Necrotizing Enteritis

by A.P. Mukherjee
MRCP (E)
Lecturer,
Department of Medicine,
W.C. Foong
MBBS (Melb), F R C S (Eng), F R C S (Edin)
Lecturer,
Department of Surgery,
and B.R. Ferguson
MD
Lecturer,
Department of Pathology,
Department of Medicine, Surgery & Pathology, University of Malaya, Kuala Lumpur.

BECKERMANN (1946), from Germany, first described a number of cases of severe illness of acute onset with abdominal pain and slight rigidity of abdomen associated with profuse diarrhoea, vomiting and mild fever. These cases were often fatal and were due to necrotic inflammation of several areas of the intestine, especially in the jejunum.

Epidemic outbreak of necrotizing enteritis had been described from New Guinea, Murrell (1967). Sporadic cases had been described from the United Kingdom, Greville Young, (1949); the United States of America, Patterson and Rosenbaum (1952); Australia, Goulston et al (1965); Indonesia, Gan et al (1962); Thailand, Headington et al (1967) and Uganda, Wright (1967). The association of the disease with pork eating has been described by Murrell (1967). A case is described here which had the features of necrotizing enteritis. Causative factors of the disease are discussed.

Case Report

A 12-year-old-female Chinese was admitted with a history of colicky abdominal pain associated with passage of mucus and loose watery stool, "red" in colour for 3 days, and vomiting of 2 days' duration. She had been perfectly healthy prior to her present illness and no one else in the family had a similar illness. On examination, she was well built but pale, dehydrated, conscious and co-operative. Pulse was 148 per minute regular, BP — 95/65 mm Hg. She was febrile with a temperature of 99°F. There was no neck stiffness. Respiratory, cardiovascular system and central nervous system were normal. Relevant physical findings were confined to the abdomen which was grossly distended and diffusely tender. Bowel sounds were infrequent. Liver dullness was obliterated. Rectal examination was normal. A diagnosis of acute gastroenteritis with paralytic ileus was made.

Investigation

Haemoglobin was 13.1 G/100 ml, WBC 3400 per μl, with neutrophils of 53 per cent, lymphocytes 44 per cent and monocytes 3 per cent, erythrocyte sedimentation rate was 6 mm/hr. (Westergren). Blood urea was 48 mg/100 ml, sodium 128 mEq per litre, potassium 3.1 mEq per litre. A random blood sugar was 108 mgm/100 ml; urine analysis was within normal limits. An electrocardiogram revealed atrial tachy-cardia with a heart rate of 180 per minute. A straight X-ray of the abdomen revealed dilated small bowel, with multiple fluid levels; the appearance was suggestive of paralytic ileus.

Blood and stool cultures and blood for Widal-Weil-Felix were done after admission. She was treated with chloramphenicol 500 mgm 6 hourly I.M., and her dehydration was corrected by intravenous therapy. Her condition progressively deteriorated after admission. She developed high fever, bleeding per rectum and severe abdominal distension. Because of the massive bleeding per rectum, she was then referred to the surgeon.

Laparotomy was carried out on the 4th June 1970. Abdomen was markedly distended and on opening the peritoneum greenish fluid was seen.

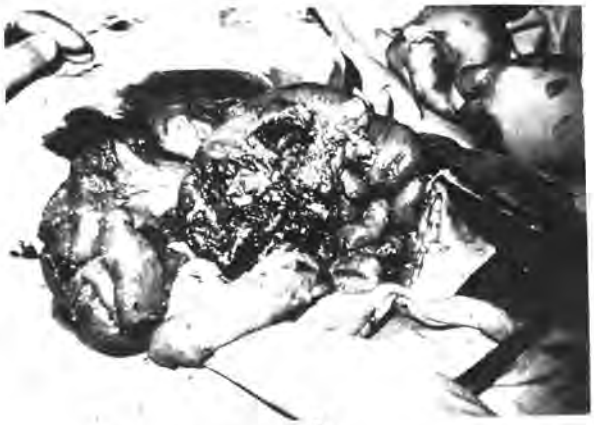

Fig 1
Portion of the small bowel with gangrenous necrosis and perforation on the second laparotomy.

