SOME OBSERVATIONS ON AMOEBIASIS IN THE COLOURED POPULATION OF DURBAN

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Durban has long been known as a hotbed of amoebic disease. What, however, has perhaps escaped general recognition is the marked variation in the severity of the disease amongst the different races locally resident. While the Native goes down in his thousands to fulminating amoebic dysentery and hepatitis, the European by comparison bears a charmed life. Where he falls ill at all, the disease is usually mild. Between these two extremes, is the intermediate picture of amoebiasis in the Coloured—seldom fatal yet frequently temporarily disabling; very common yet responding rapidly to treatment. In an attempt to throw into relief some of the highlights of this latter picture, a study was made of all cases of amoebiasis admitted to the Coloured wards of Addington Hospital over a period of 6 months.

RESULTS OF STUDY

The results of this study may be summarized as follows:

Statistics

1. Over the 6 months January to June 1955, 24 cases of amoebic dysentery and 11 cases of hepatic amoebiasis were admitted to the Coloured wards. During this period the total intake of patients (medical cases only) was about 300. That is, about 1 case in 10 was of amoebiasis.

2. In this same period, in corresponding European male and female medical wards, with about the same total intake, only 2 cases of amoebic dysentery were seen; these were both very mild clinically.

3. Sex incidence: of dysentery, 19 male to 5 female; of hepatic amoebiasis, 11 male to 0 female. This is the usual well-recognized male preponderance.

4. Concomitant dysentery and 'hepatitis': In 7 of the 11 cases of hepatic amoebiasis amoebic dysentery was present too.

Clinical Course

1. Dysentery cases: usually mildly ill. Of the 24 cases, 14 were apyrexial throughout despite macroscopic blood and mucus in the stools. Of the remaining 10 cases all but 2 were apyrexial within 2 days of starting treatment.

2. Hepatic amoebiasis: generally, all appeared ill and 'toxic'. Eight of the 11 cases had temperatures of 101-102°F. In most cases the pyrexia resolved after 4 or 5 days' treatment.

Electrocardiograms

ECGs were taken before, and immediately after, the administration of emetine, 1 gr. daily for 10 days. All were normal initially except one with left bundle branch block. Of a total of 32 such ECGs, 29 showed flattened or inverted T waves after emetine. Three showed no change. The left bundle branch block remained unaffected, otherwise than for minor T-wave changes.

Liver

1. The *liver function tests* applied were the following: Direct and indirect van den Bergh, serum bilirubin, alkaline phosphatase, zinc turbidity, cephalin cholesterol, colloidal gold, serum proteins (albumin and globulin).

(*a*) Of amoebic dysentery 16 cases were tested, of which 8 showed minor abnormalities (Table I).

TABLE I. CASES OF AMOEBIC DYSENTERY

Case No.	*Bilirubin*	*Zinc Turbidity*	*Serum Proteins*	*Albumin*	*Globulin*	*Cephalin Cholesterol*
1	1·2					
2		4·3				
3			5·56	2·16	3·40	
4		6				
5		4·5				
6*		10	7·75	3·45	4·30	+++
7		4·3				
8		8	7·10	2·97	4·13	

* An alcoholic with gross pellagra.

(*b*) Of hepatic amoebiasis all 11 cases were tested, of which 8 showed abnormalities (Table II).

TABLE II. CASES OF HEPATIC AMOEBIASIS

Case No.	*Bilirubin*	*Zinc Turbidity*	*Alkaline Phosphatase*	*Colloidal Gold*	*Serum Proteins*	*Albumin*	*Globulin*
1		9			6·80	3·29	3·51
2		8			6·79	2·76	4·03
3		7					
4		4·5					
5					6·90	3·19	3·71
6			14		5·73	2·86	2·87
7					5·7	2·4	3·3
8*	11·7	8	41	5	5·34	2·66	2·68

* Diagnosed initially as infective hepatitis. Only 24 hours before death was the correct diagnosis made. At autopsy almost the whole liver was found to have been replaced by an amoebic abscess.

Figures and tests not shown in the tables were within normal limits.

2. *Aspiration of the liver* produced 'anchovy-sauce' pus in 5 of the 11 cases of hepatic amoebiasis.

