AMOEBIASIS AS IT CONCERNS THE GENERAL PHYSICIAN

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Amoebiasis and Amoebic Dysentery

Amoebiasis and amoebic dysentery are not synonymous. The term amoebiasis usually is employed to mean infestation of the large bowel with the protozoan parasite Entamoeba histolytica without symptomatology as a result of the infection.

The organism may dwell in the lumen of the bowel on the surface of the mucous membrane, as does Entamoeba coli, living commensally with its host and producing no symptoms. Cysts are produced within the lumen of the gut and these can be recognized in the faeces. This benign equilibrium may be modified, the amoeba assuming invasive properties by secreting a cytolysin, eroding the mucosa, penetrating to the deeper tissues, and producing the typical flask-shaped ulcers which may spread widely. Under these conditions, it feeds on lysed tissue cells and may engorge red cells.

Amoebiasis is world-wide in distribution and is found quite irrespectively of climate; it is common in the inhabitants of this country who have not been out of the British Isles—at present possibly it occurs in about 5 per cent. of people continuously resident here. Only very rarely in the temperate climates does the infection cause clinical manifestations—amoebic dysentery—and the recorded number of such authochthonous cases of amoebic dysentery in indigenous inhabitants of the British Isles is very small. Why the infection should almost invariably remain non-pathogenic in these people is a matter for speculation. It is not that certain local races or strains of the parasite possess no pathogenicity, while other strains are pathogenic. Nearly all strains of the parasite, whatever their source, prove pathogenic when suitably introduced into experimental animals. Probably, active pathogenicity of the parasite in the bowel is determined by a variety of factors, of which the constitution of the associated intestinal flora is one of the most important. Asymptomatic, and so presumably commensal, parasitization of the large intestine in the indigenous inhabitants of the British Isles can safely be ignored—as in practice it invariably is; even if its presence is detected it need not be treated.

Quite otherwise is an infection acquired in an area where clinical amoebic dysentery is known to be endemic. Such an infection, whether causing obvious clinical manifestations or not, can not be disregarded in view of its potentialities. This applies to infections found in residents in Great Britain who have returned from overseas, and who therefore may have become infected abroad. Pathogenic intestinal infections with E. histolytica commonly are characterized by periods, often lengthy, of latency. An infection in a returned traveller, therefore, even when apparently causing no manifestations at the time the patient is seen, must always be suspect; it can not safely be ignored. It should be eradicated, for if allowed to remain not only may it cause a relapsing dysentery but it may originate a much more serious amoebic liver infection going on to amoebic liver abscess formation. This and similar complications of a pathogenic primary bowel infection with E. histolytica are conditions of much gravity unless diagnosed early, and promptly and efficiently treated.

At the present time, both in this country and in the tropics, there is a great deal of needless confusion about amoebiasis, and about amoebic dysentery and its complications. The primary essential to all of these is the establishment and the persistence of the parasite in the large intestine. Its presence there can only be proved, or disproved, by competent examination of the stools, in which some of the parasites escape to the exterior. If there is looseness of the bowel the parasites will be passed in the amoeboid stage; in this stage they must be sought for within a few
hours of their passage or they die and disintegrate, and so become unrecognizable. If the stools are formed the parasites will be passed in the cystic stage; these cysts are resistant to environmental change and so will survive and remain recognizable for several days. Cysts are the only stage of the parasite infective to others; they are formed solely in the lumen of the lower large intestine; they are never found actually in lesions in the wall of the bowel or elsewhere. Unfortunately, the number of parasites—particularly in the cystic stage—present in the stools fluctuates greatly. Sometimes they may be very numerous and so they can easily be found; the following day they may be so scanty that they can not be found even on long search. It follows that repeated daily stool examinations, usually over at least six days, are necessary before the presence of the parasite can with reasonable certainty be excluded.

Having found parasites in the stools it is next necessary to arrive at a decision as to the significance of the finding. In view of the foregoing it obviously is important to find out where the patient has been, not merely recently but for many years back; whether he has a history suggestive of relapsing dysentery; and what his recent and current symptomatology indicate. Amoebiasis is common and universal; it therefore does not follow that the discovery of the parasite accounts for a patient's condition, unless the history and the clinical condition conform to the recognized picture. Specific treatment for an E. histolytica infection is almost never justified until the parasite has been found and competently identified. Under reasonably good conditions for the practice of medicine, speculative treatment for the infection and a post hoc diagnosis are a confession of incompetence. A 'therapeutic diagnosis' is a thoroughly unsatisfactory one in this as in other parasitic diseases. It should not be indulged in.

