A Young Boy with Severe Leptospirosis (Weil's Disease): A Case Report

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ABSTRACT

Leptospirosis, a zoonotic disease is increasingly being recognized in developing countries. It is often underdiagnosed resulting in significant mortality as the presenting features mimic other commonly prevailing diseases like Malaria, Dengue, Acute hepatitis, Typhoid in developing world. We are reporting a case of 16 years old male student presented with acute febrile illness with icterus, hemorrhagic manifestation and pulmonary-renal involvement, diagnosed as Severe Leptospirosis (Weil's disease) confirmed by ICT method. This experience highlights the importance of considering Leptospirosis early in the diagnosis of acute nonspecific febrile illness with multi-organ involvement to cutoff mortality from this fatal disease.

Key words: *Leptospirosis; Zoonotic disease; Weil's disease; Multiorgan involvement; Fatal disease*

INTRODUCTION

Leptospirosis is a zoonotic infection caused by spirochetes of the genus Leptospira. Infection usually results when water or soil contaminated with the urine of an infected animal comes in contact with human skin or mucous membranes. Clinical manifestations of leptospirosis can range from a self-limited flu like febrile illness to a fatal illness (Weil's disease) characterized by jaundice, hemorrhage, renal failure, severe pulmonary hemorrhage and ARDS[1]. In tropical settings, leptospirosis can be indistinguishable from other febrile illnesses such as malaria, dengue, viral hepatitis, other bacterial sepsis. Leptospirosis has been reported in neighboring areas of Southeast Asia[2]. But the disease is infrequently recognized in Bangladesh where diagnostic tests for leptospirosis are less available. However, environmental factors, such as floods, humidity, and water contamination, are amenable to spread of the disease in Bangladesh. A study was conducted at 2 major hospitals in Dhaka, Bangladesh, during an outbreak of dengue fever. A total of 18% of dengue-negative patients tested positive for leptospirosis. The case-fatality rate among leptospirosis patients (5%) was higher than among dengue fever patients (1.2%)[3]. This case report will update the reader about leptospirosis, a possible diagnosis in febrile illness with multiorgan involvement.

CASE REPORT

A previously healthy 16 years old boy admitted into Apollo Hospitals Dhaka with the complaints of high grade fever, yellow colouration of eyes, scanty high coloured urine, vomiting and diarrhea for 1-2 episodes and started G-I bleeding in the form of hematemesis and hematochezia for 5 days. He had leg and back pain. He had history of playing football on bare foot during the rainy season. On admission he was found conscious but drowsy, deeply icteric with conjunctival haemorrhage, respiratory rate 24-26/ min, pulse rate 120 beats/min, blood pressure 90/60 mm of Hg, temperature 101^{O}F, bilateral lung base crepitations and tender epigastrium. Acute viral hepatitis, Malaria, Dengue, Sepsis with multiorgan failure and Leptospirosis were in the list of differentials. Investigations with results are shown in the following tables:

Table 1: Laboratory Data

Serum Chemistry	Report Result
Sodium (mmol/L)	132
Potassium (mmol/L)	4.5
Chloride (mmol/L)	102
Bicarbonate (mmol/L)	17
Creatinine (mg/dl)	6.64
Urea (mg/dl)	231
S. Bilirubin total (mg/dl)	36.4
Alkaline phosphatase (U/L)	105
SGPT (ALT) (U/L)	142
SGOT (AST) (U/L)	116
Ammonia (µg/dl)	140
CRP (mg/dl)	17
Procalcitonin (ng/ml)	35.7
LDH (U/L)	528
S. Calcium (mg/dl)	7.6
S. Albumin (g/dl)	2.4
S. Phosphate (mg/dl)	3.8
S. Uric Acid (mg/dl)	8.1
Complement C3 (g/L)	1.37
Complement C4 (g/L)	0.207