3. Liver biopsies were performed in both dysenteric and hepatic cases:

(*a*) Twelve cases of dysentery, in which clinically there was no reason to suspect 'hepatitis', were subjected to liver biopsy. Of these, 5 cases were reported as showing 'increase in iron pigment with moderate portal round-cell infiltration'. In one other case a bilharzial egg was seen. In the remaining 6 cases, nothing abnormal was found.

(*b*) Nine cases of hepatic amoebiasis were subjected to liver biopsy. Of these, 5 showed 'mild periportal round-cell infiltration and some eosinophils. Some increase in iron pigment.' One case showed 'marked oedema of portal tract with cellular infiltrate of round cells, many eosinophils and foci of neutrophils'; this from a case of proven liver abscess. Another case showed 'localized inflammatory process with liver-cell necrosis, leucocytic infiltration, and fibroblast proliferation; changes consistent with the edge of an abscess. No amoebae seen'. This case did not yield pus on needling of the liver. The remaining 2 cases showed no abnormality on biopsy. In the one fatal case of the series, in whom autopsy revealed almost the entire liver to be replaced by pus, the liver biopsy report read, 'Mild periportal round-cell infiltration'.

DISCUSSION

A few points of theoretical and practical importance emerged from this study.

The general impression of a common, disabling yet seldom fatal, and easily treated disease, was confirmed. The Addington Hospital is the only large local hospital for Coloured and European patients. Consequently direct comparison of numbers of cases of amoebiasis, for a given number of patients, in each racial group, becomes significant. Craig[1] has stated that 'it is the consensus of opinion of observers in the tropics, that natives seem to suffer less with clinical manifestations of the disease than individuals of the white race in those areas'; and he goes on to postulate partial immunity to the parasite, acquired from constant reinfections since childhood. Others[2] have agreed with this. De Bakey and Oschner,[3] however, found no difference in racial incidence. In Durban the incidence of amoebiasis in the Native is notorious; this study shows further that the disease is far commoner in the Coloured (one hospital patient out of 10 has amoebiasis) than in the European (in whom only 2 cases were seen amongst 300 patients).

At Addington most cases of amoebic dysentery are treated with emetine, 1 gr. daily for 10 days, in conjunction with Diodoquin, chloroquine and an antibiotic, usually Terramycin. In the present series electrocardiograms were taken in 32 cases, both before and after 10 gr. of emetine. In 29 of these 32 cases, marked ECG changes were present by the 10th day. This finding is at variance with the experience of others. Thus Gutch[4] reports that after 10 gr. of emetine only 13 of 35 patients showed ECG changes. Zuckerbrod and Litwins[5] noted no changes in their 3 cases. It is not known why so much higher a percentage of our cases developed abnormal tracings. If one may speculate for a moment, there is perhaps a tie-up here with the not infrequent cases of congestive cardiac failure, of undetermined aetiology, seen in the Coloured.

Despite the almost constantly induced ECG abnormalities, and the very free use made of emetine, sudden death or myocardial failure while under treatment is unheard of here—certainly during the past several years. Zuckerbrod and Litwins,[5] however, commenting on emetine toxicity, recommend twice-daily determinations of blood pressure, and alternate-day electrocardiograms. Should any change appear in the ECG, they stop emetine immediately. In view of past experience, and the very high incidence of ECG changes, the only precaution taken here is to confine the patients to bed. Repeated blood-pressure determinations and ECG tracings are not done in the absence of clinical evidence of heart disease.

Amoebic hepatitis—as distinct from amoebic liver-abscess—is a diagnosis much beloved by local practitioners. The picture is one of a (usually) middle-aged male who complains of pain below the lower right ribs. Examination reveals some tenderness, often best elicited by a light punch over the liver. Non-essentials for the diagnosis, but often present, are: a history of previous or concomitant diarrhoea; pain in the right shoulder; right-sided pleuritic pain; fine crepitations heard at the right base, often only in front; a rise in, and immobility of, the right side of the diaphragm, detected sometimes clinically, more often radiologically; a right basal effusion; fever and a polymorphonuclear leucocytosis. Trophozoites or cysts of *Entamoeba histolytica* may or may not be found in the stools. The differentiation from an actual abscess is only that the case usually appears less ill, and that needling of the liver fails to produce pus. Put to bed and given emetine or chloroquine, these patients rapidly return to normal.

