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Towering Levels of Transaminases; Acute Hepatitis versus Severe Leptospirosis: A Case with Review of Literature

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Towering Levels of Transaminases; Acute Hepatitis versus Severe Leptospirosis: A Case with Review of Literature

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I. INTRODUCTION

Leptospirosis is a zoonosis, which is an emerging public health problem usually associated with the three 'R's, i.e. rats, rains, rice fields. Clinically it has a wide spectrum of manifestations from a mild form to severe, icterohemorrhagic fever known as Weil's disease. The later, presents as jaundice, with hepatic and renal failure, pulmonary alveolar hemorrhage, myocarditis, rhabdomyolysis in some cases even with CNS manifestations.[1] The liver derangement is frequently expressed as high bilirubin and a moderate transaminitis.[2] In South-east Asia, the incidence of Leptospirosis has been estimated as 0.1-1/100000 to 10-100/10000 depending on the climatic variations.[3] In India, the endemic states are, Gujarat, Maharashtra, Kerala, Tamil Nadu and Andaman and the Nicobar Islands.[4] Few cases have also been reported from West Bengal. [5]

Here we are reporting a case of severe leptospirosis with unusually raised transaminases along with multi-organ failure and successful response to early and appropriate treatment.

II. THE CASE

32 year, male, a non-alcoholic, non-diabetic, non-hypertensive, admitted with complaints of high-grade intermittent fever for last ten days with no chill and rigor, recurrent vomiting and diarrhea for about last six days, yellowish discoloration of urine for five days. Urine output was adequate. There was no significant drug history.

On examination, GCS E2V1M4, BP 120/80, Pulse rate 108/min, icterus present. The patient was drowsy and disoriented, the bilateral planter were

extensor, bilateral pupils were mid-dilated and sluggishly reacting to light, neck rigidity was present, and Kernig's sign was positive. Rest of the systemic examinations was unremarkable.

The patient had a TLC of 18400/mm³, Total bilirubin 8.45 mg/dl, SGPT 3520 U/lit, Creatinine 2.4 mg/dl on day 1. After that, the patient had an episode of hematemesis and multiple episodes of malena. The patient was resuscitated and transfusion of three units of packed RBCs. At that time, Anti HAV IgM, Anti HBsAg, Anti HCV IgM, Anti HEV IgM were non-reactive. Serum CPK was 98 U/lit. Routine urine examination showed Albumin ++, Pus cell 8-10/hpf, plenty of RBCs. Concurrent USG of the abdomen revealed altered renal cortico-medullary differentiation suggestive of renal parenchymal disease with normal kidney size. A chest X-ray was also obtained which was unremarkable.

A provisional diagnosis of severe leptospirosis was made, and treatment was initiated with injection ceftriaxone 1 gram twice daily. On day 3 of hospital stay there was a fall in the trend of liver enzymes but a marked derangement of the renal profile (Hb 7.8 g/dl, TLC: 19700/ mm³ {N80 L15}, total bilirubin: 15 mg/dl ALP: 56 U/lit, ALT: 450 U/lit, AST: 135 U/lit, serum urea 203 mg/dl serum creatinine 7.2 mg/dl, serum sodium 133 mmol/lit, serum potassium 3.1 mmol/lit). On day four onwards, the patient developed oliguria with signs of volume overload. Early initiation of dialysis was considered, and after receiving three cycles of hemodialysis, blood picture showed progressive improvement of renal status in the form of urine volume and decreasing serum urea, creatinine as well. Meanwhile, serology for leptospirosis came with IgM positive.

The treatment was continued for two weeks, and the renal and hepatic impairment showed progressive reversal towards normal, and the patient was discharged in a hemodynamically stable condition at one month.

III. DISCUSSION

Leptospirosis is characterized by the development of endothelial injury, and inflammatory infiltrates in the vessel wall. Clinically the manifestations range from more frequent, petechiae to various degrees

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of organ involvements, markedly in the liver, kidneys, heart, and lungs. The liver involvement is noted as, intrahepatic cholestasis, Kupffer cells hypertrophy, and hyperplasia without any significant structural damage. Whereas interstitial nephritis, renal tubular damage by cellular infiltration and minor changes in the glomeruli, are the chief findings in the renal histology. Among other organs involvements, interstitial myocarditis, pericardial effusion, pulmonary hemorrhages, focal necrosis in muscle fibres by infiltration of inflammatory cells, are common pathology noted.[6] Meningeal involvement is also reported.[7]

In our case, the patient suffered from hepatic, renal, vascular endothelial and meningeal involvement as evident from clinical and laboratory pictures. Though the initial higher level of transaminases brought confusion between the actual diagnosis and viral hepatitis, negative viral serology for hepatotropic viruses and positivity of leptospiral IgM confirmed our clinical suspicion. From the literature, we found a case, reported by Neda Nozari[8] with almost similar liver enzymes still they were much higher here.

Leptospirosis accounts for 1.03 million cases and 58,900 deaths each year globally.[9] In India, according to 2005 data, the number of reported cases was 2355 and deaths were 167.[4]

The antibiotic therapy includes doxycycline for mild disease whereas intravenous penicillin or third-generation cephalosporin (ceftriaxone or cefotaxime) is prescribed for the severe form of leptospirosis.[10] Early initiation of hemodialysis has a crucial role in severe renal involvement [11].

In the above-mentioned case, the patient improved on Ceftriaxone and three sessions of hemodialysis with resolving renal and hepatic impairments and increasing urine output with no signs of meningeal impairment, no sign of bleeding. Finally, he was discharged in a stable condition and was instructed to follow up on an OPD basis. After four months of regular follow-up, he is leading a healthy life.

Multi-organ failure with transaminitis is well known in severe leptospirosis. Nonetheless, this marked rise in transaminase levels as shown here is being reported for the first time in medical literature to the best of our knowledge.

IV. CONCLUSION

Though such high transaminase levels are rarely associated with severe forms of leptospirosis, the possibility must be considered once the usual causes are ruled out. For deciphering the exact pathophysiology further scientific research is necessitated on this ground.

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