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Case Report

Mixed Diabetic Emergencies with Acute Pancreatitis and Type 2 Myocardial Infarction: A Case Report

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Abstract

Diabetic ketoacidosis and Hyperosmolar Hyperglycaemic State are both acute, life-threatening metabolic complications occurring in patients with Type 1 and Type 2 Diabetes. The presence of both these conditions simultaneously in one patient is always a challenge to the clinicians. We report such a case of a 62-year-old gentleman known to have Type 2 diabetes, presented with multiple episodes of vomiting and collapse. On arrival to the Emergency Department, he was tachycardic and physical examination revealed upper abdominal tenderness. Blood tests revealed initial pH 6.96, HCO3 6.1mmol/L, Glucose high, blood ketones 5.8mmol/L, K 4.5mmol/L, and Lactate 1.7mmol/L. His calculated serum osmolality was 340mmol/Kg, serum amylase 532U/L and Troponin 88ng/ml. He was treated as per the DKA protocol of the trust and was transferred to the High Dependency Unit. In view of his raised serum osmolality, he was diagnosed as having mixed Diabetic Ketoacidosis and Hyperglycaemic Hyperosmolar State. A CT scan of the abdomen was done to find out the underlying etiology which suggested acute pancreatitis. Additionally, he was diagnosed as having Type 2 myocardial infarction (MI). Both the surgical and cardiology teams were involved in his care. Because of his having both DKA and HHS features the management, especially fluid resuscitation was tailored accordingly. Eventually, the patient recovered and was stepped down to the ward 3 days later. Our case emphasizes that prompt and correct diagnosis of diabetic emergencies can lead to successful outcomes in patients with multiple complications as well. Timely diagnosis, thorough clinical and biochemical evaluation, and effective management are essential for the resolution of DKA and HHS.

Key words: Acute pancreatitis; Diabetic ketoacidosis; Hyperosmolar Hyperglycaemic state; Type 2 myocardial infarction.

INTRODUCTION

Diabetic Ketoacidosis (DKA) and Hyperosmolar Hyperglycaemic State (HHS) represent two extremes in the spectrum of decompensated diabetes.[1] Recent studies have shown that 12%-56% of DKA cases occur in adults with type 2 Diabetes Mellitus (T2DM). [2] HHS has high mortality and morbidity rates in elderly patients with T2DM, of which mortality is approximately 10 times higher than that of DKA.[3] DKA consists of the biochemical triad of hyperglycaemia, ketonemia, and high anion gap metabolic acidosis. [4] The hyperglycaemic hyperosmolar state may consist of moderate to variable degrees of clinical ketosis and alterations in consciousness may often be present without coma. Both DKA and HHS are characterized by hyperglycaemia and absolute or relative insulinopenia. Clinically, they differ by the severity of dehydration, ketosis, and metabolic acidosis.[5] In clinical practice, the development of diabetic ketoacidosis secondary to acute pancreatitis is rare.[6] Myocardial injury is a crucial condition related to mortality in patients with severe DKA, accounting for 28% of deaths in adult patients with DKA.[7] We report a 62 year old gentleman presented with DKA with HHS with acute pancreatitis with type 2 MI.

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CASE PRESENTATION

We report a 62 year old independent gentleman from prison, known type 2 diabetic, hypertensive and ischemic heart disease presented to the Emergency Department of Hinchingbrooke Hospital, United Kingdom with single episode of collapse. On further query, he had multiple episodes of vomiting for 3 days before admission and felt unwell. As per staffs from prison, he had one episode of loss of consciousness for 5 minutes. They did not report any seizure, no tongue bite, no urine or stool incontinence. The gentleman did not give any history of fever, cough, chest pain, shortness of breath and urinary symptoms before that. However, he reported diffuse abdominal pain that radiated to his back. He was hypothermic (34 c), tachycardic (116/min) on admission, but blood pressure and respiratory rate were stable.

In terms of his past medical history, he had Coronary Artery Bypass Grafting (CABG) done almost 15 years back. He used to smoke 20 cigarettes per day but does not drink alcohol.

He used to take aspirin, atorvastatin, bisoprolol, metformin, mixed insulin and nicorandil, however denied any miss of insulin dose or taking hypoglycaemic agent.

On arrival, on the basis of high capillary blood glucose and raised ketone, DKA protocol was started and he was transferred to High dependency unit as per DKA criteria because of very low PH.

After receiving all blood results, we found increased osmolality, markedly raised amylase with serum creatinine and high troponin.

Investigations and Management

At the time of admission,

White cell count-8.5×10^9 / L, Platelet-96×10^9 /L, Hemoglobin 104g/L, CRP-50 mg/L (Baseline 12), troponin-88 ng/ml, eGFR22 (Baseline 90), amylase 998 U/L, PH-6.96, HCO3-6. 1mmol/L, glucose- high, ketone 5.8 mmol/L, lactate-1.7mmol/L, albumin-42g/L, billirubin-6µmol/L, ALT- 7 IU/L, Alkaline phosphatase-145 IU/L, serum osmolality 340 mmol/kg, Na- 152mmol/L, calcium-2.11mmol/L

Bedside ultrasound of abdomen showed swollen and increased echogenecity in head region (Diameter 3.8 cm) of pancreas.

CT scan of abdomen with contrast suggested resolved focal pancreatitis in head region.

ECG did not show any dynamic changes.

Echocardiogram revealed normal left ventricular wall thickness, impaired left ventricular contraction with LV ejection fraction less than 30%.

He was managed conservatively with Intravenous fluids with insulin as per DKA protocol and antibiotics as he seemed septic initially. He was reviewed by the surgical team in ITU and labelled as acute pancreatitis. He was also seen by cardiology team and agreed to treat as type 2 MI. He was shifted to general ward 3 days later and discharged without any complications. At the time of discharge, all the parameters came back to normal.

DISCUSSION

In this case DKA and HHS was present in a single patient simultaneously. This presentation made the management complex and it needed judicious reduction of blood glucose. Fluid balance was also challenging. Acute pancreatitis may cause any type of diabetic complication. In a patient with diabetic emergencies, we should look for aetiology specially if there is any abdominal pain. Any critical illness can cause acute kidney injury and it need to be treated accordingly. In critical situation where there is hypoxia, type 2 myocardial infarction may happen and in this case fluid management should be dine with caution, not to cause further cardiac insult.

CONCLUSION

HHS is more typically seen in type 2 diabetes mellitus. It has high associated mortality rate (15–20%) where high volume intravenous fluid resuscitation is recommended. However, this case illustrates that DKA and HHS can co-exist in a patient with type 2 diabetes.

DKA may mask coexisting acute pancreatitis, which occurs in at least 10-15% of cases. The pathogenesis of acute pancreatitis in DKA varies, but at least in some transient and profound hyperlipidemia is an identifiable factor. Acute pancreatitis is more likely to be associated with a severe episode of DKA with marked acidosis and hyperglycaemia. The troponin increase is a phenomenon described in patients with DKA and corresponds either to a pre-existing coronary pathology unmasked by a metabolic stress or to the toxicity of acidosis, the insulin deficiency or the presence of free fatty acids on the myocyte. A troponin elevation in a diabetic patient should always be considered a coronary abnormality until proven otherwise.

DISCLOSURE

All the authors declared no competing interest.

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