INDIGENOUS AMOEBIASIS: A RECENT OUTBREAK IN ENGLAND

BY

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Active amoebiasis is rare in those who have never been out of England, although cysts of *Entamoeba histolytica* have been found in the stools of 1.5-4.7% of such people (Morton *et al.*, 1951). The cases which have been reported have all occurred sporadically, sometimes with a history of close association with a patient returned from overseas suffering from acute amoebic dysentery. The only large-scale epidemics of amoebic dysentery in a temperate climate were those in Chicago in 1933 and 1934. The first of these (Epidemic Amebic Dysentery, 1936) was due to the flooding of food stores with sewage from two broken sewers and to a cross-connexion between a waste-pipe and the water supply: 1,409 cases, with 98 deaths, were finally notified. The second epidemic followed the Chicago stockyards fire of May, 1934, during which firemen and civilian spectators drank water contaminated with sewage (Hardy and Spector, 1935).

In the small outbreak described here the facts suggest a similar origin from water grossly polluted with sewage.

History of the Outbreak

The R.A.F. station where the outbreak occurred is a permanent brick-built camp with a water supply from an Air Ministry bore-hole sunk in 1940 to a depth of 120 feet, the last 30 feet of which is through chalk, a notoriously treacherous formation, giving the bore-hole the potential dangers of a shallow well. In 1943 a sewer was laid, passing within 20 feet of the bore-hole, to serve a lavatory in the workshops (see Diagram).

Diagram of R.A.F. Camp.

In June, 1950, a routine water sample showed considerable pollution. Chlorination of the supply was started, but only in a half-hearted and haphazard manner, as shown by the fact that four water samples submitted for analysis during September all showed gross pollution. After receipt of these reports all water used in kitchens and for drinking was boiled.

In the meantime, from mid-August, cases of acute gastro-enteritis began to appear, and by the end of September 141 people had reported sick out of a total station strength of 1,042. It was noticed that the incidence was much higher in the three barracks A, B, and C than in the rest of the camp. A sewer running parallel with these three barracks had become blocked by tree roots in August (point E on Diagram). It was repaired by the end of the month, but the gastro-enteritis persisted.

On September 11 the sewer running within 20 feet of the bore-hole was excavated as a possible source of the water contamination. A soakaway taking the rain-water from the pump-house roof was found to have been placed directly over the sewer, and the water from it had so eroded the soil from beneath the sewer bed that the sewer had fractured in five places. This broken sewer led into the one which had been found blocked in August, and the dammed-up sewage could have back-flowed and escaped through the broken sections.

By the end of September the outbreak of acute gastro-enteritis was virtually over, but during October a number of cases with subacute intestinal symptoms occurred. The symptoms were chiefly those of flatulence, vague abdominal discomfort or colic, constipation or a tendency to diarrhoea, associated in some cases with blood and mucus in the stools. The clinical picture was totally different from that encountered during the outbreak of gastro-enteritis, and it was suggested by the Deputy Principal Medical Officer that the symptoms might be due to amoebic infection.

One patient who had undergone appendicectomy on September 14 complained of blood and mucus in his stools from the day following operation. He had never been overseas. Towards the end of November he was referred to the consultant in tropical medicine, and was found to be suffering from acute amoebic dysentery. Four more airmen who had symptoms suggestive of amoebiasis were then also referred for consultant opinion, and all were found to have active amoebiasis.

At the end of November a team consisting of a pathologist and three technicians, all with tropical experience, was sent to the R.A.F. station for three weeks. A minimum of three stools were examined microscopically from all those who handled food and from those who had had some form of gastro-intestinal upset during the previous four months; only one specimen was examined from each of the remainder. Bacteriological examination was abandoned after culture of stools from 100 of the cases of gastro-enteritis had failed to show pathogenic organisms.

Three months later, at the end of February, 1951, the same team re-examined all those on the camp, including 169 who had arrived after the last investigation. These 169 serve as "controls" in this report because they arrived after the source of infection was considered to have been eliminated. The rest are referred to as "contacts."

