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Case Report

A rare complication of systemic *Salmonella* infection in a young boy: a case report

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ABSTRACT

Salmonella spondylodiscitis is an uncommon extraintestinal manifestation of enteric fever and may closely mimic tubercular or pyogenic spinal infections in endemic settings. It typically results from hematogenous seeding of the vertebrae during or after a febrile illness and often presents with chronic back pain and raised inflammatory markers, while routine blood cultures may be non-contributory. We describe a young, otherwise healthy patient with subacute febrile back pain in whom spinal imaging showed lumbar spondylodiscitis, and image-guided vertebral biopsy established *Salmonella Typhi* as the etiological agent. The patient was managed successfully with prolonged, targeted intravenous antibiotics and spinal immobilization, without the need for surgical intervention. This case highlights the importance of considering *Salmonella* spondylodiscitis in the differential diagnosis of febrile back pain following enteric fever and underscores the pivotal role of early MRI and tissue diagnosis in guiding appropriate antimicrobial therapy and preventing long-term spinal sequelae.

Keywords: *Salmonella, Salmonella Typhi,* Spondylodiscitis, Extraintestinal manifestations

INTRODUCTION

Typhoid fever, resulting from infection with *Salmonella enterica* serovar Typhi, poses a persistent threat in regions with suboptimal sanitation, such as South Asia, where it annually impacts millions through contaminated water and food, compounded by rising antimicrobial resistance patterns.[1,2] Core clinical features encompass sustained high fever, abdominal discomfort, altered bowel habits ranging from diarrhea to constipation, and organomegaly, underpinned by consistent bacteremia that drives metastatic spread in a small fraction of cases.[3,4] Extraintestinal foci emerge particularly during untreated bacteremic phases, targeting sites like the skeleton, heart, lungs, or biliary tract, with bone and joint involvement representing under 1% of osteomyelitis etiologies overall.[1] Spinal manifestations, termed spondylodiscitis or vertebral osteomyelitis, account for less than 0.5% of bone infections. Spondylodiscitis, stem from hematogenous deposition into richly vascularized vertebral bodies and intervertebral discs, frequently via low-flow venous channels like Batson's plexus, with lumbar segments affected in roughly half of instances followed by thoracic levels.[3,4] Traditional associations link these to host vulnerabilities including hemoglobin disorders like sickle cell trait, metabolic conditions such as diabetes, immune suppression, or pediatric/geriatric states, though contemporary data reveal occurrences in structurally normal hosts, possibly tied to bacterial persistence post-subclinical infection.[1] Initial symptoms often include localized backache, constitutional malaise, and laboratory evidence of inflammation, frequently confounded by regional prevalences of mycobacterial alternatives.[4] Advanced imaging via MRI excels in delineating disc destruction, adjacent bone erosion, soft-tissue extension, and epidural involvement, while definitive etiology demands tissue sampling, as peripheral cultures underperform amid focal sequestration.[1,3]

CASE REPORT

A 17-year-old boy, resident of Uttarakhand and a college student, with no significant previous medical history, presented with complaints of high-grade fever with chills, associated with nausea, vomiting and generalized weakness for one week. He also complained of crampy abdominal pain for the same duration. He also complained of severe lower back pain radiating to right hip and right thigh, which was gradually progressive for the past 45 days. The patient was bedridden because of the pain for the past week. There were no complaints about headaches, abnormal bowel movements, weight loss, or loss of appetite.

On examination, he was conscious and oriented. He was afebrile and had normal blood pressure (100/64 mmHg), pulse rate (94 beats per minute), and was maintaining oxygen saturation of 98% on room air. On lower back examination, there was no point tenderness, however paraspinal tenderness was noted suggesting spasm of the paraspinal muscles. Power in both the lower limbs was 4/5, and there was no sensory or motor deficit noted in the neuromuscular examination of bilateral lower limbs. Straight leg raise test was positive in right lower limb. The rest of the systemic examination was normal. He was started on injectable ceftriaxone and oral azithromycin with the provisional diagnosis of enteric fever with lower back pain under evaluation.

Initial laboratory investigations (Table 1) showed normal leucocyte count, raised ESR (45 mm in 1st hour) and CRP (54.8 mg/dL), and elevated transaminases, alkaline phosphate and gamma-glutamyl transferase levels. Infective workup was done where; urine and paired blood cultures were negative. However, the Widal test was positive (≥1:160). Abdominal ultrasound showed mild periportal cuffing. MRI of the lumbosacral spine was done for evaluating the cause of back pain, which was suggestive of spondylodiscitis at the L3-L4 levels of the lumbar spine with pre/paravertebral soft tissue signal extending along the neural foramen with signal along the right existing nerve roots suggesting infective etiology. The patient underwent a transpedicular biopsy. The tissue biopsy showed features of acute spondylodiscitis. The tissue sent was negative of acid-fast bacilli and TB GeneXpert, ruling out tuberculosis. However, tissue culture showed the growth of *Salmonella typhi*. On further probing the patient, it was revealed that the patient had an episode of fever with loose stools around one month before the onset of pain symptoms, for which he sought treatment from a local practitioner and the symptoms relieved after 5 days of empirical treatment.

The patient was diagnosed with partially treated typhoid fever which later complicated as *Salmonella* spondylodiscitis and was treated for the same. Both intensity and frequency of the fever reduced, with some improvement in back pain. The patient was discharged in stable condition with improved laboratory parameters and advice to continue intravenous antibiotics for 1 month with complete bed rest and mobilization using Taylor's brace. The patient is doing well on follow up with significantly reduced involvement on serial MRI imaging and symptomatic improvement in back pain.