Isolated segments of small bowel extending from the jejunum to the ileum was haemorrhagic in appearance. About 1½ feet of small bowel, just 2 feet proximal to the ileo-caecal junction, was markedly haemorrhagic in appearance, with some spotty dark coloured areas. No perforation of any part of the bowel was detected. The whole of the small intestine and, to a smaller extent the large bowel, was distended. The arterial arcade of the small bowels was patent. As it was impossible to define the area of impending gangrenous bowel and in view of the widespread involvement of the whole of the small gut, no resection of the small intestine was done but decompression of the bowel was carried out to relieve the abdominal distension.

Post-operatively, she developed a high fever, partly controlled by penbritin. Soon after, she developed intermittent colicky abdominal pain, requiring frequent doses of pethidien for relief. Brown loose stools persisted. Because of the persistence of colicky abdominal pain, and the provisional diagnosis of mechancial obstruction, laparotomy was again carried out on 17th June 1970. Almost the whole segment of ileum, about 2 feet from the ileo-caecal junction, was necrotic and perforated. Multiple perforations of the jejunum were also found **(Figs. 1 and 2)**. The necrotic and non-viable small intestine was resected and anastomosed.

On the 20th June, 1970, because of the discharge of faecal material from the drainage sites, laparotomy was again carried out. Further perforations proximal to the previous anastomosis and at other sites were detected. Again non-viable gut was resected and perforation closed. On 22nd June 1970, because of

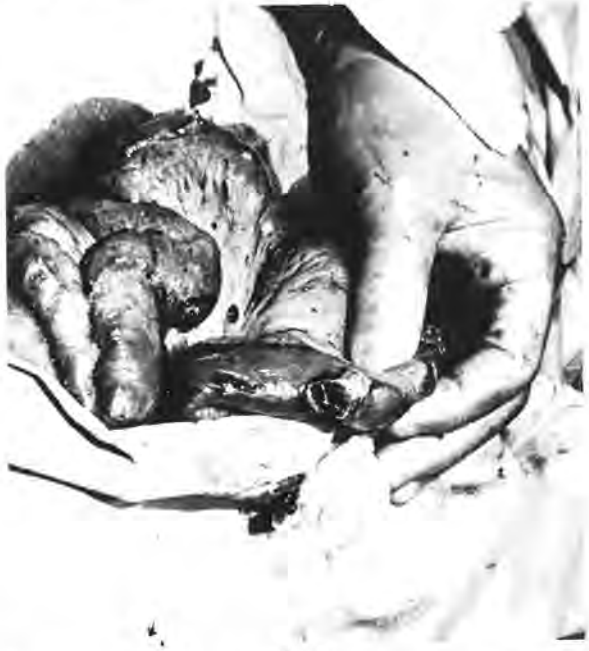

Fig 2
Appearance of the small bowel on the third laparotomy, showing the punch-out appearance of the perforation.

further discharge of faecal material through the drainage sites, laparotomy again was carried out and further perforations were detected. Resection of non-viable gut was carried out. However, the patient's condition deteriorated soon after the fourth laparotomy and died. Widal and Weil-Felix tests were available on the 2nd week and both were normal. No pathogen was isolated from stool culture.

Histological examinations of the resected gut revealed evidence of an early organising fibrinopurulent peritonitis involving the entire serosal surface. The luminal surface of the small intestine contained only a few focal patches of mucosa. Some of these consisted of only a few intestinal glands with the surface epithelium sloughed off. The submucosa was moderately thickened, the lymphatic vessels were dilated and there were a moderate to marked increase in histiocytes, plasma cells and lymphocytes.

In areas of necrosis, there were also a large number of polymorphonuclear leucocytes. The necrosis of the mucosa and submucosa in some areas extended into the inner layer of smooth muscular wall, showing degeneration of the smooth layer without accompanying inflammation. The micro-organisms seen on special stain consisted of a mixture of gram-positive rods; no gram-negative rods were seen. The histologic picture in all three specimens was that of a non-

specific necrotizing enteritis with marked submucosal oedema with early fibro-proliferative activity.

Post-mortem examination findings were mainly confined to the abdomen which revealed evidence of diffuse fibrino-purulent peritonitis. Numerous foci of necrosis were found throughout the small intestine and proximal colon. Numerous fibrous and fibrinous adhesions were found between all peritoneal surfaces. The peritoneal cavity contained thick yellow white purulent exudate. The mesentery was thickened, haemorrhagic and contained numerous large firm lymph nodes. The lungs were heavy and congested with focal areas of atelectasis. The heart was dilated and numerous subendocardio-petechial haemorrhages were found, particularly in the left ventricle.

