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Case report

LEPTOSPIROSIS COMPLICATED WITH MENINGOENCEPHALITIS AND PANCREATITIS - A CASE REPORT

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ABSTRACT

In severe leptospirosis multi organ involvement is common. Pancreatitis and meningo encephalitis are two uncommon manifestations of leptospirosis. Our patient presented with fever, jaundice, altered sensorium and subsequently developed severe pain abdomen. He was finally diagnosed as having icteic leptospirosis complicated with pancreatitis and meningoencephalitis. Simultaneous presence of these two complications in a patient of leptospirosis probably not been documented before.

Key words: Leptospirosis, Pancreatitis, Meningo encephalitis.

INTRODUCTION

Leptospirosis is a zoonosis, and is considered as a major public health problem. The clinical phenotypes of leptospirosis are diverse, ranging from mild, flu-like illness to a severe disease form known as Weil's syndrome. Severe disease is characterized by hepatic, renal and pulmonary involvement, which can lead to death. The disease may also present with some uncommon manifestations. Ascending progressive leg weakness, acalculous cholecystitis, hemorrhagic pneumonitis have been documented as atypical manifestations of leptospirosis in various literatures.[1,2,3] Pancreatitis and meningoencephalitis are uncommon manifestations of leptospirosis. There are few case reports concerning leptospirosis complicated with either pancreatitis or meningoencephalitis. However presence of both pancreatitis and meningoencephalitis in a patient of leptospirosis is extremely rare. The case reported herein describes the concomitant presence of pancreatitis and meningoencephalitis in a patient of complicated leptospirosis.

CASE REPORT

A 32 year male, admitted with a history of moderate grade, intermittent fever for 10 days associated with mild cough. Fever was associated with redness of eyes and pain in muscles, especially calf regions. The patient developed yellowish discolouration of sclera 3 days prior to admission, repeated vomiting, drowsiness and diminution of urine volume since last 1 day. On the day of admission the patient developed intense pain in his upper abdomen. There was no history of any rash or convulsion. Examination revealed a confused patient (Glasgow coma scale: 11), severe pallor, icterus, neck rigidity, positive kernig's sign, pulse rate 114/minute and a blood pressure of 112/74 mm of Hg. Abdominal examination was inconclusive apart from sluggish peristaltic sound and hepatic enlargement. Other

systems were normal. Routine blood investigations showed anemia (Hb 9.4GM/dl) with leucocytosis (15600/ cu.mm.) with neutrophilic predominance. Liver function was dearranged with a total bilirubin 11.7 mg/dl (conjugated 7.6 and unconjugated 4.1 mg/dl), SGOT 168 IU/L and SGPT 71 IU/L. Prothrombin time was normal. Serum urea 110 mg/dl, creatinine 4.1 mg/dl. CSF study showed cell count 36/cu.mm (70% lymphocyte), protein 76 mg/dl and sugar 48 mg/dl. CSF for Herpes simplex virus (HSV) and arbovirus were negative. Serum amylase (750 U/L) and lipase (3720 U/L) were elevated. CT scan of abdomen showed bulky pancreas with peri pancreatic fat stranding, suggestive of acute pancreatitis (Figure 1). Test for malaria parasite, vivax and falciperum antigen and IgM antibody for dengue were also negative. As there was a high index of suspicion of leptospirosis in this clinical setting serum Creatine phosphokinase (CPK) was sent and the value was 968 U/L (normal: 52-336 U/L). Later on leptospirosis was confirmed by a positive IgM anti leptospira antibody. The patient was treated with injection Ceftriaxone (1 GM/day i.v BD) and showed good clinical improvement over next 10 days. Follow up after 1 month showed normalization of liver function test parameters, CPK value with a normal CT scan of abdomen.

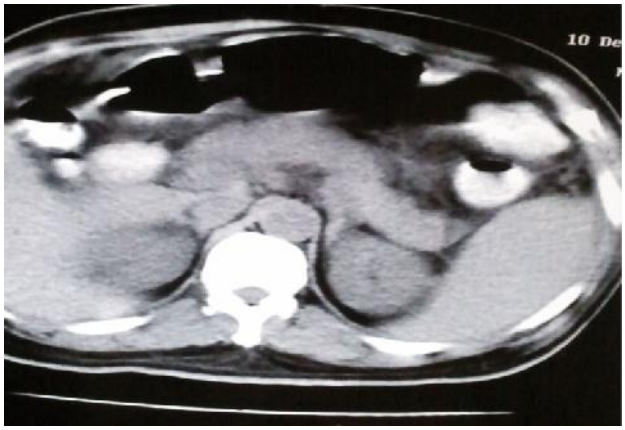

Fig 1: CT scan of abdomen at the level of pancreas showing bulky pancreas with mild peri pancreatic fat stranding suggestive of acute pancreatitis.

DISCUSSION

Leptospirosis is a zoonoses and this disease entity is frequently found in India. The first recognized leptospiral disease was described by Weil in 1886. Causative agent of Weil's disease was isolated in 1915 and named Leptospira icterohaemorrhagiae. Leptospirosis has two clinically recognizable phenotype the anicteric leptospirosis (80-90% of all cases) and icteric leptospirosis.

Apart from hepatic and renal involvement, various other organs are frequently affected in leptospirosis. Pancreatitis is an unusual manifestation of leptospirosis affecting approximately 25% of patients. There are some case reports regarding pancreatitis in leptospirosis.[4,5] In our patient pancreatitis was confirmed by both laboratory investigation and imaging and there was no other common cause of pancreatitis like gall stone, alcoholism or drug intake. Though the exact mechanism of acute pancreatitis in leptospirosis has not been clearly established, vasculitis of small vessels with ischemic injury leading to activation of proteolytic enzymes and pancreatic auto-digestion is the possible mechanism.[6]

Features of meningo encephalitis, as seen in our patient are also uncommon in leptospirosis. Currently there are not enough published data about neurological features in Leptospirosis. In one study it was found that only 5.9% patient can present with signs of meningism. [7] Another prospective study conducted in France among 62 cases of leptospirosis meningo encephalitis was documented in only 2 patients, signifying rarity of this feature. [8] In a study by Matiash VI, et al. on 120 patients with fatal icterohemorrhagic leptospirosis, neurotoxicosis was evident in almost all patients along with microcirculatory disturbances. Meningitis was found in 29.2%, meningoencephalitis in 5% of patients. Morphologic studies showed that focal serous meningitides and meningoencephalitides were significantly more common that they are diagnosed in clinical settings. They tend to develop during the second and third weeks of the course of the illness.[9] Seizures and altered sensorium are most common neurological manifestation of leptospirosis.[10] Alteration of sensorium and signs of meningeal irritation were present in our patient along with lymphocytic pleocytosis in CSF study, establishing a diagnosis of meningoencephalitis.

Concomitant presence of encephalitis and pancreatitis in a patient with leptospirosis is very rare and probably not reported before. It should be emphasized that neuroleptospirosis is an important differential diagnosis of cerebral malaria, dengue and other viral encephalitis, especially in the Indian subcontinent.

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