

## Ulcerative post-dysenteric colitis

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**EDITORIAL COMMENT** Better treatment is resulting in more severe cases of amoebic colitis surviving and these patients may have severe residual damage to the bowel resulting in ulcerative post-dysenteric colitis. This is considered to be a distinct entity.

The term 'post-dysenteric colonic irritability' was introduced by Sir Arthur Hurst (1943) to describe persistent irritability of the bowel following an acute attack of bacillary or amoebic dysentery. The early symptoms were attributed to a non-specific chronic colitis occurring after the specific infection had died out, but in the later stages were thought to be due to 'functional irritability' of the colon. Stewart (1950) found that post-dysenteric colitis was more commonly a sequel to acute amoebic dysentery and was able to recognize two forms in his patients: 1 Those with mild symptoms and no colonic ulceration, which he named 'functional post-dysenteric colitis', and (2) Those with colonic ulceration and more severe symptoms, which he termed 'ulcerative post-dysenteric colitis'.

The form with mild symptoms accords with Hurst's (1943) description of post-dysenteric colonic irritability and comprises one group of patients forming part of the 'irritable colon syndrome' (Chaudhary and Truelove, 1962). However, there are few descriptions of patients with ulcerative post-dysenteric colitis. The condition is omitted from many modern textbooks of gastroenterology and, apart from a brief account by Wilmot (1958), the more recent literature has not enlarged on Stewart's (1950) observations. Yet, to judge from requests for information, confusion exists concerning the distinction between post-dysenteric colonic irritability, ulcerative post-dysenteric colitis, and chronic, non-specific ulcerative colitis.

Among Africans in Durban both bacillary and acute amoebic dysentery are common. The former condition is usually self-limiting, responds readily to treatment and, in our experience, presents few problems. On the other hand, among the several

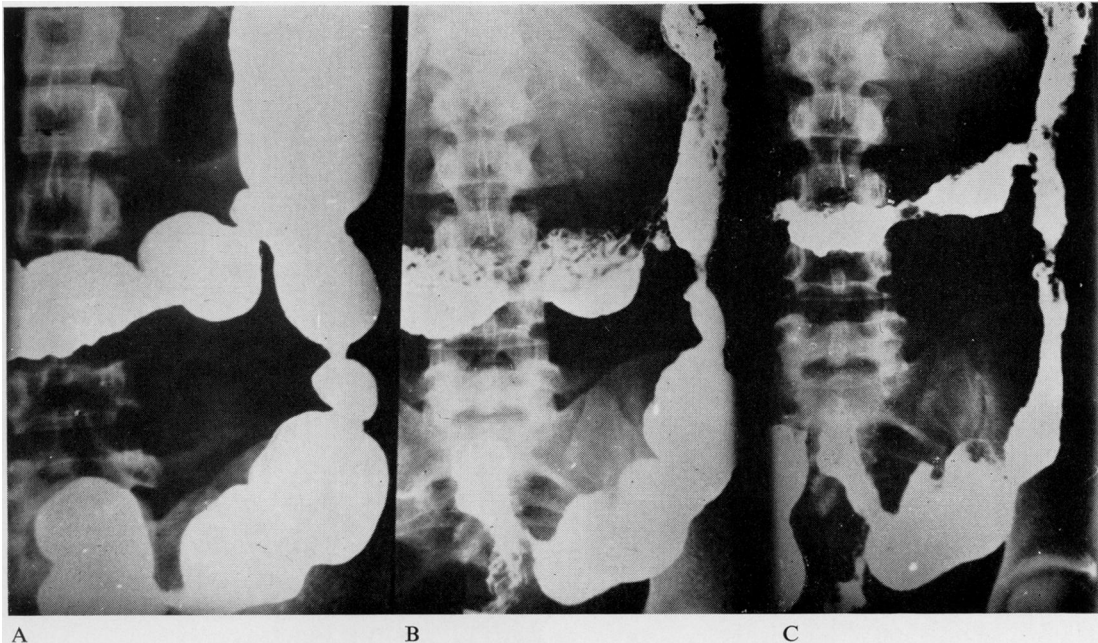
thousand patients who attend this hospital annually with acute amoebic dysentery complications are common and we have had the opportunity to study them (Wilmot, 1962). It is from this material that we have based the following report of ulcerative post-dysenteric colitis in 33 African patients observed in recent years.

