Ulcerative post-dysenteric colitis

S. J. POWELL AND A. J. WILMOT

From the Amoebiasis Research Unit1 and the Department of Medicine, University of Natal, Durban, South Africa

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,
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. Section of colon showing surface ulceration with loss of muscle layers on left side × 25.


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 × 60.
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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 reduc-
tion 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 moder-
ately delayed healing and occupies an intermediate
position between those patients with typical acute
amoebic dysentery responding readily and complete-
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 diarr-
hoeal remedies had little effect but courses of
insoluble sulphonamides seemed of value in allevi-
ating 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 sig-
moidoscopy 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 im-
provement 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 in-
distinguishable 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 exten-
sive 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 them-
selves 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 haustations, 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 dysenty, 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 re-infection 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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**REFERENCES**


