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DIABETIC KETOACIDOSIS AND MYOCARDIAL INFARCTION- A DIAGNOSTIC AND THERAPEUTIC CHALLENGE

Internal Medicine

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ABSTRACT

In patient with acute myocardial infarction with acute left ventricular failure and concomitant Diabetic ketoacidosis, diagnostic and therapeutic dilemma possess a significant challenge in management. A 62 year diabetic male presented acute onset epigastric pain and acute onset shortness of breath for 8 hours. Physical examination showed bilateral basal pulmonary crackles. ECG was suggestive of T wave inversions in antero-lateral leads and troponin levels were elevated. Random blood sugar level was 450 mg/dL and blood gas analysis showed type 1 respiratory failure with high anion gap metabolic acidosis and positive urine ketones. Severity of ketoacidosis incites one to initiate aggressive fluid therapy but the in presence of acute heart failure it can worsen pulmonary congestion. Hence, early diagnosis and treatment modification in form of restricted fluid therapy with continuous clinical and echocardiographic monitoring is required.

KEYWORDS

BACKGROUND

Diabetes is a complex metabolic disorder characterised by uncontrolled hyperglycemia leading to a magnitude of complications and a significant cause of morbidity and mortality. It is one of the leading cause of cardiovascular disorders. However, in addition to long-term effects of diabetes, acute hyperglycemic complications such as Diabetic ketoacidosis and hyperglycemic hyperosmolar state are important causes of acute mortality and morbidity in diabetic patients.
Acute myocardial infarction is an important precipitating factor of ketoacidosis and approximately 4% of patients of DKA are associated with AMI. Symptoms of both these conditions can be overlapping e.g. epigastric pain and acute onset shortness of breath. This can lead to a diagnostic dilemma for clinicians. Also, treatment of DKA requires correction of dehydration, hyperglycemia, acidosis, electrolyte imbalance, and identification of precipitating events. In the absence of cardiac compromise, isotonic saline can be infused at higherratesof 1–1.5 L during the first hour.2 Unfortunately, AMI is frequently accompanied by acute heart failure (AHF). In AMI patient with AHF concomitant with DKA, volume expansion can be the source of a crucial therapeutic dilemma.

Case Presentation

62 year old male, an ex-railway serviceman is a regular ethanol consumer with daily intake around 70-80 gram ethanol for the last 18 years, an active smoker for last 44 years with smoking index of >500, a known diabetic for a decade on irregular treatment with oral antidiabetic medications. He now presented with a history of epigastric pain for one day which was sudden onset, severe intensity, constricting type, at rest, which was non-radiating without any aggravating or relieving factors. Following this, he developed shortness of breath which was acute in onset, at rest, which increased on lying down. There was no histroy of cough, expectoration, hemoptysis, palpitations or loss of consciousness. There was no history of reduced urine output or lower limb swelling.

On presentation, patient was conscious and oriented. He was tachypneic with a respiratory rate of 26/min and heart rate of 124/ min, regular in rhythm. His room air saturation was 58% and was started on supplemental oxygen. Chest auscultations shows diffuse bilateral end inspiratory fine crackles with normal cardiac sounds.

Based on the acute presentation with a background history of long-standing diabetes mellitus and multiple risk factors, a possibility of acute coronary syndrome was kept. Patient was evaluated further on these lines. However, less likely possibility of a Pulmonary embolism or a spontaneuous pneumothorax was also kept and evaluation was done for the same.

Investigations

Haemoglobin- 13.9 mg/dl

Investigations pointing towards Acute coronary syndrome

ECG(at admission)- normal sinus rhythm, normal axis, narrow QRS complexes with ST depression in leads I, II, AVF, V5,V6
Troponin I- 40 (0.02 - 0.06 ng/mL)

2D ECHO- Regional Wall motion abnormality present with anterolateral wall hypokinesia with reduced ejection fraction (LVEF ~ 40%)

Investigations pointing towards Diabetic ketoacidosis

Blood gas analiysis- High anion gap metabolic acidosis with Type 1 respiratory failure
Urine ketones- 2+
Random blood sugar- 450 mg/dL

Differential Diagnosis

Considering the acuteness of the presentation and multiple risk factors and comorbidities, the differential was an acute coronary syndrome causing an acute left ventricular dysfunction. This was confirmed in the Emergency department with a 12 lead ECG and point of care Troponin levels.

However a reasonable differential for acute epigastric pain and acute shortness of breath with a history of non-compliance to anti-diabetic drugs would be Diabetic ketoacidosis. This was confirmed with blood gas analysis which showed high anion gap metabolic acidosis (HAGMA) and blood sugar level 450 mg/dL with urine ketones positive.

Other differential considered was a spontaneous pneumothorax on the background history of regular smoking which can occur due to rupture of emphysematous bulla. This was ruled out with auscultation and bedside lung ultrasound and later CT scan of chest did not show any pneumothorax.

Treatment

Patient was immediately started on Non-invasive positive pressure ventilation in view of type 1 respiratory failure with pulmonary edema.

Diagnosis of Non-ST Elevation MI (NSTEMI) was made and patient was immediately given a loading dose of high dose aspirin and clopidogrel with high dose atorvastatin. He was started on therapeutic anticoagulation with low molecular weight heparin.

