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Case Report

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Pseudotumor Cerebri Associated with Enteric Fever in a Child: A Case Report

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Abstract

Enteric fever is a common infection of tropical countries that can have a variety of neurological complications. Reported neurological complications are encephalopathy, eningism, spastic paralysis-cerebral origin, convulsions, meningitis, parkinsonian syndrome, sensory motor neuropathy, cerebellar involvement, and schizophrenic psychosis. The patient, who was diagnosed with enteric fever and started treatment, developed headache and blurred vision 5 days after the treatment. Cerebral venous sinus thrombosis secondary to enteric fever and related pseudotumor cerebri were detected in the patient. When additional symptoms develop despite treatment in patients with enteric fever, complications such as thrombosis and pseudotumor cerebri should be considered.

Keywords: Enteric Fever, Complications, Children

Introduction

Salmonella group of organisms causes typhoid fever. The typical symptoms may not be seen in all patients and the disease may have unusual manifestations. Fever, anorexia and headache are common symptoms of the disease. Abdominal pain, diarrhea, constipation and myalgia are other known symptoms [1].

The incidence of neurological manifestations in enteric fever varies widely. Various well-known neurological manifestations include confusional state, encephalopathy, meningism, convulsions, Guillain-Barre'syndrome (GBS), cerebellar ataxia, and isolated cranial nerve palsies [2-4]. In literature, pseudotumour cerebri (PTC) associated with enteric fever have been reported in a few cases report [5-7]. We recently had a case of enteric fever that developed PTC due to cerebral venous sinus thrombosis (CSVT) in the course of the disease.

Case Report

A 12-year-old girl was healthy until four weeks admission, when she began with fever of up to 39 °C, arthralgias, generalized purpuric rash, and a sore throat. She treated with amoxicillin in another hospital. But, her complaints had not improved. The patient was brought to our hospital with these complaints.

On the examination of the patient at the time of admission to the our hospital, she was toxic looking, febrile, and tachypneic. Her blood pressure was normal. She had severe pallor and a mild generalized petechial rash. There was no significant lymphadenopathy or icterus. The liver and spleen were not palpable. There were no cranial nerve palsies, cerebellar signs, or signs of meningeal irritation. The remainder of the physical and neurological examination were unremarkable.

In laboratory studies; hemoglobin of 10.9 g% and total leukocyte counts of 11,280/mm3 (polymorphs 55 %, lymphocytes 45 %). The ESR was 69 mm/hr and CRP was 11 mg/dl (normal 0-0.8 mg/dl). Results of renal and liver function tests were normal. Serum markers for herpes simplex virus, cytomegalovirus, varicella zoster, mumps, rubella, rubeola, Epstein-Barr viruses, Borrelia burgdorferi, and mycoplasma were all negative. Gruber-Widal test done at the time of admission showed antibody titre of 1:640 for both O and H antigens.

The diagnosis of enteric fever was made on the basis of the clinical findings and the laboratory studies. Patient was started on intravenous ceftriaxone 100 mg/kg twice a day. On five day of therapy, her complaints have diminished, but, she developed headache, and blurred vision. She was conscious and had no meningeal signs. The ophthalmologic exam revealed bilateral papilledema. The remainder of the physical and neurological examination were unremarkable. Magnetic resonance imaging of brain was normal.

We thought PTC, and performed lumbar puncture. The cerebrospinal fluid (CSF) showed no pleocytosis and normal protein and glucose concentrations. CSF openning pressure was 40 cm/H2O. We diagnosed PTC and started to acetozolamide treatment at a dosage of 10 mg/kg. Magnetic resonance venography of the brain was performed because of CSVT, which revealed a thrombus in the left transverse sinus. A hypercoagulable workup including anticardiolipin antibodies, protein S and protein C, antithrombin III antibody, prothrombin 20210, and factor V Leiden mutations did not reveal any abnormalities. We thought CSVT would be due to enteric fever. Because of CSVT secondary to enteric fever, she was only treated with ceftriaxone. A follow-up magnetic resonance venography two weeks later demonstrated recanalization of the left transverse sinus. At the time of discharge on 1 month, her neurologic examination was normal, and no recurrent symptoms.

Discussion

Enteric fever is a common infection of tropical countries that can have a variety of neurological complications with an incidence varying from 5% to 35% [8]. In the past 20 years, reports from India, Papua New Guinea, Nigeria, and Indonesia have documented a wide spectrum of neurological complications in cases of enteric fever [9]. The neurological complications of enteric fever include encephalopathy, encephalitis, meningitis, cerebellar ataxia, cerebral abscesses, cerebral edema, GBS, acute transverse myelitis, optic neuritis, CVST, and isolated cranial nerve palsies [2,3]. PTC is very rare and limited to a few cases reports in literature [5-7]. Most of the neurologic complications occur during the second week but it may manifest within the first few days of illness.

PTC is characterized by signs and symptoms of increased intracranial pressure, such as headache and papilledema, absence of an intracranial mass lesion or ventricular dilatation, usually normal findings on neurological examination except for papilledema, and an occasional abducens nerve palsy [10]. PTC may be primary or occur secondary to certain conditions. In secondary cases, some conditions have been identified as: causative agents, including certain medications, endocrine abnormalities, autoimmune disorders, anemias, infectious or postinfectious processes and cranial venous outflow abnormalities [11].

In literature, Bhatt et al. [5] reported 7-year-old boy with enteric fever who presented with PTC. Moodley et al. [6] described an 11-year-old child with PTC due to enteric fever. Balasubramanıan et al. [12] presented a 10-month-old infant girl with PTC because of enteric fever. In another case, Vargas et al. [7] reported 22-year-old patient who developed a clinical picture of PTC in the course of enteric fever. We report a case with enteric fever that was admitted because of PTC due to CSVT.

CVST in children is a rare under recognized disorder due to its wide range of clinical manifestations and diagnosis requiring specialized neuroimaging techniques. Common aetiologic factors of CSVT in children include perinatal insults, dehydration, sepsis, connective tissue disorders, and prothrombotic state like antithrombin III, protein C, or protein S deficiencies [13]. Our patient had a CVST, which was secondary to infection with salmonella species. Salmonella species have been implicated in only a few cases of CVST [14,15].

The exact pathogenesis of neurological complications of enteric fever is unclear. Metabolic disturbances, toxemia, hyperpyrexia and non-specific central nervous system changes such as oedema and hemorrhage have been hypothesized [2,3].

Management of enteric fever with ataxia requires only appropriate antibiotics. The cerebellar symptoms do not warrant any specific treatment (including corticosteroids). However, some have also recommen use of dexamethasone as an adjunct to antibacterial therapy in patients with neurological complication [14]. We used only ceftriaxone, and his symptoms were recovered succesfully.

This case high-lights the fact that enteric fever with PTC due to CSVT can be very important and the diagnosis should be suspected in febrile children presenting with PTC and/or acute neurological symptoms.

Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms.

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Conflicts of Interest

There are no conflicts of interest.

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