lesions. Hepatic transaminases were elevated—AST: 147 iu/L (normal 5–40 iu/L) and ALT: 347 iu/L (normal 5–40 iu/L). The other liver function tests were normal. The elevated transaminases normalised in the subsequent two weeks. ESR was 6 mm/h. The mantoux test was negative. Urinalysis, serum urea, electrolytes, creatinine and fasting blood sugar estimations were within normal limits. HIV antibody test, VDRL, mycoplasma antibody titre in serum, serology for Hepatitis A and B viruses and Paul Bunnel tests were negative. Visual evoked potentials were normal.

Titres of leptospira agglutination lysis tests performed on admission, 11 days later and 17 days later were 200, 200 and none respectively, thus confirming a recent leptospira infection. Urine darkground microscopy for leptospira was negative.

He was given a course of intravenous crystalline penicillin and dexamethasone. At the time of discharge, 35 days after admission, he was independently mobile with minimum support and had regained bladder control.

Discussion

Leptospirosis has a worldwide distribution, predominantly involving tropical areas. The clinical spectrum of leptospirosis ranges from a mild, anicteric febrile illness to the more serious Weil syndrome, comprising jaundice, renal dysfunction and bleeding diathesis. Our patient presented with transverse myelitis without clinical features of renal dysfunction without clinical features of renal dysfunction.

Leptospires reach the CSF and brain as early as 48 h after inoculation [2]. However, nervous system involvement is essentially immune mediated. Neurological manifestations in leptospirosis include aseptic meningitis, encephalitis, intracranial bleeding (subarachnoid and extradural haemorrhage), cerebellitis, movement disorders, myelitis, flaccid paraplegia, mononeuritis, autonomic lability and polymyositis [2]. However, it is uncommon for leptospirosis to present as a primary neurological disease without clinical features to suggest leptospirosis (primary neuroleptospirosis).

In a series of 100 hospitalised patients in Manila, Philippines, with aseptic meningoencephalitis, five patients were diagnosed to have leptospirosis [3]. In a series reported from Kerala, India, 40 patients presenting with an acute neurological disease were found to have leptospirosis [1]. None of these patients had renal dysfunction or jaundice at presentation. Papilloedema is a documented ocular manifestation of leptospirosis.

Hence, all the features in our patient were consistent with the diagnosis of primary neuroleptospirosis. Had it not been for the high index of suspicion in view of his occupational hazard of being exposed to stagnant water, and a literature search for an association between leptospirosis and myelitis, the diagnosis of leptospirosis would have been easily overlooked.

The prognosis after primary neuroleptospirosis is generally good, but altered sensorium and seizures herald a worse prognosis [2]. Crystalline penicillin has been shown to reduce severity and duration of illness, even in the immune phase [4]. The role of steroids is controversial.

References

Case report

A 9-year old girl was admitted to Lady Ridgeway Hospital complaining of fever for four days and right-sided abdominal pain for three days. She had been vaccinated against Japanese encephalitis infection. On admission she was flushed, with a positive Hess test. Her pulse rate was 96 per min and blood pressure 100/60 mmHg. The extremities were warm. The liver was tender, enlarged to 2 cm below the right costal margin.

Next day she developed three bouts of coffee ground vomitus. A diagnosis of dengue haemorrhagic fever was made. On the fifth day of illness, rapid deterioration of her sensorium occurred from nonsensical speech to decorticate rigidity (Glasgow coma scale of 5/15), despite a stable cardiovascular status. She also had generalised seizures and was transferred to the intensive care unit (ICU).

At the ICU, the peripheral circulation deteriorated, the packed cell volume increased to 48% and the platelet count dropped to 25×10^9/L. Serum transaminases (SGPT > 110 iu/L, SGOT > 105 iu/L) and prothrombin time (test 40s, control 14s) were elevated. Random blood glucose and serum electrolytes remained normal throughout.

The electroencephalogram (EEG) showed marked slowing of background activity. Computed tomography (CT) scan of the brain showed cerebral oedema but no intracranial haemorrhage. Although the cerebrospinal fluid (CSF) report showed no cells and had a protein content of 28 mg/dL, her altered consciousness associated with fever and generalised convulsions was compatible with encephalitis [2]. She was treated with intravenous mannitol, aciclovir and platelet and plasma infusions. She did not require ventilator support.

The serum taken on 10th day of illness was positive for dengue IgM antibodies. Her IgG antibody titre on the same day was < 20 HAI units for dengue virus subtype 2 which rose to 320 HAI units during the convalescent period (day 24), confirming a primary dengue infection of subtype 2. Her serum IgM was negative for Japanese encephalitis, herpes simplex, and mumps. The CSF IgM was negative for Japanese encephalitis, dengue, herpes simplex and mumps. The nasopharyngeal aspirate was negative for influenza antigens.

She remained in decorticate rigidity for a further 2 days. Thereafter, her level of consciousness gradually improved over the next 5 weeks. She was discharged from the hospital 37 days after the onset of illness with no residual motor or sensory weakness and near normal cognitive functions.

Discussion

Although neurological manifestations associated with dengue infection have received little attention in the past, there have been recently a number of cases reported [2,3]. The incidence has been reported as 1% of all dengue admissions in a study conducted in Vietnam [2]. Out of the 378 cases of suspected central nervous system (CNS) infection reported in this study, 16 cases (4.2%) were due to dengue infection.

Such manifestations have been attributed to CNS invasion by the virus, associated complications or concomitant infection of the CNS by other arboviruses [2]. The complications implicated are hypotension, microvascular or frank intracranial haemorrhage, and encephalopathy due to cerebral oedema, hyponatraemia and associated hepatic failure [2,3,4]. Clinical features of neurological manifestations reported include altered consciousness (Glasgow coma scale < 14), severe headache, neck stiffness, focal neurological signs, generalised convulsions, tense fontanelle, mono- or polynuropathies and transverse myelitis [2-5].

Neurological manifestations are commonly associated with dengue type 3 (DEN-3) infection [2]. Detection of IgM antibodies in the CSF is diagnostic of dengue CNS infection [7]. This is done using an ELISA technique. The CSF may show a pleocytosis with increase in protein level, but it may be acellular.

The outcome of patients with dengue encephalitis admitted to the Centre for Tropical Diseases in Southern Vietnam had a median coma recovery time of 3.5 days (range 1–45). In this group of 16 patients no deaths were reported but six had neurological sequelae on discharge (2). In Sri Lanka since dengue fever is endemic, the possibility of a dengue aetiology in encephalitis should be considered.

References