### CLINICAL FINDINGS

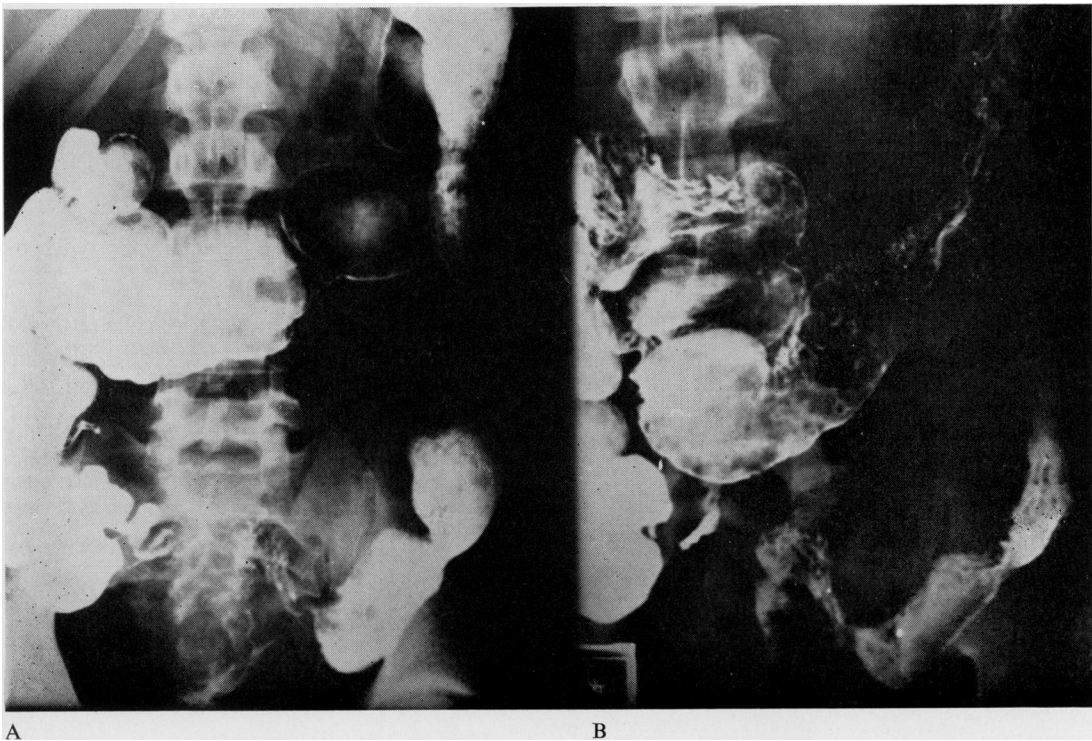
All patients presented initially with severe amoebic dysentery, sigmoidoscopic examination showing a congested, oedematous mucosa with extensive rectal ulcers the surfaces of which were covered by sloughs and exudate. In all instances culture for bacterial pathogens was negative but haematophagous trophozoites of *Entamoeba histolytica* were present in the dysenteric stools and ulcer scrapings. Apart from being more severe than average the initial picture was typical of acute amoebic dysentery.

