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A CASE OF EUGLYCEMIC PANCREATIC KETOACIDOSIS- A CASE REPORT

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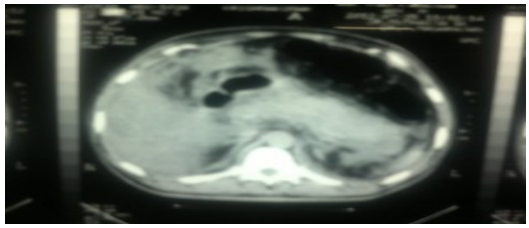
Abstract : Ketosis or ketoacidosis secondary to acute pancreatitis is a distinct, though previously unrecognised syndrome, induced and maintained by extremely high levels of pancreatic lipase in the circulation. Pancreatic lipase is responsible in induction of ketonaemia by promoting ketogenesis via peripheral adipose tissue breakdown. Ketonaemia is well documented as a consequence of prolonged starvation, acute alcoholism, and uncontrolled diabetes mellitus. Euglycemic ketoacidosis due to pancreatitis is a very rare entity and Only very few cases have been reported world wide17. Hence we report this rare entity.

Keyword : Euglycemic pancreatic ketoacidosis, pancreatitis40 yr old male was admitted on 5/7/12 with complaints of abdomen pain, distension for 5 days duration. It was sudden onset, excrutiating type, around the umbilicus , radiated to back, relieved on sitting up &stooping forwards. He was a chronic alcoholic, type2 diabetes & systemic
hypertension patient, not on regular treatment.
On examination patient was conscious oriented,afebrile, dehydrated , dyspnoeic. Pulse rate- 98/min, B.P- 130/90mmhg, Respiratory rate-38/min. Abdomen showed diffuse tenderness with shifting dullness, no rigidity or guarding, bowel sounds were hypoactive ,no hepatosplenomegaly. other system examination were normal.
Investigations revealed Hb- 8gm/dl,
leukocytosis with predominant polymorphs,
Total count- 9600/mm3, Differential count-P72%, L-26%, E-2%,
E.S.R- 10mm for first hour.,
Platelet count- 1.8 lakh/mm3,
Blood urea- 68mg/dl, serum creatinine- 1.8mg/dl
Random blood sugar(Rbs)- 170 mg/dl,
Plasma acetone – positive, urine ketone bodies - positive,
serum amylase- 316i.u,
S. Lipase-880i.u.
serum bicarbonate- 12mM/l,
S. Calcium- 7.8mg/dl,
serum triglyceride- 100mg/dl,
Serum sodium- 134mg/dl, serum potassium- 3.9mg/dl.
Ultrasound abdomen showed- enlarged pancreas with decreased
echogenicity, peripancreatic fluid collection & ascites
CT Scan abdomen showed – edematous enlarged pancreas with necrosis , other organs appeared normal

CT SCAN ABDOMEN



A clinical diagnosis of Acute pancreatitis with ketoacidosis was made.

Treatment given

Absolute bed rest
Nasojejunal feeding
Continous bladder drainage
Input /output charting Vitals
monitoring I/V fluids 2 pint
Normal saline at 5ml/kg/hr 2 pint Normal saline at 2ml/kg/hr 1 pint Ringer lactate at 2ml/kg/hr
I/V antibiotics (piperacillin Tazobactom 4.5mg i/v bd for 14 days
Inj . metronidazole 500mg i/v tds for 10 days
Inj. Sodium bicarbonate 20 cc in 100ml NS i/v bd
Inj. Pantoprazole 40mg i/v od
Blood sugar , ketone bodies were monitored daily.

