## **Case Report**

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# Acute sensorineural hearing loss in a child with typhoid fever

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#### **ABSTRACT**

Typhoid fever is a systemic febrile disease caused by Salmonella typhi and may lead to involvement of virtually any organ system in the body. Cochleovestibular involvement resulting in acute sensorineural hearing loss (SNHL) has been reported as a rare complication and may result in permanent deafness. We describe here a child who developed moderate but reversible SNHL associated with typhoid fever.

Keywords: Typhoid fever, Acute sensorineural hearing loss, Deafness, Child

#### INTRODUCTION

Infection with Salmonella typhi may result in diffuse organ involvement. Although most infections manifest with classical clinical features of enteric fever, atypical presentations have been increasingly observed over past few decades probably due to prior antibiotic use, incomplete treatment with common antibiotics and multidrug resistance in endemic areas. Of all the atypical features of enteric fever, cochleovestibular involvement resulting in sensorineural hearing loss (SNHL) has been reported rarely in children as well as adults. Most patients show complete recovery after effective antibiotics use, but permanent loss of hearing has also been reported.

It is important to recognize SNHL as a complication of enteric fever to prevent delays in clinical suspicion of disease that often happens when patients present with atypical manifestations.<sup>1</sup>

In this communication, we describe a child who presented with fever and acute severe hearing loss which reversed completely after treatment of enteric fever.

#### **CASE REPORT**

This 8 year old boy had moderate to high grade fever for 2 weeks, and developed headache with decreased hearing a day prior to presentation. The hearing loss was bilateral and sudden in onset. There was no history of ear discharge, exposure to ototoxic medications, trauma, altered sensorium or facial deviation. There was no associated vertigo, ear discharge or tinnitus. He had received cefixime in inadequate doses. On physical examination, he appeared pale and toxic and axillary temperature, pulse rate, respiratory rate and blood pressure were recorded as 103°F, 123 per min, 28 per min and 100/70 mmHg respectively. There was no eschar, icterus or lymphadenopathy. Abdominal examination revealed hepatosplenomegaly with liver palpable 4 cm below right costal margin (liver span 11 cm) and spleen palpable 4 cm below left costal margin. Rest of the systemic examination was normal. In cranial nerves examination, test for glossopharyngeal nerves were impaired bilaterally. Tuning fork test could not be performed as child was unable to comprehend instructions. Rest of the cranial nerves functions were within normal limits. There was no nystagmus. Otoscopy showed normal tympanic membranes. Based on the

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history and clinical examination, enteric fever, scrub typhus and viral infections were considered as the probable initial diagnoses.

Investigations revealed haemoglobin of 7.5 g/dL, total leukocyte count of 6000 (neutrophils 70%, lymphocyte 27%, monocytes 2%, eosinophils 1%) and platelet count of 120000/mm<sup>3</sup>. Serum sodium, potassium, urea and creatinine were 133 mEq/L (normal 135-145 mEq/L), 5.0 mEq/L (normal 3.5-5.0 mEq/L), 20 mg/dL (normal 5-18 mg/dL) and 0.5 mg/dL (normal 0.3-0.7 mg/dL) respectively. Liver function tests showed serum alanine transferase 126 U/L, serum aspartate 229 U/L, alkaline phosphatase 284 U/L, serum albumin 2.0 gm/dL and total serum bilirubin of 0.7 mg/dL. C-reactive protein was 46.8 mg/L (normal range, 0.052-3.2 mg/L). Chest radiograph did not show any abnormality. Widal test showed TH titre of 1:320 and TO titre <1:40. Blood culture was positive for Salmonella typhi which was sensitive to ceftriaxone, ampicillin and chloramphenicol. Serological tests for scrub typhus and herpes simplex virus were negative. Multiple peripheral smear examinations for malarial parasites as well as rapid card test (QDx Malaria Pv/Pf, Nicholas Piramal India) were negative.

