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Salmonella Typhi-Associated Peripheral Facial Nerve Palsy in a Child: A Case Report

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Abstract

Bell's palsy is the most common cause of acute peripheral facial nerve paralysis and is usually idiopathic; however, infectious etiologies may present with a similar clinical picture. We report the case of a previously healthy seven-year-old boy who was hospitalized with fever, profuse diarrhea, and recurrent vomiting, and who developed acute left-sided lower motor neuron facial nerve palsy during admission. Blood culture grew *Salmonella enterica* serovar Typhi. Cerebrospinal fluid analysis, neuroimaging, otologic examination, and targeted infectious evaluation did not identify an alternative cause. The patient was treated with intravenous ceftriaxone, corticosteroids, and ocular protective care, with gradual improvement and complete recovery of facial nerve function within six weeks. This case highlights a rare association between typhoid fever and peripheral facial nerve palsy and emphasizes the importance of considering enteric fever in children presenting with acute facial palsy preceded by febrile gastrointestinal illness.

Categories: Neurology, Pediatrics, Infectious Disease
Keywords: bell's palsy, pediatrics cranial neuropathy, salmonella typhi, typhoid fever, unilateral peripheral facial nerve palsy

Introduction

Bell's palsy is the most common cause of acute peripheral facial nerve paralysis, presenting as sudden unilateral facial weakness [1]. Although less frequent in children than in adults, with reported annual incidences of approximately 40 cases per 100,000 adults and 6.1 cases per 100,000 children aged 1-15 years, it remains an important pediatric diagnosis [1]. Most cases are idiopathic; however, facial nerve palsy may occur secondary to congenital or acquired conditions, including infections, trauma, neoplasms, surgery, metabolic or autoimmune disorders, and drug-related adverse effects. Infectious causes are particularly relevant, as improvement after antimicrobial therapy in selected cases suggests a possible bacterial role in some patients. Therefore, careful evaluation and follow-up are essential to identify underlying causes and monitor recovery [1-3].

Salmonella species are Gram-negative bacilli causing salmonellosis, including enteric fever and non-typhoidal gastroenteritis. Enteric fever, mainly caused by *Salmonella enterica* serovars Typhi and Paratyphi, remains a major global burden, with an estimated 20 million cases and 200,000 deaths annually. It commonly presents with sustained fever, headache, malaise, anorexia, abdominal pain, constipation, abdominal distension, nausea, and vomiting, while severe cases may develop gastrointestinal bleeding, intestinal perforation, or systemic complications [4,5].

Neurological complications of typhoid fever are uncommon but recognized, including encephalopathy, meningitis, cerebellitis, myelitis, peripheral neuropathy, and cranial neuropathies. In a large Nigerian study of nearly 1,000 hospitalized patients, infranuclear facial nerve palsy occurred in approximately 0.5% of cases, emphasizing the rarity of this manifestation. Proposed mechanisms include direct bacterial invasion, immune-mediated injury, neurotoxin production, and systemic inflammatory responses. In particular, infection-related immune activation may trigger inflammation of the facial nerve, while associated edema within the narrow facial canal may contribute to nerve compression and impaired conduction. These mechanisms could explain the development of peripheral facial nerve palsy in the absence of direct central nervous system infection. However, the precise pathophysiological mechanism remains uncertain. Given this rare association, reporting cases of *Salmonella*-related facial nerve palsy may help expand knowledge of the neurological spectrum of salmonellosis and improve clinician awareness of this unusual complication [6].

Case Presentation

A previously healthy seven-year-old boy, born at term by uncomplicated vaginal delivery, was admitted to the pediatric department with a one-week history of fever, profuse diarrhea, and recurrent vomiting. On the first day of hospitalization, his mother noted the sudden onset of left-sided facial weakness. There was no history of facial trauma, recent vaccination, tick exposure, previous neurological illness, or contact with sick individuals. His family history was noncontributory.