7/7/12	152/170	+
8/7/12	160/190	+
9/7/12	80/140	+
10/7/12	96/152	+
11/7/12	120/146	+

12/7/12 110/148 +13/7/12 120/156 +

14/7/12	140/160	+
15/7/12	106/140	+
16/7/12	100/146	+
17/7/12	98/130	+
18/7/12	86/146	+
19/7/12	96/126	+
20/7/12	80/130	+

We observed that despite good glycemic control, plasma acetone was persistently positive . After 2 weeks of intense therapy and care, serum amylase came back to normal but lipase was still elevated with evidence of ketosis

20/7/12 (after 2 weeks)

s. lipase	640 iu
Blood sugar	80/130
Plasma acetone	+
s. calcium	7mg/dl

Patient improved symptomatically over a period another 1 week. Hydration status was

good. Respiration came back to normal. Oral diet was started, Serum lipase became normal -40 i.u, blood sugar – Fbs 96mg/dl, PPbs 126mg/dl. Plasma acetone became negative. Urine ketone bodies were absent

date	Blood sugar	Plasma acetone
21/7/12	86/140	positive
22/7/12	78/136	absent
23/7/12	90/144	absent
24/7/12	90/138	absent
25/7/12	90/130	absent

Insulin was not started for this patient as patient's glycemic control was good even from the initial phase.

DISCUSSION

Causes of Euglycemic ketoacidosis

Starvation
vomiting
pregnancy
cirrhosis
Alcohol intoxication
pancreatitis
sepsis
Lactic acidosis
RENAL TUBULAR ACIDOSIS
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Ketoacidosis in our patient cannot be attributed to a lack of oral intake, since ketonaemia and/or ketoacidosis is not a frequent manifestation of starvation[2]. Furthermore, starvation could not be responsible for the persistence of ketonaemia in patient , since abundant quantities of glucose were infused initially followed by resumption of oral intake during the period of observation and patient had good glycemic control through out the course. Alternatively, ketoacidosis is frequently attributed to ethanol ingestion. However, alcoholic ketoacidosis is unlikely since serum ketone titers are negative or only minimally elevated in this syndrome and cannot persist for longer periods[5]. Moreover ketoacidosis cannot be attributed to diabetes in our patient since plasma acetone was persistently elevated despite good glycemic control for more than 2 weeks[3] . Therefore, it seems likely that the acute pancreatitis itself was responsible for ketoacidosis in this case, since both ketonaemia and ketoacidosis resolved with recovery from the disease.

Ketosis or ketoacidosis secondary to acute pancreatitis is a distinct , though previously unrecognised syndrome, induced and maintained by extremely high levels of pancreatic lipase in the circulation[11]. Pancreatic lipase is responsible in induction of ketonaemia by promoting ketogenesis via peripheral adipose tissue breakdown ,since low serum Ca++ levels in this patient indicates the presence of digestion of mesenteric fat stores by the leaked pancreatic lipase.Therefore, we believe that our patient manifested ketoacidosis secondary to high circulating lipase levels induced by acute pancreatitis. The occurrence of mild hyperglycaemia in the same patient may confuse the physicians with the diagnosis of diabetic ketoacidosis as also previously described in some patients with alcoholic ketoacidosislack of requirement of insulin or any other antidiabetic therapy following resolution of acute pancreatitis suggests that both hyperglycaemia and ketoacidosis were secondary to acute pancreatitis.

Explanations for reviewer's suggestions

1.CRP is a predictor of severity in acute pancreatitis. Values more than 150 mg/dl is a predictor for necrotizing pancreatitis. CRP was not evaluated in our case because severity was evaluated based on direct evidence of necrosis in CT and other clinical and laboratory parameters ,. - (Yamada's Text book of Gastroentorology ,5th edition)

2.Patient was treated in Intensive care unit for 3 weeks. The details of management are given above.

3. Serum ketone bodies were qualitatively assessed daily. Serial monitoring is given in the table provided above. Serum lipase was not monitored on a daily basis. It was not available in our institution , moreover we had to sent the sample outside and the patient was not able to afford such a costly investigation daily. Instead we checked it periodically during the first week- 880 i.u, 2nd week- 640 i.u and 3rd week - 40 i.u. when ketone bodies disappeared, serum lipase was found to be in normal range.

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