On pure tone audiometry (PTA) hearing loss was found to be 57/62 db in right ear and 57/57 db in left ear, indicating bilateral moderate SNHL (Figure 1a). Tone decay test was normal, thereby ruling out any retrocochlear pathology. Child was started on intravenous ceftriaxone, oral acyclovir and prednisolone. Acyclovir was omitted after culture showed positivity for Salmonella typhi. Repeat PTA 7 days after starting treatment showed improvement (42/45 db in right ear and 42/42 db in left ear (Figure 1b). Improvement in general well-being and appetite occurred over the first few days and he became afebrile by day 8 of hospitalization. Serial hematological and biochemical parameters also showed consistent improvement. Child was discharged on oral cefixime to complete the antibiotics course for 2 weeks. Audiogram repeated 1 month later showed essentially normal hearing (Figure 1c).

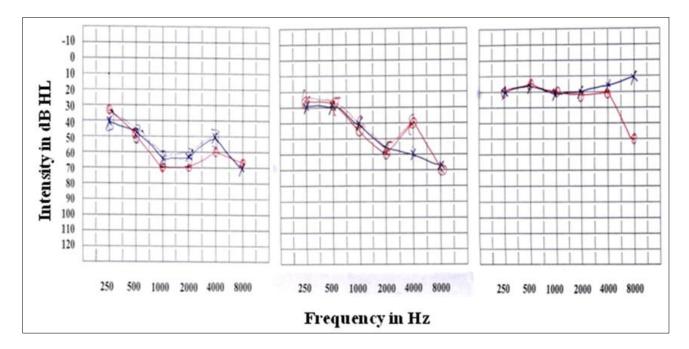


Figure 1: Pure tone audiograms at presentation (a), after 1 week (b) and 1 month (c).

## **DISCUSSION**

Sudden SNHL is a rare manifestation of typhoid fever.<sup>1,2</sup> Only a few cases have been described in children.<sup>1-4</sup> The hearing loss usually occurs from second to third week of illness, and may occasionally become permanent.<sup>2,3</sup> In our patient, hearing loss appeared on day 13, started showing improvement from day 21, and recovered completely by day 44 of illness. Similar hearing loss is reported more commonly in our country in patients with scrub typhus,

which we excluded by serological tests.<sup>5</sup> Other causes of sudden SNHL, such as viral infection of the labyrinth or cochlear nerve, vascular insult, perilymphatic hypoxia, intralabyrinthine membrane rupture, inflammatory and metabolic causes, and immune-mediated inner ear disease are usually reported in adults.<sup>6</sup>

The exact mechanism of hearing loss in typhoid fever is not established. In a study of six cases of pathologically confirmed cochleovestibular lesions due to typhoid fever, host susceptibility, endotoxins, arteritis, and ischemia were the factors believed to have resulted in development of these lesions and the consequent hearing loss.<sup>2</sup> Cochleo-vestibulitis may also occur secondary to endotoxinemia and cytokine mediated damage.<sup>7</sup>

The treatment of SNHL in typhoid fever involves effective antibiotic therapy for the infection. Corticosteroids have a limited beneficial effect on hearing recovery and their role is debated. Oral, intratympanic or pulse steroids are commonly used in idiopathic SNHL which is presumed to be due to subclinical viral labyrinthitis in most cases. Since the etiology of SNHL was unclear initially, we had initiated oral prednisolone in our patient presuming a viral infection of the labyrinth or cochlear nerve. It is possible that the hearing recovery was entirely due to the effective antibiotic use, but the contribution of steroids to suppression of cochleovestibular inflammation and hastening recovery of SNHL cannot be completely excluded in our patient.

In conclusion, we report a rare complication of SNHL in a child with typhoid fever. This communication should serve to alert the treating physicians to consider typhoid fever in the differential diagnosis of a child with fever and hearing loss, and initiate antibiotic therapy promptly.

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