CLINICAL TROPICAL DISEASES

by

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INTRODUCTION

FOR some time we have felt the need for a handbook giving that information essential for the clinical diagnosis and the treatment of those diseases which occur primarily in the tropics. It is to fill this need that we have ventured to produce this volume. Our aim has been to supply essential facts as dogmatically and concisely as we can; where possible we have avoided speculation. We have made no attempt to provide information of an encyclopaedic nature. None other than the most brief descriptions are given of the organisms causing disease, their identification, and the vectors that convey them; such information is readily available in the text books on bacteriology and parasitology.

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AINHUM

DEFINITION

AINHUM is a name of South American origin applied to a fissured constriction, of unknown aetiology, which affects usually the fifth or fourth toes, and less commonly other digits of the feet or occasionally of the hands. The progress of the lesion is very slow, and ultimately it causes sequestration of the distal part of the affected digit.

GEOGRAPHICAL DISTRIBUTION

Ainhum occurs widely throughout the tropics and subtropics in the dark-skinned races. It is essentially a disease of negroes, and true ainhum apparently does not occur in the white-skinned races. It may develop in negroes who have been resident in the temperate climates for many years. Adult males are far more commonly affected than are adolescents or females.

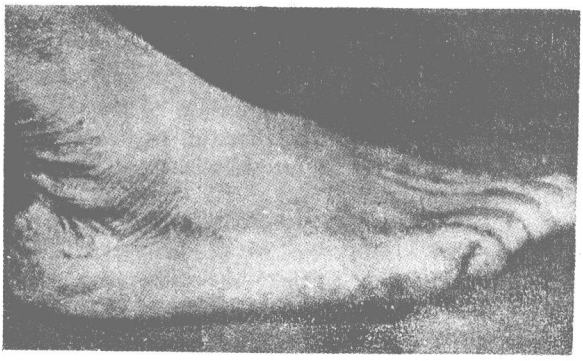

FIG. 1 a

PATHOLOGY AND CLINICAL PICTURE

The causation is unknown, but the tendency to ready keloid formation in negroes may be a contributory factor.

Ainhum starts as a fissure usually on the plantar and outer surface of the fifth toe. There is marked hyperkeratinization around the fissure, which in due course extends round the affected digit. A constricting fibrous band finally completely encircles the digit, commonly at the level of an interphalangeal joint, but sometimes in the middle

of a bony phalanx. The distal part of the digit becomes bulbous, misshapen, and everted; though painless it causes mechanical inconvenience and is prone to be traumatized. In time, often after many years, the distal part spontaneously sequestrates. One toe only, and then usually the fifth, may be involved; or the condition may be bilateral; or several toes, and very occasionally a finger, may be affected.

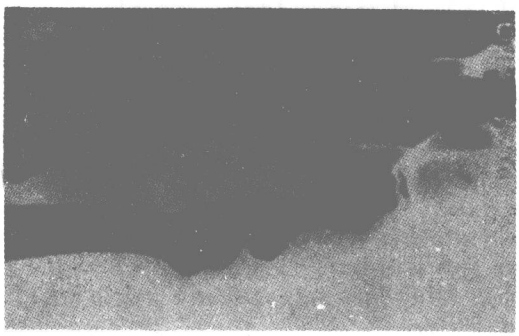

FIG. 1 b

FIG. 1 a and b. Ainhum of the fifth digit of the foot of an African.

TREATMENT

There is no specific treatment. Division of the constricting band and other conservative surgical methods of treatment are ineffective; amputation of the digit is the only satisfactory way of ending the inconvenience ainhum causes.

II

AMOEBIASIS

DEFINITION

AMOEBIASIS is the name applied to a state of infection of the large intestine with the protozoan parasite *Entamoeba histolytica*. In some cases the infection secondarily extends elsewhere from this primary location, for example to the liver.

GEOGRAPHICAL DISTRIBUTION

Entamoeba histolytica infection of man is of world-wide distribution. It is most prevalent in ill-sanitated areas, particularly in warm climates.

