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Case Report

Enteric fever resembling Malawi-Mozambique outbreak

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ABSTRACT

A 47-year-old male with no known co-morbidities presented with hematochezia and multiple episodes of vomiting. He had a history of high-grade fever with chills, rigor, vomiting, and abdominal pain 3 weeks ago, for which he was treated with 5 days of IV antibiotics in another hospital. Examination revealed tremor, rigidity, hyperreflexia, bradykinesia, and hypophonic monotonous speech. Colonoscopy showed ileocolonic ulcers. Widal came positive. Treated as a case of Enteric fever with neurological complications such as parkinsonism, UMN signs, and gastrointestinal bleeding secondary to an ileal ulcer.

Keywords: Enteric fever, Parkinsonism, Ileal ulcer, Gastrointestinal bleeding, UMN signs

INTRODUCTION

Enteric fever is a systemic disease characterized by fever and abdominal pain. It is caused by the dissemination of gram-negative bacteria: *Salmonella typhi* or *Salmonella paratyphi*. It is usually spread through food or water with fecal contamination by ill or asymptomatic chronic carriers.[1] The patient will present with a high grade step ladder fever, relative bradycardia, nausea, vomiting, diarrhea, and abdominal pain. Constitutional symptoms like myalgia, headaches, and arthralgia may present. Rose spots—salmon-colored macule seen during the second week of illness. During the 3rd week of illness, the patient may develop hepatosplenomegaly, intestinal bleeding, and perforation.[1]

Routine blood investigations may reveal leukopenia with a shift to left, neutropenia, elevated liver enzymes, and elevated inflammatory markers. Definitive investigation is culture and sensitivity. Blood, stool, bone marrow, rose spot samples, and intestinal secretions can be used for culture. Maximum sensitivity (>80%) is with bone marrow culture.[3] The widal tube agglutination test is a simple, rapid test, but it has less sensitivity and specificity.

The patient may develop complications during the 3rd–4th week of the illness. Common ones are GI bleeding and perforation. With many reports of multidrug-resistant typhoid fever outbreaks from India, attention has been drawn to its newer facets, such as neuropsychiatric manifestations like UMN signs, ataxia, Parkinsonism, encephalopathy, muttering delirium, and coma vigil.[2]

Less common complications are DIC, HLH, pancreatitis, pyelonephritis, myocarditis, endocarditis, and pneumonia.[2]

Prompt antibiotic treatment decreases mortality to <1%. Fluroquinolones are most effective, with cure rates of approximately 1%. However, because of the high prevalence of strains with decreased susceptibility, they are no longer used for empirical treatment in India.[4] For treatment, ceftriaxone or azithromycin are preferred. In ceftriaxone-resistant cases, meropenem and imipenem can be used.[1]

CASE REPORT

A 47-year-old male came to the hospital with bleeding per rectum, abdominal pain, and vomiting for 2 days. Bleeding

was spontaneous, fresh blood, 2 episodes, approximately 100 ml, associated with abdominal pain, and had no relation to defecation. He had a history of high-grade fever with chills, rigor, vomiting, and abdominal pain 3 weeks ago, for which he took treatment from an outside hospital in the form of an IV antibiotic for 5 days, and he became better. He gives a history of frequent travel between the cities of Kerala and food intake from hotels daily. On general examination, he was hemodynamically stable. Pallor and Tremor were present.

Systemic examination

For CNS, tremor, masked like facies, braykinesia, hypophonic monotonous speech, rigidity, hyperreflexia, for GIT, diffuse mild tenderness, and for CVS, RS-normal.

Evaluation

Colonoscopy

The scope passed up to the terminal ileum. Multiple large, deep ulcers are seen in the terminal ileum, one of which shows a non-bleeding, visible vessel. Ulcerations are noted in the ileocaecal valve. Two to three discrete ulcers (~1 cm) are noted in the ascending colon. The rest of the visualized colonic mucosa appeared normal. No growth/polyp seen.

Lab parameters

Lab parameters included- hemoglobin: 7.3 gm%, total leucocyte count (TLC): 14600 cells/mm^3; N79 L17, erythrocyte sedimentation rate (ESR): 5 mm/hour.

Platelet count: 4.2 lakh, Red cell indices: reduced MCV, MCH, and MCHC, CRP: 10 mg/l, total bilirubin: 1.32 mg/dl; total protein: 5.5 g/dl; albumin: 3.3 g/dl; globulin: 2.2 g/dl SGOT: 96 U/l; SGPT: 47 U/l; urea: 45 mg/dl; creatinine: 1.2 mg/dl; sodium: 108 mmol/l; potassium: 3.1 mmol/l.

Lepto card test-negative, dengue card test-negative, HIV, HepB, and C were negative. Ultrasonography abdomen and pelvis: grade 1 fatty liver.

