THE CLINICAL SPECTRUM OF AMOEBIC COLITIS*

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The importance of a clinical classification in both the acute and chronic forms of ulcerative colitis has been recognized as providing a useful approach to the therapy and prognosis of the disease. The acute attack of ulcerative colitis is commonly referred to as being mild, severe or fulminating. In its chronic form, the disease has been described as running a chronic continuous or chronic intermittent course.[1]

The clinical presentation of acute amoebic colitis and its subsequent course is in many respects similar to that seen in ulcerative colitis, and in the absence of demonstrating *Entamoeba histolytica* in the warm stool, rectal scrapings or rectal biopsy, it may be virtually impossible to distinguish the two diseases.[2-4] To date, however, no classification for the clinical presentation of amoebic colitis has gained widespread acceptance.

The purpose of the present article is to suggest a clinical classification for amoebic colitis based on selective representative cases seen in this hospital, and to demonstrate its therapeutic and prognostic usefulness (Table I).

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CASE REPORTS

1. Acute Amoebic Colitis (Mild Form)

Case 1. A 35-year-old Coloured male presented to the Medical Outpatient Department with a 2-week history of diarrhoea with blood and mucus in the stools. He was otherwise well. A warm stool was taken for microscopy and culture, tetracycline 250 mg every 6 hours was prescribed, and he was referred to the gastro-intestinal sigmoidoscopy clinic. The report of the warm stool examination was obtained when he arrived for sigmoidoscopy 2 days later; numerous *E. histolytica* having been found. By this time his symptoms had largely regressed, sigmoidoscopy showed a diffusely reddened mucosa and a rectal scraping showed the presence of myriads of dead amoebae. Emetine-bismuth-iodide, Diodoquin and chloroquine were added to his therapy and he made an uneventful recovery.

TABLE I. CLASSIFICATION OF AMOEBIC COLITIS

	Distribution	*Complications*	*Treatment*	*Frequency*
Acute amoebic colitis				
Mild	Rectal or rectosigmoid. Caecal involvement—not clinically apparent	Rare. Amoeboma occasionally	Emetine or metronidazole, tetracycline, Diodoquin—outpatient treatment	Common
Severe	Often diffuse with right-sided involvement	Local perforation with abscess formation occasionally amoeboma	Hospitalization and daily assessment. Emetine, metronidazole, tetracycline, Diodoquin (?chloroquine). Attention to fluids and diet	Less common
Fulminating	Usually diffuse with right-sided involvement (including terminal ileum)	Paralytic ileus. Mucosal sloughing. Toxic or inflammatory megacolon. Free or local perforation. Haemorrhage	Hospitalization with intensive care. Nasogastric suction. Intravenous rehydration and electrolyte repletion. Emetine and intravenous antibiotics. Surgery if: (*a*) toxic megacolon unresponsive to conservative treatment; (*b*) perforation; (*c*) severe unresponsive electrolyte depletion; (*d*) uncontrolled bleeding	Infrequent. Common in Bantu or elderly subjects
Chronic amoebic colitis				
Continuous or intermittent	Often recto-sigmoid. May be diffuse	Debility. Protein loss. Anaemia	Ambulant oral or hospitalization for combined therapy if severe	Infrequent
Amoeboma	Localized, usually caecum or rectosigmoid	Confused with carcinoma	Conservative. Avoid surgery	Infrequent
Postamoebic colitis				
Mild	Usually localized	Rare	Salazopyrin. Rectal steroids	Rare
Severe and fulminating	Diffuse	Debility, protein loss, anaemia. Perforation. Toxic megacolon	Systemic and rectal steroids. Salazopyrin. Colectomy	Rare

Case 2. An 11-year-old White boy presented with mild diarrhoea of 3 weeks' duration. The stools contained blood and mucus. He was apyrexial and the physical examination was normal. The erythrocyte sedimentation rate and white blood cell count were normal. Sigmoidoscopy showed a diffusely reddened, friable mucosa and a barium enema was normal. A diagnosis of ulcerative colitis was made and prednisolone enemata were started. He was referred to the sigmoidoscopy clinic 4 days later and the sigmoidoscopic appearances were unchanged. However, rectal scrapings showed the presence of actively motile amoebae and he was admitted for Emetine and Diodoquin therapy. He made an uneventful recovery.

Case 3 (amoeboma). A 60-year-old Coloured male presented with a 3-week history of passing blood in his stools. Physical examination was normal. Rectal examination showed a rectal mass of cartilagenous consistency at 5 cm and a confident diagnosis of rectal carcinoma was made, and barium enema showed a rectal mass with an abnormal descending colon. Despite 3 negative rectal biopsies the patient was prepared for operation on the basis of rectal carcinoma or ischaemic colitis. The day before operation he was sigmoidoscoped in the Gastro-Intestinal Clinic and rectal scrapings showed motile amoebae. His symptoms subsided rapidly on metronidazole but it took some weeks for the barium enema and sigmoidoscopy to return to complete normality.

