Nigerian Journal of Paediatrics 1992; 19(1): 15-17

Typhoid perforation in a neonate: a case Report

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Summary

Doyin Fagbule, Odigie M and Duze AT. Typhoid perforation in a neonate: a case report. *Nigerian Journal of paediatrics 1992;* **19**:15. A case of typhoid perforation in a 26-day old neonate is reported. The baby Presented with a high grade and continuous fever, increasing abdominal distension, and vomiting. Intestinal perforation was confirmed radiologically and at laparatomy. Histopathology of the perforated ileum showed acute necrotizing ileitis. Baby's serum was positive for both flagellar and somatic antigens. She responded well to combined chemotherapy, consisting of intravenous ampicillin, chloramphenicol and metronidazole (Flagyl).

Introduction

TYPHOID fever remains a major public health problem in many parts of the world,[1] but more so in developing countries with low socio-economy and poor environmental sanitation. *Salmonella* infection is endemic in Nigeria and *Salmonella typhi* in particular has been reported to be more prevalent in Ilorin than all other serotypes, put together.[2] Perforation of the bowel is the most serious complication of typhoid fever.[3 4] Previous reports on *Salmonella* infections in Nigeria have not documented any perforation in neonates.[5-9] Peritonitis resulting from typhoid perforation in a severely ill neonate can be rapidly progressive and fatal unless suspected early and treated vigourously and appropriately. Surgical intervention offers the best hope of survival.[3 4] The present case of typhoid perforation in a neonate is reported because of its rarity, and as a reminder that this condition should be considered in the differential diagnosis of acute abdomen in the newborn period.

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Case Report

A 26-day old full-term female neonate was referred to the University of Ilorin Teaching Hospital (UITH) from a peripheral hospital where she was admitted for two days prior to the referral. There was a 5-day history of a high grade and continuous fever, progressive abdominal distension, constipation and vomiting. Vomiting was copious and bile-stained, but not projectile. There was no haematemesis. The baby refused all feeds and was crying excessively. There was also a cough. Both parents are butchers and the baby was the fourth in the family. All family members were well. The mother did not receive any antenatal care; delivery was at home. Baby passed meconium and urine within 12 hours of delivery. From the second day of life, baby was placed on prophylactic traditional concoction against convulsions. Physical examination revealed a toxic, moderately dehydrated, ill-looking baby, weighing 3.10kg. There was

tachypnoea (respiratory rate of 72/min.). The temperature was 39.5°c and pulse rate 152/min. There was mild pallor. There was no visible peristalsis, but there were distended superficial veins. The abdomen was tense and abdominal organs were difficult to palpate due to tenderness. Percussion note was highly tympanitic and bowel sounds were absent. Rectal examination showed an empty rectum. Examination of the lung fields as well as other systems was normal. The provisional diagnosis was neonatal septicaemia and paralytic ileus.

Treatment comprised intravenous fluid (10% dextrose in half-strength Darrow's solution) intravenous cloxacillin (200mg/kg/day) and intramuscular gentamycin (5mg/kg/day). A strict input-output chart was kept. A nasogastric tube was inserted and nil per oris regime instituted. She was reviewed hourly by the physicians, while the vital signs were recorded quarter-hourly and the abdominal girth measured 4-hourly.

Plain abdominal radiograph revealed gaseous distension of the small bowels, absence of any gas in the colon and multiple fluid levels. There was also pneumoperitoneum. Cultures of blood, urine, cerebrospinal fluid and stool yielded no organism. Haematological investigations showed a packed cell volume (PCV) of 36% and white blood cell count (WBC) of 18 x 10^9/L, with absolute neutrophillia and a left shift.

A surgical review of the case and the radiographic findings suggested an intestinal peforation. On the morning of the third day the baby was taken to the theatre for laparotomy. Operative findings included two small circumscribed perforations (0.5 - 1cm in diameter), a few centimetres from the ileo-caecal valve at the antimesenteric border of the terminal ileum. There was free pus in the peritoneal cavity with lots of fibrinous adhesions and a distended bladder. The perforation was closed and a thorough cleaning of the abdomen undertaken. Specimen of perforated ileum was sent for histopathology studies which revealed a neorotic tissues surrounded by polymorphs, eosinophils and giant lymphocytes. No granulomata were seen. The muscle coat was diffusely infiltrated by lymphocytes. Based on these findings a morbid anatomical diagnosis of an acute typhoid necrotizing ileitis was made.