All those found to be passing *E. histolytica* in their stools were referred to the consultant in tropical medicine, and only those whose stools were confirmed as positive were accepted for this report. On the basis of the consultant's clinical examination the cases were classified into two main groups: (a) those with active amoebiasis, and (b) symptomless cyst-carriers. The criteria of classification were:

(a) *Active Amoebiasis*.—Patients who were passing either active or cyst forms of *E. histolytica* and who also had clinical symptoms which could be attributable to amoebiasis—such as irregularity of bowel action, lower abdominal pain, flatulence, lassitude, and in some cases blood and mucus in the stools. Those who had active *E. histolytica* and

blood and mucus in their stools, in addition to the other features of this group, formed a subgroup of acute amoebic dysentery.

(b) *Symptomless Cyst-passers*.—Those in whose stools *E. histolytica* cysts were found but who had no symptoms attributable to amoebiasis; single short-lived attacks of diarrhoea were ignored.

The presence of crateriform pitting on sigmoidoscopy was noted, but was not counted as evidence of active amoebiasis.

Treatment

The schedules of treatment used were those described by Morton (1949). Those with acute amoebic dysentery were treated with Halton course II, except one patient who was treated with an experimental amoebicide. In the latter case there was healing of ulceration, but *E. histolytica* persisted in the stools until after treatment with Halton course I. The rest of the patients with active amoebiasis were treated with Halton course I.

Halton Course I.—Sulphaguanidine, 100 g. during the first five days, with emetine hydrochloride, 1 gr. (65 mg.) daily, subcutaneously, given concurrently. Emetine bismuth iodide, 3 gr. (0.2 g.) every night by mouth, and "diodoquin," 3 tablets three times a day, are given for the next six days, and until this is completed the patient is confined strictly to bed. Diodoquin, 3 tablets three times a day, is then given for a further 16 days and the patient gradually allowed up.

Halton Course II.—Sulphaguanidine, 100 g., plus penicillin, 1,500,000 units, during the first five days; then emetine hydrochloride, 1 gr. (65 mg.) daily, subcutaneously, and diodoquin, 3 tablets three times a day by mouth, for seven days, followed by diodoquin, 3 tablets three times a day for 15 days. Finally, emetine bismuth iodide, 3 gr. (0.2 g.) every night, and 2½% chiniofon retention enemata for 10 days.

All symptomless cyst-passers were treated with diodoquin, three tablets (3.2 gr.—0.2 g.—each) thrice daily for three weeks. One of these relapsed and presented as a case of acute amoebic dysentery two months later. The faeces of all patients were checked one month and three months after treatment, three specimens being examined on each occasion. There were no relapses up to three months except for the two cases mentioned above.

Results of Investigations

Analysis of the August and September epidemic of gastro-enteritis showed a much higher incidence of infection in barracks A, B, and C than in the rest of the camp (see Table I).

TABLE I.—Incidence of Gastro-enteritis

Group	No. in Group	% with Symptoms
Barracks A, B, and C	333	23.4
Rest of camp	709	8.9

The total number of contacts found to have *E. histolytica* in their stools was 43 (4.1%), and 60% of these were revealed by a single examination of faeces. All had a large strain of *E. histolytica* (> 10 µ), and many had more than one size of strain present. Six (3.5%) symptomless *E. histolytica* cyst passers were found in the "controls."

Of the 43 positive contacts, 36 were found at the first investigation—24 in the first specimen examined, 6 in the second, 5 in the third, and 1 in the sixth. The remaining seven positive contacts were found at the second investigation—two had not previously been examined, three had had one negative specimen, and two had had three negative specimens at the first investigation; all seven were found positive on their first specimen at the second investigation.

Seventeen of the positive contacts were classified as having active amoebiasis, and six of these had acute amoebic dysentery. Five of the six acute cases had never been overseas. Six of the remaining 11 with active amoebiasis showed crateriform pitting on sigmoidoscopy, and two of

these had active *E. histolytica* in their stools; the mucosa was normal in the other five, but the stools of three of them contained active *E. histolytica*.

Thirteen of those with active amoebiasis lived in barracks A, B, and C; of the remaining four, two lived in the sergeants' mess, and one in each of barracks X and Y.