Coagulation profile	Report Result
PT	13.2
INR	1.1
aPTT	34

Hematology	Report Result
Hb (gm/dl)	6.8
WBC (10^9/L)	11.54
Neutrophil	91.3%
Lymphocyte	6.5%
PLT (10^9/L)	40
RETICULOCYTE COUNT	0.44%
ESR (mm - 1st hour)	92

Urine R/E	Report Result
pH	7.0
S. gravity	1.015
Protein	++
RBC (/HPF)	Numerous
Puscell (/HPF)	15 - 20
Casts	Nil

ABG	Report Result
pH	7.4
PCO_2	27.8
PO_2	87.5
HCO_3-	16.9

Table 2: Microbiological investigation.

Serological Test	Interpretation
Anti-HAV IgM	Negative
HbsAg	Negative
Anti-HCV	Negative
Anti-HEV IgM	Negative
Dengue Chikungunya RTPCR	Negative
ICT for Malaria	Negative
Triple antigen/Fibrile antigen	Negative
IgM antileptospiral antibody (ICT)	Strongly Positive
Blood culture	No growth
Direct Coombs Test	Negative
Anti-Nuclear Antibody	Negative

Table 3: Subsequent lab data.

Serum Chemistry	Report Result
Sodium (mmol/L)	144
Potassium (mmol/L)	4.0
Chloride (mmol/L)	102
Bicarbonate (mmol/L)	21
Creatinine (mg/dl)	1.07
Urea (mg/dl)	38

S. Bilirubin total (mg/dl)	7.8
Alkaline phosphatase (U/L)	103
SGPT (ALT) (U/L)	80
SGOT (AST) (U/L)	68
Ammonia (µg/dl)	69
CRP (mg/dl)	6.0

Serum Chemistry	Report Result
Hb (gm/dl)	9.4
WBC (10^9/L)	20.63
Neutrophil	59.6%
Lymphocyte	24.1%
PLT (10^9/L)	160

On Chest X- Ray P/A view there was bilateral diffuse patchy opacity and sinus tachycardia on ECG, USG of abdomen revealed mild hepatomegaly with acute renal parenchymal changes. The patient was managed in ICU with Inotropes support, oxygen inhalation, maintenance of nutrition, hydration with I/V fluid, intravenous Ceftriaxone 1.5gm 12 hourly, Capsule Doxycycline, I/V antiemetic, I/V proton pump inhibitor with strict monitoring. Two units of fresh frozen plasma and one unit of whole blood were given. On subsequent days creatinine had fallen gradually to normal level and output increased satisfactorily. Serum bilirubin had fallen slowly and patient had significant clinical recovery with normalization of hematological and biochemical parameter over a period of around 3 weeks. Then he was discharged on 18th days of illness with stable hemodynamics. He was advised to come for follow-up at OPD with repeat liver and renal function tests after one week. Following figure shows patient's toxic appearance during admission (Fig. 1) and stable condition while discharge (Fig. 2).

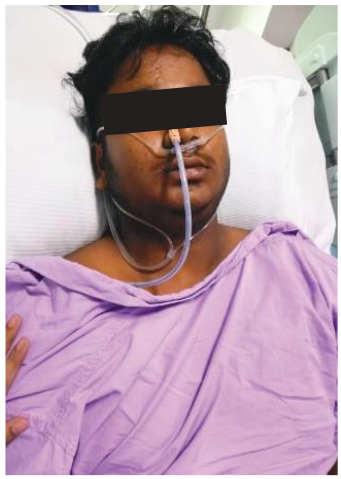

Fig. 1: During Admission

Fig. 2: During Discharge

DISCUSSION

Leptospirosis has recently been recognized as a re-emerging infectious disease among animals and humans that has the potential to become more prevalent with anticipated global warming[4,5]. Leptospirosis peaks during the monsoon and post monsoon months and occurs more commonly where poor sanitation and low hygienic conditions are prevalent[6].

