

Unusual Neurological Complication Of Typhoid Fever

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Citation

S Vidyasagar, S Nalloor, U Shashikiran, M Prabhu. *Unusual Neurological Complication Of Typhoid Fever*. The Internet Journal of Infectious Diseases. 2004 Volume 4 Number 1.

Abstract

A 36-year-old male with typhoid fever presented with conduction aphasia and parietal lobe dysfunction due to an infarct in the left posterior parieto-temporal cortex documented by CT Scan. This case highlights an unusual neurological complication of typhoid fever hitherto not reported in the literature.

INTRODUCTION

Typhoid fever caused by Salmonella group of organisms has a high prevalence in tropical countries of Asia and Africa. Classically described clinical manifestations are rarely encountered due to early diagnosis and institution of antibiotic therapy. Of all the complications described in typhoid fever, the neuropsychiatric manifestations are the most varied and fascinating for the medical world. Here we present a case of typhoid fever developing cortical infarction with aphasia and parietal lobe dysfunction.

CASE REPORT

A 36-year-old male was admitted to the hospital with 20 days history of fever and headache. He was receiving treatment before admission to our hospital as typhoid fever based on positive Widal test (initial titre 1:80 later 1:320) with ciprofloxacin and gentamycin. On the day of admission, there was history of sudden onset of giddiness with altered sensorium for one hour. Following this, the patient became responsive but was unable to communicate freely due to reduced word output for which he was brought to the hospital.

Clinical examination showed an anxious, febrile (39.6° C) patient with mild splenomegaly. The patient was conscious, well oriented, with well-preserved comprehension for spoken words, but had severely impaired naming and repetition. No focal motor deficit was present. All primary modalities of sensations were intact. However, tactile localization and two-point discrimination were impaired on right half of the body. Parietal lobe dysfunction was documented by presence of dyscalculia, ideational apraxia with inattention to tactile and auditory stimulation. Right to left disorientation and finger anomia were also present.

Reading and writing could not be tested in detail, as the patient was not literate, and could write only his name. No visual field defect was documented.

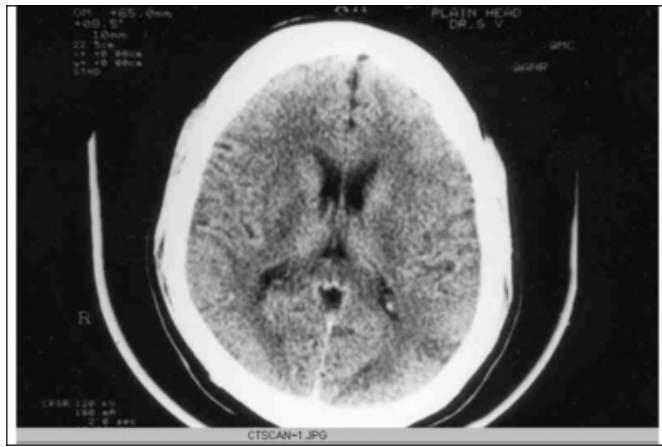
Haemoglobin concentration was 15.3g/dL, white blood cell count was 6900cells/mm³, platelet count was 2,18,000/mm³ and ESR was 20mm in the first hour. Biochemical parameters were normal. Chest X-ray was normal. Mantoux test and serology for HIV were negative. Malaria and urinary tract infections were ruled out.

A diagnosis of typhoid fever was considered in view of Widal test being strongly positive (1:640 titer after admission for both somatic and flagellar antigen). A rising titer was also documented. Blood cultures were sterile, probably due to prior antibiotic therapy.

CT scan of the brain revealed a hypo-dense lesion involving the left posterior parieto-temporal cortex suggestive of an early infarct (Figure 1). Lumbar puncture showed normal opening pressure and CSF analysis revealed no abnormality.

Figure 1

Figure 1: Plain CT scan study of the brain showing a hypo-dense lesion involving the left posterior parieto-temporal cortex suggestive of an early infarct.



The patient received ceftriaxone and gentamycin following which he became afebrile. He was discharged with aspirin 325 mg per day. Neurological assessment at follow-up 15 days later showed markedly improved parietal lobe functions with persistence of language deficit. 6 months after discharge from the hospital, patient was asymptomatic and speech was normal.

DISCUSSION

Neurological complications in typhoid fever are not uncommon and range from 5 to 35 % in various studies. ¹ Of these typhoid encephalopathy is the most common (9.6 to 57%) followed by meningismus (5 to 17%). ^{1,2} convulsions (1.7 to 40%), spasticity (3.1%), Focal neurological deficit (0.5%) and Meningitis (0.2%) are frequently described. ^{1,2,3} Other rare complications like Parkinson's syndrome, Motor-neuron disease, Transient amnesia, Symmetrical sensory-motor neuropathy, schizophreniform psychosis and cerebellar involvement are also described. Aphasia as a complication of typhoid fever is described in 2 to 7.4% in various studies. ² Case-reports documenting this rare complication have also been published. ^{1,4,5,6} However, focal parietal lobe involvement has not been documented in literature (Medline search).

Most of the neurological complications described were seen during the course of illness, at height of fever or during defervescence. Some occurred during convalescence like neuropathy, amnesia and psychosis. Others like motor neuron disease, scholastic deterioration occurred well after recovery. ^{1,3} In our patient, the neurological deficit occurred during the course of the illness after one week of fever.

The mechanisms responsible for the neurological manifestations of typhoid fever have been variously described. Possible mechanisms implicated are hyperpyrexia (>43°C), fluid and electrolyte disturbances, typhoid neurotoxin, vasculitis with peri-vascular cuffing, autoimmune mechanism, pressure effect on blood vessels resulting in cerebral infarction and acute disseminated encephalomyelitis. ^{1,3,7} CSF analysis in most of these cases revealed no abnormality except for an elevated opening pressure. CT scan wherever done has failed to document any lesion. Typhoid neurotoxin causing damage in the speech area has been put forth as the most likely explanation for aphasia. ³ In our case, the patient had sudden onset of decreased word output with documented parieto-temporal lobe infarct on CT scan most probably pointing to arteritis as a cause for his neurological deficits.

Since our patient had well preserved comprehension and fluency, but severely impaired repetition and naming, we made a diagnosis of conduction aphasia, probably due to posterior parieto-temporal infarct as seen on the CT scan. Common causes of lesion in this region include embolic stroke, neoplasms or trauma. So far no cases of enteric fever with conduction aphasia and parietal lobe dysfunction have been reported. Most of the earlier reported cases were of motor aphasia. ^{4,5,6}

The prognosis of neurological deficits in enteric fever is usually good. In most of the cases the recovery is slow and complete, but in some cases the deficit may persist for long. ^{1,3} Our patient showed gradual improvement in his parietal lobe functions such as sensory and auditory inattention but nominal aphasia and impaired repetition were persisting even after he became afebrile after treatment with antibiotics.

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