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CASE REPORT

Intestinal Perforation in a Child with *Shigella dysenteriae* type 1 Infection: A Rare Complication

JENA DERAKHSHANI HAMADANI, MUHAMMAD TAREK AZAD, JANNAT JAHAN CHOWDHURY, AND IQBAL KABIR

International Centre for Diarrhoeal Disease Research, Bangladesh; GPO Box 128, Dhaka 1000, Bangladesh

INTRODUCTION

Shigellosis or bacillary dysentery is associated with a number of severe and often life-threatening complications, including haemolytic-uraemic syndrome, toxic megacolon, septicaemia, severe malnutrition, hyponatraemia and hypoglycaemia (1).

Intestinal perforation due to shigellosis is a rare complication and only a few cases have been reported (2). Perhaps, for being a rare complication, not much attention has been given to its management. We present here a case of intestinal perforation in a patient with bacteriologically proven shigellosis.

CASE REPORT

A 5-year old boy was admitted into the Clinical Research and Service Centre of the International Centre for Diarrhoeal Disease Research, Bangladesh with the complaints of frequent passage of loose stools with blood and mucus, straining during defecation, fever, and vomiting for 15 days and anorexia for 5 days.

Physical examination revealed a severely malnourished child weighing 9.9 kg. (wt/age 50% of National Centre for Health Statistics median), with rectal temperature of 39°C, radial pulse of 136/min and respiratory rate of 40/min. The abdomen was soft, not distended, but tender. On auscultation of the chest occasional crepitations were heard in both the lung fields. Periferal blood examination revealed a total white cell count of 11,000/mm^3; platelet count was 75,000/mm^3, and haematocrit was 31%. The patient had a serum sodium concentration of 110 mmol/l, potassium of 2.9 mmol/l, chloride of 75mmol/l, total CO_2 of 24.7 mmol/l, total protein of 54 g/l, and the chest X-ray showed patchy opacities in both the lung fields. Microscopic examination of stool showed pus cells of >50, red cells of 21-50 and macrophages of 1-5 per high power field. No helminth or protozoa was seen. Serological test for *Salmonella typhi* infections was negative. Both rectal swab and blood culture taken on admission grew *S. dysenteriae* type 1 which was susceptible to pivmecillinam, ciprofloxacin, ceftriaxone and gentamicin, but resistant to ampicillin, chloramphenicol, cotrimoxazole and nalidixic acid.

The child was initially treated with nalidixic acid suspension but two days later, on receiving rectal swab culture results, nalidixic acid was stopped and parenteral pivmecillinam was started in a dose of 60 mg per kg body weight per day. Additionally, ampicillin was administered in a dose of 75 mg/kg/day for treating lower respiratory tract infection on the day of admission. A hypertonic solution of 3% NaCl was also infused in a dose of 12 ml/kg body wt. over 3 hours as the patient was severely hyponatraemic.

On the third day after admission, a repeat peripheral blood count showed a drop of haematocrit from 31% to 17%, and platelet count from 75,000/mm^3 to 40,000/mm^3. RBC fragmentation was 0.7%. He had a serum sodium concentration of 124 mmol/l, potassium of 3.45 mmol/l, chloride of 86 mmol/l, total CO_2 of 29.8 mmol/l and serum protein of 42 g/l. On receiving blood culture result on day 4, parenteral ceftriaxone in a dose of 75 mg/kg/day was started for covering *Shigella* septicaemia, and three units of whole human blood were transfused on day 4, 6, and 7 in a dose of 10 ml/kg.

On day 7, patient's abdomen became distended and tender and bowel sound was sluggish. Food and oral medications were stopped, and a plain X-ray of abdomen in erect posture on day 8, showed free gas under both domes of the diaphragm (photograph) indicating intestinal perforation. The child was transferred to a general paediatric hospital where he was successfully operated and discharged after complete recovery. After communication with the surgeons, it was found that a perforation was detected in the transverse colon. No further details could be obtained.

DISCUSSION

Shigellosis and its complications are major causes of diarrhoeal disease morbidity and mortality in the developing countries perhaps due to poverty and poor sanitation.

Fig. Plain X-ray of abdomen and chest in erect posture showing free gas shadow under both domes of the diaphragm

Infection with *Shigella* is mainly confined to the mucosal and submucosal layer of the colon. One of the intestinal complications of *Shigella* infection is toxic megacolon i.e. acute dilatation of the colon which has been associated with a high mortality rate (3).

Another complication is acute intestinal obstruction which is associated with greater risk of death (33.3%) when compared to fatalities without intestinal obstruction (8.2%) (4).

Instances of intestinal perforation due to shigellosis, are rare. Although the mechanism of perforation is not yet fully understood, it has been speculated that it is not the extension of the mucosal ulceration which causes the perforation but the diffuse vasculitis of the small vessels which results in necrosis of the gut wall extending through the serosal layer (5). During the epidemic of shigellosis in the central America, autopsy studies in few cases showed that in the patients with toxic megacolon, there was deposition of fibrin thrombi in the arterioles and veins of the submucosa and lamina propria (6), and perforation was found in 3 of 173 cases that were examined post-mortem (7).

In our case, the child, in addition to having *Shigella*-colitis and sepsis, had radiologically confirmed broncho-pneumonia, leukemoid reaction and hypokalaemia which are known risk-factors for death (1). In spite of appropriate treatment with antimicrobials, the child developed intestinal perforation indicating that perforation is not always preventable, especially in malnourished children who are presumed to have thin bowel walls. Therefore, the role of other supportive measures in the treatment of shigellosis needs to be further evaluated. Efforts should be made to reduce the risks of perforation in shigellosis. For instance, prevention of hypovolaemic shock is very important since this can cause intravascular fluid-shift toward the vital organs. The splanchnic vessels are then compromised causing necrosis of the bowel wall which may result in perforation. Correction of electrolyte imbalance including hypokalaemia which may lead to paralytic ileus causing stasis in the gut is also an important measure to be taken.

However, early diagnosis and early initiation of therapy is definitely a major step for preventing intestinal perforation. In this regard, health education to the mothers to recognize the seriousness of dysenteric illness and to consult a physician immediately, is very important. In our case, surgical intervention at an early stage saved the life of the child.

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