On examination, the patient was mildly dehydrated but alert and hemodynamically stable. His temperature was 38°C, heart rate 100 beats/min, blood pressure 110/70 mmHg, and oxygen saturation 98% on room air. Neurological examination demonstrated left-sided lower motor neuron facial nerve palsy, with facial asymmetry at rest, drooping of the left side of the mouth, loss of the left nasolabial fold, and incomplete closure of the left eye. The palsy was classified as House-Brackmann grade IV [7].

Examination of the external auditory canals and auricles revealed no vesicular lesions, and there was no postauricular swelling or tenderness over the mastoid or parotid regions. Otoscopic examination was normal bilaterally, with no evidence of otitis media or other ear disease. No oral lesions suggestive of herpes simplex virus or varicella-zoster virus infection were present. There was no skin rash, lymphadenopathy, or meningeal irritation, and Kernig and Brudzinski signs were negative. The remainder of the neurological, cardiovascular, respiratory, and systemic examinations was unremarkable.

A comprehensive diagnostic evaluation was performed to identify a secondary cause of the facial nerve palsy. Investigations included a complete blood count, erythrocyte sedimentation rate, C-reactive protein, renal and hepatic function tests, serum electrolytes, urinalysis, urine culture, stool analysis, and stool culture. Given the acute neurological presentation, lumbar puncture was performed to evaluate for central nervous system infection. Cerebrospinal fluid (CSF) analysis revealed a white blood cell count of 0 cells/µL, a mildly elevated protein concentration of 55 mg/dL, and a glucose level of 65 mg/dL. The absence of pleocytosis and the preserved CSF glucose concentration, despite mild protein elevation, did not suggest typical bacterial meningitis and supported the diagnosis of an isolated peripheral facial neuropathy in the appropriate clinical context. Additional infectious testing included serology for Epstein-Barr virus, cytomegalovirus, herpes simplex virus, *Brucella* species, and SARS-CoV-2, together with Widal testing, which were negative. The laboratory findings are summarized in Table *1*.

Test	Result	Normal Range
Hemoglobin	13 g/dL	13-18 g/dL
WBC count	12.08 × 10^3/µL	4-11 × 10^3/µL
Neutrophils	49%	40-75%
Lymphocytes	39%	20-45%
ESR	74 mm/hr	0-20 mm/hr
SGPT (ALT)	21 U/L	7-56 U/L
CRP	79.38 mg/L	<5 mg/L
CSF WBC	0	0-5 (cells/µL)
CSF protein	55 mg/dL	50-70 mg/dL
CSF glucose	60 mg/dL	50-75 mg/dL
Blood culture	*Salmonella Typhi* (positive)	No growth
Widal typhoid O	1/320	<1/320
Widal typhoid H	<1/320	<1/320
Paratyphoid A	<1/320	<1/320
Paratyphoid B	<1/320	<1/320
EBV IgM	0.3 (negative)	Negative
CMV IgM	0.35 (negative)	Negative
HSV	Negative	Negative
Brucella antibodies	Negative	Negative
Indirect Coombs test	Negative	Negative
Urine analysis	Few WBCs, glucose negative, bilirubin negative	No WBCs, glucose negative, bilirubin negative
Urine culture	No growth	No growth
Stool analysis	No parasites detected	No parasites
Stool culture	Negative	Negative

TABLE 1: Laboratory investigation results

WBC: white blood cell; ESR: erythrocyte sedimentation rate; SGPT: serum glutamic-pyruvic transaminase; ALT: alanine aminotransferase; CRP: C-reactive protein; CSF: cerebrospinal fluid; EBV: Epstein-Barr virus; IgM: immunoglobulin M; CMV: cytomegalovirus; HSV: herpes simplex virus

Neuroimaging was performed to exclude structural and central nervous system pathology. Contrast-enhanced brain MRI, including dedicated facial nerve sequences, was unremarkable. CT of the chest, abdomen, and pelvis showed no evidence of malignancy, lymphadenopathy, or other significant abnormalities, and echocardiography was normal. Blood culture subsequently grew *S. enterica* serovar Typhi. With no alternative infectious, autoimmune, traumatic, or structural etiology identified, a diagnosis of *Salmonella*-associated peripheral facial nerve palsy was established.