2. Severe Colitis

Case 4. A Spanish sailor presented with a 1-week history of explosive diarrhoea. He had passed up to 25 stools a day. On examination he was found to be moderately dehydrated and there was tenderness to palpation in the left iliac fossa. Sigmoidoscopy was impossible due to the profuse diarrhoea, the stool containing blood and pus. A warm stool at the time showed numerous red blood cells and pus cells, but no amoebae were seen. He was hospitalized, given adequate fluids by mouth but no other food, and 3 further stool examinations were done. These also failed to demonstrate amoebae. There was no change in his condition and after a further 4 days a repeat sigmoidoscopy was attempted. Stool resembling 'anchovy sauce' poured from the sigmoidoscope, but a brief view of the rectal mucosa showed a diffusely reddened mucosa with black, necrotic ulcers 2 - 6 cm in diameter undermining the mucosa at 15 cm. Rectal scrapings were taken from the area and these showed numerous amoebae. Intensive oral rehydration was given together with intramuscular Emetine, tetracycline and Diodoquin. He made a rapid recovery and was able to board the next tanker for Spain.

Case 5. A 36-year-old White male was admitted to hospital from a mental institution. He had been a compulsive alcoholic and had a 3-month history of passing blood in his stools. For 9 days there had been severe, bloody, mucoid diarrhoea and he claimed to be having bowel actions almost continuously. Abdominal pain had been present for 1 day. Examination showed that he was ill, sweating with a temperature of 100·2°F and a pulse rate of 120/minute. The abdomen was distended and diffusely tender with slight generalized guarding and rebound tenderness. An X-ray of the abdomen showed a dilated colon. The erythrocyte sedimentation rate was 50 mm/hour (Westergren), white blood cell count 12 000/mm³ and haemoglobin 12 g/100 ml. Serum sodium and potassium were 132 mEq/litre and 2·6 mEq/litre respectively. Blood cultures were negative. Proctoscopy showed a diffusely reddened and friable mucosa but no ulcers. Rectal scrapings were positive for amoebae. Because of his toxic state and the abdominal signs, oral feeding was stopped and intravenous therapy commenced with careful attention to electrolyte replacement. The surgeons were notified and intramuscular Emetine and chloroquine were given. His general state was monitored twice daily and daily X-rays of the abdomen were taken. His condition remained unchanged for 48 hours and then started improving. The abdominal signs subsided, the pulse rate and temperature gradually diminished and he was able to take oral fluids as well as metronidazole, chloroquine and Diodoquin. A repeat sigmoidoscopy 13 days later showed scanty healing ulcers with oedematous surrounding mucosa. Recovery was complete.

3. Fulminating Colitis

Case 6. A 60-year-old White male was admitted with a 3-week history of bloody diarrhoea. The stools had become progressively more frequent and he became acutely ill 2 days before admission. On admission he was dehydrated, pyrexial and had tachycardia of 120/minute. The abdomen was moderately distended and tender. The haemoglobin was 10 g/100 ml, erythrocyte sedimentation rate 55 mm/hour (Westergren) and white blood cell count 14 500/mm³. Serum sodium was 135 mEq/litre and serum potassium 3·0 mEq/litre. Two warm stools were sent for examination and showed blood and pus but no amoebae. He was rehydrated by intravenous therapy, but oral foods were not discontinued. Proctoscopy was difficult because of the faecal stream, but the mucosa appeared reddened. Rectal scrapings were not taken. While awaiting the results of further stool examinations tetracycline was given, but his condition deteriorated with increasing abdominal distension and tenderness. Two days later he became acutely ill and, despite intravenous therapy and nasogastric suction for 24 hours, he became hypotensive and died. Autopsy showed a diffuse amoebic colitis with areas of extreme sloughing of the mucosa and perforation of the colon.

Case 7. A 73-year-old Bantu male was admitted with a 3-week history of diarrhoea with blood and mucus. He was dehydrated, pyrexial and toxic. The abdomen was grossly distended and a radiograph showed a few fluid levels in the small bowel with a grossly distended colon. Proctoscopy showed an ulcerated mucosa and rectal scrapings showed the presence of actively motile amoebae. Despite rehydration, blood transfusions and a 6-day course of Emetine, he remained acutely ill. Repeat rectal scrapings failed to show amoebae. The serum potassium levels were found to be in the region of 2 mEq/litre after 100 mEq of intravenous potassium. Hypokalaemia was corrected after the administration of a further 200 mEq potassium intravenously, and Emetine, chloroquine and metronidazole were given without improvement. An X-ray of the abdomen showed distended small bowel and marked dilatation of the descending colon (Fig. 1). At this stage the addition of corticosteroids to the regimen was advised but not