Four of the 26 classified as symptomless cyst-passers did in fact show crateriform pitting on sigmoidoscopy; but as this finding has not yet been generally accepted as indicative of active amoebiasis they have been included in the group of symptomless cyst-passers.

One patient, originally classified as a symptomless cyst-passer with a normal mucosa on sigmoidoscopy in December, was sent on leave with instructions to take three diodoquin tablets thrice daily for 21 days. In mid-January he noticed blood in his stools, and this continued until the beginning of February, when he was found on sigmoidoscopy to have typical amoebic ulceration.

The original patient, whose symptoms started immediately after appendicectomy, was almost certainly a case of misdiagnosed amoebiasis, but unfortunately his appendix was not submitted for section. One other patient presented with symptoms and signs of subacute appendicitis, and would almost certainly have been operated upon had he not come to the notice of one of the members of the investigating team before being transferred to hospital.

Only two who handled food were infected—one a cook in the sergeants' mess and the other a batman in the officers' mess; both were symptomless cyst-passers and neither had ever been overseas. One slept in barrack block Y and the other in the batmen's quarters of the officers' mess.

One further patient, not included in any of the figures presented in this paper, was referred to the consultant in tropical medicine in July, 1951. He was on leave from the affected station during the first investigation, and had been posted to another unit before the second. He had had diarrhoea in October, 1950, and again in January, 1951, which continued, gradually getting worse, until he was seen in July and found on sigmoidoscopy to have acute amoebic ulceration. This man belonged to barrack block A and had never been overseas.

TABLE II.—Analysis of Cases of Gastro-enteritis and Amoebiasis

Group		No. in Group		Active Amoebiasis		Cyst Passers	
		H	O	H	O	H	O
Barracks A, B, and C	With gastro-enteritis	60	18	6	2	1	2
	No gastro-enteritis	204	51	5	Nil	3	3
Rest of camp	With gastro-enteritis	42	21	1	1	Nil	Nil
	No gastro-enteritis	424	222	1	1	5	12
Controls*		126	43	Nil	Nil	2	4

* Those arriving at camp after December, 1950. H=Those who had never been overseas. O=Those who had at some time been overseas.

The main findings are summarized in Table II, which separates those who had and those who had not been overseas. In Table III the incidence of gastro-enteritis in August and September and that of amoebiasis have been expressed as percentages of the number at risk in each

TABLE III.—Incidence of Gastro-enteritis and Amoebiasis in Various Groups

Group		No. in Group	% with Active Amoebiasis	% Cyst Passers
Barracks A, B, and C	With gastro-enteritis	78	10.2	3.8
	No gastro-enteritis	255	2.0	2.3
	Total	333	3.9	2.7
Rest of camp	With gastro-enteritis	63	3.0	Nil
	No gastro-enteritis	646	0.3	2.6
	Total	709	0.6	2.4
Controls*		169	Nil	3.5

* Those arriving at camp after December, 1950.

group. The main points of interest shown by Tables II and III are the higher incidence of active amoebiasis in groups A, B, and C, and the positive correlation between active amoebiasis and gastro-enteritis.

Discussion and Conclusions

The finding of *E. histolytica* in the stools of 4.1% of those examined is not in itself outside the normal expectation in the population of this country; but the occurrence of six cases of acute amoebic dysentery and a further 11 cases classifiable as active amoebiasis within a three-months period in a population of approximately 1,000 people is so very unusual that it may properly be termed a minor epidemic, and a common source of infection may be postulated with reasonable certainty. There is a positive correlation between the incidence of gastro-enteritis and amoebiasis which makes a common source of infection still more probable.

The time relationship between the onset and control of the gastro-enteritis and the contamination of the water supply and its control strongly suggests that the gastro-enteritis, and therefore the amoebiasis, were water-borne. This view is further strengthened by the finding of a potent source of contamination and by the fact that barracks A, B, and C, which are nearest to the source of water-supply, were most affected.

In the case of the amoebiasis this view is further strengthened by the fact that the only four active cases not in barracks A, B, and C were in barracks X and Y and in the sergeants' mess, which are the next nearest living quarters to the source of water-supply.

