

Atypical Presentation of Leptospirosis

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Abstract

Leptospirosis, a disease of protean manifestations occurs sporadically throughout the year with a peak seasonal incidence during the rainy season. We hereby present a case that had clinical features of nephrotic syndrome with massive proteinuria. Leptospirosis was detected on ELISA testing. Patient was cured with antibiotics and diuretics.

Introduction

Leptospirosis is a zoonotic disease caused by pathogenic *Leptospira* of genus *Leptospira*, spread by rodents, mice, voles, dogs, deer, rabbits, hedgehogs, cows, sheep, raccoons, possums, skunks and even certain marine mammals. *Leptospira* comprises of two species- *L. interrogans* and free-living saprophytic *L. biflexa*. On the basis of antigenic composition, they are divided into about 200 serovars.¹ In 1907, Stimson described the micro-organisms in the renal tubules of a patient who died of yellow fever. In its mild form, leptospirosis may present similar to an influenza-like illness with headache and myalgia. Severe leptospirosis is characterized by jaundice, renal dysfunction and hemorrhagic diastases i.e. Weil's Syndrome (human icterohaemorrhagic fever). In this report we describe a 30 year old male patient who presented as nephrotic syndrome with substantial proteinuria and no other classical symptoms of leptospirosis.

Case Report

A 30 year old male sewage worker in the Bombay Municipal Corporation, presented with chief complaints of swelling all over the body and breathlessness since 10 days. He was apparently healthy 15 days back when he showed up with fever without chills but with myalgia for 2 days which subsided on taking medications from his family physician. A few days later he noticed swelling of his feet as he had difficulty in wearing his footwear. The swelling gradually progressed over his entire body in a couple of days. He then felt short of breath for which he was admitted in our hospital. He was a chain smoker and a chronic alcoholic. There was no major illness in the past. He was afebrile with a pulse rate of 90/min. and a respiratory rate of 30/min. His blood pressure was 160/100 mm. of Hg. He had anasarca with pitting edema of the legs. JVP was not raised. There was no icterus, pallor, cyanosis, clubbing and lymphadenopathy. Respiratory system examination revealed reduced breath sounds over the lung bases bilaterally. Other system examinations were unremarkable. The investigations revealed a hemoglobin level of 12.5 gm%, White Blood Cell count of 7400/microlitre (Neutrophils-69, Lymphocytes-27, Eosinophils-03, Monocytes-01) and a platelet count of 1.5 lacs/microlitre. His liver function tests showed a total protein count of 5 g/dl (albumin- 3 g/dl, globulin- 2 g/dl), total bilirubin of 0.5 mg/dl (direct- 0.3 mg/dl and indirect- 0.2 mg/dl) and SGOT and SGPT values of 24 I.U. and 20 I.U. respectively. The value of Serum Creatinine was 0.8 mg/dl & that of Blood Urea Nitrogen (BUN) was 14 mg/dl. His abdominal sonography was normal

but the ultrasonography of the chest revealed bilateral pleural effusion. The right cardiophrenic angle showed about 900cc. pleural fluid with underlying collapse and consolidation of the lung. The left cardiophrenic angle showed an accumulation of around 700 cc. of pleural fluid. The serum calcium level was at 7.4 mg/dl and serum sodium, potassium and chloride levels were at 139 mEq/L, 4 mEq/L and 111 mEq/L respectively. *Leptospira* IgM was positive by ELISA method. Dengue IgM was negative. His lipid profile was normal. A 24-hour urine analysis revealed 4+ proteinuria with a volume of 1500 milliliters. The proteins excreted in urine were 3.84 grams/24 hrs. The Electrocardiogram and 2-D Echo were within normal limits. Arterial blood gases revealed a pH of 7.36, pCO₂ of 28.3 and pO₂ of 87.2. Pleural fluid tapping was done and the fluid analysis showed proteins at 1.7 g% and sugar at 109.5 g%. The total cell count of the fluid was 120 cells/ml. (Polymorphonuclear Neutrophils- 60%, Lymphocytes- 40%).

Discussion

Leptospirosis is a disease with protean clinical manifestations, the incubation period being 2 to 26 days. There are two distinct phases of leptospiral infection in the body – first: septicemia and second: the immune phase. The septicemic phase is a result of vascular injury.² The immune phase is due to the immune complexes deposition leading to endothelial cell damage. Main organs affected are kidneys, liver, heart, lungs and the meninges. In the kidney the main histopathological finding is tubulointerstitial inflammation. Tubular necrosis is also a frequent finding. The glomeruli show mild hyperplasia of mesangial cells.³ In our case the patient presented in the late immune phase, characterized by leptospiruria and correlates with the appearance of IgM antibodies in the serum. In spite of a low clinical score (part A of the Faine's criteria),⁴ leptospirosis was diagnosed on the basis of IgM ELISA positivity. The patient however refused any further investigations like kidney biopsy and immunological tests (immunofluorescence studies for C1q, C3, C4, IgG, IgD etc.) and immunochemical methods for detection of plasma proteins in urine (Radial immunodiffusion in gel, Radioimmunoassay, Immunoenzyme techniques). Following the administration of furosemide and antibiotics (Ceftriaxone) the patient improved dramatically within four to five days as against other varieties of nephrotic syndrome requiring steroids. The pleural effusion was a transudate which disappeared completely after a week. The patient was completely symptom-free within 10 days and we discharged him on the 11th day. Since the patient was improving he refused any further investigations such as complement levels and kidney biopsy. Two weeks after discharge the patient was asymptomatic with normal blood pressure and 1+ proteinuria. He was only on vitamin supplements.

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