For DKA, his potassium levels were measured and was started on human regular insulin (HIR) with a bolus dose of 0.1 mg/kg followed by continuous infusion. His random blood glucose (RBS) was monitored hourly and insulin infusion was titrated accordingly to maintain target RBS of 150-250 mg/dl. Dextrose-Normal saline infusion was given to achieve target RBS and was continued till

resolution of HAGMA. After acidosis resolved, HIR was stopped after overlap with subcutaneous long acting insulin.

The real challenge faced was the fluid resuscitation. As DKA requires aggressive fluid therapy with saline, the same could precipitate further worsening of cardiac function due to increased preload. The patient was monitored with clinically with visual signs of dehydration such as tongue dryness and skin turgor along with JVP measurement andregular chest auscultation to look for resolution or worsening of chest crackles. Other method used was Inferior vena cava (IVC) diameter and serial lung ultrasound to look for B-lines and serial bedside screening 2D-ECHO to monitor cardiac contractility and end diastolic volumes. Fluid administration was one of the most critical therapeutic component in the management of this case.

Outcome And Follow-up

Patient showed gradual improvement and was discharged after 7 days of admission. He was advised for cardiology follow-up for further management of coronary artery disease.

He was followed telephonically after one month and was symptom free. He was advised for cardiac rehabilitation.

DISCUSSION

Diabetic ketoacidosis (DKA) is an acute metabolic complication of diabetes mellitus characterized by uncontrolled hyperglycemia, metabolic acidosis, and increased total body ketone concentration. This results from an insulin deficiency- either absolute or relative, and increase in counter-regulatory hormones. It results in morbidity and mortality in diabetic population. Treatment of DKA consists of dehydration, elevated glucose levels, and electrolyte disturbances; identification of comorbid precipitating events; and above all, frequent patient monitoring. Initial fluid therapy is directed toward expansion of the intravascular, interstitial, and intracellular volume, which are reduced in hyperglycemic crises. In the absence of cardiac compromise, isotonic saline (0.9% NaCl) is infused at a rate of 15–20 $ml \cdot kg\ body\ wt^{-1} \cdot h^{-1}$ or 1–1.5 L during the first hour. (1)

A myocardial infarction is a injury attributed specifically to ischemia, i.e., with clinical evidence of a rise in troponin and at least one of the following: ischemic symptoms or electrocardiographic changes, development of pathologic Q waves, imaging evidence of new loss of viable myocardial or regional wall motion abnormalities consistent with ischemia. (2) Treatment of acute coronary syndrome-NSTEMI involved loading with dual antiplatelet agents along with high dose statins, anticoagulation and early revascularization. (3)Acute coronary syndrome is the most common cause of acute left heart failure(AHF). (4)

In a diabetic patient presenting with acute onset shortness of breath and epigastric pain, common differentials include an Acute myocardial infarction (AMI) and DKA. However they are can coexist and further complicate the clinical diagnosis. It is important to identify these conditions as treatment protocols need to be adjusted according.

AMI is a rare but a well-known cause of DKA. In patients with DKA, AMI should always be kept as a possible precipitating event. Early recognition of AMI is important in making crucial and early therapeutic decisions.

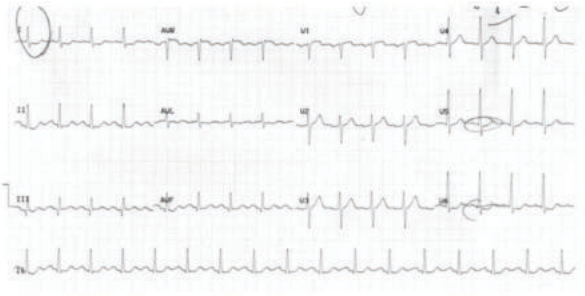
In AMI patients with AHF with concomitant DKA, management can be very challenging. Correction of dehydration in DKA with the standard volumes can put additional stress on the failing left ventricle and can worsen the pulmonary congestion. (5) Currently there are no consensus guidelines for fluid therapy in these scenarios. In a study by Monnet et al, it was suggested that isotonic saline can be started at a lower rate of 250-500ml/hour. In this setting, continuous monitoring of cardiac performance and volume status is crucial. (6)

Echocardiography is increasingly recommended for the diagnosis and evaluation of patients with AHF. A study by Öhman et al. found that using cardiothoracic ultrasound (CaTUS)-guided therapy for AHF showed better decongestion during shorter hospitalization without increased adverse events. CaTUS protocol combine echo cardiographic assessment of cardiac filling pressures, that is, medial E/e′ ratio and inferior vena cava index.(7) In a study by Nagueh et al, it was suggested that a estimated PCWP from echocardiography examination could predict LV filling pressure and E/e′ ratio >15 is associated with increased LV filling pressures (PCWP >15 mmHg).(8)(9)

In conclusion, AMI and DKA possess a diagnostic as well as therapeutic dilemma. Early recognition and treatment modification is required for managing when these conditions coexist.

Learning Points

- Clinical features of DKA and AMI can have overlap. Clinicians should keep a broad differential and appropriately rule-in/out to arrive at the complete diagnosis
- Fluid therapy in concomitant DKA and AMI with acute left ventricular failure is challenging and requires a lower rate of fluid administration along with continuous clinical as well as echocardiographic monitoring.



Figure

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