It could perhaps be argued that the amoebiasis was due to activation of previous cyst-passers by the gastro-enteritis, but this would not account for the seven cases of active amoebiasis without gastro-enteritis. It is far more probable that the correlation between gastro-enteritis and amoebiasis was due to a common source of infection than to any synergistic action between the two infections.

The presence in the community of a relatively large number of people who had served overseas may well have been an important epidemiological factor, since there is evidence (Hoare, 1950) that overseas strains of *E. histolytica* are more pathogenic than home-grown varieties.

It seems likely, therefore, that the important factors in the epidemiology of the present outbreak were gross contamination of the water-supply with sewage, the short length of water-piping from the site of contamination to the points of distribution, and the use of the contaminating sewer by a community containing a high percentage of people who had served overseas.

Clinically the epidemic once more demonstrated the insidious onset of amoebiasis and the way in which it may simulate almost any other subacute abdominal upset and even acute appendicitis. These features account for the considerable delay before its true nature was realized, even though several of the doctors concerned were familiar with amoebiasis. This leads to speculation whether similar outbreaks masked by a concurrent acute intestinal infection may be relatively common.

Laidlaw (1918), when particularly looking for them, found three cases of amoebiasis at necropsy at Guy's Hospital in one year; none of these persons had ever been overseas and none of the cases had been diagnosed during life.

On the other hand, we have been able to find reports in the literature of only 33 cases of indigenous amoebiasis in England, and we have known one further case ourselves which has not been published. The earlier reports, which included 11 cases, were summarized by Dobell (1921); Morton *et al.* (1951) mentioned records of 12 more and reported a further three of their own. Coles (1923), Russell (1926), and Simpson (1926) each reported one case. Adams and Seaton (1949) reported a further four cases. Nearly all these occurred singly, sometimes with a history of close contact with a patient who had contracted the disease over-

seas, but often with no such contact discovered. In no case was there any record of previous involvement in an outbreak of gastro-enteritis.

The probable explanation is that a short water-distribution system contaminated by sewage from a population containing a large proportion of people who have recently been overseas forms a combination of circumstances very unlikely to be found in civilian communities in this country.

On the other hand, massive infection with the less pathogenic home-grown varieties of *E. histolytica* might give rise to an even more insidious and subacute form of the disease, which might well remain unrecognized indefinitely. It is therefore felt that wherever a water-borne epidemic disease occurs through gross faecal contamination of the supply the population at risk should be carefully watched for the appearance of patients with symptoms attributable to amoebiasis, and a sample of those suffering from the original disease should be examined for the presence of amoebic infection.

Summary

An epidemic of acute gastro-enteritis and amoebiasis on an R.A.F. station is described.

In a population of 1,042 at risk, 141 suffered from acute gastro-enteritis, 6 developed acute amoebic dysentery, and 11 had active amoebiasis.

Five of the six with acute amoebic dysentery had never been overseas.

Twenty-six symptomless cyst-passers were found.

Among 169 controls on the same camp, six symptomless cyst-passers were found, but none with active amoebiasis.

Both the gastro-enteritis and the amoebiasis are thought to have been water-borne, contamination of the station bore-hole having occurred from a broken sewer.

The main factors responsible for the epidemic are considered to have been gross faecal contamination of the water-supply, short water-distribution system, and the presence of a relatively large number of people in the community who had been overseas.

The insidious and protean nature of the disease is recalled and the possibility of similar epidemics being missed is discussed.

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Waiting for the doctor may soon become a real pleasure. Studies are under way at University of Chicago clinics on the possibility of using movies in out-patient departments and doctors' waiting-rooms as a medium for health education and entertainment. Existing film libraries provide a wealth of material ranging from instructive cartoons to formal educational films. A portable machine, easy to set up, is used. It has an 11 by 15-in. (28 by 38-cm.) screen and is about the size of an average suitcase when closed. No operator is required once the machine is put in action. A continuous loop film makes possible repeated showings of 16-mm. colour or black and white film without rewinding or rethreading. A 300-watt projection lamp makes viewing easy even in a lighted room.