Case Report

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Atypical Diabetic Ketoacidosis in a Young Female

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Abstract

Diabetic ketoacidosis is a life threatning complication of Diabetes Milletus and can be catastrophic if left untreated.Being one of the most common complication but sometime with no clear history and symptoms the diagnosis become difficult. Hence emergency physicians should keep a broad vision while dealing with young patients with high glucose level. Diabetic ketoacidosis may be the first symptom of previously undiagnosed Diabetes.It is diagnosed with combination of Hyperglycemia, Acidosis and Ketonuria. Patients with prolonged acidosis and tendency to hypokalemia should be investigated for consumption of synthetic cannabinoids. No literature till date have showen Metabolic acidosis with PCO2 4.7 mmHg and HCO 3 1.3 mmol/L.

Keywords: Diabetic Ketoacidosis; Hyperglycemia; Acidosis; Ketonuria; Cannabinoids.

Introduction

Diabetic ketoacidosis is an acute serious complication of Diabetes Milletus (mainly Type 1) and still a major cause of morbidity and mortility in young patients. The criteria for diagnosis of Diabetic Ketoacidosis is Hyperglycemia >11mmol/L or 200mg/dl PH <7.3 mmol/L , Bicarbonate < 15 mmol/L And Ketonuria [1].

Further classification by American society of diabetology is Based on bicarbonate as Mild Moderate and Severe. The typical symptoms are as vomitting, nausea, deep gasping breathing (kausmaul respiration), Pronounced thirst, abdominal pain, malaise and generalised weakness [2].

The patient may aslo present with altered concoiusness, disorientation, confusion or occasionally coma when condition is severe [2]. Treatment includes correction of dehydration, hyperglycemia, and electrolyte imbalance.

Case History

A 24 years female Brought in Emergency Department early morning with complaint of altered sensorium from last 2 days which is gradually increasing and history of nausea and vomitting for last 10-12 days and Anorexia for 20 days. Patient was hospitalized for 3 days in some other local nursing home where symptomatic management with IV fluid and antiemetic given. Patient is having back ground history of Oligomenorrhoea and PCOD for which patient is taking Metformin.On examination patient was drowsy but arousable GCS (E3V3M6) vitals as BP- 90/60 mmhg Pulse 101b/min. SpO2 96% on RA Temp. 98.8F and CBG 314mg/dl and patient was dehydrated and mild diffuse abdominal tenderness was present. Urine ketone was positive (++). Rest of the examination was normal. ABG was done which shows:

Patient was diagnosed as a case of Diabetic acidosis and treatment was started accordingly. 2 litre IV Fluid NS bolus given in 1 hour and insulin infusion was