AETIOLOGY

Though essentially a parasite of man, naturally acquired, *E. histolytica* infections have occasionally been recorded in other primates, in rats, in dogs and, rarely, in other animals. Infection, in nature, is acquired by swallowing the cysts of the parasite passed in the formed stools of those infected with it. The cysts if kept moist survive for some days in faeces; if washed free from faeces and kept at low temperatures they survive longer. They will not withstand drying or high temperatures, and are readily destroyed by disinfectants.

Foodstuffs faecally-contaminated by uncleanly habits, or indirectly by the agency of flies, are the usual medium of infection; water is much less commonly the vehicle. Cysts on being swallowed are incubated in moist surroundings as they pass down the intestine. In the lowermost part of the small intestine or in the uppermost part of the large intestine each viable cyst hatches and liberates a four-nucleated amoeba. The latter gives rise to a number of single-nucleated entamoebae which establish themselves in the large intestine, their precise location in some cases being in doubt (see PATHOLOGY).

The motile vegetative entamoeba is the only form of this organism which parasitizes man; it multiplies by simple division. When there is diarrhoea some entamoebae are carried rapidly down the bowel to the exterior, and are to be found in the fluid faeces in the amoeboid form. When there is no diarrhoea amoebae being conveyed slowly down the bowel round up into a sphere, secrete around themselves a cyst wall, and appear in the formed stools as the morphologically characteristic *E. histolytica* cysts. It is the time spent in passage to the exterior in the faecal stream that determines whether the parasites are voided in the amoeboid or in the cystic state. Amoebae, therefore, are normally

found only in loose stools, and cysts in formed stools. A sudden sharp purge obviously may result in a loose stool containing cysts; but if the purgation continues amoebae only are to be found in the continuing loose stools, the cysts already formed now having been evacuated from the lower bowel and insufficient time being allowed for others to develop. Cysts once formed in the lumen of the bowel do not excyst again in the same host; the cysts, therefore, are not parasitic, and they are harmless to the host in whose bowel they are formed; their presence is an indication of the infection with amoebae from which they derive.

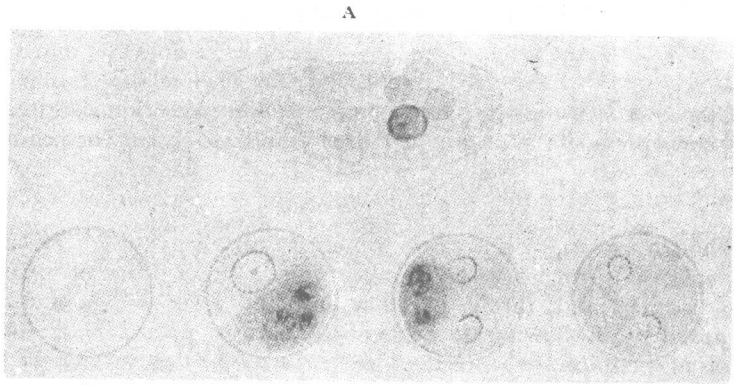

FIG. 2.

Entamoeba histolytica. A, Vegetative amoeba; B, Unstained cyst showing chromidial bar; C, D, E, Iodine-stained cysts, C and D showing glycogen mass. (Diameter of cysts, 14 μ.)
[From E. Noble Chamberlain, *A Textbook of Medicine*, John Wright & Sons Ltd., Bristol, 1951]

PATHOLOGY

The effect of a bowel infection with *E. histolytica* varies from individual to individual. Infections acquired in the temperate climates rarely cause clinical manifestations of their presence; infections acquired in the tropics commonly do so, and classically cause amoebic dysentery.

