



Neurological complications of gastrointestinal shigellosis in a child: A case report

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Abstract

Introduction. Shigellosis is an invasive bacterial gastroenteritis transmitted between humans or via the fecal-oral route through food and water, which can lead to extraintestinal complications, particularly neurological ones.

Observation. This observation reports the case of a 4-year-old boy admitted for incomplete febrile meningitis, generalized tonic-clonic seizures, altered consciousness, osteotendinous areflexia, weakness in the lower limbs, and drowsiness, all occurring in the context of a deterioration in his general condition. Bloody mucous diarrhea, vomiting, and diffuse abdominal pain appeared secondarily. Brain CT scan, cytobacteriological and biochemical examination of CSF, and neuromeningeal PCR were normal. Laboratory tests showed a CRP of 397 mg/L, hepatic cytolysis (ALAT 234 U/L, ASAT 589 U/L), renal abnormalities (urea 0.79 g/L; creatinine 7 mg/L), and hypoglycemia. Gastrointestinal PCR was positive for *Shigella*/enteropathogenic *Escherichia coli* (EIEC). The child received rectal diazepam (0.5 mg/kg) during seizures and a third-generation cephalosporin (C3G) at a meningeal dose followed by a conventional dose, with rapid clinical and biological improvement within 48 hours.

Conclusion. Seizures associated with shigellosis occur predominantly in children and are based on multifactorial mechanisms (fever, metabolic disorders, possible toxins). Early recognition of the digestive link allows for optimized management and avoids unnecessary neuromeningeal investigations in uncomplicated cases.

Keywords: Shigellosis; Children; Seizures; Encephalopathy; EIEC

1. Introduction

Shigellosis is an invasive enteric infection caused by the genus *Shigella* [1-4], responsible for acute dysentery and systemic complications [5]. Neurological manifestations (convulsions, encephalopathy with lethargy, confusion, headaches, meningism, hallucinations) are well described in children, but rarer in adults [6]. Seizures are the most common extraintestinal manifestation, reported in 12 to 45% of affected children, mainly under the age of 15 [1,2]. We report a pediatric case illustrating the clinical sequence and diagnostic and therapeutic challenges.

2. Observation

2.1. Clinical data

Boy, 4 years old, admitted for incomplete febrile meningitis syndrome (meningeal rigidity, temperature 38.5°C), generalized tonic-clonic seizure, altered consciousness, osteotendinous areflexia, functional impotence of the lower

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limbs, and drowsiness, in a context of asthenia and anorexia. Bloody mucous diarrhea, vomiting, and diffuse abdominal pain appeared during hospitalization. The mother's interview revealed that digestive symptoms preceded the neurological signs.

2.2. Neuromeningeal investigations

The brain scan showed no contraindications to lumbar puncture and was normal. The cerebrospinal fluid (CSF) was clear; cellularity < 3 cells/mm³; red blood cells 80/mm³; glycorrachia 0.50 g/L (concomitant blood glucose 0.10 g/L); proteinorrachia 0.17 g/L; direct examination negative; sterile culture. The neuromeningeal polymerase chain reaction (PCR) was negative.

2.3. Biological assessment and additional imaging

C-reactive protein (CRP) 397 mg/L; hepatic cytolysis (alanine aminotransferase ALAT 234 U/L ≈ 5 N; aspartate aminotransferase ASAT 589 U/L ≈ 12 N); renal abnormalities (urea 0.79 g/L, creatinine 7 mg/L); lymphopenia 1,340/mm³; monocytosis 1,279/mm³. Normal serum electrolyte and urine culture. Chest X-ray unremarkable. Note hypoglycemia (0.10 g/L).

2.4. Digestive etiology

Given the early onset of digestive symptoms, a multiplex gastrointestinal PCR test was positive for *Shigella*/enteropathogenic *Escherichia coli* (EIEC).

2.5. Management

Three recurrent seizures with post-critical deficit led to the administration of diazepam (0.5 mg/kg) rectally. Given the initial suspicion of meningoencephalitis, a third-generation cephalosporin (C3G) at a meningeal dose and acyclovir were started. After *Shigella*/EIEC was detected, acyclovir was discontinued and C3G was continued at a conventional dose.

2.6. Progression

Rapid improvement within 48 hours: decreased stool frequency and abdominal pain, neurological recovery (consciousness, walking, and deep tendon reflexes (DTR) returned to normal), and improved laboratory values (CRP 397 $\rightarrow$ 150 mg/L; AST 589 $\rightarrow$ 14 U/L; ALT 234 $\rightarrow$ 5 U/L).

The parents' informed consent was obtained for the publication of this case, and the patient's anonymity was respected in accordance with ethical recommendations.

3. Discussion

Shigellosis is endemic in many resource-limited countries and is a major cause of dysentery [7]. Neurological manifestations occur predominantly in children under 5 years of age [6,8]. The reported incidence of neurological complications varies widely between series (approximately 5 to 50%), with seizures occurring in 12 to 32% of cases [9]. Neurological signs are more common with *Shigella* than with other organisms, occurring 3 to 4 times more frequently in shigellosis than in digestive salmonellosis [10,11].

The pathophysiology of seizures is multifactorial: fever, metabolic disturbances (particularly hydroelectrolytic and glycemie disorders) and the possible role of bacterial toxins [10,14-16]. The contribution of Shiga toxin is not consistent [6,12,14,17,18-20] and direct invasion of the central nervous system (CNS) is rare [7]. Electrolyte disturbances (particularly hyponatremia), hypocalcemia, and hypoglycemia, when present, must be corrected quickly [20]. In our observation, the temporal sequence of "gastrointestinal involvement $\rightarrow$ neurological signs," hypoglycemia, and young age, in a context of fever and dysentery, are plausible factors triggering seizures.

The neuromeningeal assessment is usually normal or of little significance (CSF most often non-inflammatory) [12,13]. Thus, in the absence of evidence of CNS infection or signs of severity (persistent focal deficits, prolonged coma, status epilepticus, major impairment), systematic invasive investigations are not always necessary. Conversely, rapid identification of a digestive etiology (stool culture and/or multiplex PCR) can guide treatment.

Management is based on: (i) seizure control (acute benzodiazepines if necessary), (ii) correction of metabolic disorders and dehydration, (iii) probabilistic then targeted antibiotic therapy (C3G are among the first-line options depending on

local ecology and resistance), and (iv) close clinical monitoring. The outcome is usually favorable with no sequelae when treatment is early and appropriate [10,14,21,22].

4. Conclusion

In children, shigellosis can be complicated by neurological manifestations, primarily convulsions, which are usually benign and transient. Recognizing the dysenteric context and performing a digestive microbiological test (PCR/culture) can avoid repeated neuromeningeal investigations and optimize antibiotic therapy. Educating families and quickly correcting metabolic disorders help prevent adverse outcomes.

4.1. Practical points

- Consider shigellosis in cases of febrile convulsions with digestive symptoms, especially in children under 5 years of age.
- Systematically check for and correct hypoglycemia and fluid and electrolyte imbalances.
- CSF is often normal; prioritize rapid digestive microbiological tests.
- Antibiotic therapy adapted to the local context; clinical improvement expected within 48–72 hours.
- Inform parents of the generally favorable prognosis.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that they have no conflicts of interest in relation to this article.

Statement of informed consent

Informed consent was obtained by all participants in this study.

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