Table 1: Hematological and biochemical investigations.

Parameters	Result
Haemoglobin	12.1 g/dl
Platelet count	3.3 lakh /µl
Total leukocyte count	5.3 ×10³/µl
Erythrocyte sedimentation rate (ESR)	45 mm/hr
CRP	54.8 mg/dl
Aspartate aminotransferases (AST)	70 U/l
Alanine aminotransferases (ALT)	80 U/l

DISCUSSION

Enteric fever, caused by *Salmonella enterica* serovar Typhi, typically presents as a systemic febrile illness with gastrointestinal symptoms, but it can occasionally spread beyond the gut through persistent bacteremia. Although musculoskeletal involvement occurs in fewer than 1% of cases worldwide, however, in endemic regions like India the incidence is roughly 2%.[1,4] Among skeletal complications, vertebral osteomyelitis is the most frequent, responsible for about 40% of adult Salmonella bone infections. It usually develops gradually, with patients presenting with back pain and elevated inflammatory markers, often resembling pyogenic or tuberculous infections especially when empirical treatment causes further diagnostic delays.[1-3] Within these cases, *S. Typhi* accounts for nearly 28% of osteomyelitis reports, surpassing non-typhoidal strains in tropical areas. The lumbar spine is most commonly affected (around 50%), and symptoms tend to evolve slowly over weeks to months after the initial infection.[1,4] The current case of *S. Typhi* spondylodiscitis at L3-L4 in an otherwise healthy patient, confirmed by transpedicular biopsy, highlights how this unusual hematogenous complication can arise even after apparent resolution of a prior enteric illness that had been treated conservatively a few weeks earlier.[1,4] The one-month gap between the initial fever-diarrhea episode and the onset of radicular back pain aligns with the subacute timelines described in the literature (typically 1-6 months). During this period, incomplete bacterial clearance can allow *S. Typhi* to persist inside macrophages, creating reservoirs that later seed the vertebrae even in patients without classic risk factors such as hemoglobinopathies.[2,3] While outpatient therapy may have reduced systemic bacterial load, it likely permitted small persister populations dormant and stress-tolerant to silently disseminate, a phenomenon linked to relapse and focal complications in settings with high quinolone use.[1] Diagnosis in this case was established through MRI, which identified features of discitis and prompted an orthospinal-

guided biopsy. This approach is strongly supported in the literature: biopsy yields *S. Typhi* in nearly 100% of cases, compared to only 68% positivity with blood cultures in adult cohorts.[1,3] Such findings emphasize the importance of obtaining tissue samples in chronic osteoarticular infections where bacteremia may no longer be detectable. Sensitivity results then guided targeted intravenous therapy, helping avoid the pitfalls of empirical treatment in regions with significant drug resistance. Notably, the involvement of the lumbar spine mirrors the distribution reported in about half of typhoid spinal infections, many of which are initially mistaken for Pott's spine and managed with antitubercular therapy before the correct diagnosis is made.[4]

Treatment relies primarily on prolonged beta-lactam therapy. Third-generation cephalosporins, such as ceftriaxone, are preferred due to their intracellular activity and favorable resistance profile. These are typically administered intravenously for at least six weeks before switching to an appropriate oral agent (such as cotrimoxazole, if susceptible). This regimen corresponds with a 78% success rate across 69 adult cases treated without surgery.[1,4] In uncomplicated cases without neurological compromise or abscess formation similar to the present scenario conservative therapy works in about 80% of patients, whereas 55% of those requiring surgical intervention undergo procedures for instability, including debridement or fusion.[3,4] The average treatment duration approaches 11 weeks, with declining inflammatory markers serving as a useful indicator of recovery. Drug-resistance patterns remain a major consideration, as nearly 38% of *S. Typhi* isolates show multidrug resistance, underscoring the need to tailor therapy and to confirm oral efficacy before de-escalation.[1] Follow-up experiences from comparable cases show sustained improvement at 6-12 months, with resolution of pain, normalization of neurological findings, and stabilization on imaging. Nonetheless, a recurrence rate of 5-10% has been reported when treatment duration is insufficient.[4]

This case reinforces the importance of asking about prior enteric fever when evaluating patients with febrile back pain in endemic areas, and it highlights the role of MRI followed by biopsy in preventing delays that could lead to deformity or neurological deficits. A complete course of therapy is critical to eliminate persisters and prevent further spread.[2,3] Emerging reports in immunocompetent children further broaden the differential for spinal infections, reminding clinicians that *S. Typhi* remains a relevant cause of vertebral disease in many parts of the world.[1]

CONCLUSION

S. Typhi spondylodiscitis is a rare but important extraintestinal complication of enteric fever that can occur even in otherwise healthy individuals after apparent resolution of the primary infection. A history of recent enteric fever, persistent or new-onset back pain and elevated inflammatory markers should raise suspicion for vertebral involvement, particularly in endemic regions. Early MRI followed by image-guided biopsy and microbiological confirmation is essential for establishing the diagnosis and enabling targeted antimicrobial therapy. Prolonged, susceptibility-guided treatment can result in favorable outcomes when neurological compromise, instability, or significant abscess formation is absent. Maintaining a high index of suspicion may help prevent misdiagnosis as pyogenic or tuberculous spondylodiscitis and reduce delays in appropriate management.

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