The patient received intravenous fluids, intravenous ceftriaxone at a dose of 1 g once daily (50 mg/kg/dose) for 14 days, and oral prednisone at a dose of 20 mg twice daily for 10 days, followed by 20 mg once daily for three days and then 10 mg once daily for an additional three days before discontinuation. Supportive treatment included antipyretics and ocular protection with frequent lubrication and nighttime eye patching. Gastrointestinal symptoms improved progressively, and no worsening of the neurological deficit occurred during hospitalization.

Initial improvement in facial nerve weakness was observed two days after treatment initiation, accompanied by progressive improvement in gastrointestinal symptoms. The patient was discharged after four days with

continued corticosteroid tapering and ocular care. At the two-week follow-up, facial weakness had improved substantially, and complete recovery without residual neurological deficits was documented six weeks after symptom onset.

Discussion

Typhoid fever, caused by *S. enterica* serovar Typhi, affects an estimated 22 million people annually and accounts for approximately 216,000 deaths worldwide. Clinical manifestations commonly include fever, headache, abdominal pain, vomiting, and diarrhea, while severe disease may be complicated by gastrointestinal, cardiovascular, pulmonary, skeletal, or neurological involvement. Neurological complications occur in approximately 3-5% of cases and include delirium, meningitis, aphasia, ataxia, seizures, peripheral neuritis, and cranial nerve palsies [5]. These complications are thought to result primarily from immune-mediated nerve injury, systemic inflammation, and toxin-related effects rather than direct bacterial invasion. Similar post-infectious mechanisms have been proposed in other infection-associated cranial neuropathies [8].

The Widal test has long been used as an inexpensive and widely available serological test for suspected enteric fever, particularly in resource-limited settings. However, because it detects antibodies against *Salmonella* O and H antigens rather than the organism itself, it cannot confirm active infection. Its diagnostic accuracy is further limited by prior infection or vaccination, background antibody levels in endemic regions, cross-reactivity with other infections, antibiotic exposure, and variation in local cut-off values [9,10]. Consequently, the CDC does not recommend Widal testing for routine diagnosis, and WHO defines confirmed typhoid fever by culture or molecular detection of *S. Typhi* from a normally sterile site [11,12]. Studies from endemic regions have also demonstrated poor specificity and positive predictive value compared with blood culture [13,14]. Accordingly, in our patient, the diagnosis was established by positive blood culture, while the Widal test was considered supportive rather than diagnostic.

Cranial nerve palsies are rare neurological manifestations of typhoid fever. Reported cases include palatal palsy, abducens nerve palsy, and lower motor neuron facial nerve palsy. Joshi et al. described three pediatric patients with culture-confirmed *S. Typhi* infection who developed cranial neuropathies, including two cases of palatal palsy and one of abducens nerve palsy [15]. Suri et al. reported an 11-year-old child with typhoid meningitis and concomitant lower motor neuron facial nerve palsy confirmed by positive blood and CSF cultures [16]. In adults, Gkrania-Klotsas et al. described abducens nerve palsy in a traveler with typhoid fever, while Ramapriya and Vetriselvan recently reported lower motor neuron facial nerve palsy associated with *Salmonella* infection [17,18]. Similar to these reports, our patient developed peripheral facial nerve palsy in temporal association with culture-confirmed *S. Typhi* infection after exclusion of alternative etiologies. As in previously reported cases, neurological recovery was complete following appropriate antimicrobial therapy and supportive care, suggesting a favorable prognosis for this rare complication. The reported cases are summarized in Table *2*.

