CLINICAL VIGNETTE

Management of Diabetic Ketoacidosis with Concomitant Cardiogenic Shock Secondary to Non-ST Elevation Myocardial Infarction

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Case

A 72-year-old man with uncontrolled diabetes (A1c 13.7) and chronically untreated atrial flutter presented with twelve hours of dyspnea, orthopnea, and lower extremity edema. He was afebrile, hypertensive to 158/107 mm Hg, tachycardic to 130 beats per minute, tachypneic to 28 breaths per minute, and hypoxic requiring 3L nasal cannula. Physical exam was remarkable for cool extremities, elevated jugular venous distension at 13 cm, bibasilar lung crackles, and 1+ bilateral lower extremity edema. Initial labs were remarkable for a serum pH of 7.26 with a bicarbonate of 17 mM (ref: 20-30 mM), anion gap of 21 (ref: 8-19), glucose 639 mg/dL (ref: 65-99 mg/dL), lactate 54 mg/dL (ref: 5-16 mg/dL), beta-hydroxybutyrate 16 mg/dL (ref: <3 mg/dL), high sensitivity troponin 2418 ng/L (ref: <4 ng/L), and BNP of 1838 pg/mL (ref: <100 pg/mL). Electrocardiogram demonstrated typical atrial flutter with rapid ventricular response and no clear evidence of ischemia. He was diagnosed with acute non-ST elevation myocardial infarction (non-STEMI), diabetic ketoacidosis (DKA), and cardiogenic shock.

He was admitted to the CCU for DKA management prior to cardiac catheterization. He was started on an insulin drip, and diuresed with parenteral furosemide for CHF in lieu of aggressive intravenous fluid replacement for DKA. Within 12 hours, his fluid intake/output was net negative 2 liters with normalized serum anion gap, lactate, and pH. High sensitivity troponin remained stable between 2000-4000 ng/L. Left heart catheterization demonstrated 90% stenosis of the ramus intermedius (RI) artery and 80% stenosis of the mid-RCA. He underwent percutaneous coronary intervention with a drug eluting stent to the RI. Right heart catheterization demonstrated a mean right atrial pressure of 16 mm Hg, right ventricular pressure of 35/18 mm Hg, pulmonary artery pressure of 36/32 mm Hg, with a mean PAP of 35 mm Hg, a pulmonary capillary wedge pressure of 20 mm Hg, and a cardiac index 1.7 L/min/m^2. Transthoracic echocardiogram (TTE) demonstrated diffuse hypokinesis with ejection fraction of <20%. Cardiogenic shock was managed with further diuresis, dobutamine (weaned off within 24 hours of initiating), and successful TEE cardioversion of atrial flutter with RVR. Cardiogenic shock resolved by hospital day 5 with repeat TTE demonstrating mild improvement in EF to 20-25%. He completed staged PCI of the mid-RCA without complication on hospital day 9. On hospital day 12, he was stable for downgrade from the CCU for further guideline directed medical therapy and physical rehabilitation. Two months post discharge, he remained stable with no acute medical events or hospitalizations. He was initiated on sacubitril-valsartan, carvedilol, spironolactone, and dapagliflozin, and repeat TTE showed improved EF of 30-35%.

Discussion

Standard of care in DKA management includes IV fluid replacement, insulin, and monitoring/treating of electrolyte shifts. In usual DKA, IV fluid replacement is critical as patients typically present with hypovolemia and hyperosmolarity from glucosuria. However, DKA patients may present with volume overload including cardiogenic shock.

There is a paucity of management guidance of DKA in volume overloaded states. A consensus statement published by the America Diabetes Association in 2006, and updated in 2009, states that in the presence of cardiac or renal compromise, frequent hemodynamic and clinical monitoring should be performed during fluid resuscitation.[1] A reasonable approach to the management of DKA in volume overloaded states is to triage all urgent/emergent medical problems and stabilize the patient to prevent decompensation. On initial triage of our patient, highest concern was his cardiogenic shock with evidence of worsening hypoxia. Thus he was admitted to the CCU for close hemodynamic monitoring and diuresis prior to urgent left heart catheterization.