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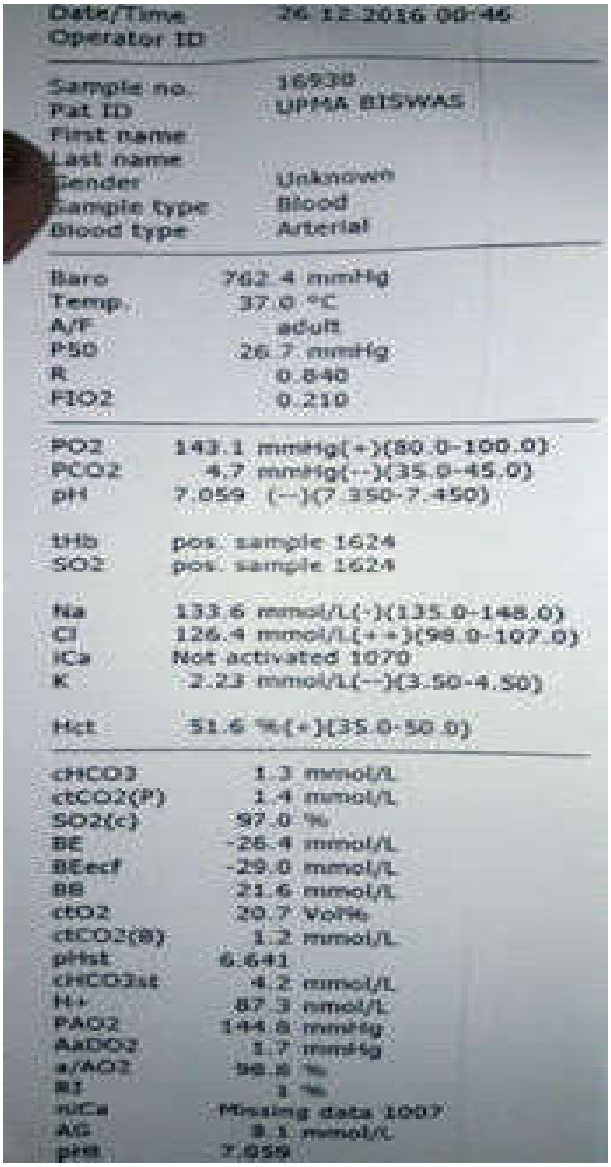
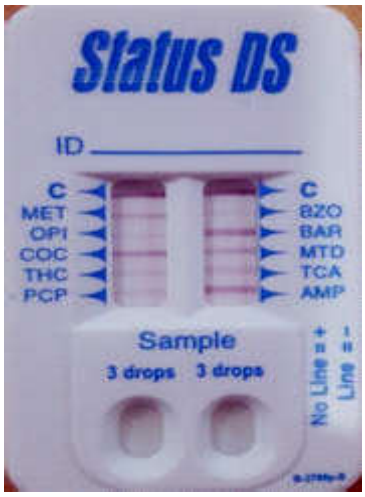
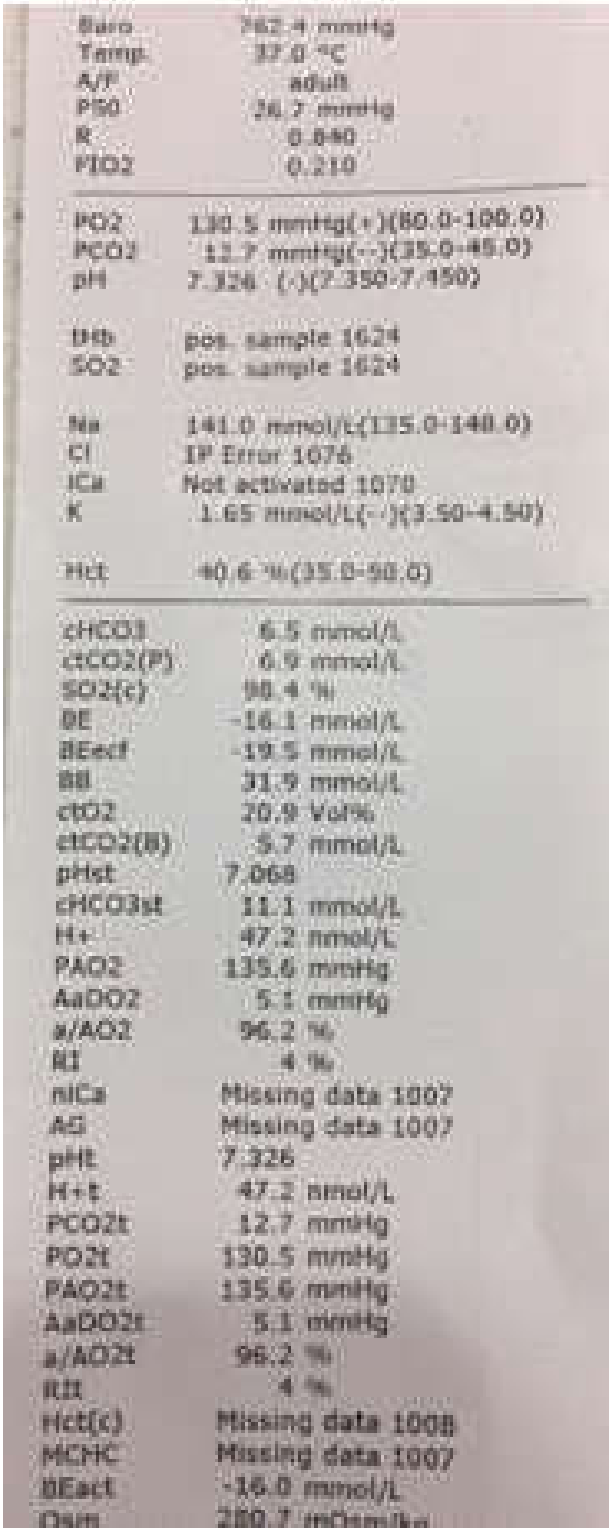


PO2 -143; PCO2- 4.7; PH – 7.059; HCO3- 1.3



Urine status DS done which was negative and urine pregnancy test as also negative.

started @ 0.1U/kg/hr. Patient was given Bicarbonate infusion as per the calculation and all blood investigation forwarded and patient was admitted in ITU and shifted and before shifting the patient almost after 1 hour a repeat ABG was done which shows slight improvement.



