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Acute Myocardial Infarction and Diabetic Ketoacidosis: The Lethal Duo

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ABSTRACT

Diabetes Ketoacidosis in association with acute myocardial infarction is quite frequent but is also associated with higher morbidity and mortality. These two can trigger each other, different hypothesis have been proposed to explain this phenomenon but still it is difficult to know which one appears first. We report a referred case to our centre with acute Myocardial Infarction and diabetic ketoacidosis promptly initiated treatment of diabetic ketoacidosis along with primary PCI.

Keywords: Cardiogenic shock; diabetic ketoacidosis; metabolic acidosis; myocardial Infarction

INTRODUCTION

Diabetic ketoacidosis (DKA) is an acute, major, life-threatening complication of diabetes characterized by hyperglycemia, ketoacidosis, and ketonuria. Approximately 4% of patients DKA Associated with Acute Myocardial Infarction (MI) and the mortality rate in these patients to nearly 85%.[1] MI is the major cause of all deaths occurring within 48 hours of hospitalization for ketoacidosis.[2]

There is a paucity of published reports in our context. We report a case of 70 years female presented to us with DKA associated with acute MI in cardiogenic shock and managed with DKA treatment and Primary PCI.

CASE REPORT

A 70 years female, hypertensive, was referred to our centre with the suspicion of acute coronary syndrome. Patient had complaints of sudden onset epigastric pain associated with sweating. On arrival patient was dyspnoeic, BP 92/65mmHg, pulse 94/min RR 22/min, wheeze on chest auscultation. ECG showed RBBB morphology with ST elevation in v5, v6, I and aVL as shown in figure. She had no significant past medical history like diabetes mellitus, hypertension and was not under any medications. Her Random blood sugar was 27.9, ABG suggestive of severe metabolic acidosis, urine acetone was positive, CPK MB187U/L and Troponin I was positive Screening echocardiography showed hypokinetic posterior wall, mild MR with LVEF 45%. DKA management was started immediately in the meanwhile patient went into bradycardia and oxygen saturation dropped, managed according to ACLS protocol, food particles were present during intubation suspicious of aspiration, patient was transferred to cath lab. A coronary angiography revealed a total occlusion of the proximal left circumflex artery and 60% stenosis in right coronary artery. Primary angioplasty with drug-eluting stent (DES) was done in left circumflex and RCA was planned for medical management. Patient was transferred to CCU and kept on ventilator with inotrope support along with dual antiplatelet therapy (DAPT), Statin, broad coverage antibiotics, and fluid and electrolyte resuscitation along with insulin infusion. On the second day, patient suddenly developed bradycardia, we performed cardiopulmonary resuscitation, as per ACLS protocols. Unfortunately, after 30 minutes of resuscitation, patient passed away.

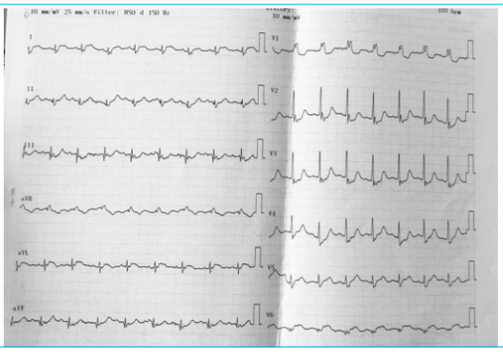

Figure 1. ECG suggestive of Sinus rhythm with RBBB with ST elevation in v5, v6, I and aVL.

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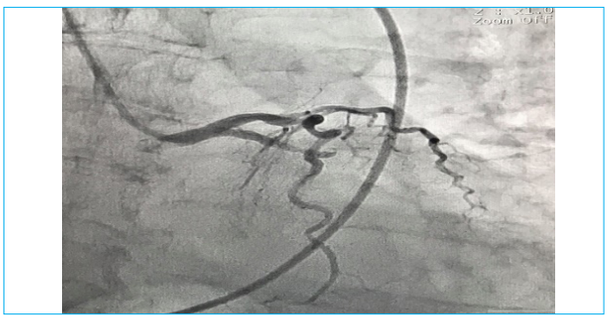

Figure 2. LAO cranial view showing total occlusion of proximal LCX.