While in the more severely ill the differentiation between hepatitis and actual abscess becomes academic, milder cases present a problem. As understood here, the only essentials to the diagnosis of amoebic hepatitis, are an ill patient and a tender liver—a coincidence which may occur not infrequently and from a variety of causes. Irritated by the facility with which this diagnosis is often made, and piqued by the observation that the occasional case responded as quickly to bed rest alone as to active treatment, one awaited the results of the aspiration liver-biopsies with particular interest.

In the 'literature' varying pathological descriptions were found, each described as specific for amoebic hepatitis. Thus Sir Leonard Rogers,[6] in a classical paper, described 'presuppurative amoebic hepatitis', in which he envisages a few amoebae reaching the liver from slight or latent amoebic colitis, becoming entangled in blood clot in the small interlobular veins, and so causing acute congestion of the liver. He differentiates this phase sharply from that of amoebic liver-abscess. More recent authors[3] are content to include within the diagnosis of hepatitis, single or multiple liver-abscesses, too small for the presence of pus to be detected clinically. Thus Sherlock[2] describes the liver, in amoebic hepatitis, as 'presenting a moth-eaten appearance in which necrotic areas are surrounded by zones of congestion'. Classically, trophozoites of *E. histolytica* are found in the cytolysed areas. These small lesions may heal by connective-tissue replacement, in which case focal areas of scar tissue will be found scattered throughout the liver. In chronic amoebic hepatitis, Palmer[7] describes increased portal connective-tissue, often with a noticeable increase in lymphocytes and monocytes in the portal tracts.

Aspiration liver-biopsies were performed on 12 cases of proven amoebic dysentery in which there was no reason to suspect the presence of 'hepatitis', in 5 cases of amoebic liver-abscess proven by the aspiration of

'anchovy-sauce' pus; and in 4 cases of classical amoebic hepatitis. In only one case of hepatitis did the biopsy yield anything other than 'mild periportal round-cell infiltration'. In this single case changes consistent with the edge of an abscess were seen, but amoebae were absent. One other biopsy, in a case of abscess, showed oedema and cellular infiltration of the portal tract. In none of the cases of dysentery, nor in the remaining 7 cases of hepatic amoebiasis, were any significant changes present. It is of interest that in the one fatality of the series, in which at autopsy almost the entire liver was found to have been replaced by pus, the biopsy report read only 'mild peri-portal round-cell infiltration'. At post-mortem examination the track left by the biopsy needle was seen to be within an inch of the abscess.

On the whole, then, aspiration liver biopsy proved a sore disappointment as an aid in the positive diagnosis of amoebic hepatitis.

Liver function tests were performed in 16 cases of dysentery, and 11 cases of hepatic amoebiasis. As can be seen in the tables, the incidence of abnormalities found was greater in the latter, but the changes noted were not marked enough to be helpful. The one case showing marked biochemical aberration was the fatality referred to above. Reports of liver function tests in hepatic amoebiasis are few[8, 9] but the consensus of opinion is that, while they often show slight impairment, they are not worth doing. This is in accord with our experience.

Sigmoidoscopy and rectal biopsy were also performed on all cases of hepatic amoebiasis, as on all cases of dysentery. In the absence of clinical dysentery, however, no help was forthcoming here either in the positive diagnosis of amoebic hepatitis.

It will be seen then that the diagnosis of amoebic hepatitis remains a clinical one, the differentiation from amoebic abscess, on the one hand, residing only in the fact that pus cannot be demonstrated; and from the other causes of a tender liver, on the other hand, in the different history clinical course.

SUMMARY

1. Attention is drawn to the high incidence of amoebiasis in the Coloured population of Durban. This is in contrast to the relative immunity in the European.

2. Aspiration liver-biopsy and liver function tests were found to be of little value in the positive diagnosis of amoebic hepatitis.

3. The frequency and nature of electrocardiographic changes following the administration of emetine, are briefly reviewed.

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