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A CASE REPORT ON FLUMINANT LEPTOSPIROSIS WITH HEPATORENAL INVOLVEMENT

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ABSTRACT

Leptospirosis is an important zoonotic disease humans and animals that are caused by pathogenic spirochetes of the genus *Leptospira*. Leptospirosis is endemic in most areas where dengue virus is transmitted and may be mistaken for dengue, which is typically more common. Leptospirosis has protean manifestations ranging from a flu-like illness to fulminant hepatic and renal failure culminating in death. A 40 years old male patient came with complaints of fever and myalgia for past 4 days. He had history of yellowish discoloration of skin and sclera, decreased urine output, malena and petecheial rashes all over the body for past 2 days. On examination the patient was febrile, icteric and petecheial rashes were seen. The investigation parameters showed high levels of ESR, Blood urea, LFT, Creatinine and Leptospira AB IgM – positive. The patient was treated with INJ. CEFTRIAXONE, T.DOXICYCILLINE, T.BILASTINE, T.ACETAMINOPHEN, and T.CILNIDIPINE. After the 10 days of treatment in hospital the patient was discharged as he was symptomatically improved. Leptospirosis should be diagnosed as early as possible since early antibiotics are beneficial in the treatment.

KEYWORDS: Leptospirosis, zoonotic, spirochetes, fulminant, malena, petecheial, icteric.

INTRODUCTION

Leptospirosis is a zoonosis of global distribution caused by infection with Leptospira interrogans, a pathogenic spirochete. The most important reservoirs are rodents, predominantly rats. Urinary shedding of organisms from infected animals is the most significant source of infection. The majority of patients present a mild, anicteric febrile illness, but few of patients develop a severe form with hepatorenal involvement.[1] Fluminent leptospirosis also known as Weil's disease is characterized by multiorgan dysfunction and presents with high fever, significant jaundice, kidney failure, hepatic necrosis, pulmonary diseases (especially pulmonary hemorrhage), shock, hemorrhagic diathesis.[1]

CASE REPORT

A 40 years old male patient came with complaints of fever and myalgia for past 4 days. He had history of yellowish discoloration of skin and sclera, decreased urine output, malena and petecheial rashes all over the body for past 2 days. On examination the patient was febrile, icteric and petecheial rashes were seen. The investigation chart revels ESR-80mm/hr, Blood urea-151mg/dl, CRP-274mg/dl. LFT parameters shows: T.Bilirubin:16.2mg/dl, T.Direct Bilirubin:14.1mg/dl, AST-68U/L,ALT-60U/L, Alkaline phosphatase-193U/L, Total protein-5.4g/dL, Total albumin-2.7g/dL, Creatinine kinase-279U/L, Creatinine-8.2mg/dL. Electrolytes levels: Na^+-130mmol/L, K^+-3.88mmol/L. Urine analysis shows the presence of Albumin-++, Bilirubin-++, Pus level-10-12HPF, RBC-numerous.

The differential diagnosis include: Malarial Antigen Rapid- negative, Dengue Ag/ IgM/IgG-negative, HBV Ag/Av, Anti HCV- nonreactive. The Leptospira Ag IgM was positive indicating the presence of Leptospirosis infection. The blood culture revels sterile but urine culture revels the presence of insignificant bacteriauria. The ECHO showed grade 1 diastolic dysfunction. The urine protein Creatinine ratio shows protein creatinine ratio-3.83, Urine protein-144.9mg/dL, Urine creatinine-37.8mg/dL.

During the 10 days of hospital stay, the patient was admitted in the ICU for the 1st 4 days and later shifted to the ward. In ICU the patient was started with INJ.CEFTRIAXONE 1g BD and INJ.PANTOPRAZOLE 40mg. The patient was initiated on HD for 2 days due to the worsening of azotemia and tremor. The patient was on fluid resuscitation therapy. The patient was administered with T.DOXYCYCILINE 100mg BD, T.CILINIDIPINE 20mg OD, T.BILASTINE 20mg BD, T.ACETAMINOPHEN 650mg SOS and T.PANTOPRAZOLE 4Omg OD.