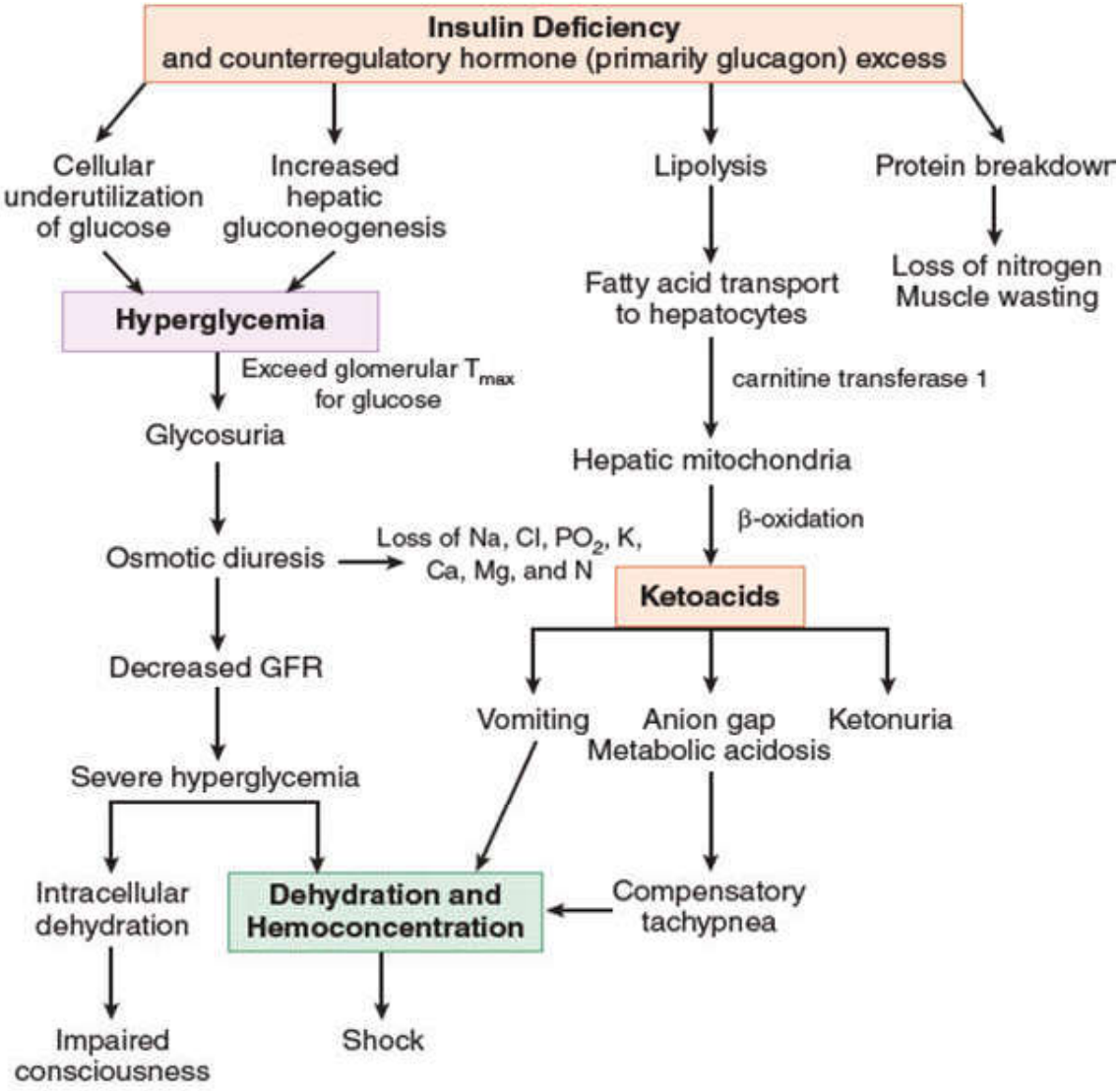


Fig. 4: Pathophysiology of DKA

Other reports are as: Hb- 15.3 g/dl PCV 44.5 TLC 13300, Platelet 2.15 Na^+ - 135 K^+ -3.3 Ca - 9.17 mg - 2.80. Lactate 2.1 creatnine -1, CRP -18.5 TSH 1.18 Urine protein 2++ , Glucose 3+++ and Ketone present. Hb A1C–14. LFT was normal. Echocardigram was normal and LVEF was 60%.

During her course in ITU the patient initially responded well but the her ABG detorated and urine output was less and had hypoventilation and patient was intubated and ventilated and central line insertion was done and patient was put on inotrophic support. patient responded well to the treatment and symptoms subsided and was extubated and shifted to general ward and discharged in a stable condition after 13 days.

Discussion

Diabetic ketoacidosis (DKA) is an acute, life-threatening complication of diabetes mellitus. DKA occurs predominantly in patients with type 1 (insulin-dependent) diabetes mellitus, but 10% to 30% of cases occur in newly diagnosed type 2 (non–insulin-dependent) diabetes mellitus, especially in African Americans and Hispanics [3,4]. A better understanding of the pathophysiology of DKA and an aggressive, uniform approach to its diagnosis and management have reduced mortality to <5% of reported episodes in experienced centers [5]. However, mortality is higher in the elderly due to underlying renal disease or coexisting infection and in the presence of coma or hypotension.

There are several causes of DKA are as; Omission or reduced daily insulin injections, Dislodgement/ occlusion of insulin pump, catheter Infection, Pregnancy, Hyperthyroidism, pheochromocytoma, Cushing's syndrome, Substance abuse (cocaine), Medications: steroids, thiazides, antipsychotics, sympathomimetics, Heat-related illness, Cerebro-vascular accident, GI hemorrhage, Myocardial infarction, Pulmonary embolism Pancreatitis, Major trauma Surgery.

Clinical Features

