to explain on the basis of only one case. Nevertheless, it *is* possible for *E. histolytica* to reach the ascitic fluid by the blood or lymphatic circulation or by infiltration of the peritoneum.

The striking improvement in the patient's condition after anti-amoebic therapy also confirms the nature of the disease. The therapeutic test often gives the only valid evidence of amoebic disease. In explaining why no abscess developed in the liver during the long course of the disease it must be remembered that many different factors are related to the formation of the lesions of hepatic amoebiasis. Seneca (1956) mentioned that liver lesions produced by malaria, syphilis, alcohol, malnutrition, or trauma may predispose to the development of liver abscess. It must also be assumed that the majority of amoebae do not survive in the liver when the organ is intact.

Summary

An unusual case of chronic non-suppurative diffuse amoebic hepatitis has been described. *Entamoeba histolytica* was found in needle and surgical liver biopsies as well as in a liver abscess which developed in an experimental animal after injection of ascitic fluid. The mechanism of the disease process is discussed.

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AMOEBIC LIVER ABSCESS IN GREAT BRITAIN

BY

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It has been variously stated that the carrier rate for *Entamoeba histolytica* cysts in the faeces of groups of people who have never been out of Great Britain ranges between 1.6% (Morton *et al.*, 1951), 2.7% (Dobell, 1921), and 4.7% (Matthews and Smith, 1919). As Morton *et al.* pointed out, considerable differences are likely to arise from the methods used and the criteria adopted, notably pathogenicity tests, there still being some doubt about the pathogenicity of small race strains. With increasing travel, and the effect of infected personnel returning from two world wars, the indigenous carrier rate may be expected to rise; for example, Morton *et al.* found a rate of 8.8% in airmen returning from overseas. The same authors recorded three instances of clinical dysentery in patients who had never been abroad.

Manson-Bahr (1943), in an extensive review of the literature, quotes seven authenticated cases of amoebiasis arising indigenously in the British Isles. Four of these had abscesses of the liver (Saundby and Miller, 1909; Wenyon, 1916; Simpson, 1926; Gilroy, 1928), and in a personal communication in 1959 Air Vice-Marshal Sir Aubrey Rumball has told me of a further case. In France, Garin and Lépine (1924) recorded 208 cases of indigenous amoebiasis, two of them with liver abscesses.

Case 1

A youth of 18 was admitted to hospital on March 31, 1959. A diagnosis of retrocaecal appendicitis had been made, but at laparotomy the appendix was normal, and a manual search had failed to reveal any abnormality in the paracolic or subdiaphragmatic areas. The white-cell count was 22,400 (polymorphs 90%), and urine examination negative. A pre-operative film showed the right diaphragm to be raised with some paracardiac collapse of the right lower lobe. "Crystamycin" 1 ampoule b.d. was prescribed. The next day he was transferred by the surgeon to my care; though very ill, he was able to give the following history.

At school early in February, 1959, he had "influenza," but afterwards never felt really well. Three weeks later he had further pyrexia, which was also thought to be influenza, and with it he had pain in the right lower chest posteriorly. Shortly before admission he had severe pain in the right upper abdomen. Movement and breathing caused the pain to be intense, and lying down gave him pain in the right shoulder-tip. His temperature was 103° F. (39.4° C.) and pulse 112; his tongue was dry and furred when the surgeon saw him. A provisional diagnosis was made of retrocaecal appendix with a leak into Morison's pouch.

The patient had always lived in the United Kingdom, apart from one holiday in Dinard and one in Brunswick. His father had spent a year in Palestine, but the patient had not been in touch with him for 12 years. He had once spent a few weeks' holiday with an aunt who had lived in India.

Examination on the day after his laparotomy (April 1) revealed a very toxic patient experiencing very severe pain over the right lower ribs on movement or breathing deeply. Lying on his left side exacerbated the pain. He was tender to percussion over the right lower chest and on palpation subcostally. A further antero-posterior chest film and a lateral view showed that the whole diaphragm was raised, but especially anteriorly.

A tentative diagnosis of non-specific abscess of the liver was made in the absence of residence in the tropics or subtropics. Penicillin was increased to two mega units t.d.s., and chloramphenicol, 500 mg. q.i.d., was added. By April 2 his temperature had fallen to 101° F. (38.3° C.), but his condition had deteriorated and he was having repeated drenching sweats. The pain in the right subcostal area increased, and any movement exhausted him. On the morning of April 4 he was weaker and had had two rigors during the night. His condition was grave, and occasioned great anxiety. Emetine hydrochloride was initiated, 1 gr. (65 mg.) intramuscularly daily. Rigors and sweats ceased from that time, and 18 hours after his first injection he was less tender, and 36 hours later he was asking for food for the first time, and was able to turn in bed. He made an uninterrupted recovery and was afebrile by April 9. He had emetine for six days, and then chloroquine by mouth for three weeks. At the time of writing he was very fit, but his diaphragm was still elevated.