DISCUSSION

Leptospirosis, a zoonosis caused by Leptospira interrogans, a spirochete. It occurs worldwide, but is more common in tropical regions. Wild or domestic animals, including rats, mice, sheep, cattle, pigs, dogs, raccoons, and goats, are its reservoir.[2,3] Leptospires often survive and grow in warm, humid environments and in neutral to slightly alkaline water and soil (chlorine, sea water, gastric juices and acidic pH destroy the leptospire.[4,5] However the incidence of disease and infection is probably greater and extends far beyond the tropics. In endemic regions, leptospirosis can account for 5 to 15% of diagnoses in febrile diseased patients.[6,7]

The natural course of leptospirosis comprises of two distinct clinical stages: septicemic and immune. People usually become sick within 7 to 12 days following leptospires exposure. The first stage is called the septicemic phase (leptospiremic phase) and the bacteria may be isolated from blood cultures and cerebrospinal fluid (CSF). This phase is characterized by a nonspecific flulike illness with sudden onset of high temperature, headache, myalgias (especially in the paraspinal, calf and abdominal muscles)[8] and conjunctival suffusion. Conjunctival suffusion (reddening of the eye surface) is a significant characteristic in leptospirosis, and its presence in a nonspecific febrile ill patient should raise suspicion for diagnosis.

The second stage is called the immune phase (leptospiruric phase) and circulating antibodies can be detected and the bacteria can be isolated from the urine. This stage occurs as a result of immune response by producing immunoglobulin IgM antibodies and can last more than a month. Specific organ damage can be seen during this stage.[9] Severe Leptospira infections are manifested by acute renal and liver failure, and shock. Death mostly occurs usually in the second week of illness.[10] Renal symptoms, such as uremia, azotemia, pyuria and hematuria, may occur while Acute renal failure remains to be the most common complication of severe leptospirosis. Renal leptospirosis is usually depicted as a combination of acute tubular damage and interstitial nephritis.[9] An increase in liver enzymes (up to 5 times normal) with a disproportionately high total bilirubin level is considered as a prognostic indicator in leptospirosis.[11] Hepatic dysfunction is usually mild and reversible. Varying degrees of jaundice, pancreatitis, hepatomegaly and myocarditis can also be observed.

Thrombocytopenia have also been reported with leptospirosis and the pathogenesis of thrombocytopenia and hemorrhagic diathesis in leptospirosis is not well understood.[9]

The diagnosis of leptospirosis requires a high degree of clinical suspicion because the numerous manifestations of disease can mimic other tropical infections or any nonspecific febrile illnesses, as well as noninfectious diseases such as small vessel vasculitides, systemic lupus erythematosus (SLE) or even Cancers. Leptospira pathogen are hard to isolate in pure culture from clinical specimens such as blood, CSF (during first 4-10 days) and urine (after first week).[4] Several laboratory methods can help to prove the diagnosis. In the intial stage, cultures of blood, urine, and even cerebrospinal fluid can grow the spirochete in special media. On the other hand, demonstrating the presence of IgM antibodies or a fourfold rise in IgG titers between acute and convalescent sera by EIA[12,4] is confirmatory diagnostic.

Antimicrobial therapy is mainly indicated for the severe form of leptospirosis, whereas its use is controversial in mild form. Mild leptospirosis is treated with antibiotics such as Doxycycline, Ampicillin, or Amoxicillin. For severe leptospirosis, intravenous penicillin G has been the drug of choice over years, although the third-generation Cephalosporins Cefotaxime and Ceftriaxone have become widely used nowadays. The use of Ceftriaxone and Doxycycline in this case has helped to reduce the severity, hospital stay and provided a better quality of life Alternative regimens are Ampicillin, Amoxicillin, or Erythromycin. In severe cases, Supportive care and careful monitoring and management of renal, hepatic, hematologic, and central nervous system complications are important. Patients should be managed in a monitored setting as their condition can promptly progress to CVS collapse and shock. Renal function should also be evaluated routinely and dialysis should be considered in cases of renal failure. The routine monitoring of CBC and RFT and intiation of 2 hemodialysis has significantly assisted to reduce the severity of renal failure in this case.

CONCLUSION

Leptospirosis remains to be a great burden of infection, and mortality rate is significant which is related to lack of a rapid, reliable diagnostic test and the need for a high degree of clinical suspicion. An accurate and rapid diagnostic test is warranted in the interest of the individual patient, as well as public health. The case of fulminant leptospirosis presented here should serve to alert the health care providers and the general public to the clinical importance of this severe, sometimes fatal, disease. Recognition of fulminant leptospirosis is especially important because early use of antimicrobial agents in this case has helped to reduce its severity and duration as well as lead to a favorable outcome of this potentially lethal condition.[9]

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