RAISED SERUM LACTATE DEHYDROGENASE ASSOCIATED WITH GANGRENOUS SMALL BOWEL VOLVULUS : A CASE REPORT.

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ABSTRACT

Usually CPK, CK-MB and lactate dehydrogenase (LDH) enzymes are measured in blood during the period of myocardial infarction. The changes in LDH assay indicates duration of the infarction.

The level of lactate dehydrogenase is 500 times greater in tissue than those found in serum. A small mass of damaged tissue causes leakage of enzyme and increases its level in serum.

Any cause of tissue break down/hemolysis which is sufficiently severe can produce LDH pattern similar to that in myocardial infarction.

We report this case of small bowel volvulus showing significant increase in LDH.

It may be due to tissue necrosis which increases the level of LDH in serum.

KEYWORD

Serum Lactate Dehydrogenase, small bowel volvulus.

INTRODUCTION

Lactate dehydrogenase transfers hydrogen using NAD⁺ as hydrogen acceptor thus catalyzing the oxidation of L-lactate to pyruvate. LDH activity is present in all the cells of the body predominantly in cytoplasm of the cell(1). Thus tissue levels are 500 times greater than those in serum, thus even a small mass of damaged tissue causes leakage of enzyme and increasing its level in serum significantly.

Small bowel volvulus is a rare but life threatening surgical emergency. The etiology may be primary or secondary depending upon predisposing factors. Early preoperative investigation and further surgical treatment is required to prevent bowel infarction(2) in a case of volvulus. Here is a case of small bowel volvulus with gangrene where the lactate dehydrogenase level was very high. Increased level of LDH may be due to necrosis of small bowel.

CASE REPORT

A 30 yr. old male was admitted in the emergency ward. He complained of epigastric pain radiating to whole abdomen without radiation to the back. The pain was associated with vomiting since two days. Constipation was also present since two days. There was no past history of fever, hematemesis, melena and jaundice. He was non-alcoholic, and there was no history of severe illness in the past. There was history of mild abdominal pain for one year. On general examination, the patient was dehydrated pulse - 120/min, B.P. - 110/90 mm Hg. On auscultating chest and Cardio Vascular System- No abnormality was detected. On per abdominal examination, there was tenderness all over the abdomen.

On investigation - Hb% - 10.3 gm/dl, total leucocyte count - 8400/cmm, neutrophil - 64%, lymphocyte - 32%, eosinophil - 04% and ESR - 76 mm/hr.

Serum bilirubin - Total 1.5mg/dl, Direct - 0.9 mg/dl, SGOT - 66 U/L, SGPT - 36 U/L, total protein - 5.5 g/dl, Albumin - 2.7 g/dl, BUN - 16.8 mg/dl, Creatinine - 0.9 mg/dl, Random Blood Sugar - 215 mg/dl, Na⁺ - 128 mmol/l, K⁺ - 3.6 mmol/l, Ca 2⁺ -

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8.0 mg/dl, Amylase - 126 u/dl, LDH - 1562 U/L. On ultrasound examination Abdomen showed Ascites ++ with increased gas shadow in abdominal cavity obscuring pancreatic visuality. CT Scan was done to rule out pancreatitis. A classical sign like whirl sign (due to rotated mesentry) and peacock tail sign (due to torsion of small bowel) was present. There was bowel wall thickening, intramucosal air and intraperitoneal fluid which suggested small bowel ischemia or infarction. Both lower quadrant were tapped, the Ascitic fluid was brownish with foul smell. The Ascitic fluid was investigated and the results were total protein - 2.9 g/dl, albumin - 2.2 g/dl, sugar - 115 mg/dl and amylase - 213 u/dl. It also supported the radiological diagnosis.

On laparotomy there was small bowel volvulus and gangrene of 6" of small bowel from terminal ileum, Whole of small bowel was studded with round worm. Resection of gangrenous bowel was done along with ileocolic anastomosis. Part of jejunum was blackish due to loading of worm, that portion was also resected and jejunojejunal anastomosis was done.

CONCLUSION

Small bowel volvulus affects children and young adults. The delay in its diagnosis leads to development of gangrene.

According to Bizer *et al* (3) and Sarr *et al* (4) WBC count > 18,000/cmm was co-related to presence of gangrenous bowel, but on contrary to Bizer *et al* WBC count in our case was only 8400/cmm.

Till recently, increased LDH along with CPK and CK-MB was taken as a common parameter to indicate myocardial infarction and its duration from onset. Besides other conditions raised LDH level has been shown to increase in the case of SBV with gangrene.

Muchas (5) reported increased serum lactate dehydrogenase levels in 86% of patients with gangrenous small bowel volvulus, in contrast to 4% in those with other causes of small bowel obstruction.

According to Thompson (6), lactate dehydrogenase is one of the markers of intestinal ischemia.

One group (7) examined the lactate dehydrogenase level in patients with acute abdominal disease. They reported a sensitivity of 100 % and specificity of 42% for ischemia of intestine.

Dr. Neil R. Feins (8) in his article has stated that lactate dehydrogenase may be taken as criteria for small bowel obstruction.

Thus the above authors finding go in favour of our case. In addition it may be stated that sequential estimation of LDH is needed to know whether there is an increase in the enzyme thus indicating development of ischemia and gangrene, so that intervention may be done and gangrene prevented.

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