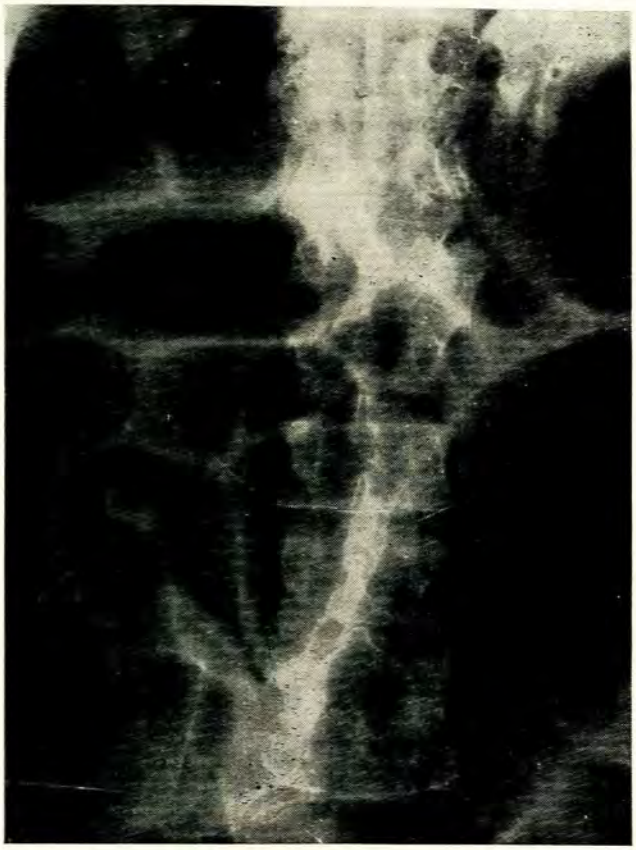

Fig. 1. Case 7. Radiograph of abdomen (supine) showing small and large bowel distension with gross dilatation of sigmoid colon in patient with acute fulminating amoebic colitis ('toxic megacolon').

instituted, and surgery was contemplated pending some improvement in his general condition. This did not occur and he died 10 days after admission. Necropsy showed a diffusely ulcerated colon with hardly any recognizable mucosa.

Case 8. A 36-year-old Bantu male was admitted to the surgical wards with a rectal abscess. This was incised and drained and he was discharged. He was readmitted one week later with pain in the left side of the abdomen and passing blood and mucus in the stools. On examination he was extremely ill with a high fever and a tender mass in the left iliac fossa. The haemoglobin was 9·5 g/100 ml, erythrocyte sedimentation rate 94 mm/hour (Westergren) and white blood cell count 8 700/mm^3. *E. histolytica* were found in the warm stool. Two days later he started vomiting and developed colicky abdominal pain and the abdomen became distended with excessive bowel sounds. A radiograph of the abdomen showed a dilated transverse colon but a normal descending colon. Nasogastric suction, intravenous fluid and electrolyte replacement and intravenous tetracycline with intramuscular Emetine were given, and he was transferred to a medical ward. He remained extremely ill and approximately 3 weeks later passed a 230-mm cast of bowel mucosa which included circular muscle layer when examined histologically. A Gastrografin enema showed a very large dilated transverse colon but no obstruction (Fig. 2). He was maintained on intravenous therapy and nasogastric suction, and laparotomy was seriously considered to exclude the presence of an obstruction. However, his condition improved before surgery was undertaken. The abdominal signs subsided and he made a good recovery. When last seen, some 2 months after discharge, he was well but complained of mild attacks of diarrhoea. Unfortunately he has been lost to further follow-up study.

Case 9. A Bantu male was admitted severely ill with a 4-day history of abdominal pain and bloody diarrhoea. On examination his pulse was 120/minute and temperature 99°F and the haemoglobin level was 9·5 g/100 ml. The abdomen was distended and amoebae were not found in the stool. Two days later he developed generalized abdominal pain and the abdomen became markedly distended. An abdominal radiograph showed a dilated transverse colon and small bowel fluid levels. Laparotomy showed turbid free fluid in the abdomen and several necrotic patches in the rectum and sigmoid flexure. The upper rectum and sigmoid, descending and transverse colon was excised and the rectal stump closed. The colon showed vast numbers of amoebae and severe sloughing. Despite anti-amoebic therapy the ascending colon sloughed at the colostomy and 5 days later the right hemi-colon and 460 mm of distal ileum was resected. Histology showed severe amoebic