In dysenteric amoebiasis of average severity appropriate amoebicidal therapy almost invariably results in the cessation of symptoms and disappearance of *E. histolytica* from the stools and ulcer scrapings within approximately five days. Complete healing of ulcers takes a little longer but by the tenth day of treatment the rectal ulcers, if not entirely healed, are clean and free of exudate, some degree of epithelialization is present, and the intervening mucosa is no longer congested. However, in the patients under study, on the tenth day dysentery persisted, despite intensive amoebicidal therapy consisting of emetine hydrochloride or dehydroemetine in full doses combined with diiodohydroxyquinoline and either tetracycline or penicillin and phthalysulphathiazole. Although *E. histolytica* could no longer be found there was little change in the mucosal picture apart from some lessening of exudate on the ulcers. In 12 patients peritonitis supervened within the first 10 days but, after successful treatment by conservative measures,

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**FIG. 1.** Radiograph of left transverse colon, splenic flexure, descending and sigmoid colon in different degrees of distension with barium. *A*, fully distended colon; *B*, after partial evacuation; *C*, after further evacuation. Note constancy and rigidity of strictures.



**FIG. 2.** *A*, air-barium contrast demonstrating rigidity of strictures. *B*, mucosal relief film demonstrating mucosal irregularity (pseudo-polypoid appearance) and evidence of ulceration, best seen at junction of descending and sigmoid colons.

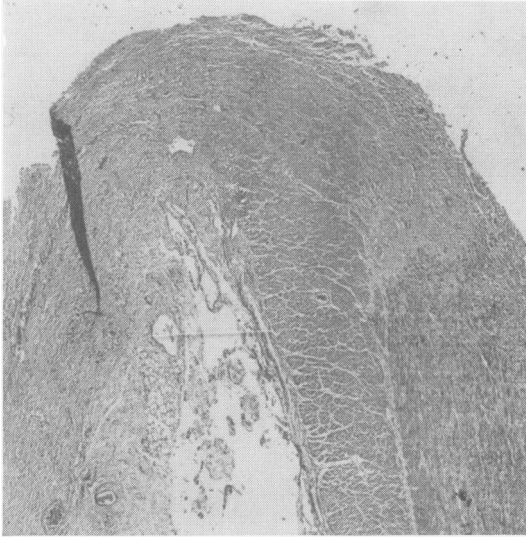


FIG. 3



FIG. 4



FIG. 5

FIG. 3. Section of colon showing surface ulceration with loss of muscle layers on left side  $\times 25$ .

FIG. 4. Higher power of a relatively normal area of bowel with post-mortem autolysis of surface. Muscle layers intact  $\times 60$ .

FIG. 5. Higher power of an ulcerated area showing disruption of mucosal surface, endarteritis of a vessel, destruction of muscle layer, and a rather mild inflammatory infiltrate of plasma cells and lymphocytes  $\times 60$ .

dysenteric symptoms continued. It is also noteworthy that in many patients during the initial, acute episode of dysentery large sloughs of necrotic bowel mucosa were passed and, when sigmoidoscopic examination could be done, extensive, raw, bleeding areas were visible at the site of separation.

The subsequent course of the condition appeared to depend on the degree of damage to the bowel. Although repeated search failed to reveal amoebae after initial treatment, all patients received some form of additional amoebicidal therapy at the tenth day without much symptomatic response. However, the least severe cases began to show a progressive reduction of dysentery and by approximately the 30th day in 13 patients the stools had become normal. Except for some granularity and hyperaemia at the site of previous ulcers the rectal mucosa returned to normal. Thereafter these patients remained free of symptoms. This group may be summarized as showing moderately delayed healing and occupies an intermediate position between those patients with typical acute amoebic dysentery responding readily and completely to amoebicidal therapy and those who develop chronic colitis.

The remaining 20 patients formed the latter group, in whom dysentery continued, in some instances for over nine months, with little tendency for the rectal ulcers to heal. Fever, anaemia, leucocytosis, and elevation of the erythrocyte sedimentation rate persisted and the problem in management became one of maintaining the patient despite intractable dysentery. Antispasmodics and non-specific diarrhoeal remedies had little effect but courses of insoluble sulphonamides seemed of value in alleviating symptoms. Blood transfusion was frequently necessary to correct anaemia, and in some patients severe protein, fluid, and electrolyte loss required correction by intravenous infusions. In 14 of these patients symptoms slowly abated. Repeated sigmoidoscopy throughout their long illnesses showed a slow tendency towards healing of the ulcers and a gradual reduction in mucosal fragility but, at sites where ulceration had been deep or sloughing had occurred, some narrowing of the bowel lumen was common, and in 11 instances final healing was accompanied by scarring and stricture formation. A common site was at the recto-sigmoid junction. In these patients the mucosa eventually became abnormally pale and avascular but, although in most the strictures were sufficient to prevent the passage of a sigmoidoscope, symptoms of obstruction developed in only one instance. These were mild and have recurred during the past six months but it has not been necessary to resort to surgery.