Case 2

A Pole aged 44, who had lived most of his life in Paris before the war and had never been further south than Marseilles, where he had once spent a three-months holiday, developed pneumonia and a right pleural effusion in 1943 while serving with the Polish forces in Scotland. The effusion took several months to resolve. In 1946 he was admitted to the Middlesex Hospital with a recurrence of pain and signs of effusion in the right lower chest, a raised diaphragm, fever, and leucocytosis. He failed to respond to penicillin and sulphonamides. He spoke little English, but on being approached with a sigmoidoscope to search for amoebic ulceration he announced that he was "very small." This was confirmed by the finding of a rectal stricture. With the aid of an interpreter there emerged a story of diarrhoea and what sounded like a mucosal slough while in Paris before the war. I carried out exploratory

needling of the liver and produced 30 ml. of "anchovy" pus. Some air was returned, and *x*-ray films showed the abscess cavity. He made an uneventful recovery on emetine.

Discussion

Amoebic abscess of the liver is relatively common in this country: apart from the two patients recorded above, I have seen 15 further cases in civilian practice since 1946, 14 of these in Ipswich since 1949. For comparison during the same period I have seen two examples of liver abscess, apparently not amoebic; one contained *Haemophilus influenzae* the other *Staphylococcus aureus*. Of the series of 15 patients all but one were men, all had been in the tropics or subtropics at some time, though in many instances the "incubation period" was long enough to raise doubts whether their infection may not have been indigenous in origin.

Cases of Liver Amoebic Abscess Seen During 1946–60

Sex	Age	Incubation Period	Leucocytosis	Lobe of Liver
M	39	8 years	0	Right
M	38	15 ,,	0	,,
F	53	8 ,,	+	,,
M	52	11 ,,	0	,,
M	39	7 ,,	0	,,
M	33	6 months	+	,,
M	56	20 years	+	,,
M	72	50 ,,	0	,,
M	55	35 ,,	+	,,
M	21	6 months	0	,,
M	35	11 years	+	,,
M	43	7 ,,	+	,,
M	29	2 ,,	0	,,
M	47	25 ,,	+	Left
M	?	10 ,,	+	Right

The Table gives the age of these patients and the time interval since their last residence abroad. In only one case did the abscess present in the left lobe of the liver, and in seven there was absence of leucocytosis—a common feature of the disease, but one to which attention cannot be drawn too often because the diagnosis of hepatic amoebiasis is often ruled out of consideration by doctors without tropical experience, because of a normal white blood count.

There is little doubt that unrecognized amoebiasis exists in Great Britain, both as amoebic dysentery under the guise of ulcerative colitis, of which I have seen six samples, and under the diagnosis of non-specific, or cryptogenic, liver abscess.

When one recalls the attention to detail required in the collection of specimens and in laboratory identification, even in hot climates, it would not be surprising if many vegetative *E. histolytica* or cysts were missed in our colder country, with, for the most part, its inexperience in techniques. But, of course, it is exceptional to find the faeces positive in the hepatic form of the disease.

I believe that amoebiasis is rarely thought of when liver abscess arises in patients who have never been abroad, possibly with tragic results; I therefore recommend a therapeutic trial of emetine and chloroquine as of equal importance to antibiotics in all patients when liver abscess is suspected, and before resort is made to surgical drainage.

Summary

A case of amoebic abscess of the liver is recorded arising indigenously in a boy who had never left this country apart from two holidays in Northern Europe. A second case is described in a man who lived in Northern Europe all his life except for a three-months holiday in Marseilles.

Amoebic liver abscess is not rare in Great Britain, and reference is made to 15 further examples seen between 1946 and 1960, all of whom had lived at some time in accepted endemic areas.

In several of them the incubation period between residence in the tropics and onset was a long one. A trial of emetine and chloroquine, as well as antibiotics, is recommended in hepatic abscess of unknown aetiology before surgical intervention.

Case 2 was under the care of Dr. G. E. Beaumont, and I thank him for permission to publish the clinical details.

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PATHOGENESIS AND TREATMENT OF FISTULA-IN-ANO

BY

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[WITH SPECIAL PLATE]

Descriptions of fistula-in-ano are found in the oldest known medical writings. The condition would appear to have been commoner in ancient and mediaeval times than it is to-day, since it occupies more space in old manuscripts than its present frequency would justify. John of Arderne gave an excellent account of the disease and its treatment in the fourteenth century; his writings have been quoted frequently since the transcription of the manuscripts in the British Museum by D'Arcy Power (1910). From ancient times to the present day, treatment has remained the same—namely, operation in the form of a "lay-open" with knife or cautery or the use of a seton.

Perhaps the most widely held theory concerning the cause of fistula is that infection penetrates the wall of the anal canal through a fissure or other wound and that the infected track, once established, is maintained by faecal contents entering the internal opening. The "lay-open" procedure is based on this concept of aetiology. Any attempt at simple excision of the fistula is doomed to failure, because the surgical wound would also be affected by faecal contamination and would not heal. The only possible treatment, on this view, is to incise the track so that it becomes part of the anal canal. Provided that epithelization occurs from the edges the wound will become a new segment of the anal wall. Should the wound edges close over and heal prematurely the fistula will re-form deep to them.

However, infection rarely crosses the wall of any part of the large bowel spontaneously unless there is a free channel in the form of a diverticulum or ulcer. One of the most cogent arguments against the theory that infection is caused by an anal wound is the extreme