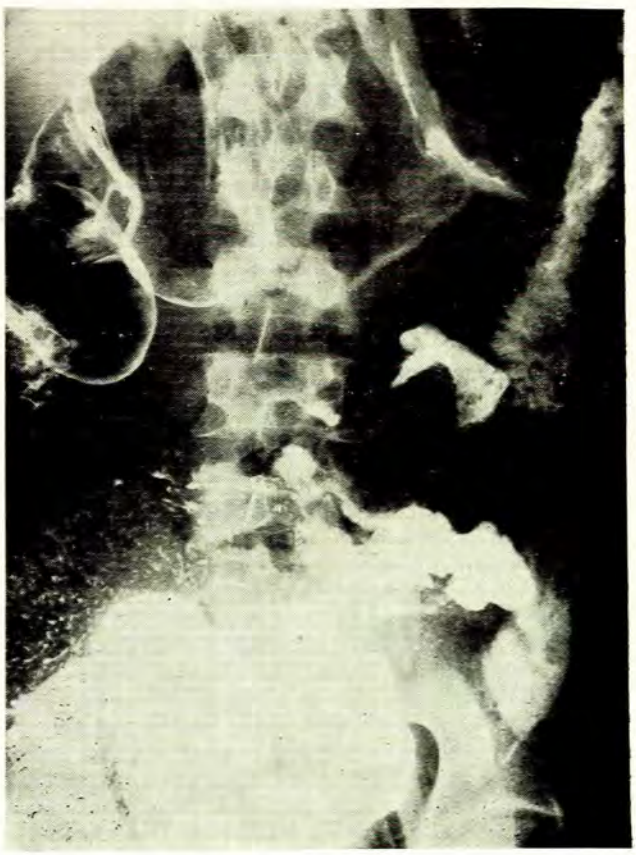

Fig. 2. Case 8. Gastrografin enema on patient with acute fulminating amoebic colitis showing gross distension of transverse colon.

ulceration of the colon and distal ileum. His postoperative course was stormy but with intensive medical measures he made a slow recovery to complete normality after the drainage of a liver abscess. He was fitted with an ileostomy bag and 1 year later a barium enema showed that the rectal stump consisted of a tube of fibrous tissue. Surgery was a life-saving procedure in this patient and the appearance of the rectal stump leaves little doubt that he would have suffered from severe postamoebic colitis had he survived without colectomy.

Case 10. A 7-month-pregnant Coloured female aged 23 years presented with a 3-week history of bloody diarrhoea. Sigmoidoscopy revealed amoebae and she was started on metronidazole, chloroquine and Reverin. Forty-eight hours after admission she developed generalized abdominal pain and laparotomy revealed a free perforation of the hepatic flexure. The perforated bowel was exteriorized as a colostomy and showed large necrotic ulcers with normal intervening mucosa. After extensive medical treatment the ulceration of the mucosa healed; she delivered a 30-week premature infant (which subsequently died) and the exteriorized colon was resected and an end-to-end anastomosis carried out after 6 weeks. She made an uncomplicated recovery.

4. Chronic Amoebic Colitis

Case 11. A 13-year-old Coloured boy was admitted with a 10-year history of diarrhoea. He had passed from 3 to 9 stools daily with continued blood and mucus on most occasions. The family had accepted this as virtually normal and he had occasionally been given costive medicines. The physical examination was non-contributory. The haemoglobin level was 10·5 g/100 ml, erythrocyte sedimentation rate 6 mm/hour and white blood cell count 20 000/mm³. Sigmoidoscopy showed well-defined ulcers with normal intervening mucosa, and a rectal scraping confirmed the presence of amoebae. Rectal biopsy showed areas of chronically inflamed rectal mucosa with *E. histolytica* attached to surface mucus. The serum sodium was 134 mEq/litre, potassium 3·3 mEq/litre and plasma albumin and globulin were 3·3 and 2·2 g/100 ml respectively. He was treated with Emetine, chloroquine, tetracycline and Diodoquin, and after a few days he passed his first normal stool for 10 years. A repeat sigmoidoscopy showed a reddened mucosa only, and a barium enema was completely normal.

Case 12. A 47-year-old White male was referred for investigation of diarrhoea. A barium meal, stool cultures and blood count carried out elsewhere were normal. The diarrhoea had been present intermittently for 17 years, and at its worst he had passed 5 or 6 mucoid stools a day. Sometimes the stools had contained blood. Examination showed a physically well man; he was not anaemic and the abdominal examination was negative. A barium enema showed mild ulcerative changes of the rectum and sigmoid colon, and in addition there was a smooth filling defect in the transverse colon (Fig. 3). Sigmoidoscopy showed numerous small ulcers with a reddened mucosa, and rectal scrapings were positive for *E. histolytica.* Treatment with intramuscular Emetine, tetracycline and Diodoquin was given with rapid recovery. Since then the patient has been so well that he has refused a repeat examination to assess the possibility of an amoeboma in the transverse colon.

