

Invasive *Shigella flexneri* Bacteraemia in an Immunocompetent Child: A Rare Case Report

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ABSTRACT

Shigella flexneri is a common cause of bacillary dysentery in children, especially in developing countries. However, bacteraemia due to *Shigella* species is rare, especially in immunocompetent and well-nourished children. A five-month-old male child presented with a 4-day history of high-grade fever and multiple episodes of watery diarrhoea, accompanied by abdominal pain for two days. On examination, the child showed signs of moderate dehydration but remained haemodynamically stable. Laboratory investigations revealed elevated inflammatory markers. Blood cultures grew non-lactose fermenting Gram-negative bacilli, which were identified as *Shigella flexneri* using an automated system. The isolate was susceptible to ceftriaxone, cefepime, cotrimoxazole, and azithromycin, but resistant to fluoroquinolones. The patient was treated with appropriate supportive care and intravenous ceftriaxone antibiotics that lead to gradual clinical improvement and successful recovery. Clinicians should consider *Shigella* bacteraemia in paediatric patients presenting with severe febrile diarrhoea. Timely blood culture and appropriate antimicrobial management are crucial for favourable prognosis, even in atypical presentations without known risk factors.

Keywords: Bacillary dysentery, Blood culture, Dehydration, Diarrhoea

CASE REPORT

A five-month-old male child from Central Delhi was admitted to a paediatric emergency in a tertiary care hospital with a history of multiple episodes of watery diarrhoea and high-grade fever for four days, accompanied by abdominal pain for two days. The fever was continuous and was not associated with chills or rigours. There was no history of vomiting, seizures, or altered sensorium. The child was exclusively breastfed, and there was no significant antenatal history. There was no significant past medical history or previous hospitalisation, and no significant family history of chronic illness or recurrent infections.

On admission, the child appeared moderately dehydrated. He was irritable but conscious. Clinical assessment revealed sunken eyes, dry oral mucosa, and decreased skin turgor with delayed skin pinch return time of approximately two seconds. On physical examination, the abdomen was soft, tender, and non-distended, with audible bowel sounds and the presence of tenesmus. The liver and spleen were not palpable clinically. The child was haemodynamically stable with a heart rate of 121 beats/min, respiratory rate of 24 breaths/min, blood pressure of 111/68 mm Hg, and axillary temperature of 99.6°F. Anthropometric assessment revealed a weight of 5.7 kg (expected weight for age: approximately 5.2 kg), length of 63 cm (expected: approximately 63-67 cm) head circumference 39 centimetre. Respiratory system examination demonstrated bilateral air entry without added sounds. Cardiovascular examination revealed normal S1 and S2 heart sounds without murmurs. Neurological assessment showed a Glasgow Coma Scale score of E4V5M6 [1], equal and reactive pupils, normal tone, and power in all four limbs, and no signs of meningeal irritation or cranial nerve deficits. Ultrasonography of the abdomen reveals no significant finding.

A provisional diagnosis of acute dysentery was made. Empirical management included intravenous fluid therapy for correction of dehydration and parenteral ceftriaxone at a dose of 50 mg/kg/day intravenously. Supportive care included maintenance of hydration

and electrolyte balance, continued feeding, antipyretic therapy for fever, and close monitoring of vital signs, urine output, and clinical response.

Haematological investigations showed haemoglobin of 11 g/dL, total leucocyte count of 8,700 cells/mm³ (neutrophils 40.7%, lymphocytes 45.8%, monocytes 11.3%, eosinophils 2%, basophils 0%), and platelet count of 318×10³/μL. Peripheral smear examination did not reveal any haemiparasites. Liver function tests were within normal limits, including a total bilirubin level of 0.38 mg/dL. Renal function parameters were normal. C-reactive protein was elevated at 34.1 mg/L, indicating an ongoing inflammatory response. Serum electrolytes were as follows: sodium (Na⁺) 138 mmol/L, potassium (K⁺) 4.4 mmol/L, ionised calcium (Ca²⁺) 0.79 mmol/L, and chloride (Cl⁻) 125 mmol/L.

In view of the persistent fever and elevated inflammatory markers, septicaemia was considered, and blood cultures were obtained. Blood samples were collected under complete aseptic conditions from two separate sites (central line and peripheral arterial line) and inoculated into aerobic blood culture bottles. After 24 hours of incubation, subcultures were performed on MacConkey agar and 10% sheep blood agar. After overnight incubation at 37°C, both sets of cultures appearing as non-lactose fermenting translucent colonies (1-2 mm in size) on MacConkey agar. Similar colony morphology was observed on blood agar, though the colonies were non-haemolytic. The isolate was non-motile on motility testing. On manual biochemical characterisation, Triple Sugar Iron (TSI) agar showed an alkaline slant with acid butt (K/A) reaction without hydrogen sulphide production, consistent with a non-lactose fermenting enteric pathogen. The isolate was identified as *Shigella flexneri* by the VITEK® 2 automated identification system (bioMérieux), and antimicrobial susceptibility testing was performed. The isolate demonstrated susceptibility to cotrimoxazole, cefepime, ceftriaxone, and azithromycin. Resistance was observed to ciprofloxacin, and levofloxacin. Susceptibility to azithromycin was determined using

the Kirby-Bauer disk diffusion method and interpreted according to established guidelines.

