Sugar and Stress: Takotsubo Cardiomyopathy Induced by Severe Diabetic Ketoacidosis

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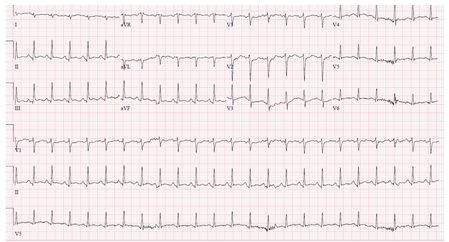
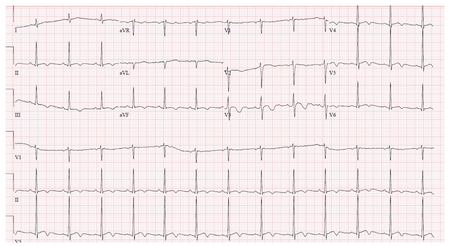
Background

Takotsubo cardiomyopathy, also known as stress-induced cardiomyopathy, is characterized by transient left ventricular (LV) dysfunction. Patients often present with symptoms of acute coronary syndrome however are noted to have normal coronary arteries. Various stressors have been known to precipitate Takotsubo cardiomyopathy. We present a case of diabetic ketoacidosis (DKA) inducing cardiac arrest and subsequent Takotsubo cardiomyopathy in a young patient.

EKG



EKG following cardiac arrest showing Qtc prolongation



EKG next day with new T wave inversions, without pathologic Q waves

Case Description

Patient is a 19 year old male with past medical history of type 1 diabetes mellitus who presented to the emergency room due to altered mental status for one day. He was tachycardic to 124, with blood glucose of 1,123 mg/dl, positive acetone in the serum and ketonuria. Venous blood gas showed a pH of 6.79 and chemistry showed an anion gap of 44. Patient was started on IV fluids, insulin and bicarbonate infusions and admitted to the medical ICU. Labs showed gradual improvement over the next 12 hours, however he remained lethargic. Approximately 18 hours after admission, patient went into pulseless ventricular tachycardia and ACLS protocol was initiated. He was shocked once and given 1 dose of epinephrine, with ROSC achieved after 4 minutes. At the time, his labs were notable for pH of 7.49, potassium 3.6 mEq/l, magnesium 2.0 mg/dl and phosphorus 4.3 mg/dl. A bedside transthoracic echocardiogram (TTE) showed significantly decreased left ventricular function of 20%, compared to prior 58%, with global hypokinesis and regional wall motion variation. The troponin level was 0.03 ng/ml. The patient was started on dobutamine infusion and transferred to the cardiac intensive care unit. Over the next several days, his DKA resolved and he was weaned off of the dobutamine. A cardiac MRI was performed, which did not show any areas of perfusion defect. Subsequent TTE four days later demonstrated improvement of ejection fraction to 49%.

Conclusion

Takotsubo cardiomyopathy can be caused by various physical or emotional stressors. In this case, our patient presented with severe DKA and had a subsequent cardiac arrest with extreme dysfunction of his left ventricle. This patient was young and had no other risk factors for cardiovascular disease or acute coronary syndrome. A normal cardiac MRI and subsequent improvement in cardiac function support this diagnosis. He was also noted to have a prolonged QTc on his EKG prior to the cardiac arrest, which is of the most common findings in Takotsubo cardiomyopathy, along with T wave inversions that tend to deepen following the initial stressor or event. In this patient, better glycemic control and correction of electrolyte imbalances led to a rapid improvement in left ventricular function. This case highlights the importance of treating the underlying stressor contributing to Takotsubo cardiomyopathy.

References

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