PATHOGENICITY

Various theories have been advanced to account for these facts. It has been suggested that strains of *E. histolytica* vary in their pathogenicity; those producing cysts of less than 10 μ in diameter rarely give evidence of pathogenicity. Large cyst-producing races of the parasite by no means constantly cause clinically overt infections in man, though invariably they are pathogenic when suitably introduced into experimental animals. It has been stated that *E. histolytica* always causes

invasive lesions in the bowel of man and that it is the number, the extent, and the distribution of these that determine clinical patency; but post-mortem studies fail adequately to support this view. Again, it has been postulated that *E. histolytica* is normally a commensal parasite of the large bowel, and that it lives on the surface of the mucosa as does *E. coli*; when lesions occur in the mucosa from some other cause, as when inflammatory changes occur in it, or for some yet unexplained reason, the amoebae become haematophagous and invade the tissues of the bowel wall causing pathological lesions. There is at least some indication that *E. histolytica* in the bowel may alternate between pathogenicity and commensalism; the classical course of amoebic dysentery tends to support this view. Finally, it has been claimed that the establishment and maintenance of an *E. histolytica* infection in the large bowel is dependent on the presence of a suitable accompanying bacterial flora. In support of this view are the facts that the parasite has not satisfactorily been maintained in culture in the absence of bacterial growths; and that modification of the intestinal flora by treatment with antibiotics, and other anti-bacterial drugs themselves not amoebicidal, may be followed by the disappearance of an amoebic infection. Against this view is the fact that amoebic abscesses in the liver and elsewhere flourish though they remain bacteriologically sterile.

LOCATION AND SPREAD

The primary *E. histolytica* infection is always located in the large intestine. Secondarily it may spread locally to surrounding structures within the abdomen, and it may extend directly by artificial or natural orifices to the exterior. Additionally, the infection secondarily may be spread extra-intestinally by blood-borne embolism, commonly to the liver; thence it will extend directly to neighbouring structures, and it may even be further conveyed embolically elsewhere.

In the primary infection in the large intestine the lesions are found most plentifully at the points of stasis, that is in the caecum and at the flexures. In those with a minimal symptomatology they may be confined to these points. In more severe infections they occur more widely; in extremely severe infections enormous numbers of amoebae will be found in the mucosa and submucosa of the large bowel throughout its entire length. In such cases the submucosa commonly is extensively fenestrated by contiguous and communicating lesions beneath large irregular areas of sloughing mucosa.

HISTOLOGY

In every instance the essential pathological process is one of amoebic invasion causing a bacteriologically sterile colliquative necrosis. The amoebae secrete a ferment which liquifies the cells with which it comes

into contact; the amoebae subsist on the pabulum so created, and also engorge cellular particles, more especially red cells. The lesions are not inflammatory; they contain no true pus, but consist of the lysed debris of the affected tissue. An amoebic liver abscess exemplifies on a gross scale the characteristic histology of any amoebic abscess, even the minute abscesses seen microscopically in the submucosa of the large intestine. The contents of an amoebic liver abscess are broken-down

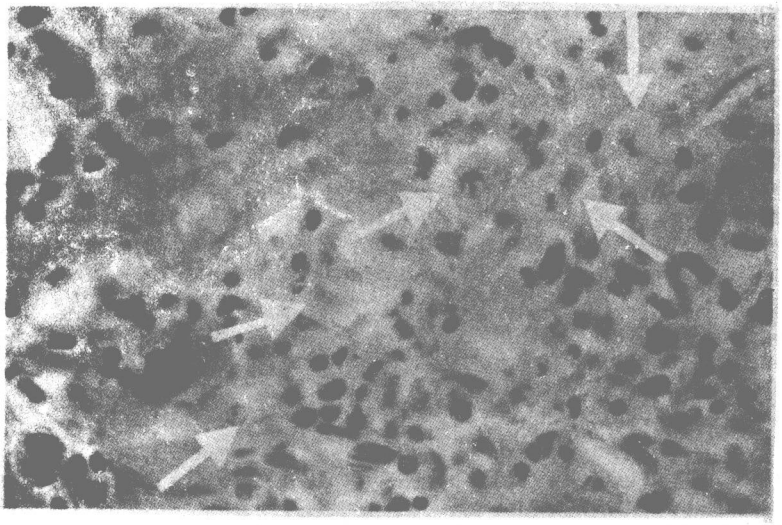

FIG. 3. A section of the submucosa of the colon containing many tissue-invading *E. histolytica*.