The patient demonstrated gradual clinical improvement following targeted antimicrobial therapy. Child remained haemodynamically stable and was discharged in satisfactory condition after a five-day hospital stay. At the two-week follow-up, the child was clinically stable with no complain of fever and diarrhoea and no recurrence of symptoms.

DISCUSSION

Shigella flexneri is a Gram-negative facultatively intracellular bacteria responsible for bacillary dysentery in human beings. *Shigella* infections contribute significantly to global mortality, particularly among children in developing countries, accounting for 1.5 million deaths annually. The pathogenesis of *Shigella* spp. depends on the ability of this organism to invade the colonic epithelium where it induces severe mucosal inflammation [2]. Transmission commonly occurs through the fecal-oral route, often through the ingestion of contaminated potable water. Some important species of *Shigella*, i.e., *Shigella dysenteriae*, *S. flexneri*, *S. boydii*, and *S. sonnei*, also known as groups A, B, C, and D, respectively [3].

Shigella spp. contributes to approximately 10% of all diarrhoea and one-third of dysentery cases worldwide. The highest incidence is observed between one year of age and four years, infrequently affecting adults [4]. Although extraintestinal complications are uncommon, with an incidence of bacteraemia reported in 0.4-7.3% of cases [5]. Clinically shigellosis present as an acute infectious condition characteristic by fever, abdominal cramps, tenesmus and blood stool, resulting from the invasion of the colonic epithelium by *Shigella* species and the release of potent enterotoxins. Worldwide, an estimated 188 million people are infected annually, with the majority of case occurring in young children and infant from resource-limited setting, primarily through the ingestion of contaminated food and water source [6].

Shigella primarily affects gastrointestinal system, where it primarily causes localised mucosal inflammation. In contrast, extraintestinal dissemination with bloodstream invasion is rare and found approximately 0.4% to 7.3% of infected patients [6]. *Shigella*-associated bacteraemia should be considered a serious infection, as it is associated with notable rise in mortality [7]. Among the all *Shigella* species, *Shigella flexneri* is considered the most virulent serotypes and are frequently associated with severe, invasive forms of shigellosis, including progression to septicaemia [8,9]. In present case, *S. flexneri* demonstrated susceptibility toward cotrimoxazole, cefepime, ceftriaxone and resistance to ciprofloxacin, and levofloxacin. So, the initial empirical treatment with ceftriaxone was successful. A study done by Sadredinamin M et al., demonstrated resistance to ampicillin, cefotaxime, ceftriaxone, cefixime, and cotrimoxazole [10]. In contrast, some studies indicated that ceftriaxone remains an appropriate option for treating *S. flexneri* bloodstream infection [11,12]. A study by Delgado M et al., in Argentina reported two cases of *Shigella flexneri* bacteraemia in infant, one of them presented with primary immunodeficiency, whereas the other had no any predisposing factor. Both patients presented with fever and diarrhoea [13].

Similarly, a study from Mumbai reported four cases of septicaemia due to *Shigella* in children admitted to Lokmanya Tilak Municipal General Hospital. All of these four cases, three were attributed to *Shigella dysenteriae* serotype 1 and one to *S. flexneri*, with an overall mortality rate of 75% [8]. The high mortality observed in these patients may be attributable to infection with multidrug-

resistant *Shigella* strains, particularly *S. dysenteriae* type 1, which is most virulent serotype. Antimicrobial susceptibility testing demonstrated that all isolates were sensitive to gentamicin, amikacin, norfloxacin, and nalidixic acid, while resistance to the amoxicillin, chloramphenicol, tetracycline, and cotrimoxazole [9]. Additionally, a rare case of fatal septicaemia caused by multidrug-resistant *Shigella flexneri*, followed by an episode of gastrointestinal infection, has been reported in a six-month-old infant from New Delhi [14]. Shigellosis has been predominantly described in malnourished infants and young children; Another study from New Delhi documented a rare case of *Shigella flexneri* bacteraemia in a 65-year-old adult male with non-diabetic, hypertensive male who presented with history of dysentery and significant co-morbidities including chronic kidney disease and dilated cardiomyopathy, also had undergone permanent pacemaker implantation seven years subsequently developed *Shigella* bacteraemia [15].

In the present case, septicaemia was due to *Shigella flexneri*, which was isolated from both blood sample, hereby confirming systemic dissemination from a gastrointestinal system. The isolate demonstrated susceptibility to multiple antimicrobial agents. The patient was managed with supportive therapy and appropriate antibiotics. Clinical improvement was observed, and the patient was discharged in stable condition after 5-day hospitalisation, with significant recovery after that.

CONCLUSION(S)

The present study highlights the consequence of *Shigella* infection into the differential diagnosis of patients presenting with severe sepsis in accompanied by diarrhoea. Although *S. flexneri* bacteraemia is rare, it occurs mainly in children under five years of age. Early and timely diagnosis, intensive treatment is essential for decline the severity. The optimal duration of antimicrobial therapy in cases complicated by bacteraemia remains to be clearly defined. Further prospective and comparative studies are required to establish evidence-based recommendations regarding the duration of therapy in such case.

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