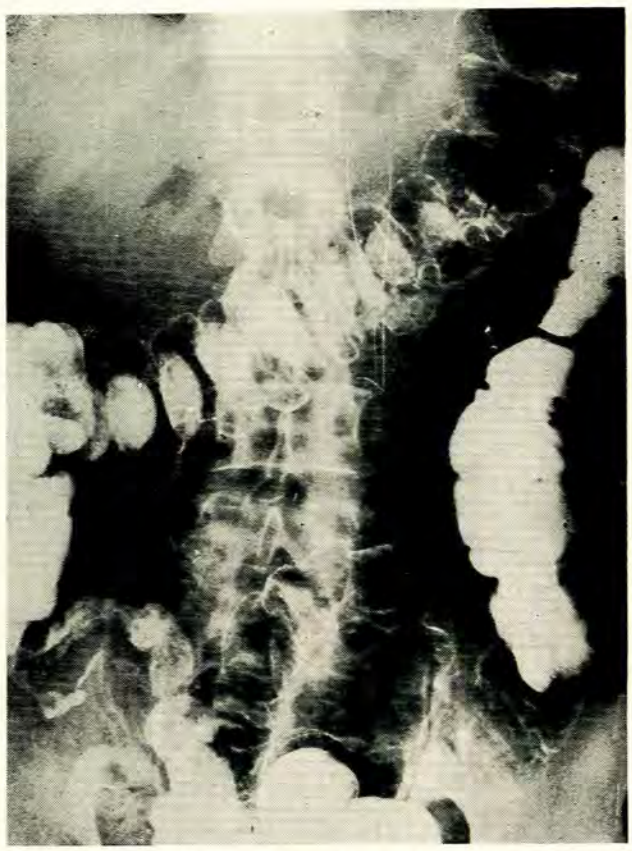

Fig. 3. Case 12. Barium enema in a patient with chronic intermittent amoebic colitis showing a filling defect (presumed to be an amoeboma) in the transverse colon.

Case 13. A 35-year-old Bantu male was admitted with a 9-year history of recurrent rectal bleeding with the passage of mucus. The episodes lasted about 3 weeks, followed by a period of well-being for approximately a month. Periumbilical cramp had been present for 1 year and there had been recurrent episodes of haematemesis. On examination he was pyrexial, showed evidence of weight loss, and there was guarding over the colon. The haemoglobin level was 6 g/100 ml, erythrocyte sedimentation rate 3 mm/hour (Westergren) and white blood cell count 21 000/mm³. Sigmoidoscopy showed an oedematous bowel but there were no ulcers, and rectal scrapings were positive for amoebae. Total serum protein was 4·4 g/100 ml, the albumin being 2·3 g/100 ml. The serum iron was low and an X-ray of the abdomen showed gross gaseous distension only. A barium enema showed ulceration in the descending colon. He was transfused and given metronidazole, Emetine, tetracycline, chloroquine and Diodoquin. By the third day he felt well, the pyrexia settled and he was discharged.

5. Chronic Amoeboma

Cases 14, 15 and 16. Three non-White patients were admitted to one of the surgical wards within a period of 2 months. Each presented with rectal bleeding and was thought to have a rectal carcinoma. Sigmoidoscopy showed

a firm, raised non-ulcerated area at 6, 8, and 10 cm respectively, and biopsies showed this to consist of a chronic inflammatory reaction with *E. histolytica* embedded in the fibrous tissue. Treatment with Emetine and chloroquine was given in all cases and one of the patients was referred to the Gastro-Intestinal Clinic for further follow-up study due to slow improvement in his symptoms. Repeat sigmoidoscopies over 4 months in this patient showed a persistently raised, hard area at 10 cm, and biopsies were all of a chronic inflammatory nature. There was a slow regression to virtual normality thereafter.

6. Postamoebic Colitis

Case 17 (mild form). A 23-year-old Bantu female presented to the Medical Outpatient Department with a 2-month history of diarrhoea with blood and mucus in the stools. Proctoscopy showed a reddened mucosa with small ulcers and a warm stool examination confirmed the presence of motile *E. histolytica*. She was admitted for intramuscular Emetine treatment for 10 days and given chloroquine, Diodoquin and tetracycline in addition. She improved on this regimen and was discharged. However, at follow-up one month later, she still complained of mild diarrhoea with occasional bloody mucoid discharge and was referred to the Gastro-Intestinal Clinic for reassessment after examinations of warm stools were shown to be negative. A barium enema now showed mild ulcerative changes in the rectosigmoid region, the rest of the bowel being normal (Fig. 4). Sigmoidoscopy showed a hyperaemic and friable mucosa, but no ulcers were seen and rectal scrapings for amoebae were negative. Histologically the mucosa was indistinguishable from the changes seen in ulcerative colitis. Metronidazole failed to influence the course of the disease, but rectal prednisolone produced a marked improvement in her symptoms. She now attends the ulcerative colitis clinic at the hospital, and remains well on maintenance doses of Salazopyrin and prednisolone enemas when necessary.