liver substance, the classical 'anchovy sauce' content characteristic of such an abscess; most of the amoebae are invading fresh tissue around the margins of the lesion; there are but few in the central necrotic area. There is no cellular walling off of the lesion; no fibrous tissue capsule is formed around it; its contents are not under pressure.

A contaminant bacterial infection in due course may appear in amoebic lesions, especially those in the bowel wall; this obviously will lead to modification of the histological appearances, but this modification is solely a result of the secondary bacterial infection.

PROGRESS

The course of the intestinal manifestations in all but fulminating infections is characterized by extreme chronicity, with periodic exacerbations and, often, lengthy remissions. The reason for this is not clear. It may be that some humoral or similar defensive mechanism checks

the activities of the parasites and leads to the restriction, if not the disappearance, of the intestinal lesions during the quiescent periods. It may be that the parasite becomes nonpathogenic and lives commensally within the lumen of the bowel during the intermissions, and that the exacerbations are manifestations of recurring pathogenicity and tissue invasion.

EXTRA-INTESTINAL INFECTIONS

An established extra-intestinal infection, however, rarely spontaneously retrogresses or resolves in the absence of specific treatment. An amoebic abscess of the liver, for example, steadily extends radially beyond the liver into the abdomen and its viscera, or through the diaphragm into the chest and its contents, or to the exterior through the chest or abdominal wall. The direction of its extension is governed solely by its initial location in the liver. In addition to extending locally amoebae in a liver abscess may be conveyed elsewhere embolically. Amoebic abscesses due to embolism of the parasites have been described in the lungs, pericardium, brain, spleen and more rarely in other tissues.

Any tissue in contact with an amoebic infection, or subject to contamination with a discharge containing amoebae, may become infected with *E. histolytica*. A fistula or fissure *in ano* may become infected; in debilitated incontinent persons the skin of the perinaeum and buttocks may become involved. Drainage wounds, such as a colostomy, may be followed by a spreading cutaneous amoebiasis around the opening.

CLINICAL PICTURE

ASYMPTOMATIC INFECTION

Asymptomatic intestinal infections are the rule in those acquiring their infections in temperate climates. Nevertheless it is not unusual to ascribe a most diverse symptomatology, both intra-abdominal and widely remote from the abdomen, to such infections. There is no adequate justification for this.

AMOEBIC DYSENTERY

Asymptomatic infections also are acquired in the tropics, but there true amoebic dysentery is very prevalent. The symptoms of this may appear within a week or two of infection or be delayed for months or years. The onset of an attack of amoebic dysentery is associated with looseness of the bowel usually with evacuation of up to six or eight, but rarely more than a dozen, mucoid blood-stained motions a day. Colic and tenesmus are unusual unless there is a lesion immediately inside the anus. On physical examination there may be no signs of significance. Occasionally, and especially during more acute attacks,

there is palpable thickening, with tenderness on pressure, of the caecum or of the descending colon and sigmoid flexure. There is no fever or toxaemia and little prostration, so the patient if he wishes to do so usually is able to continue his activities. The duration of an attack of amoebic dysentery of ordinary severity may be a few days or it may last for some weeks; it then usually subsides spontaneously. There follows a period of remission which may last days, weeks, months, or even years; during this the patient not uncommonly is constipated. In the quiescent period any digestive disturbances and bowel discomforts are commonly ascribed to the infection; often it is doubtful whether they can truly be regarded as due to it. Another attack of dysentery then follows. This sequence of attacks of dysentery followed by intermissions associated with constipation, which may continue for years and even for the duration of the patient's life, constitutes the classical picture of amoebic dysentery. At any time complications, especially an amoebic liver abscess, may develop; they do so in about one-fifth of neglected cases.

