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Case Report

Weil's Disease with Severe Anemia and Jaundice in a 19 years Old Boy

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Abstract

Weil's disease is a infection caused by the bacteria Leptospira interrogans. Most cases present as mild to moderate category and self-resolving but few are complicated with multiorgan involvement and can lead to death. We present a case of leptospirosis in a young boy of Nankana sahib who presented with haemorrhagic, hepatic and renal manifestations. The purpose of this case report is to raise awareness in physicians regarding consideration of leptospirosis in patients with hepatic and renal involvement. It also emphasizes on primary prevention in population with zoonotic exposure.

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Introduction

Leptospirosis is a disease that can affect both animals and humans. Bacteria involved in this disease belongs to genus Leptospira. Mode of transmission to humans is getting in contact with water or soil contami-nated with the urine of infected rodents and mammals[1] it presents as variety of symptoms. Some patients may have no symptoms. 10 percent of the patients develop severe spectrum of the disease and mortality rate is 5 to 15% in severe disease[2]. It has incubation period of 2 to thirty days.[3] Most of the cases 90% recover completely.[2] It has early or Leptospiaremic phase having flu-like syndrome sudden high grade fever, chills, headache, muscle aches, vomiting and conjunctivitis, Second or immune phase in which Leptospires disappear from blood due to action of phagocytes, IgM and complement, it can lead to kidney and liver failure, Widespread hemorrhages occur leading to anemia, coma and finally death mainly in elderly or due to virulent strain, "Red eye" conjunctival suffusion due to immune reaction is also seen in this phase.[4] We Present you a classical patient of Weils syndrome with anemia, renalfailure, hyperbilirubinemia and Septicemia.

Case Report

A 19 years old boy belonging to Ahmed wala from district Nankana sahib presented to Emergency Department of Mayo Hospital with fever(high grade), Vomiting and pain in abdomen from 7 days, prior presentation to Emergency department he visited Quack in his village who gave him oral antibiotics and anti-emetics but his symptoms did not resolve. From last 2 days he has been noticing that his urine has turned dark reddish and his eyes have turned yellow. He denied of having diarrhea, pain in right hypochondrium, photophobia, rash and itching. Physical examination revealed conjuctival suffusion, pallor and jaundice .Vital signs recorded were Blood pressure of 100/60 mmHg respiratory rate 26/min and body Temperature 101F. Initial Emergency treatment included one liter of Intravenous Ringer lactate, antipyretic, antiemetic and PPI(proton pump Inhibitor). All baseline blood tests were ordered. Patient was admitted to Medical ICU with provisional Diagnosis of fulminant Hepatic failure. Differential diagnosis included acute viral hepatitis, drug induced hepatitis.

Laboratory test revealed Hemoglobin 4.6gm/dl, Leukocyte count 35000 with predominant neutrophils Hyperbilirubinemia 2.83mg/dl, ALT 110U/L, AST 80U/L , compensated metabolic acidosis with acute renal injury creatinine 2.6mg/dl, Urinalysis showed microscopic hematuria with RBCS 20-25/HPF , Urgent abdominal Ultrasonography was ordered that did not show cholecystitis, or any pathology of biliary tract and any evidence of renal calculi. With markedly low hemoglobin, Leukocytosis and hyperbilirunimia all cultures were sent including blood and urine. Indicators of hemolysis were also ordered which revealed markedly raised LDH 3346U/L, reticulocyte count 16 and peripheral blood smear showing macrocytes and occasional nucleated RBCS. Fractioned bilirubin showed primarily unconjugated hyperbilirubinemia. Patient was initially put on tazobactum and piperacillin Intravenous 2.25 g TDS with cultures awaited. All indicators were suggestive of definite hemolysis.

To rule out cause of hemolysis first malarial parasite slide was ordered which turned out to be negative, autoimmune hemolysis was also excluded by negative direct and indirect coombs test. Still cause of hemolysis was uncertain. Further probing into history of patient revealed that he helps his father at his cattle farm and had laceration on hand while working with cattles 10 days prior to developing illness. That raised the suspicion of any zoonotic infection like leptospirosis so ELISA for antileptospiral antibodies was sent. Meanwhile patient was transfused one pint of whole blood and 2 volume of packed cells supportive therapy included IV tazobactum, PPI and fluids. With blood transfusions and supportive therapy patient's lab parameters showed marked improvement on the third day, abdominal pain settled,urinary parameters turned back to normal and oral intake improved.

On fifth day of admission ELISA for antileptospiral antibodies IGM turned out to be positive raising probability of spirochete infection. Next day awaited blood cultures showed leptospiral growth which confirmed the diagnosis of Leptospirosis. After completing one week of IV tazobactum patient lab parameters were reassessed, response to the treatment was satisfactory with all lab parameters showing significant improvement. Patient was subsequently discharged on 8th day on oral Cephlosporins (cefixime) for 7 days and asked for follow up after one week. Family was counselled about the mode of transmission of disease and was advised to minimize this exposure.

Table 1: *Results of Test Parameters*

Parameter	First day	3rd day	7th day
Hb	4.6	8.2	9.1
TLC	35	21.6	15
Bilirubin	2.83	1.1	0.9
Creatinine	2.6	1.6	1.1

Discussion

Leptospirosis is a morbidity with a different grades of severity. Neurological involvement is seen in up to 25% of patients and manifests as aseptic meningitis; however, cerebellitis, intracranial hemorrhages, and myelitis have been seen in patients.[1] Renal failure has been seen in 16%–40% of patients where oliguria holds prognostic importance.[2] The anemia in Weil's syndrome is caused due to bleeding, acute renal failure and ill-defined hemolytic process.[5] Anemia due to hemolysis in animals is well documented however in humans only 2 cases have been reported.[5] Solmazgul et al. cited a case of hemolytic anemia which was coombs positive that was treated by injecting immunoglobulins.[6] Referring to all studies in animals it is believed to be caused by hemolysins with phospholipase activity.[5,6] Another patient cited by Trowbridge et al. the hemolysis in that patient did not stop by any means.[6] From all in vitro studies, it is proposed that hemolytic process is caused by Red blood cells damage by enzyme phospholipases of the infecting bacteria Leptospira.[6] In our case coombs test was negative so it wasn't immune mediated and course of illness was self-limiting with supportive therapy and early initiation of antibiotics. So the degree of hemolysis is also related to severity of spirochete infection and prompt treatment with Intravenous antibiotics. So hemolysis should also be considered as the cause of anemia in patient with Weils disease.

Conclusion

Leptospirosis is a zoonotic infection mainly occurring in tropical countries with variety of manifestations including hemorrhagic, hepatic and renal symptoms. Mode of transmission to humans is direct contact with water and soil contaminated with urine of infected animals. In country like Pakistan which is badly struck with flood last year, suspicion index of leptospirosis should be kept high in all patients presenting with renal and hepatic involvement. Cause of hemolysis should always be ruled out in severe disease whether immune mediated or not because immune mediated may need prompt treatment with steroids and immunoglobulins. Early initiation of antibiotics may prevent severe course of the disease and anemia.

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