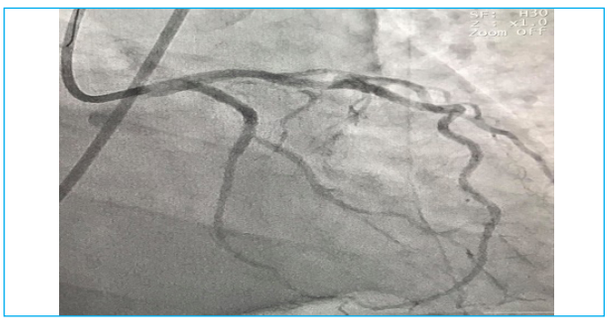

Figure 3. RAO caudal view after stent implantation in LCX.

DISCUSSION

DKA may be the precipitating event for the occurrence of acute MI, serious arrhythmia and pulmonary edema, while MI is also a well-known precipitant factor for DKA. If these two combined, it has a very high mortality rate.[3] MI due to DKA has a complex pathophysiology and exact mechanism is still unclear but there are few proposed mechanisms which including a rise in counter-regulating hormones such as adrenaline, cortisol and glucagon leads to supply-demand mismatch, resulting in myocardial necrosis.[4] A high circulating level of free fatty acid during DKA leads to the incorporation of fatty acids into the lipid structure of the myocyte membrane with the formation of micelle with destabilization and rupture of this membrane. Also, insulin deficiency is associated with a high level of free fatty acids and ketone bodies, which inhibits the glucose uptake by the cell and thus deprives the myocardium of its energy substrate.[5]

Myocardial infarction should always be considered as a possible precipitant of DKA in diabetic patients. In order to avoid unnecessary diagnostic procedures and prompt treatment, clinicians must be able to recognize electrocardiographic abnormalities in DKA patients suggestive of myocardial ischemia like shortening of QTc interval, ST-elevations without reciprocal changes which is related to metabolic derangements like hyperkalemia, hypercalcemia. On the other hand clinicians should also promptly recognize those patients with actual on-going ischemia that may benefit from a coronary percutaneous intervention or Intravenous thrombolysis.[6,7]

A pseudomyocardial infarction pattern has been described in patients with DKA and hyperkalemia with no echocardiographic or angiographic finding suggestive of real myocardial ischemia, and there was a complete resolution of electrocardiographic abnormalities after metabolic improvement.[8,9]

Myocarditis in uncontrolled diabetes has been described in the literature; however, in all the cases, a viral cause was responsible for the clinical findings.[10] Dehydration in DKA patient may be responsible for leading to subepicardial injury which can present with pericarditis or myopericarditis. Ketoacidemia may contribute to elevations in cardiac enzymes.[11]

A retrospective observational study published in 2019 in IJC Heart & Vasculature with 745 patients who had a concomitant diagnosis of STEMI and DKA showed higher overall mortality, cardiac arrest, cardiogenic shock, and acute kidney injury as well as longer ICU stay and total length of stay and ultimately higher cost of care.[12]

CONCLUSIONS

We present a case with diagnostic and therapeutic dilemma as patient presented with acute STEMI and DKA together. Acute cardiovascular changes can also occur due to rise in counter-regulating hormones, free fatty acid release and metabolic acidosis and myocardium ischemia can be further exacerbated during acute MI. When combined, DKA and MI had high overall morbidity and mortality. But, clinicians also must be able to recognize ECG abnormalities due to metabolic derangements suggestive of myocardial ischemia in order to avoid unnecessary diagnostic procedures and treatments.

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