Introduction

Leptospirosis is a zoonosis prevalent in Kerala with considerable mortality in farmers and agricultural workers. Causative organism is a Spirochete-Leptospira interrogens (1). After a short incubation period it results in clinical manifestations in two phases, initial leptospiremic phase and later immune phase with multiorgan involvement. Most severe infections with renal and hepatic involvement, thrombocytopenia and bleeding tendency is known as Weil’s syndrome (2). Neurological manifestations are not common in Leptospirosis. Here we present a patient of leptospirosis admitted to our medical ward with dominant neurological symptoms.

Case Report

A 35 year old male, paddy field worker presented with fever, headache and myalgia of 4 days duration. He had a course of amoxicillin prescribed by local consultant; fever subsided, but after 3 days he had progressive holocephalic headache, altered behaviour and one episode of generalised seizures. He was brought to our medical department. There was no history of head injury, seizures, diabetes, stroke and hypertension. On examination patient had mild confusion, bilateral conjunctival suffusion, subconjunctival bleed, mild icterus, blood pressure 110/80 mm Hg, pulse rate 80 beats/min regular. He had a partially healed wound over right big toe. Calf and thigh muscles were tender. Nervous system examination was normal except for bilateral extensor plantar and positive signs of meningeal irritation.

A clinical impression of meningoencephalitis was made from history and examination. Being in an endemic area for leptospirosis and a susceptible patient, possibility of leptospirosis with neurological manifestations was also
considered. He was investigated and findings were Hb-13.4 gm%, TLC 18,200 P80 L20, ESR-110 mm/hr, platelet count 1.3 lakhs, urine R/E-albumin trace, sugar nil, pus cell 30-40/HPF, SGOT 86 IU/L, SGPT 100 IU/L, bilirubin 2.8 mg/dl, blood urea 48 mg/dl, creatinine 1.2 mg/dl. CT Scan brain –plain and contrast was normal, serum IgM (ELISA), leptospira >100 eu and CSF study revealed TLC 210 cells P80 L20, sugar 46 mg/dl, gram stain negative. Routine CSF culture was sterile. CSF antileptospiral antibody IgM Dri-dot test was positive. Patient was treated with crystalline penicillin and chloremphonicol, improved clinically and was discharged on the 14th day.

Discussion

Leptospirosis is prevalent worldwide and is endemic in Alappuzha district of South Kerala. Causative organism Leptospira interorgans a long thin spirochete is usually excreted by reservoir hosts like rats, rodents, cattle, dog etc. in urine. It can survive in contaminated soil and water for weeks. Population groups at risk of infections are farmers, agricultural workers, veterinarians, sewage workers, slaughterhouse employees and workers in fishing industry (3). Organism enters the body via intact mucosa and abraded skin. After entry organism multiplies in blood and tissues. Initial Leptospiremic phase of illness is manifested. Average incubation period is 7-10 days. Leptospirae bind to capillary endothelium and results in vasculitis which is an important cause of multisystem involvement (3).

Kidney and liver are the major organs involved (4,5). Hepatic involvement causes centrilobular necrosis, intrahepatic cholestasis and kupfer cell hyperplasia. It can also cause pulmonary hemorrhage, ARDS (acute respiratory distress syndrome) and myocarditis. Subclinical infections are common. Clinically evident infections can take two forms; anicteric form and Weil’s syndrome (6). Anicteric (90%) of infection presents as acute influenza like illness - fever, headache, myalgia of calf and thighs. Conjunctival congestion and subconjunctival bleed are important diagnostic clues. Person usually becomes asymptomatic in 5-7 days; this is the leptospiremic phase where organism is isolated in all tissues. After an interval of 3 days, symptoms reappear with less fever and myalgia; Neurological symptoms like meningism, iridocyclitis, chorioretinitis are also common in immune phase. In many patients immune phase is not manifested.

Neurological manifestations are described in leptospirosis, about 15% of anicteric forms have symptomatic meningitis. Many have asymptomatic pleocytosis. Signs of meningism disappears by 2-3 days. CSF abnormality resolve slowly. Neurological involvement is purely an immunological reaction as the meningism corresponds to appearance of antibodies in CSF and serum. Common neurological manifestations are aseptic meningitis, myelitis, meningoencephalitis, polyradiculoneuritis and seizures. When 100 consecutive patients were studied with meningoencephalitis 5 of them had high value of IgM antibody for leptospirosis indicating that it is an overlooked cause of meningoencephalitis in endemic areas (7). We suggest that in an endemic area in a susceptible person, leptospirosis should be considered as a cause of meningoencephalitis.

References