Transmission of leptospiral infections results from direct or indirect exposure to the urine of infected animal. Meningitis, Acute Renal Injury (AKI) pulmonary hemorrhage and Acute Respiratory Distress Syndrome (ARDS) may occur. This most severe form of leptospirosis is the Weil's disease[7].

Our patient presented with leptospirosis in its severe form, i.e. icteric-hemorrhagic illness with multiorgan dysfunction (Weil's disease). Initially he had nonspecific symptoms of Flu like illness, later he developed symptoms of multiorgan involvement (Jaundice, G-I bleeding, hemoptysis, oliguria) which let us think about the rare but fatal disease leptospirosis. The infected animals (most commonly rat). The natural course of leptospirosis comprises of two distinct clinical phases: septicemia and immune phase. Seven to twelve days post exposure patient develops nonspecific flu-like illness with sudden onset of high fever, headache, myalgia and conjunctival suffusion (Septicemia phase)[7]. While body's immunologic response leads to production of immunoglobulin M antibodies and specific organ damage can be observed (Immune phase)[7]. The gold standard for diagnosis of leptospirosis is isolation of the organism by culture of clinical specimens (Blood, CSF, urine) during the first 7 to 10 days of the illness. However, this method requires special media and more than 16 weeks because initial growth may be slow and has a low sensitivity and specificity. The majority of leptospirosis cases are diagnosed by serological method, MAT

(Microscopic Agglutination Test). MAT is unavailable in our laboratory. Here diagnosis was done by IgM anti leptospiral antibody detection with ICT method. ICT test is moderately sensitive but highly specific[8].

Major cause of death in patients with Weil's disease is severe pulmonary hemorrhagic syndrome[9]. Our patient had pulmonary involvement in the form of cough and hemoptysis along with the radiological evidence of diffuse, ill-defined ground glass density in both lung field, that has been described in leptospirosis[8]. This radiologic abnormality was suggestive of alveolar hemorrhage because of faster resolution (Within a week) in comparison to other forms of bacterial pneumonia which resolve slowly.

Hepatic dysfunction is usually not severe in leptospirosis and reversible. But in severe leptospirosis, liver dysfunction can be seen as conjugated serum bilirubin levels may increase to above 80 mg/dl, accompanied by moderate elevations in transaminases, which rarely exceed 200 U/L[10]. Conjugated hyperbilirubinemia and mild elevation of SGPT but normal Alkaline phosphatase were evident in this case as well. Renal impairment is frequently seen in Weil's disease. Azotemia, oliguria, and anuria commonly occur during the second week of the illness but may appear as early as 3 to 4 days after the onset[11]. In this case there was hematuria, pyuria, proteinuria and AKI. Serum creatinine was raised up to 6.4 mg/dl and following single episode of dialysis gradually became normal.

The current treatment of choice for mild leptospirosis includes oral doxycycline and amoxicillin. In cases of fulminant leptospirosis parenteral high-dose penicillin G has long been considered as the treatment of choice. Recent clinical trials have proved the acceptability of third generation cephalosporins: cefotaxime and ceftriaxone agents for patients with severe leptospirosis[12]. We empirically started inj ceftriaxone and capsule doxycycline. The patient responded with satisfactory clinical improvement.

Overall, mortality rate of Weil's disease is 5% to 10%. The mortality is caused mainly by renal failure, cardiopulmonary failure and widespread hemorrhage[13]. So, this fatal disease needs early notification, diagnosis and prompt initiation of specific antimicrobial.

CONCLUSION

Diagnosis of Leptospirosis is often a challenge as the presentations mimic other common diseases. A high index of suspicion is crucial if a patient presents with acute febrile illness with hepatic and renal dysfunction and prompt antibiotic therapy should be initiated on the basis of clinical judgement as laboratory confirmation can be delayed. Availability of a quick, reliable diagnostic method (ICT test) should be ensured at local hospital for early diagnosis.

DISCLOSURE

All the authors declared no competing interest.

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