

# Necrotizing Enteritis In A Nigerian Child: Lessons To Learn

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## Abstract

Necrotizing enterocolitis is an uncommon gastroenterological condition. We report a 14 -year old male who was admitted with a working diagnosis of gastroenteritis. Rapid deterioration in the clinical condition culminating in death despite adequate fluid therapy and monitoring necessitated post-mortem request and hence the final diagnosis.

## CASE HISTORY

A 14-year old male was admitted with vomiting, diarrhoea, abdominal pains and moderate dehydration following voracious feeding on under-cooked beef and pork, left-over foods and confectionaries at a social gathering. Complaints of vomiting which started 10 hours prior to presentation was treated at home with oral Promethazine (Phernegan®) to no avail.

Physical examination revealed a drowsy child, not pale, anicteric and afebrile with a temperature of 37.5 °C. He weighed 43kg and was moderately dehydrated. The blood pressure was 100/ 50mmHg and the heart sounds were normal. He had hepatomegaly of 4cm but the spleen was not palpably enlarged. Other systems were essentially normal. Sexual maturity rating was 2. A diagnosis of gastroenteritis with moderate dehydration was made with bacterial food poisoning as a possibility. Full blood count, serum electrolytes and urea assay and haemoglobin electrophoresis were done. He was planned to have 3 Litres of intravenous infusion of Ringer's Lactate (with 20mL of 50%Dextrose added to each 500mL bag of infusion) over 4 hours. Intravenous chloramphenicol was to be given as 500mg 6 hourly and the vital signs were to be monitored 2-hourly.

About two hours after commencement of therapy when he had had about one and half litres of intravenous infusion, he developed rigors, cold extremities, impalpable peripheral pulses and a blood pressure of 50/?. Anti-shock dose of the intravenous Ringer's Lactate and 200mmg of hydrocortisone was given in addition to intranasal oxygen therapy. He developed generalized tonic-clonic seizures by the fourth hour of admission and these were aborted with 10mL of intramuscular paraldehyde. He thereafter lapsed into

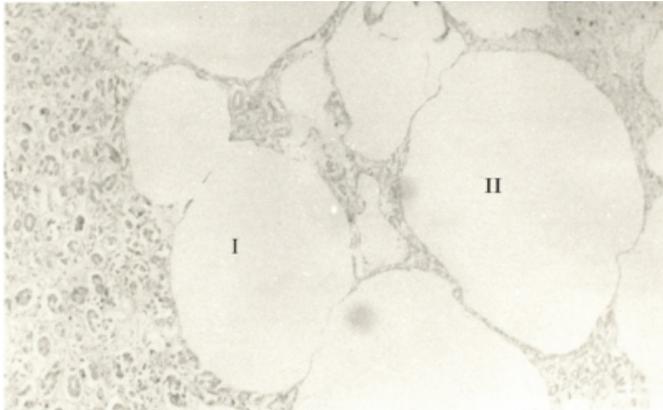
unconsciousness. Despite more than 3 Litres of intravenous fluid, the patient made no urine. At about the sixth hour of admission, he succumbed to the illness.

The Cerebrospinal fluid was macroscopically normal and the results of the other investigations were not available till after the demise of the patient. However, the haemoglobin genotype was AC and the only serum electrolyte derangement was bicarbonate of 18mmol/L. The major autopsy findings were in the gastrointestinal tract where there was complete loss of rugae along the lesser curvature of the stomach with fine nodularity of the mucosa. These raised mucosal lesions felt doughy. There was also segmented gangrene of the jejunum and ileum while the colon was not remarkable. There were numerous enlarged para-aortic and mesenteric lymph nodes. The spleen was enlarged with areas of fibrosis. There was also evidence of cerebral oedema. All the other organs were not remarkable.

Histology of the gastrointestinal tract showed numerous gas filled cysts within the mucosa of the stomach and small intestine (Figure 1). These gas filled cysts were also present within the mesenteric lymph nodes. The muscle coat was not remarkable. Cause of death was necrotizing enteritis from *Clostridium perfringens* infection.

