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CASE REPORT

Acute Cerebellitis and Bilateral Sensorineural Hearing Loss Following Typhoid Fever

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ABSTRACT

Background: Typhoid fever, caused by *Salmonella enterica* serovar Typhi, is a systemic infection primarily affecting the gastrointestinal tract. Common complications include internal bleeding, intestinal perforation, cholecystitis, bronchitis, and osteomyelitis. Rarely, neurological manifestations such as cerebellitis and bilateral sensorineural hearing loss (SNHL) have been described.[1,2] We report a rare case of a 20-year-old female with a prior history of idiopathic intracranial hypertension (IIH) who developed acute ataxia and bilateral SNHL following typhoid fever.

Case Presentation: A 20-year-old woman presented with fever, diarrhea, and vomiting, followed by progressive imbalance and hearing loss. She was diagnosed with typhoid fever and started on oral antibiotics. Despite therapy, her neurological symptoms worsened. MRI and CSF studies excluded other etiologies, and audiometry confirmed moderate to severe bilateral SNHL. She received intravenous antibiotics and intravenous immunoglobulin (IVIG), which improved her balance, though hearing loss persisted with only partial audiometric recovery.

Conclusion: This case illustrates an uncommon post-infectious complication of typhoid fever and highlights the importance of early recognition and prompt management of neurological sequelae. Clinicians should maintain vigilance for atypical manifestations, particularly in endemic regions.

KEYWORDS

• Intravenous • Manifestations • Typhoid fever • Bilateral • SNHL • Typhoid Cerebellitis • IVIG

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INTRODUCTION

Typhoid fever, caused by *S. Typhi*, is a systemic infection that primarily affects the gastrointestinal tract. While typical complications include gastrointestinal bleeding, perforation, and gallbladder involvement, neurological complications, though rare, are increasingly recognized.[1] These may include encephalopathy, meningitis, and, less commonly, cerebellitis.[2] Cerebellitis presents with ataxia, dysarthria, and nystagmus and is an unusual but potentially disabling complication.[2,3] Similarly, sensorineural hearing loss due to typhoid fever is scarcely documented, with possible mechanisms including inflammatory injury to the cochlear nerve or labyrinthine structures.[4] We present a rare case of acute cerebellitis with bilateral SNHL in a young woman with typhoid fever, emphasizing the role of clinical vigilance and early intervention.[5]

Case

A 20-year-old woman presented with ataxia and bilateral hearing loss for 4 days. She had a history of idiopathic intracranial hypertension (IIH), diagnosed 2 years earlier, managed with acetazolamide. Sixteen days before presentation, she developed watery diarrhea, vomiting, and fever with rigors. Typhoid fever was diagnosed based on a positive Widal test and Typhi Dot IgM, and oral cefixime was initiated on day 7 of illness. Despite treatment, her symptoms persisted.

By day 12, she developed progressive unsteadiness requiring support. On day 14, bilateral hearing loss appeared and worsened. She presented to the emergency department on day 16 due to worsening neurological symptoms.

On examination, she was conscious and oriented. Vitals were stable. Neurological exam showed horizontal gaze nystagmus, scanning speech, and bilateral cerebellar signs with ataxic gait. Motor power and reflexes were preserved.

ECG revealed sinus tachycardia with mild global ST depression. Blood tests showed mild anemia, respiratory alkalosis with mild metabolic acidosis, and hypokalemia (K+: 2.0 mEq/L, Na+: 125 mEq/L), likely due to acetazolamide use. IV antibiotics and potassium supplementation were started.

MRI brain with contrast showed bilateral optic atrophy and left transverse sinus hypoplasia but no cerebellar lesion or hydrocephalus. Lumbar puncture revealed normal opening pressure and biochemistry. CT temporal bone was normal, while pure tone audiometry showed moderate to severe bilateral SNHL. Blood culture and meningitis/encephalitis panel were negative. Repeat Widal was strongly positive, and gastrointestinal BioFire PCR detected *Salmonella* and enteropathogenic *E. coli*.

Other infections (malaria, rickettsial disease) were excluded. Considering a diagnosis of typhoid cerebellitis, IVIG was initiated on hospital day 3.

Her ataxia improved by day 5 and diarrhea subsided, though low-grade fever persisted. After completing IV antibiotics, she stabilized and was discharged. At follow-up, hearing loss persisted but audiometry showed partial improvement.

DISCUSSION

Acute ataxia and hearing loss are uncommon presentations that warrant urgent evaluation because of their broad differential diagnoses, which include vestibular neuritis, cerebellar stroke, multiple sclerosis, autoimmune inner ear disease, and vestibular schwannoma.[6] Infectious causes such as labyrinthitis, meningitis, and viral encephalitis must also be considered. Careful neurological examination, MRI, and audiometry are crucial in differentiating central from peripheral etiologies and guiding management.[6]

Neurological involvement in typhoid fever, often referred to as "typhoid encephalopathy," is a recognized but rare complication, with manifestations ranging from confusion to cerebellitis.[1,7] The pathogenesis is thought to involve direct bacterial invasion, immune-mediated inflammatory injury, or metabolic derangements.[7,8]

In our patient, the absence of structural lesions on MRI with clinical cerebellar signs suggests an immune-mediated cerebellitis. Bilateral SNHL is exceptionally rare in typhoid fever, with only isolated Case Report attributing it to inflammatory or vascular mechanisms affecting the cochlea.[4,9] Most reported cases of typhoid cerebellitis have been managed with

intravenous antibiotics such as ceftriaxone, and corticosteroids are sometimes added to hasten neurological recovery.[2,7,10] In our case, IVIG was chosen in view of a suspected immune-mediated mechanism, with good response in terms of balance recovery, though hearing recovery remained incomplete.

CONCLUSION

This case highlights the importance of recognizing rare neurological manifestations of typhoid fever, such as cerebellitis and SNHL, which can significantly increase morbidity. Early diagnosis, antimicrobial therapy, and consideration of immunomodulatory treatment (steroids or IVIG) are critical for improving outcomes. Further research is required to clarify mechanisms and establish evidence-based management strategies.

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