Oral prednisolone was given to the remaining six patients after dysentery had persisted for two to

three months. In one patient there was prompt improvement but relapse occurred on two occasions when the dosage was reduced below 60 mg. daily. It was only possible to discontinue steroids after several weeks but cure, when achieved, appeared permanent. A second patient responded more slowly to 30 mg. daily of prednisolone and, after a month, was able to return home although the rectal mucosa remained grossly abnormal and strictures were present. He returned to hospital six months later with a recurrence of diarrhoea. The sigmoidoscopic appearance was unchanged and barium enema on two occasions showed persistent multiple strictures. The course and findings in this patient were indistinguishable from those of chronic, non-specific ulcerative colitis.

In the remaining four patients there was little or no response to prednisolone, and one followed a steady downhill course until he died three months after the initial attack of dysentery. Necropsy showed extensive ulceration throughout the colon but amoebae were absent. The final three patients were also given hydrocortisone enemas without effect. In one a rectosigmoid fistula, demonstrated by barium enema, was probably responsible for persisting symptoms. Two of these patients eventually discharged themselves from hospital although they were not free of diarrhoea. The last patient, who has shown little response to any form of treatment, remains extremely ill and the outcome is doubtful.

In 10 patients barium studies were done after the acute attack had settled down. In seven severe damage to the colon, loss of haustrations, and multiple strictures were demonstrable.

We have little follow-up information but, of the nine patients who have attended again at periods of one month to one year after discharge, in seven the disease has remained quiescent although there has been little, if any, change in the degree of stricture visible at sigmoidoscopy. One patient, previously mentioned, had a recurrence of symptoms and the condition now shows great similarity to chronic non-specific ulcerative colitis. A further patient returned with a recurrence of dysentery several months after discharge. Despite intensive amoebicidal therapy on the previous occasion he was shown to have suffered a recurrence of amoebic dysentery on his second admission. This again responded to amoebicides. Although the rectal ulcers healed the strictures persisted unchanged and have produced mild obstructive symptoms.

#### DISCUSSION

The term 'post-dysenteric colonic irritability' is more accurate than 'functional post-dysenteric colitis', as

Chaudhary and Truelove (1962) have shown that inflammatory changes are absent in this condition. This distinguishes it and all other forms of the irritable colon syndrome from both ulcerative post-dysenteric colitis and chronic, non-specific ulcerative colitis. A further distinction is that, in contrast to the predominantly functional nature of all forms of the irritable colon syndrome, the origin and course of ulcerative post-dysenteric colitis is associated with structural changes in the bowel. In our experience it has been confined to patients with severe amoebic dysentery, in some cases complicated by peritonitis. The colitis was an immediate sequel to invasion by *E. histolytica* and its duration ranged from mere prolongation of the normal healing time of amoebic dysentery to a protracted course of more than nine months. Strictures were common in the more severe cases and in one instance persistent symptoms were probably due to the development of a recto-sigmoid fistula. Such intestinal fistulae are a recognized, although rare, complication of severe amoebic dysentery (Dinner and Bader, 1961).

In our series the severity and degree of post-dysenteric colitis appeared to be related to the severity and extent of the initial damage to the bowel mucosa during the original infection. With two exceptions there was slow, at times exceedingly slow, improvement, but, despite residual scarring and strictures, once the rectal ulcers had healed relapse was infrequent. In most the course progresses towards lasting recovery but some progress equally steadily downhill, and occasional cases may follow a course indistinguishable from that of chronic, non-specific ulcerative colitis. In the latter instance it is important to exclude relapse or reinfection by *E. histolytica*.