Conclusion The findings were that of necrotizing enteritis of obscure aetiology.

Discussion

Since the first description of the disease from Germany by Beckermann (1946), similar cases have been reported from other parts of the world. Different authors have used different names to describe what appears to be the same disease. Greville-Young (1949) used the term acute jejunitis, whereas Patterson and Rosenbaum described it as enteritis necroticans. Goulston et al preferred to use the term ulcerative jejunitis.

Although there still exists a greal deal of controversy about the terminology of this disease, there is also no uniformity about the aetiology of the disease. Oakley thought it to be due to Clostridium Welchii type F., whereas Schutz believed it to be due to Cl. Welchii type A., Murrell et al postulated Cl. Welchii type C. to be the cause of this disease. Although most of the authors tend to link this disease to the infection by some strains of Cl. Welchii, Greville-Young was of the opinion that this is a variant of Crohn's disease. Kravetz and Brazenas thought it to be due to a virus and related this disease to enteritis gravis which is sometimes associated with infectious hepatitis. In the case reported here, there was no evidence of Clostridium Welchii infection. The culture of the stool and intestinal contents of post-operative material did not grow Cl. Welchii. However, antibody titre was not estimated against Cl. Welchii.

Although the terminology of the disease and the causative mechanism of necrotizing enteritis have not been firmly settled, it is important to recognise the condition, as early diagnosis and correct management may reduce the mortality and morbidity. In the tropics, it is important to differentiate this disease from the Salmonella enteritis and in children, it can simulate intussusception.

The surgical treatment of this pathological state is the correction of dehydration, electrolytes imbalance, hypovalaemic shock and resection of necrotic gut. However, in the above reported case, the disease process at the time of laparotomy was widespread involving multiple segments of the small gut, without well demarcated or obvious gangrene of the small bowel. Post-mortem findings revealed more extensive involvement than at first realised, in that the disease process extended throughout the whole gastrointestinal tract, involving patches of the oesophagus, stomach and colon.

Whether radical resection of the diseased small bowel, or any portion of the small bowel that had necrosed mucosa, with or without normal seromuscular layered coat during the first operation, would have helped remained uncertain. However, from the study of this case, it is felt that earlier resection is strongly recommended, especially when the area of involvement is not extensive and this might prevent the occurrence of perforation.

Summary

A case of necrotizing enteritis in a young Chinese girl is described here. The aetiology and the management of the disease is discussed.

Acknowledgement

We wish to thank Dr. M. Somasundaram, Senior Lecturer in Medicine, for allowing us to investigate the patient under his care and to Che Rohani for secretarial assistance.

References

1. Beckermann, F. and Lass (1946). Arzt. Wschr. *1,* 329.
2. Gan, G.H.; Sukono, D., Satari, S.; Sujudi, R. and Njoo, S.M. (1962). *Madj. Kedokt. 12,* 9.
3. Goulston, K.T.; Skyring, A.P.; McGovern, V.J. (1965). *Ann. Int. Med. 14;* 57.
4. Greville Young (1949). *Lancet. 1,* 1005.
5. Headington, J.T.; Sathornsumathi, S.; Simark, S. and Sujatanon, D.W. (1967). *Lancet, 1,* 802.
6. Kloos, L. and Brummond, W. (1951). *Z. Hyg. 132,* 64.
7. Kravetz, R.E.; Brazenas, N.V. (1963). *Arch. Int. Med. 112,* 179.
8. Murrell, T.G.C.; Eggerton, J.R.; Rampling. A.; Samels, J. and Walker, P.D. (1966). *Lancet. 1,* 217.
9. Murrell, T.G.C. (1967). *Aust. Ann. Med. 16,* 4.
10. Oakley, C.L. (1949). *Brit. Med. J. 1,* 269.
11. Patterson, M. and Rosenbaum, H.D. (1952). *Gastroenterology. 21,* 110.
12. Schutz, F. (1948). *Dtsch. Med. Wschr. 73,* 176.
13. Wright, D.H. (1967). *Lancet. 1,* 953.