The clinical manifestations of DKA are related directly to hyperglycemia, volume depletion, and acidosis. The metabolic alterations of DKA tend to evolve within 24 hours [5]. Osmotic diuresis gradually leads to volume loss in addition to renal losses of sodium, chloride, potassium, phosphorous, calcium, and magnesium. Initially, patients may compensate by increasing fluid intake, and polyuria and polydipsia are usually the only symptoms until ketonemia and acidosis develop. As acidosis progresses, ventilation is stimulated physiologically by acidemia to diminish the PCO_2 and to counter metabolic acidosis. Acidosis combined with the effects of prostaglandins I_2 and E_2 leads to peripheral vasodilation despite profound levels of volume depletion. Prostaglandin release is also felt to play a role in unexplained nausea, vomiting, and abdominal pain that are seen frequently at presentation, especially in children. Vomiting, which may be a maladaptive physiologic response to diminish the acid load, unfortunately exacerbates potassium losses. As volume depletion progresses, poor absorption of SC insulin renders its administration ineffective. Impaired mental status may develop and is most likely multifactorial, related to metabolic acidosis, hyperosmolarity, low extracellular fluid volume, and poor hemodynamics. Tachycardia, orthostasis or hypotension, poor skin turgor, and dry mucous membranes result from volume depletion.

Kussmaul respirations, increased rate and depth of breathing, may be observed. Acetone produces the characteristic fruity odor on the breath found in some patients. The absence of fever does not exclude infection. Hypothermia is present occasionally because of peripheral vasodilation. Abdominal pain and tenderness associated with DKA generally correlates with the level of acidosis. Pain can be due to gastric dilatation, ileus, or pancreatitis, but any other acute abdominal disorder can also develop. Due to the frequency of abdominal pain and the presence of an elevated serum amylase or lipase level in both DKA and pancreatitis, distinguishing these two conditions may be difficult. An elevated serum lipase level is more specific to pancreatitis, but it may also be elevated in DKA.

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