Although some degree of selection in our series may have favoured investigation of the more severe cases it is noteworthy that no instances of the milder condition of post-dysenteric colonic irritability were seen during the period of observation. Moreover no sequelae to bacillary dysentery were observed. Functional disturbance of the gastrointestinal tract is infrequently diagnosed in the local African population but it is not known if this is a true reflection of its incidence as the Africans are reticent about minor disturbances of function. However, there is no doubt that our African medical students, who are not typically representative of the general African population, are just as subject to nervous diarrhoea under examination stress as students elsewhere.

Whereas, in the patients we have seen, distinction from post-dysenteric colonic irritability can be readily made, it is more difficult to differentiate ulcerative post-dysenteric colitis from chronic non-specific ulcerative colitis. Although apparently rare in

Africans, reports of the latter condition have appeared recently (Billinghurst, 1964; Pillay, 1964), and we have observed such cases. More frequent are patients in whom the aetiology of the initial attack was assumed to be amoebic and, following amoebicidal therapy, it has not been possible to distinguish the two conditions with certainty. In such instances the general clinical picture, sigmoidoscopic and radiographic findings may be identical to chronic, non-specific ulcerative colitis. Nevertheless, in most patients with the post-dysenteric form the disease is not characterized by a sequence of remissions and relapses. The local complications of the two conditions are similar although it is our impression that whereas fibrous stricture is more common in ulcerative post-dysenteric colitis, carcinoma is rare. However, the latter observation may merely be a reflection of the infrequency of carcinoma of the large bowel in general among Africans. The remote complications of chronic, non-specific ulcerative colitis such as conjunctivitis, iritis, skin eruptions, arthritis, and erythema nodosum were not seen in our patients.

It has been shown that antibodies against *E. histolytica* are detectable by the gel-diffusion technique in approximately 90% of cases of acute amoebic dysentery (Maddison, Powell, and Elsdon-Dew, 1965; Powell, Maddison, Hodgson, and Elsdon-Dew, 1966) and they were demonstrable in the 11 patients in whom they were sought in our series. In one instance there was a reduction and eventual disappearance of antibodies over a period of six months although the colitis was still active, so that it is probable that negative results will be obtained in some longstanding cases of ulcerative post-dysenteric colitis. As sera from patients with chronic, non-specific ulcerative colitis have failed to show precipitins against *E. histolytica* (Maddison, 1965; Powell *et al.*, 1966), serological methods may be of value in diagnosis, at least in the earlier stages of the post-dysenteric form.

Our limited experience with steroids in ulcerative post-dysenteric colitis is similar to that reported by Rankin, Goulston, Boden, and Morrow (1960) in fulminating ulcerative colitis, and response to this form of treatment cannot be used to distinguish the conditions. Most patients with the post-dysenteric form do not require steroids but, in view of the prompt improvement in one patient, they warrant a cautious trial when other measures have failed and the patient is deteriorating. We have not used sulphasalazine but we feel that it deserves a trial.

Improved methods of treatment have resulted in increased survival in acute amoebic dysentery, including those patients in whom peritonitis has supervened (Powell and Wilmot, 1966). Recent experience

suggests that consequently more patients are now seen in whom there is severe residual damage to the bowel resulting in ulcerative post-dysenteric colitis. The condition therefore deserves more widespread recognition than it has attained in the past.

## SUMMARY

The findings in 33 patients with ulcerative post-dysenteric colitis are reported. The condition was a direct sequel to severe amoebic dysentery and its severity was related to the degree and extent of damage to the bowel mucosa caused by the initial infection. The distinction from post-dysenteric colonic irritability and chronic, non-specific ulcerative colitis is discussed. With increased survival from severe amoebic dysentery the condition may be recognized more frequently.

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