Case 18 (severe postamoebic colitis). A 32-year-old Bantu male was admitted on 10 March 1967 with a 3-week history of bloody, mucoid diarrhoea, abdominal cramps and vomiting. Examination showed a well-looking man with no dehydration, but his abdomen was distended and tender along the distribution of the colon. Examinations of the stools showed large numbers of *E. histolytica* and he was treated with Emetine, chloroquine and intravenous tetracycline. Profuse bloody diarrhoea persisted and a surgical opinion was obtained on the third day because of increasing abdominal distension, generalized abdominal tenderness and radiological evidence of an ileus. Sigmoidoscopy showed a grossly oedematous mucosa. Decompression with nasogastric suction was commenced and blood transfusions were given. After 4 stormy days the abdominal signs settled, and blood disappeared from the stools. Repeated warm stools were negative for amoebae. However, the diarrhoea continued unabated, and now consisted of 7 or 8 foul-smelling liquid stools per day, containing large quantities of pus. Attempts at a barium enema failed due to incontinence, but one film showed a 'double mucosal line' as seen in acute ulcerative colitis. After an episode of left ileofemoral thrombosis the patient continued to lose weight and developed dependent oedema. The erythrocyte sedimentation rate remained elevated, and despite repeated blood and albumin transfusions and a variety of treatments, including 2 further courses of anti-amoebic therapy, the haemoglobin level dropped and the serum albumin decreased to 1·9 g/100 ml with a globulin level of 4·6 g/100 ml. Prednisolone enemata appeared to produce some temporary improvement but failed to halt the downward course of his illness. Colectomy was advised but not carried out. He continued to deteriorate, developed evidence of protein-calorie malnutrition and died on 14 July 1967, 4 months after his initial amoebic infection.

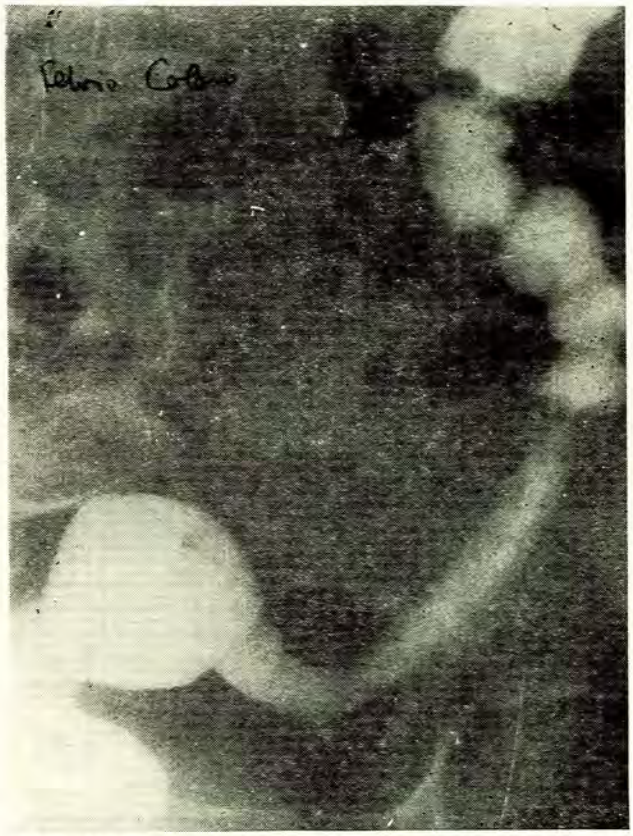

Fig. 4. Case 17. Barium enema in patient with mild post-amoebic colitis showing an ahaustral (lead pipe) sigmoid colon.

Case 19. A 45-year-old Coloured female was admitted with a 3-week history of bloody diarrhoea and a high fever. *E. histolytica* were found in the rectal scrapings and she was started on metronidazole 800 mg three times a day. There was no improvement in her general or gastro-intestinal symptoms after 5 days and a stool examination at this stage again showed abundant amoebae. The treatment was then altered to Emetine 1 gr. intramuscularly daily. Her general condition improved by the third day and amoebae could no longer be found in the stool by the 5th day or thereafter. However, despite a reduction in the number of stools she continued to have bloody diarrhoea about three times a day and started a brisk rectal haemorrhage on the 10th day. Although there was slight abdominal distension and tenderness at this stage an abdominal X-ray did not

show any gross colonic distension. Sigmoidoscopy showed a diffusely erosive colitis and rectal scrapings were negative for amoebae. Although colectomy was seriously considered at this stage, it was decided to temporize with medical management. She was started on nasogastric suction, intravenous replacement, requiring 6 units of blood in all, and given prednisolone enemata. The bleeding ceased but the rectal mucosa now showed the changes of 'ulcerative colitis'. Four weeks after admission she still had diarrhoea and a barium enema showed features indistinguishable from non-specific ulcerative colitis, and at 6 months marked improvement had occurred. However, the year after her initial admission, she was still having 3 or 4 watery stools a day and barium enema showed colitis up to the splenic flexure.

