abdominal pain, shortness of breath, or any other symptoms at the time of interview. Upon physical exam, patient was afebrile and observed to be significantly fatigued, with a heart rate of 111 bpm, blood pressure of 118/79 mmHg, respiratory of 20 bpm, and saturating 100% on room air. Remainder of the exam was unremarkable. Laboratory studies showed leukocytosis of 21,500/mm3 without bandemia, potassium of 6.2 mmol/L, BUN of 58 mg/dL, creatinine of 1.06 mg/dL, blood glucose of 1,252 mg/dL with a normal anion gap, lactic acid of 2.5 mmol/L, serum osmolality of 380 mOsm/kg confirming hyperosmolar hyperglycemic syndrome. Additionally, troponin was elevated to 0.49 ng/dL with initial EKG showed no acute ST-segment or T-wave abnormalities.

The patient was administered three liters of normal saline and started on an insulin drip. However serial troponin revealed a rise 3.13 ng/dL without any dynamic EKG changes, and full ACS protocol (aspirin, clopidogrel, atorvastatin, and low-molecular weight heparin) was started. Serial EKGs progressed to show ST elevation in leads V2 and V3 and troponin increased to greater than 40 ng/dL. Blood glucose continued to improve, reaching 583 mg/dL. Patient denied any chest pain and remained hemodynamically stable during this time, however given development of STEMI nitroglycerin drip was initiated for vasodilation. Emergency coronary angiogram showed no obstructive coronary artery disease.

Conclusion

Hyperglycemic hyperosmolar state is a complication of uncontrolled diabetes associated with major morbidity and mortality, despite treatment. The pathophysiology of HHS involves osmotic diuresis resulting in dehydration and hyperosmolar, viscous state. Hyper viscous states are known to affect tissue perfusion and may even be prothrombotic. In our patient, she denied the typical chest pain associated with a myocardial infarction. Despite this, patient was found to increasing troponin levels accompanied with ST-segment changes. An emergency catheterization demonstrated no occlusive disease, leaving the hyperosmolar state as the culprit in our patient.