Serum Amylase and Lipase Levels in Diabetic Ketoacidosis: A Common Misdirection

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Serum amylase and lipase levels are used as diagnostic indicators for pancreatitis. However, there are a host of other causes which may lead to significant elevation of these two blood parameters. Many of these other causes can present with abdominal symptoms like pancreatitis. We here report such a non-pancreatic cause of hyperamylasemia and lipasemia.

A 45 year old male patient, a known diabetic on insulin (30/70) was admitted with dengue fever (IgM +ve) to our institution. At admission, his vitals were stable except for a temperature of 101ºF and his blood glucose varied between 250 mg/dl and 300 mg/dl. He was not on any other drugs. He was non-alcoholic. Initial blood tests revealed Hb 13.4 gm/dl, total leukocyte count of 2700/µL and platelet count of 130 mEq/L. Immediate arterial blood gas study revealed a pH of 7.05 with a serum bicarbonate of 3.5 mEq/L. Urine ketone (by dipstick) was +. Immediately, aggressive fluid management with normal saline was started with i.v. insulin. I.v. sodium bicarbonate was also given. A repeat amylase level came as 1129 IU/L and lipase level 4128 IU/L. Liver function test was normal. Serum sodium came as 165 MEq/L.

The patient slowly responded to our management protocol with normalization of blood parameters as shown in the following table: -

<table>
<thead>
<tr>
<th>Day</th>
<th>Platelet count (µL)</th>
<th>Amylase (IU/L)</th>
<th>Lipase (IU/L)</th>
<th>pH</th>
<th>Sr sodium (mEq/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1</td>
<td>72000</td>
<td>ND</td>
<td>ND</td>
<td>7.21</td>
<td>130</td>
</tr>
<tr>
<td>Day 2</td>
<td>80000</td>
<td>1129</td>
<td>4128</td>
<td>7.4</td>
<td>140</td>
</tr>
<tr>
<td>Day 3</td>
<td>52000</td>
<td>139</td>
<td>506</td>
<td>7.05</td>
<td>ND</td>
</tr>
<tr>
<td>Day 4</td>
<td>10000</td>
<td>ND</td>
<td>ND</td>
<td>7.43</td>
<td>141</td>
</tr>
<tr>
<td>Day 5</td>
<td>15000</td>
<td>ND</td>
<td>ND</td>
<td>7.43</td>
<td>141</td>
</tr>
<tr>
<td>Day 6</td>
<td>85000</td>
<td>364</td>
<td>3510</td>
<td>7.4</td>
<td>141</td>
</tr>
<tr>
<td>Day 7</td>
<td>85000</td>
<td>263</td>
<td>1980</td>
<td>7.4</td>
<td>141</td>
</tr>
</tbody>
</table>

ND: Not done

Table 1: Table showing the blood parameters of the patient (day 1=day of admission)

that evening came as 1200 IU/L. He was put on nil per mouth protocol and fluid management was started.

However, the very next morning, the patient became unconscious. Capillary blood glucose was recorded as >700 mg/dl. Immediate arterial blood gas study revealed a pH of 7.05 with a serum bicarbonate of 3.5 mEq/L. Urine ketone (by dipstick) was +. Immediately, aggressive fluid management with normal saline was started with i.v. insulin. I.v. sodium bicarbonate was also given. A repeat amylase level came as 1129 IU/L and lipase level 4128 IU/L. Liver function test was normal. Serum sodium came as 165 MEq/L.

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After stabilization, a repeat ultrasonography of abdomen and CT scan were done. Both were normal. There was also no evidence of ascites or gallstones. Blood lipid profile was normal. Serum calcium was 10 mg/dl. Thus, the raised amylase/lipase values were attributable to the diabetic ketoacidosis state only.

Various non-pancreatic intra-abdominal pathologies may lead to high serum amylase and lipase. Case-control studies have shown that high lipase levels can be found in acute or chronic renal diseases. Diabetes per se or diabetic ketoacidosis (DKA) have been shown as causes of raised amylase and/or lipase in some studies.

The exact cause of raised lipase in DKA is not known. Possible pathogenetic factors include reduced renal clearance of lipase enzyme, pancreatic hyperperfusion with consequent cellular stress and finally, non-pancreatic release of these enzymes. Also another viable hypothesis is that the insulitis of endocrine pancreas in diabetes may “spill over” into exocrine pancreas with consequent release of pancreatic enzymes into blood.

There is no upper limit of elevation of serum amylase or lipase in DKA. In one reported case series of DKA from USA, the serum amylase varied from 400-1000 IU/L and serum lipase varied from 2000-3500 IU/L. In both of these cases, extensive imaging failed to show any pancreatic pathology and the serum enzyme levels became normal with resolution of the DKA.

The most landmark study on this topic showed that serum pancreatic enzymes may be elevated in up to 25% of cases of DKA. The raised enzymes correlated with blood pH or osmolality.

The present case, along with previously reported cases, show that serum amylase and lipase are not reliable markers of pancreatitis in certain settings. However, it must also be noted that acute pancreatitis may precipitate DKA. Hence, when DKA is diagnosed, the raised amylase/lipase should not be assumed to be due to the DKA only and imaging studies for acute pancreatitis must be undertaken quickly. While overdiagnosis of pancreatitis may lead to unnecessary food restriction and possible aggravation of the dehydration in DKA, undiagnosed pancreatitis is also equally harmful.

References