Case 20 (fulminating postamoebic colitis). A 27-year-old Coloured female was admitted with a 2-week history of severe diarrhoea with mucus and small amounts of blood in the stool. She was dehydrated, pyrexial and toxic. Abdominal examination showed a moderately distended, tender abdomen and amoebae were found in the rectal scrapings, but not in the stool or mucosal biopsy. A severe initial electrolyte imbalance with hypokalaemia and acidosis were temporarily corrected with intravenous replacement solutions, and Emetine, chloroquine and tetracycline were given. On this therapy, frequent subsequent examinations of stools and rectal scrapings were free of amoebae. However, yellow, mucoid diarrhoea continued, passing up to 15 stools a day, and on the 7th day after admission she passed a colonic cast. Eleven days after admission she developed temporary renal failure in spite of intensive replacement therapy which failed to contain the recurring electrolyte problems. Despite the negative stool examinations, a course of metronidazole was also given without benefit. Proctoscopy now showed a friable, reddened mucosa indistinguishable from ulcerative colitis. Because of her deteriorating general condition with conservative management, surgical intervention was suggested and she was transferred to a surgical ward on the 35th hospital day for subtotal colectomy. This was never carried out as the patient continued to have severe diarrhoea, developed a left-sided pulmonary infarct and died while being prepared for surgery. A limited autopsy showed an extensive proctocolitis with severe ulcerative changes of most of the bowel mucosa.

DISCUSSION

The classification of amoebic colitis presented in this article (see Table I) is suggested as a guide to the therapy and prognosis of this disease. Although previous authors have referred to amoebic colitis as mild, severe, or fulminating and even relapsing, no definite attempts at classifying the disease have been made, and the various forms of postamoebic colitis have not been delineated.

One of the problems in attempting such a classification is that it is often difficult to know when amoebic colitis passes into the stage of postamoebic colitis. In the usual case of postamoebic colitis the clinical symptoms and ulcerative changes persist for some time after careful examination of stools, rectal scrapings and mucosal biopsies have excluded the presence of amoebae, and after adequate anti-amoebic measures have been given. However, the more fulminating progression of postamoebic colitis (case 19) may occasionally leave one in doubt as to whether the bowel changes merely constitute a delayed healing due to extensive mucosal infarction and ulceration, rather than a distinct entity. As mucosal regeneration is usually very rapid after treatment, it is suggested that persistence of symptoms with sigmoidoscopic, histological, and occasionally radiological changes in the colon after 2 - 3 weeks of intensive amoebicidal treatment, coupled with persistently negative stool examinations, rectal scrapings and biopsy evidence of amoebae when these were previously found, can conveniently be regarded as denoting the time when postamoebic colitis supersedes. In addition, there is sometimes a temporary clinical remission between the acute disease and postamoebic colitis (case 17). The latter may then become difficult to distinguish from non-specific ulcerative colitis.

Difficulty has also been experienced in ascertaining the relative incidence of the various grades of acute amoebic colitis, chronic amoebic colitis and postamoebic colitis. Clearly, postamoebic colitis is uncommon with modern therapeutic measures and Powell and Wilmot[5] reported 33 cases among several thousand attending each year with acute amoebic dysentery. However, Craig[6] states that the 'attacks of diarrhoea frequently continue due to the loss of mucous membrane and its replacement by fibrous scar tissue' and Schaffer *et al.*[7] note that an appreciable number of patients in whom the infection is eradicated continue to have short, sharp recurrences of their symptoms. A knowledge of the frequency of mild, severe and fulminating forms of acute amoebic colitis is hampered by the difficulty in assessing the number of mild cases occurring in one area. Many patients are treated without a positive diagnosis, or are treated on an outpatient basis if the diagnosis is established. It is likely that the acute fulminating and chronic stages of the disease are relatively infrequent, but these two groups do constitute an important problem because of the high mortality in the former, and the diagnostic difficulty in the latter. The possible danger of using corticosteroids in amoebic colitis, erroneously diagnosed as non-specific ulcerative colitis, has been noted,[3,8] and the chronic phases of the disease are particularly liable to receive this treatment if this is not appreciated. The reason why some patients have mild, fulminating or chronic forms of the disease is not readily apparent. Host-parasite resistance, socio-economic factors, nutrition and age of the patient may be of importance.[9]

Treatment

The therapeutic implications of this classification are perhaps the most important. In the absence of hepatic or other complications, the milder acute attacks or the chronic forms of amoebic colitis may be treated on an outpatient basis with metronidazole or Emetine-bismuth-iodide, usually given in combination with tetracycline and Diodoquin. Chloroquine is often given in addition to combat undetectable liver involvement. Sigmoidoscopy and rectal scrapings should be repeated after 7 - 10 days to ensure recovery of the mucosa and the absence of amoebae. The more severe forms of the disease require hospitalization, combined with anti-amoebic therapy, oral fluids or even

intravenous fluids for a few days to allow 'the bowel to rest and recover' and a daily assessment of the general and local physical signs to ensure that there is steady improvement. The response to therapy is very gratifying in the vast majority of cases. However, an occasional patient, in whom large areas of sloughing or deep ulceration of the bowel wall has occurred, may develop a localized pericolic abscess and regress to fulminating colitis or develop postamoebic colitis.