With the history of fever and ileocolonic ulcers, enteric fever was suspected. Blood culture and sensitivity, stool culture and sensitivity, and the Widal test were sent, and the patient was started empirically on injection Ceftriaxone 2 mg IV once daily.

Magnetic resonance imaging (MRI) brain with screening of the cervical spine was normal.

In view of features of parkinsonism and deranged liver function, an evaluation was done to rule out Wilson's disease, which came out negative (serum copper: 154 154 mcg/dl serum cerulplasmin: 30 mg/dl).

Widal came positive with a titre of *Salmonella typhi* O antigen 1:320 and *Salmonella typhi* H antigen 1:160. Blood and stool cultures showed no growth.

Diagnosis

Enteric fever with gastrointestinal bleeding secondary to an ileal ulcer, neurological manifestations (parkinsonism, UMN signs).

Treatment

Two hemoclips (INSC-7-230-S) were applied over the non-bleeding visible vessel. Haemostasis attained.

Injection Ceftriaxone 2 gm IV OD given for 10 days followed by tablet Ciprofloxacin 500 mg 1-0-1 for 1 week.

Tablet Levodapa 100 mg + Cardidopa 10 mg 1-0-1.

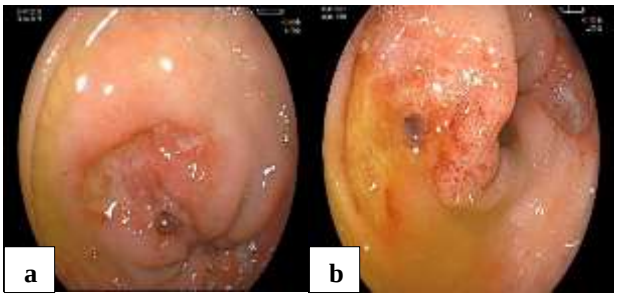

Figure 1: (a) and (b) Ileal ulcers.

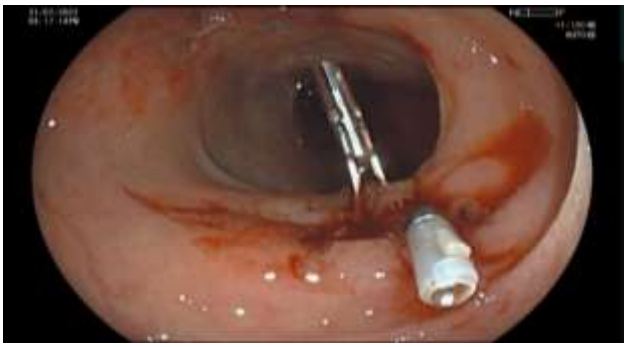

Figure 2: Hemoclip in situ.

DISCUSSION

The outbreak of typhoid fever in an area along the Malawi-Mozambique border (South-East Africa) in 2009 was associated with a range of early neuropsychiatric manifestations.[3] The most common manifestations were related to upper motor neuron dysfunction, including spasticity, clonus, and hyper-reflexia; a bradykinetic rigid syndrome; and ataxia. 303 cases of typhoid were reported between March and November of 2009. Among this, 43% had UMN signs including spasticity and hyperreflexia, 22% had ataxia, and 20% had parkinsonism.[1,2] With many reports of multidrug-resistant typhoid fever outbreaks

from India, attention has been drawn to its newer facets, such as neuropsychiatric manifestations.[4] The presence of variable neurological manifestations suggests that typhoid produces dysfunction at numerous sites within the nervous system. The mechanism by which typhoid fever may produce neurological illness is unknown, and a better understanding of the underlying pathophysiological mechanisms is needed. However, the possible mechanism is that *Salmonella typhi* endotoxins could transiently interfere with cholinergic-dopaminergic control of the basal ganglion in parkinsonian rigidity.[3,4] Gastrointestinal bleeding is the most common symptom, and it occurs in up to 10% of patients. It results from the erosion of a necrotic Peyer's patch through the wall of an enteric vessel.[5] In the majority of cases, the bleeding is slight and resolves without the need for a blood transfusion. In 2% of cases, however, bleeding is clinically significant and can be rapidly fatal if a large vessel is involved.

Gastrointestinal bleeding in typhoid fever usually occurs from the ulcers in the ileum or the proximal colon, and the most common colonoscopic manifestations are multiple variable-sized punched-out ulcerations.[6] The shape of the ulcers is usually ovoid, with the longest diameter parallel to the long axis of the gut, so that stricture formation does not occur after healing.[7] The edges are soft, swollen, and irregular, but not undermined. The floor is usually smooth and is formed by the muscular coat. Near the ileocecal valve, where perforation occurs more commonly, ulcers become deeper than elsewhere.[8]

CONCLUSION

The case report presents a patient presenting in the 3rd week of enteric fever with gastrointestinal bleeding secondary to an ileal ulcer and neurological complications like UMN signs and Parkinsonism.

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