In a study of type 1 diabetics presenting with STEMI with or without DKA, patients with DKA had greater than two times all-cause mortality, cardiogenic shock, and cardiac arrest.[2] Due to the excess catecholamine surge in DKA, it is possible that the metabolic changes of a hypoinsulinemic state deprives myocardium of glucose which results in greater demands for anaerobic respiration during significant ischemia.[3] Given cardiogenic shock, coronary angiography was the most important investigation for this patient.[4] After sufficient DKA

stabilization, left heart catheterization revealed two vessel significant obstructive coronary artery disease and acute myocardial infarction, supporting coronary artery disease responsible for his cardiogenic shock. Longstanding untreated atrial flutter may have also contributed to cardiomyopathy, which was addressed with cardioversion prior to discharge.

There are few published cases of concomitant DKA and cardiogenic shock. One case similar to our patient described a 57-year-old female presenting with STEMI, acute heart failure, and moderate-severity DKA. She was triaged immediately for left heart catheterization with intervention on a distal LAD obstructive lesion followed by fluid resuscitation at 250 cc/hr for DKA with hemodynamic monitoring in the CCU with serial echocardiography. The rate of fluid resuscitation was reduced when worsening volume overload emerged, and DKA indices normalized with usual DKA management.[5] There are two published cases of DKA-induced stress cardiomyopathy with cardiogenic shock, however without acute coronary syndrome. In both cases, standard DKA management was performed including IV fluids. However, both patients had declining clinical status including escalating vasopressor requirements and extracorporeal membrane oxygenation likely due to the severe metabolic derangements of DKA and stress cardiomyopathy.[6,7] These two cases differed from our patient with non-obstructive left heart catheterization findings.[6,7] Regardless of clinical differences, these cases demonstrate the importance of close hemodynamic monitoring while managing DKA in hypervolemic states.

REFERENCES

1. **Kitabchi AE, Umpierrez GE, Miles JM, Fisher JN**. Hyperglycemic crises in adult patients with diabetes. *Diabetes Care*. 2009 Jul;32(7):1335-43. doi: 10.2337/dc09-9032. PMID: 19564476; PMCID: PMC2699725.
2. **Dangl M, Albosta M, Sanchez CAV, Hernandez RJ, Colombo R**. Impact of diabetic ketoacidosis on st-elevation myocardial infarction outcomes in patients with type 1 diabetes. *J Am Coll Cardiol*. 2023; 81(8_Supplement):1258-1258. doi:10.1016/S0735-1097(23)01702-3. Available at: https://www.jacc.org/doi/abs/10.1016/S0735-1097(23)01702-3.
3. **Gandhi MJ, Suvarna TT**. Cardiovascular Complications in Diabetic Ketoacidosis. *Int J Diab Dev Countries*. 1995;15:132-3. Available at: https://rssdi.in/newwebsite/journal/1995_oct-dec/article5.pdf.
4. **Vahdatpour C, Collins D, Goldberg S**. Cardiogenic Shock. J *Am Heart Assoc*. 2019 Apr 16;8(8):e011991. doi: 10.1161/JAHA.119.011991. PMID: 30947630; PMCID: PMC6507212.
5. **Zuhri E, Iryuza N**. Management of Diabetic Ketoacidosis Concomitant With Acute Myocardial Infarction With Acute Heart Failure: A Case Report. *CardioMetabolic Syndrome Journal*. 2022;2(1):71-76. doi:10.51789/cmsj.2022.2.e9. Available at: https://e-cmsj.org/DOIx.php?id=10.51789/cmsj.2022.2.e9.
6. **Gurreri RA, Poommipanit P, Yamamuro M, Alghamdi A**. Cardiogenic Shock Secondary to Stress Induced Cardiomyopathy Precipitated by Severe Diabetic Ketoacidosis. In: *B42. CARDIAC AND PULMONARY CASES*. American Thoracic Society; 2022:A2869-A2869. doi:10.1164/ajrccm-conference.2022.205.1_MeetingAbstracts.A2869. Available at: https://www.atsjournals.org/doi/abs/10.1164/ajrccm-conference.2022.205.1_MeetingAbstracts.A2869.
7. The Perfect Storm: Severe Acidosis, Cardiomyopathy, Cardiogenic Shock, And Extracorporeal Membrane Oxygenation. Accessed March 16, 2024. Available at: https://shmabstracts.org/abstract/the-perfect-storm-severe-acidosis-cardiomyopathy-cardiogenic-shock-and-extracorporeal-membrane-oxygenation/