### Figure 1

Figure shows numerous gas filled cysts (I) within the mucosa of the stomach (II)(H & E)



### DISCUSSION

Gastroenteritis is one of the leading diagnoses in a typical children emergency ward in developing countries. (1) The prevalence of gastroenteritis had reduced considerably following the introduction of Oral Rehydration Therapy by the World Health Organization (WHO) in 1990. (2) Experiences have shown that most cases of childhood gastroenteritis are self-limiting once dehydration is prevented or promptly corrected.

Bacterial food poisoning was suspected when gastroenteritis started suddenly with abdominal cramps but without fever. (3) This was especially so when the gastroenteritis started within 6-18 hours of taking those meals. (4) This may be caused by the enterotoxins of *Staphylococcus aureus* or clostridia organisms especially *Clostridium perfringens*. *Clostridium perfringens* type C is particularly known to cause necrotizing enteritis. (4) This disease follows the ingestion of large meals hence bowel distension and bacterial stasis may be contributory factors. (3)

Necrotizing enteritis is common in Papua New Guinea during pig feasts (3,4) and in the low social class where sweet potato is the main diet. (4) Although, scattered cases of necrotizing enteritis caused by *Clostridium perfringens* have been reported in Western countries, (3) it is however, not commonly diagnosed in this part of the world. This report is meant to create awareness about this disease in Nigeria and alert physicians to this possibility when managing gastroenteritis. The diagnosis of necrotizing enteritis is not commonly made in our environment probably because the self-limiting viral diarrhoeal diseases are most commonly seen in our clinical practice. Therefore, the rapid deterioration of this patient despite seemingly adequate fluid

therapy was unusual and that got us worried. The diagnosis of bacterial food poisoning was also considered because of the initial predominance of vomiting but the fact that the patient was the only person affected in the household made that diagnosis un-likely.

Stool samples were not cultured aerobically and anaerobically because it was not a routine investigation in acute watery diarrhoea in our clinical practice. Perhaps, it would have been helpful in this case. *Clostridium perfringens* was shown to cause watery diarrhoea and dysentery in a case-control study in Bangladesh but none of the subjects developed necrotizing enteritis. (5) Although, the organism was not identified in this case, the clinical and pathologic features in the patient were typical of *Clostridium perfringens* infection. The patient probably contracted the infection from eating large quantities of under-cooked beef and pork which were probably contaminated with the bowel contents of the animals as suggested by Rood and Cole. (3)

The altered sensorium at presentation and the rapid progression to shock may be due to the overwhelming effect of toxins produced by the organism. While Asmuth et al (6) felt that the mechanism by which the toxins cause shock and end organ failure is enigmatic; Wallace et al (7) ascribed death from enterotoxin-induced shock to various inter-related immunologic mechanisms. It was difficult reversing the shock in this case because of the rapidity of deterioration. It was also interesting that the patient developed cerebral oedema and anuria despite the meticulous fluid therapy. It is not clear why most of the fluid administered was diverted to the brain but it is speculative that this may also explain the altered sensorium and convulsion. However, these would be of research interest in the future.

Intravenous chloramphenicol was used in this patient because it is one of the drugs recommended for infective diarrhoea in the developing world. (2) It also has an additional advantage of high bioavailability when administered orally. The fact that the mother administered promethazine at home implied that she must have bought it off the counter. This practice of unguided drug dispensary and self-medication is common in the developing world. The regulatory bodies like the National Agency for Food and Drugs Administration and Control (NAFDAC) in Nigeria have a lot of task in this regard.

We are reporting this case because it is an uncommonly diagnosed clinical problem in this part of the world.

Awareness needs to be created about this infection because it can readily be passed for the usual viral gastroenteritis that is commonly seen in clinical practice, hence may be repeatedly missed. In addition, efforts must be made to address the problem of cultural disapproval of post-mortem examination in this part of the world in order to unravel the causes of death. This would improve the knowledge of the physicians and inform the general populace about the need to institute preventive strategies.

Food handlers and the general populace must understand the dangers involved in serving and consuming under-cooked meat. Fast foods and food left over must be re-warmed prior to consumption. Specifically, the culture of warming foods to at least 75°C in order to destroy toxins and storing them to below 4°C to prevent toxin production must be adopted. Public health departments may also need to re-institute the sanitary inspectorate system whereby physical inspection and certification of foods meant for public consumption are compulsory.

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