**Amoebic Hepatitis and Liver Abscess**

In individuals who have harboured the organism as a pathogen in the colon, often for some years, liver infection with the parasite may occur. It is probable that amoebae from time to time are released into the portal blood stream from intestinal lesions and are carried embolically to the liver. They may not always establish themselves in the liver and it has been suggested that they only do so when the gut infection has produced in the liver a state of tissue sensitivity. However this may be, at times they cause a number of small scattered foci of infection in the liver; and the great mass of liver parenchyma between these foci remains singularly unaffected. As they enlarge and spread adjacent foci of amoebic colonization in the liver fuse, so forming an irregular abscess, which continues to expand peripherally.

The earliest stage of liver infection clinically is referred to as that of amoebic hepatitis. This is of insidious onset, with intermittent or remittent fever and pronounced sweating, and it may progress slowly over weeks, with a steady loss of weight and of condition. The liver is enlarged and tender, and there is a moderate degree of polymorphonuclear leucocytosis. At this stage the clinical signs respond rapidly to treatment with emetine or with chloroquine. The later stage of frank amoebic abscess may follow evidence of an amoebic hepatitis, but in some cases there is only a history of ill health and dyspepsia with bowel irregularity, and but little direct clinical evidence of the antecedent liver infection. The localizing signs of an amoebic liver abscess are dependent upon the site and size of the abscess. Most abscesses develop in the right lobe; probably they arise from infection from lesions in the caecum and ascending colon, which drain to the right lobe through the superior mesenteric vein. The contour and movement of the diaphragm commonly are modified. When the infection extends through the diaphragm changes occur within the chest due to invasion of the pleura and the lung. An amoebic liver abscess may extend into the peritoneal cavity, the gut and other abdominal organs, the pleura and lung or pericardium, or directly through the chest or abdominal wall. The direction of its extension is dependent on its location in the liver, and in every case the organ or tissue involved is the site of an extension of the amoebic infection.

**Treatment**

The clinical arrest of an acute attack of amoebic dysentery is a simple matter. One grain of emetine hydrochloride, subcutaneously or intramuscularly, daily for three or four days will achieve this. But neither this dosage nor any other of emetine, however long continued, will do more than stop the attack. Emetine alone will not sterilize the bowel infection, and so when it is stopped the infection continues and clinical relapse of the disease can occur. As emetine is a toxic drug it should not be continued needlessly.

Having stopped the dysentery with emetine further treatment is needed to eradicate the residual bowel infection, and a variety of amoebicidal drugs such as EBI chiniofon and Diadoquin are usually given simultaneously to achieve this end. Their selection and methods of administration are matters of choice and experience, but competently given over a period of from two to three weeks successful sterilization of the bowel infection is obtained in all but a very small
minority (less than 5 per cent.) of cases. It is a not unusual practice to give amoebicidal drugs continuously for many weeks or months but such a practice has nothing to commend it if the drugs are in fact effective; if they are not effective there is no point in prolonging their use.

The small minority of cases not cleared of the bowel infection by three weeks treatment with the recognized amoebicides should be retreated with a second course of these with the addition of one of the tetracycline antibiotics. Such treatment very rarely demands repetition. The gauge of cure is absence of parasites on daily examination of the stools over a couple of weeks, done some few weeks after the completion of treatment.

The extra-intestinal infections with amoebae, such as an amoebic hepatitis or amoebic liver abscess, are more susceptible to treatment with emetine than is the primary bowel infection. A course of daily emetine injections should be given for 10 to 12 days, and this almost invariably will sterilize the extra-intestinal lesions of their parasites. If there is a large amount of liver destruction—a big abscess—drainage of this by aspiration may be desirable, but this is not always necessary, especially if the patient is treated before the abscess becomes large.

An alternative to emetine for the treatment of liver amoebiasis (but not effective at other sites of infection) is the antimalarial drug chloroquine. This is concentrated and stored in the liver, and there very effectively destroys amoebae. It is given orally in doses of $\frac{1}{2}$ to 1 g. daily for five to seven days.

Finally, when the extra-intestinal infection has successfully been dealt with by emetine, or chloroquine, treatment of the still persisting primary bowel infection must not be overlooked. If allowed to remain dysentery and its complications can recur; unfortunately this is allowed to happen only too frequently after a patient makes an excellent recovery after specific treatment of an amoebic liver abscess. His treatment is incomplete while his intestinal infection remains, and this must never be forgotten.
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