Post-operatively, attempts were made to confirm typhoid infection as well as to trace the source of infection. Cultures of baby's blood, stool, peritoneal pus, as well as maternal blood, stool, urine and breast milk yielded no organism. Maternal blood serum showed no agglutination, but baby's blood serum was positive for both flagellar and somatic antigens.

Post-operatively, the antibiotics were changed to intravenous ampicillin (200mg/kg/day), metronidazole (7.5mg/kg/dose 8 hourly) and chloramphenicol (100mg/kg/day); nasogastric sunctioning and nil per oris were maintained.

Post-operative PCV was 32%, and the baby was transfused with packed cells. Temperature settled at 37°c two days post-operative. On the fourth post-operative day, the patient was commenced on clear fluids orally; subsequently expressed breast milk and later breastfeeding was commenced.

The patient passed normal, formed stools on the fifth post-operative day. She was discharged on the 12th post-operative day.

Discussion

Perforations of the bowel in the newborn due to causes other than typhoid have been documented in the literature.[2,3] Small bowel perforation may be spontaneous in apparently well infants, or secondary to perinatal asphyxia with artificial resuscitation, birth trauma, or diagnostic procedures. It may also complicate necrotizing enterocolitis, meconium ileus, Meckel's diverticulum or gastrogenic cysts. Most perforation in the new born tend to be gastric or duodenal.[1]

Neonates may not present the classical features of peritonitis and board-like rigidity of a perforation is unusual. Thus, the diagnosis could be very difficult and therefore, a high index of suspicion is essential in a patient with a distended and silent abdomen presenting with a history of diarrhoea preceeding the acute outset of the illness. Diagnosis may be confirmed by radiographic evidence of free air in the peritoneal cavity.[10]

In a poor socio-economic environment, bacterial and parasitic agents must also be considered in bowel perforation.[11] Typhoid perforation produces a fulminant generalized peritonitis, as in the present case. The resulting peritonitis is frequently faecal in nature.[2] The perforations are rarely sealed, because omental migration to the site is unusual on account of the rudimentary or underdeveloped nature of the greater omentum in the newborn period. Cellular response also differs in the newborn. The peritoneal cavity contains pus and pathogenic organisms in virtually all cases,[11] hence the need to consider gram negative anaerobic organisms and bacteroides in addition to *salmonella typhi*. This was the rationale for the post-operative antibiotics combination given to the patient.

References

1. Woodward TE and Woodward WE. A new oral vaccine against typhoid fever. J Inf Dis 1982; 142: 289-91.
2. Onile BA and Ougbemi Tolu. Salmonella serotypes in Ilorin, Nigeria. W Afr J Med 1986; 5: [illegible].
3. Roger B and Hupley J. Intestinal perforation in typhoid fever - a historical and state of the art review. Rev Inf Dis 1985; 7: 191-9
4. Forrest C, Eggleston and Santoshi B. Typhoid perforation - a choice of operation. B J Surg 1991; 68: 341-2.
5. Akinkugbe OO, Lewis EA, Montefiore D and Okunbadejo OA. Trimethoprim and sulphamethoxazole in typhoid. Br Med J 1968; 3: 721-2.
6. Alausa KO, Montefiore D, Sogbetun AO, [illegible] and Sobayo O. Septicaemia in the tropics: A prospective epidemiological study of 146 patients with a high case fatality rate. Scand J Inf Dis 1977; 9: 181-5.
7. Laditan AAO and Alausa KO. Problems in the clinical diagnosis of typhoid fever in children in the tropics. Annals Trop Paediat 1981; 1: 191-5.
8. Elegbeleye OO. Typhoid fever in Lagos. W Ind Med J 1976, 42: 39-42.
9. Olurin EO, Ajayi OO and Bohrer SP. Typhoid perforations. J Coll Surg Edin 1972; 17: 353-633.
10. Avery EM. Gastrointestinal disorders. In Avery EM and [illegible] HW, eds. Schaffer's Diseases of the newborn. Philadelphia, London, Tokyo. WB Saunders (Publishers) Ltd; 1984: 344-89.
11. Eggleston FC, Santoshi B and Singh CM. Typhoid Perforations of the bowel: experiences in 78 cases. Ann Surg 1979; 190: 31-5.