Fulminating amoebic colitis requires intensive intravenous electrolyte and fluid replacement with particular attention to serum potassium levels and systemic acidosis, which may be extreme. Nasogastric suction should be instituted if abdominal tenderness or distension are present, and intramuscular Emetine and antibiotics administered until such time as combined oral therapy can be given. Monitoring of the general and local abdominal signs should be undertaken twice daily with special attention to increasing diarrhoea, severity of rectal bleeding and increasing abdominal tenderness or distension. Abdominal radiography is required on admission and daily thereafter, if distension is marked, in an attempt to assess the presence of a generalized ileus, perforation or more particularly a 'toxic megacolon' picture. Extreme mucosal sloughing with the passage of colonic casts makes it very likely that mucosal regeneration will be prolonged, or that a phase of postamoebic colitis may supervene with continued diarrhoea, electrolyte difficulties and ultimately mucosal fibrosis. Repeated rectal scrapings, biopsies and stool cultures should be done to ensure the complete disappearance of amoebae. The role of surgery is a controversial issue, but we feel it may have a definite place in patients with acute or subacute deterioration in abdominal signs, or in those with frank perforation.[10]

The treatment of postamoebic colitis would appear to be the same as that currently in use for ulcerative colitis, which it may closely resemble. It is not unusual for these patients to have repeated courses of anti-amoebic therapy without symptomatic benefit. We feel that mild forms should be treated with Salazopyrin for prevention of recurrences, and rectal steroids for acute episodes. The severe form of the disease may necessitate oral corticosteroids, periods of hospitalization, Salazopyrin and careful follow-up study. No response to treatment and continued severe symptoms with anaemia, hypoproteinaemia and general ill-health may necessitate colectomy for intractability. Fulminating postamoebic colitis should be treated in the same way as fulminating ulcerative colitis; with intensive fluid and protein replacement, nasogastric suction, systemic corticosteroids and constant clinical assessment. An inability to contain the symptoms, or the lack of evidence of definite improvement within 2 - 5 days may necessitate emergency colectomy.

Prognosis

The prognosis of amoebic and postamoebic colitis is almost entirely dependent on the clinical severity and extent of the disease. The prognosis of amoebic colitis may, of course, be altered by the development of liver or more remote abscesses during any stage of the disease. While there are no definite figures for the mortality of the disease, Wilmot[9] estimates it at 3% among the Bantu in Durban and it is evident that the cause of mortality in the vast majority of cases is impending or actual colonic perforation and peritonitis. In addition, the prognosis of free perforation or fulminating amoebic colitis is so bad that reported survivals are exceptional. The prognosis of the more severe cases of postamoebic colitis has been reported to be poor,[5] but it would appear that these cases followed severe attacks of amoebic colitis and therefore tended to be diffuse and severe. Powell and Wilmot[5] did not find corticosteroids very helpful in controlling the disease.

Whereas it is suggested that the indication for surgery in postamoebic colitis should be similar to those in acute non-specific ulcerative colitis, the feasibility of surgery in fulminating amoebic colitis, with or without perforation, is far less definite. By and large a nihilistic attitude towards surgery has been adopted in these cases. However, the mortality in the fulminating variety of the disease is so formidable that surgery would appear to be the only course which may be life-saving. With modern amoebicidal treatment, careful selection of patients and timing of operation, together with the present methods of postoperative intensive care, colectomy or lesser operations may secure a lower mortality for this lethal form of the disease.

While sporadic reports of carcinoma and chronic amoebic colitis occurring concurrently have appeared, the causal relationship between the two remains speculative. Little is known of the long-term follow-up of postamoebic colitis, but it is not unreasonable to presume that this disease may carry all the hazards of chronic ulcerative colitis, and this may include the development of carcinoma.

SUMMARY

A clinical classification of amoebic colitis is presented and its therapeutic and prognostic usefulness outlined by representative case reports.

Severe and fulminating amoebic and postamoebic colitis are potentially lethal forms of the disease requiring intensive general care and daily monitoring of the abdominal signs. The place of surgical intervention in these subgroups is discussed.

Postamoebic colitis is virtually indistinguishable from nonspecific ulcerative colitis and it is suggested that similar treatment be adopted.

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