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Clinical and Autopsy Findings of a Six-year Old Girl with Malnutrition, Amoebic Colitis and Peritonitis

CASE 3, 1991

Presentation of the Case

A 6 – year old, severely malnourished girl was admitted to the Dhaka Hospital of the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDR,B) with intermittent fever of 20 days and bloody, mucoid diarrhoea of 17 days.

The child belonged to a poor family; the father is a daily labourer earning about $70 per month. The family lived in the city slum. The girl was said to have had no major illnesses in the past and had not received any vaccinations. She was breastfed until the age of 6 months and also given a dilute preparation of cow's milk, rice powder, sugar, and water at home. She received a normal family diet including rice curry, which consists predominantly of carbohydrate and less than adequate protein and fats. She was the third child in the family; 2 other siblings are reported to be in good health at home.

The present illness started 20 days before admission with intermittent fever but was not associated with chills and rigor. The patient had suffered from measles 20 days before admission along with fever, cough, and redness of the eyes and photophobia. For the last 17 days, there was passage of bloody, mucoid, foul – smelling stool, 6 – 8 times a day without any vomiting. The patient became anorectic and refused to take food for 5 days before admission. Her mother noticed a gradual swelling of her legs during the last 5 days.

On physical examination, the patient's body weight was 7.2 kg. and she had bilateral pitting oedema of the feet. Her weight for age was 37% of NCHS median (1). Her pulse rate was 110 per minute and was regular; respiratory rate was 30 per minute. Her rectal temperature was 37.8 degrees celcius. The child was moderately dehydrated, lethargic with sunken eyes, and appeared pale and wasted. She had no cyanosis or jaundice; the liver was just palpable below the right costal margin and appeared soft. Fine crepitations were heard on both sides of her chest. Multiple black marks of measles rash were visible on her body; oral moniliasis was also present. A provisional diagnosis of post – measles bronchopneumonia with marasmic kwashiorkor and thrush was made on admission.

The patient was rehydrated initially with intra – venous acetate solution (Na 133 mmol/l, K 13 mmol/l, Cl 98 mmol/l, HCO_3, 48 mmol/l) 400 ml, and parenteral ampicillin was started to treat bronchopneumonia. Her serum electrolyte profile on admission showed mild hypokalaemia (K 2.82 mmol/l), which was corrected by adding 17 mmol KCl/l in IV acetate solution and giving potassium by mouth. On the 3rd hospital day, stool microscopy showed vegetative forms of *Entamoeba histolytica*, and metronidazole was started. On the fourth hospital day, the patient was in shock and her abdomen became markedly distended, tense, shiny, and tender with absent bowel sounds, suggesting development of toxic colitis. Liver dullness was not obliterated. The patient was then managed in the intensive care unit. Continuous nasogastric suction was given and adequate fluid ration was maintained and nothing was given by mouth. At that time, a blood culture was drawn and parenteral gentamicin was added to cover probable Gram – negative septicaemia. Parenteral metronidazole was given. A complete blood count showed polymorphonuclear leukocytosis which may have been due to severe bacterial infection leading to sepsis. Her platelet count was normal (115,000/cmm). Serum protein and electrolytes on the fifth day of hospitalization showed hyponatraemia (Na 122 mmol/l) and severe hypoproteinaemia (serum total protein 49 g/l) which correlated with severe malnutrition along with pedal oedema. On the 5th and 6th hospital days, the patient received two units of fresh whole human blood (10 ml/kg). The fresh blood transfusions were given for correcting severe hypoproteinemia and improving her general condition.

Radiological examination of the abdomen taken in an erect posture, done on the 4th and 8th days, showed marked distension of the large bowel loops with thin walls, multiple fluid levels, and gas shadows, suggestive of intestinal obstruction. There

was no radiological evidence of perforation. Liver dullness was obliterated on the 8th day of hospitalisation. On the same day, she became severely thrombocytopenic (platelet count 15000/cmm) and she received the 3rd unit of fresh blood. In spite of all the above measures taken, the patient could not be saved and she expired on the 9th day of hospitalisation.

DISCUSSION

Dr. Hasan Ashraf. The patient stayed in the hospital for 9 days before death. During this period, the following problems were encountered: (1) diarrhoea, (2) bronchopneumonia, (3) severe malnutrition (marasmic Kwashiorkor), (4) amoebiasis, (5) toxic colitis, (6) probable intestinal obstruction, perforation, peritonitis, (7) severe hypoproteinaemia, (8) severe thrombocytopenia, (9) hyponatraemia, and (10) mild hypokalaemia.

It is probable that this patient became severely malnourished at home either by receiving foods of low calorie or from the consequences of prolonged invasive diarrhoea which causes considerable loss of micronutrients and macronutrients, or a combination of both. A decreased intake of food due to loss of appetite, withholding of food, and increased catabolism during diarrhoea might further have added to the development of severe malnutrition in this child.

Acquisition of infection leading to severe bronchopneumonia and suspected septicaemia and electrolyte abnormalities can all be explained by the severity of malnutrition. The interaction between the nutritional status and resistance to infectious diseases has long been recognised (2). The child probably had a poor immune status related to malnutrition. It is likely that the offending organism could easily colonise and invade the gastrointestinal and respiratory epithelia, giving rise to invasive diarrhoea, bronchopneumonia, and septicaemia.