Reference (Year)	Age	Sex	Presentation	Type of Weakness	Diagnosis
Present case	Seven years	Male	Fever, diarrhea, vomiting	Left peripheral facial nerve palsy (CN VII)	Blood culture positive for *Salmonella Typhi*
Joshi et al. (2011) [15]	Four years	Male	Fever and gastrointestinal symptoms of typhoid fever	Palatal palsy	Blood culture positive for *S. Typhi*
Joshi et al. (2011) [15]	Five years	Female	Fever and gastrointestinal symptoms of typhoid fever	Palatal palsy	Blood culture positive for *S. Typhi*
Joshi et al. (2011) [15]	11 years	Female	Fever and gastrointestinal symptoms of typhoid fever	Abducens (VI) nerve palsy	Blood culture positive for *S. Typhi*
Gkrania-Klotsas et al. (2009) [17]	25 years	Female	Diarrhea and fever (38.7°C) after travel; left hand cyanosis; no meningeal signs	Abducens (VI) nerve palsy	Serology positive for *Salmonella enterica* serovar Typhi; antibiotic sensitivity to ampicillin and chloramphenicol
Suri et al. (1992) [16]	11 years	Male	Fever (39.5°C), vomiting, altered sensorium, positive meningeal signs	Left LMN facial nerve (VII) palsy	Blood and CSF culture positive for *S. Typhi*
Ramapriya and Vetriselvan (2025) [18]	32 years	Male	Fever with abdominal pain for one week; at the end of the third week, developed mouth deviation to the left and impaired right eye closure	Right LMN facial nerve (VII) palsy	*Salmonella* infection confirmed by blood culture

TABLE 2: Reported cases of cranial nerve palsies associated with typhoid fever

CN: cranial nerve; LMN: lower motor neuron; CSF: cerebrospinal fluid

Facial nerve palsy is the most common acute mononeuropathy and has a broad differential diagnosis. Although Bell's palsy is the most common cause, secondary etiologies should be considered, particularly in children. Infectious causes include bacterial pathogens such as *Borrelia burgdorferi*, *Mycoplasma pneumoniae*, and *Haemophilus influenzae*, and viral infections including herpes zoster virus, Epstein-Barr virus, cytomegalovirus, adenovirus, human herpesvirus 6, coxsackievirus, and SARS-CoV-2. Other causes include otitis media, mastoiditis, inflammatory disorders (e.g., Kawasaki disease and Guillain-Barré syndrome), neoplasms, trauma, metabolic disorders, and autoimmune diseases [19,20]. In children, infections are the most common identifiable cause of non-idiopathic facial nerve palsy, followed by trauma and congenital disorders, whereas Bell's palsy remains the most frequent overall diagnosis. Prognosis is generally favorable, with most pediatric patients achieving complete recovery [1,19,21].

Facial nerve dysfunction in our patient was graded using the House-Brackmann classification and was classified as grade IV, indicating moderately severe dysfunction [7]. Bell's palsy is diagnosed clinically and typically presents as an acute unilateral lower motor neuron facial palsy involving both the upper and lower facial muscles. It may be accompanied by otalgia, mastoid discomfort, hyperacusis, neck stiffness, or altered facial sensation [20].

In this patient, the close temporal relationship between culture-confirmed *Salmonella* infection and the onset of peripheral facial nerve palsy, together with exclusion of alternative infectious, structural, autoimmune, and traumatic causes, supports a probable *Salmonella*-associated cranial neuropathy. The preceding febrile gastrointestinal illness also favors a secondary infectious mechanism rather than idiopathic Bell's palsy. However, although these findings suggest a possible association, a direct causal relationship cannot be definitively established.

Management depends on the underlying cause and severity. When a specific etiology is identified, treatment should target that condition. In children with Bell's palsy, oral corticosteroids are generally recommended within 72 hours of symptom onset, commonly as prednisone 1-2 mg/kg/day for 10 days followed by tapering. Ocular protection, including frequent lubrication and nighttime eyelid closure, is essential to prevent corneal injury [1,19,21].

Conclusions

Acute peripheral facial nerve palsy in children has a broad differential diagnosis and requires careful evaluation for secondary causes. Although idiopathic Bell's palsy remains common, infectious etiologies should be considered when facial weakness is preceded by fever and gastrointestinal symptoms. This case highlights a possible association between *S. Typhi* infection and peripheral facial nerve palsy, particularly in endemic settings. Although complete neurological recovery was observed following antimicrobial and corticosteroid therapy, the individual contribution of each treatment remains uncertain. Prompt recognition, appropriate antimicrobial therapy, ocular protection, and close follow-up may be associated with favorable neurological recovery.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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