A case study on anion gap metabolic acidosis secondary to Diabetes ketoacidosis.

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INTRODUCTION

Diabetic ketoacidosis is a clinical syndrome that results when the triangle of aniongap metabolic acidosis, hyperglycemia, and ketosis is present and is caused by asubstantial insulin deficiency. It is a medical crisis, with an overall mortalityrate less than 5% if patients receive swift and suitable medical treatment. Themainstream of episodes is preventable, and many of the deaths also are avertiblewith proper attention to detail during management[1-2].

CASE STUDY

A-14-year-old girl is brought to the emergency department of a Sungai petani hospital by her mother with the complaints of vomiting, weakness, abdominal pain and confusion since last night. The mother reports the patient has always been healthy and has no significant medical history, but she has lost 15lb body weight in recent times without dieting and has been complaining of fatigue for 2 or 3 weeks. The patient had attributed the fatigue to sleep disturbance, as recently she has been getting up several times at night to urinate. She felt excessively thirsty and would drink almost 6 liters of
water in a day for last one month. This morning, the mother found the patient in her room, complaining of abdominal pain, and she had vomited. She appeared confused and did not know that today was a school day.She was going through severe emotional trauma Because of sudden loss of her 5 years old younger brother. On examination, the patient is lean, markedly dehydrated, lying on a stretcher with eyes closed, but she is responsive to questions. Her body temperature was normal and has a heart rate 112 bpm, blood pressure 125/84 mm Hg, with deep and rapid respirations at the rate of 26 breaths per minute.She appears dehydrated with a dry tongue, and her breath has a 'fruity' odor, and her neck veins are flat.

Her funduscopic examination of eyes is Normal. Her chest is clear to auscultation. Her abdomen is soft with active bowel sounds and mild diffuse tenderness, but no guarding or rebound. Her neurologic examination reveals drowsy and confused.

Laboratory studies include serum Na: 136 mEq/L (135-150), K: 6 mEq/L (3.5-5.0), Cl: 100 mEq/L (98 – 106), HCO_3: 13 mEq/L (24 – 30), measured anion gap is 32 (12±4), Blood urea nitrogen (BUN) : 8.5 mmol/l. (2.5-8) Creatinine kinase: 128 umol/l. (70-120). Arterial blood gas reveals pH 7.12 with PCO_2: 16 mm Hg, PO_2 : 92 mm Hg. Urine drug screen and urine pregnancy test are negative, and urinalysis shows no hematuria or pyuria, but 3+ glucose and 3+ ketones. Chest radiograph is read as normal, and plain film of the abdomen has nonspecific gas pattern but no signs of obstruction. She is diagnosed as anion gap metabolic acidosis secondary to Diabetes ketoacidosis.

Patient is managed with the goal of restoration of metabolic homeostasis with correction of precipitating events and biochemical deficits, which consists of replacement of fluid losses with improvement of circulatory volume, correction of hyperglycemia and, in turn, plasma osmolality, replacement of electrolyte lossesand clearance of serum ketones.

DISCUSSION

Diabetes ketoacidosis (DKA) occurs as a result of severe insulin deficiency and may be the initial presentation of diabetes mellitus, as in this patient. In all patients with DKA, one must be alert for precipitating factors, such as infection, pregnancy, or severe physiologic stressors,
such as myocardial infarction. Polyuria, polydipsia, weight loss, visual blurring, and decreased mental status are related to hyperglycemia and osmotic diuresis. Nausea, vomiting, abdominal pain, fatigue, malaise, and shortness of breath may be related to the acidosis. Typical signs include reduced skin elasticity, dry mucous membranes, hypotension, and tachycardia related to volume depletion[3]. Kussmaul respirations, deep and rapid breathing, represent hyperventilation in an attempt to generate a respiratory alkalosis to compensate for the metabolic acidosis.
One may also note the fruity breath odor typical of ketosis. The combination of insulin deficiency with excess of its hormonal antagonists leads to the parallel processes. In the absence of insulin, hepatic glucose production accelerates, and peripheral uptake by tissues such as muscle is reduced. Rising glucose levels lead to an osmotic diuresis,

loss of fluid and electrolytes, and dehydration. In parallel, rapid lipolysis occurs, leading to elevated circulating free fatty-acid levels[4]. The free fatty acids are broken down to fatty acyl-CoA within the liver cells, and this in turn is converted to ketone bodies within the mitochondria.

Accumulation of ketone bodies produces a metabolic acidosis. Vomiting leads to further loss of fluid and electrolytes. The excess ketones are excreted in the urine but also appear in the breath, producing a distinctive smell similar to that of acetone. Respiratory compensation for the acidosis leads to hyperventilation[5].

Intravenous glucose and insulin are continued until the patient feels able to eat and keep food down. The drip is then taken down and a similar amount of insulin is given as four injections of soluble insulin subcutaneously at meal times and a dose of intermediate-acting insulin at night and discharge for home.

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