As the patient was severely malnourished she was most likely to be immunocompromised and was susceptible to infection due to bacterial, viral, and protozoal agents. She had had an attack of measles about 20 days previously and subsequently developed fatal complications, i.e. post-measles broncho-pneumonia and invasive amoebiasis. So, measles infection along with its severe complications led to more malnutrition and an immunocompromised condition. The fatal complications of amoebiasis include toxic megacolon, intestinal obstruction, intestinal perforation with peritonitis, and intestinal haemorrhage. Epidemiologic and clinical features of invasive amoebiasis in a case-control study in Bangladesh by Chris Wanke et al. (3) showed that amoebiasis was associated with prolonged dysentery, measles, malnutrition, hypoproteinemia, hyponatraemia, and hypokalaemia; all these factors were present in this patient.

Post mortem studies reported from Bangladesh by Butler et al. (4) showed that 22 fatal cases of segmental necrotising enterocolitis (SNE) were associated with prolonged diarrhoea, blood and mucous in stool, abdominal distention or tenderness, shock and hypoproteinemia. All the above mentioned risk factors of SNE were also present in this case.

In spite of the clinical management, the patient's condition did not improve during the illness. The explanations for this may be: (1) long duration of dysentery prior to admission (17 days in this case), (2) severe malnutrition (37% wt./age) with oedema, (3) prior measles infection, (4) hypoproteinaemia, (5) hyponatraemia, and (6) hypokalaemia.

Dr. A. K. Azad. What is the cause of thrombocytopenia in this patient?

Dr. Hasan Ashraf. The thrombocytopenia in this patient may be due to disseminated intravascular coagulation as a result of endotoxic shock leading to consumption of platelets by the ongoing intravascular clotting process.

Dr. Mohammad Ali. The platelet count was only 15,000/cmm on day 8 after the hospital admission. Was there any evidence of bleeding in the patient?

Dr. Hasan Ashraf. No, there was no clinical evidence of bleeding in the patient. But we don't know whether there was any internal bleeding.

Clinical Diagnosis

1. Marasmic Kwashiorkor
2. Bronchopneumonia
3. Amoebic Colitis with peritonitis

Pathological discussion

Dr. Moyenul Islam. A postmortem examination was carried out 27 hours after death on this very malnourished child. Residual marks of measles rash were not visible. Approximately 50 ml of thin purulent peritoneal fluid was present in the hypogastric and right paracolic areas of the abdominal cavity. The serosal surface of distal ileum showed reddish purple discolouration, and 10 cm proximal to the ileo-caecal junction two white necrotic patches, up to 1 cm in diameter each, were present in the ileal wall. The caecum was lightly adherent to the surrounding tissue and the rectosigmoid was covered by thin purulent fluid. The small bowel mucosa was ulcerated over the necrotic areas. The mucosa of the rectosigmoid showed large ulcers with irregular outline, the intervening mucosa being oedematous. The proximal colon showed smaller and fewer ulcers; the caecum showed only red dots of pin-head size. Other abdominal organs appeared unremarkable. The lower lobes of both lungs were congested. A circumscribed haemorrhagic

area, up to 2 cm in diameter, was present in the posterior aspect of the right lower lobe of the lung.

Microscopic examination revealed deep undermining ulcers in the colon (Fig. 1) consistent with amoebiasis, but trophozoites of *E. histolytica* were not seen since the child was already treated with metronidazole. The distal ileum showed full thickness necrosis of the wall and there was focal suppurative inflammation on the serosal surface (Fig. 2). The proximal jejunal mucosa showed a small number of trophozoites of *Cryptosporidia* attached to its brush-border surface. The haemorrhagic area in the lung showed a fresh infarct and an adjoining pulmonary artery contained a recent thrombus (Fig. 3).

Figure 1. Undermining colonic ulcer; no *E. histolytica trophozoites* are seen. H and E stain (33x)

Figure 2. Full thickness necrosis of wall of distal ileum and acute inflammatory infiltration. H and E stain (33x)

I would agree with Dr. Ashraf that following an attack of measles, this child became severely malnourished and immunocompromised and later developed amoebic colitis. Although she received treatment for amoebiasis, she succumbed to its complications, i.e. suppurative peritonitis and perhaps sepsis. The small pulmonary embolus (and infarct) was a terminal event in this patient.

Figure 3. Antemortem thrombus in pulmonary artery with fresh haemorrhagic infarct in adjoining lung. H and E stain (66x)

Prof. M.R. Khan. Was *E. histolytica* found in the brain?

Dr. M. Islam. We do not know whether *E. histolytica* was present in the brain substance because it was not examined.

Dr. Jena Hamadani. Vegetative haematophagus trophozoites of *E. histolytica* were found in the stool on the 3rd hospital day but *E. histolytica* trophozoites were not found in the colon tissue. How do you explain this?

Dr. M Islam. In our previous series of post-mortem examinations, no *E. histolytica* could be detected after the patients had already received metronidazole for more than 24 hours. In this case, the patient received metronidazole for 7 days and so likewise *E. histolytica* was not found in the colon tissue.

Anatomical Diagnosis

1. Kwashiorkor
2. Amoebic colitis
3. Suppurative peritonitis

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