Fulminating attacks of amoebic dysentery rarely are seen in those otherwise well; but they occur frequently in debilitated subjects, who are suffering from malnutrition and from a variety of concomitant infections such as chronic malaria, hookworm disease, and other intestinal infections. In such cases the destruction of the mucosa and submucosa, and even the muscular layers and serous coat, throughout the large bowel is extensive; the severity of the attack is in proportion; the mortality, in the absence of prompt specific treatment, is correspondingly high. Complications of all kinds are prone to occur in these fulminating cases.

COMPLICATIONS

Local — The direct complications of an intestinal infection are haemorrhage, often considerable, from erosion of a large vessel in the bowel wall; extension of the infection through the bowel wall, with the formation of amoebic granulomata (amoebomata); and frank sudden perforation. All are rare in the classical case of average severity. Amoebomata are usually the result of extension of the amoebic together with an accompanying bacterial infection. Hard inflammatory tumours therefore form at the site of the lesion, and these develop and extend in the abdomen with an accompanying pyrexia. This complication is a serious one, and once established does not readily respond to specific anti-amoebic treatment.

Remote — Of the remote complications of the intestinal infection by far the most common is embolic spread of the amoebic infection to the liver. This occurs in from 5 to 15 per cent of all neglected cases of amoebic dysentery; it may appear even in those who, on questioning,

give no history of an attack of dysentery. It is most unusual for evidence of amoebiasis of the liver to appear during a frank dysenteric attack; it most commonly makes its appearance during a remission, when there is no symptomatic evidence of the intestinal infection. Its development often is slow and takes several weeks; occasionally it is extremely rapid and a large abscess may develop within a week or two. The diagnosis of hepatic amoebiasis is always difficult; it is particularly so in its earliest stages.

The first stage of an amoebic infection of the liver is the lodgment of amoebae, usually at several points, in the liver, most often in the right lobe. Multiple small focal amoebic abscesses form, as colonies of parasites develop from these amoebae and extend their activities radially. At this early stage the patient suffers discomfort and fullness in the liver area; there is usually liver tenderness; and the liver becomes engorged and enlarged, particularly in the affected lobe. The temperature is irregular; the patient progressively feels more unwell; he suffers from sweats at night; and there is some leucocytosis. There is never jaundice. This stage has been dubbed 'amoebic hepatitis'. It has been suggested that it is much more common than at present is recognized, and that in many or even in most cases this early infection spontaneously dies out in the liver and the condition subsides; it is doubtful whether there are adequate grounds for this contention.

The multiple small amoebic abscesses steadily enlarge and eventually impinge and coalesce, giving rise to an extremely irregular single abscess. This steadily progresses peripherally until it destroys the whole of a liver lobe, or extends through the liver margin to adjacent tissues. Discomfort and even pain are now continuous over the liver area; pain is referred to the tip of the right or left shoulder when the abscess is located high in one or other lobes of the liver, and there is commonly an irritant cough. The amount of liver destruction and the extrahepatic extension of the abscess, and so the local signs and symptomatology, are governed by its initial location in the liver. In any event the patient becomes prostrated and gravely ill; there is a swinging intermittent or remittent temperature; and there is a moderate leucocytosis of from 15,000 to 25,000 cells per cmm, the character of which is not specific. The patient suffers from drenching sweats, especially at night when sharp peaks of high temperature occur. Bulging of the liver may be felt below the costal margin; it may be seen in the upper abdomen; it may be visible in the intercostal spaces; or it may only be detected on x-ray screening of the diaphragm, which will be found to be raised, often irregular, and much limited in movement over the site of the abscess. If the condition is neglected the abscess ultimately will erode its way into the abdomen, through the anterior abdominal or through the chest wall, or through the diaphragm into the pleura