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RESEARCH ARTICLE

ELEVATED SERUM LIPASE IN DIABETIC KETOACIDOSIS: HOW SIGNIFICANT IS THE FINDING A CASE REPORT

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ABSTRACT

Aim: Asses the significance of elevated serum lipase in Diabetic ketoacidosis
Methadology: Serum sample of a female patient known to have Diabetes Mellitus, presenting to ER with complaints of recurrent episodes of vomiting, abdominal pain was taken and routine biochemical tests including a spot urine dipstick test, an arterial blood gas analysis along with serum amylase and lipase was sent. Patient was subjected to ultrasound imaging. The results obtained were analysed.
Result: It was found that along with the presence of an elevated blood glucose (304mg/dl), urine was positive for ketone bodies with low bicarbonate levels. The serum amylase was found to be normal but significantly the serum lipase was found to be three times the upper reference range (180 U/L). The abdominal ultrasound was found to be normal without any evidence of pancreatic inflammation.
Conclusion: Diabetic ketoacidosis is one of the most common complications of diabetes mellitus (especially type 1). It can often be the presenting symptom in a previously undiagnosed patients. Though occurance of pancreatitis is uncommon phenomenon in the setting of DKA but it is not rare either. Studies conducted have shown that both DKA can precipitate pancreatitis and vice versa. But a very significant finding has been that in 16-25% of patients having DKA can have an elevated serum lipase (≥ 3 times the URL) which is considered diagnostic of acute pancreatitis with or without an elevated serum amylase.

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CASE

A 23 year old newly married Muslim female presented to the ER of our hospital (Bowring & Lady Curzon hospital, Bangalore) with the complaints of intractable nausea and vomiting along with pain in abdomen since three days. She did not complain of any fever, diarrhea or constipation. Her medical history was significant for type 1 diabetes for which she was on insulin treatment since last 5 years. Her family history was also positive in that her parents and both her siblings were diabetic. Patient told that due to some marriage preparations she was unable to take her regular insulin doses for almost a week. She was also not on any oral hypoglycemic. Patient also gave h/o of a similar episode 1 year back for which she was admitted to a local hospital, details of which could not be produced. On examination patient was conscious and oriented in time place and person and appeared to have tachycardia. Other vital signs were normal. Her abdomen was soft with epigastric tenderness. There was no evidence of organomegaly. Rest of the systemic examinations were normal. Preliminary blood examinations revealed elevated WBC counts of 12800/mm^3. Biochemistry panel was found to be in normal limits. Her blood glucose level at the time of admission was 304mg/dl. But significantly her urine examination was positive for ketone bodies with elevated urinary glucose. Another important thing to be noted was that her serum lipase was elevated (180 u/l) 3 times the upper reference limits but amylase levels were found to be normal. Also her serum calcium levels were found to be in normal range. Significantly her lipid profile was also found to be normal. An arterial blood gas analysis was conducted for the patient which revealed metabolic acidosis with compensatory respiratory alkalosis. A diagnosis of diabetic ketoacidosis was made with a query of acute pancreatitis due to high lipase levels. The ultrasound evaluation was found to be normal without any evidence of any pancreatic inflammation.

INTRODUCTION

Diabetic Ketoacidosis (DKA) is an acute metabolic complication that occurs mainly in type 1 diabetes mellitus . Although more common among patients with type 1 diabetes, it can also occur in patients with type 2 diabetes. The risk factors are omission of insulin, infection, trauma and acute pancreatitis. Acute pancreatitis can be a precipitating factor for

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DKA in patients with diabetes and vice versa. Several hormonal derangements contribute to the hyperglycemia. There is a significant drop in insulin production because of the pancreatic damage (Powers, 2015; Drew *et al*., 1978). This is associated with an increase in glucagon levels along with other counter regulatory hormones such as cortisol, catecholamines, and growth hormone (Donowitz *et al*., 1975). The decreased ratio of insulin to glucagon promotes gluconeogenesis, glycogenolysis, and ketone body formation in the liver, as well as increases in substrate delivery from fat and muscle (free fatty acids, amino acids) to the liver. Markers of inflammation (cytokines, C-reactive protein) are elevated in both DKA (Powers, 2015). Besides the lipolysis effect of decreased insulin the elevated lipase level causes breakdown of local adipose tissue (Drew *et al*., 1978). DKA, on the other hand, has also been reported to cause acute pancreatitis. The exact mechanism is unclear. In a study by Nair *et al*. (1995) 11% of the patients with DKA developed acute pancreatitis. Of the 11 patients with acute pancreatitis 4 had hypertriglyceridemia (triglyceride levels > 500 mg/dl) and 4 had no identifiable etiology, and 3 had other causes (alcohol, drugs) more likely to cause acute pancreatitis. Based on their observations, the authors concluded that hypertriglyceridemia induced by the DKA might be responsible for pancreatic inflammation in some cases (Nair *et al*., 1995). The acidotic state itself might contribute to pancreatic cell injury (Nair *et al*., 2000). Because > 95% of serum lipase comes from the pancreas, as opposed to 40–50% of amylase, lipase is considered a more specific marker for pancreatitis (Nair *et al*., 1995). Elevation of lipase to levels more than three times the upper limit of normal is considered diagnostic for acute pancreatitis (Maclean *et al*., 1973). But studies have been done which have shown the presence of hyperlipasemia in patients of DKA without radiological evidence of pancreatitis. The values are normally less than three times the upper reference levels but values higher than this have also been encountered. The cause for the hyperlipasemia in the setting of DKA is unclear, but some possibilities suggested are its accumulation secondary to suboptimal excretion in the urine (Yadav, 2009), release of a number of pancreatic lipolytic enzymes (Owen, 1981), and immunological injury to pancreatic acinar cells (Frank, 1999). Hyperlipasemia by itself may not be sufficient for diagnosing acute pancreatitis in this setting (Maclean *et al*., 1973). In the present case the patient was having DKA which was precipitated by the noncompliance to the daily insulin dose and that DKA itself was the cause of the high lipase value. The patient improved with regular insulin dosage and was discharged with advice.

DISCUSSION

The elevation of amylase and lipase nearly too three times the normal value may occur in 16-25% patients. For the diagnosis of acute pancreatitis just these enzymes may not be sufficient in the setting of DKA. Though it has to be said that both acute pancreatitis as well as DKA can precipitate each other, the exact mechanisms involved are still under evaluation.

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