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Bilharziasis: A Serious Ailment*

BY

MICHAEL GELFAND, C.B.E., M.D., F.R.C.P.
*Professor of Medicine, University of Rhodesia
(with special reference to Africa).*

Is bilharziasis a serious disease? We continually ask ourselves this question. Those who practise in Rhodesia have never failed to warn the medical world that it is a disease which can carry a very real risk to life. We have reminded others that its attack on the ureters and bladder is of the highest importance, but for some reason, much of what we have said has been doubted. Perhaps this was because in territories outside Rhodesia little or no autopsy material was studied; and secondly, few African hospitals possessed facilities for proper urological investigation. Another error was the judgment of the disease in Europeans only, in the vast majority of whom I must admit its effects, although not to be scoffed at, are not very serious. It was not until recently that we have come to appreciate that bilharziasis in its early invasive stage may prove dangerous on account of the allergic non-immune type of response seen in the European. We now know for certain that in his so-called Katayama phase, and for several weeks after the ova are first passed, the patient, particularly a child, may suffer a severe attack on his nervous system with an encephalopathy, focal paralysis or even a paraplegia. I personally have not met with a death in the European, but recently I treated two very ill children who were so toxic that I feared for their lives. I believe they were saved by Ambilhar. They were desperately ill with severe long-continued dysentery, weakness and loss of weight.

When the disease is mainly localised to a particular part, such as the bladder regions or even intestines (some months or longer after it is contracted), the effects vary greatly, but are generally mild, e.g., lassitude. In a few patients the debility is severe and interferes with school work. That in itself is serious as it may affect the career of the sufferer. There is no doubt

that a fair number of Europeans, mostly in the 20-40-year-old age group, suffer from the more damaging effects of the disease on the lower ureter. In practice in Rhodesia we continually meet stenosis or dilatation of the lower third of the ureter on one or other side, less frequently on both, and in a few cases the blockage is sufficient to cause some degree of hydronephrosis or a pyelectasia of the kidney.

Mr. R. M. Honey has kindly allowed me to quote these figures which show that this type of lesion is not uncommon in European practice.

Table 1

TYPE OF UROLOGICAL LESION FOUND IN 22
EUROPEANS INVESTIGATED BY R. M. HONEY,
SALISBURY, RHODESIA

	No. of Cases
Calcification in bladder wall	2
Carcinoma of bladder arising in area of old bilharzioma	1
Fibrous contracture of bladder	0
Ureteric dilatation without stricture	94
Ureteric stricture with dilatation, but without hydronephrosis	66
Ureteric stricture with dilatation (27 on left, 22 on right, 9 bilateral)	58
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Other serious complaints, however, are rare. I have seen three European patients with bilharzial cirrhosis of the liver, one of whom, a man of 45, also had cor pulmonale.

I recently had a European lad aged 12 years admitted from Selukwe, where Dr. Saunders found him ill with a marked ascites and an enlarged, irregular and hard liver.* He had a blood eosinophilia of 25 per cent. However, we could not demonstrate any bilharzial ova despite a careful search, which included a cystoscopy and snip of the rectal mucosa. At laparotomy the liver was coarse and irregular and a wedge section of it revealed the presence of bilharzial lesions with increased fibrosis in the portal tracts. The parenchyma was unaffected.

* I have recently learnt from Dr. J. Shee that this patient was admitted to Bulawayo Central Hospital and found to have a constrictive pericarditis.

* Paper read at the First Rhodesian Science Conference in May, 1967.

I personally have never met with cancer of the bladder in a European with a bilharzial bladder and Mr. Honey states that he has seen only one case. A calcified bladder is encountered only exceptionally in the European.

In the African, on the other hand, the clinical picture is dominated by two very important factors, each as significant as the other. In the first place we have a people who have lived in contact with the infection for centuries, just as they have with malaria. The African is less sensitive to the disease than the white man and so we do not expect to meet the earlier symptoms I have just described in the Katayama phase of the disease. Thus we do not expect any of the serious attacks like encephalopathy, hemiplegia or a paraplegia—all of which are seen in the earlier phases of the disease. The African at this stage is not so seriously disturbed by the disease.

However, in the later phases, within a few years of contracting the disease, owing to his much heavier worm load from repeated exposure, there is a heavy ovideposition which in turn leads to much fibrosis. The gravity of the disease, as a rule, can be attributed to the local effects of this heavy deposition of ova in the tissues. We may find a small contracted fibrotic bladder causing pain and discomfort over the bladder itself, with an inability to retain urine for any length of time. According to Honey, this may also account for a high tension in the bladder, which in turn is reflected in the kidney tissue.

Recently in Salisbury we have found very strong evidence of what might be a very close association between a chronically infected bilharzial bladder and the subsequent development of cancer in that organ. We have known that most cancers of the bladder in the African here are of the squamous type rather than of the carcinomatous variety. Further, we know that the African here develops bladder cancer at a much earlier age than the European. We have also been able to show that Africans with a calcified bladder are more liable to develop cancer in that organ than those who do not have this change in their bladder. Bladder calcification is a sign of the presence of a severe grade of bilharzial infestation. Another valuable finding is that bladder cancer in the African is associated with a much greater frequency of ovideposition by *S. haematobium* in the rectum than in a control group of Africans who did not have cancer of the bladder (Gelfand, Weinberg and Castle, 1967).

There is already strong evidence accumulating in Salisbury that bilharziasis cannot be dismissed as a possible cause of hypertension (Gelfand, 1962, 1964). Even if the link between pyelonephritis and hypertension is still not proven, although its relationship is more than likely, there can be no doubt that in Africans bilharziasis produces a severe effect on the ureter, particularly in its lowermost portion, where it enters the bladder. This may be associated with pain and back pressure leading to pyelectasia and hydronephrosis on the affected side. This complication of either dilatation or stricture, due to fibrosis following replacement of tissue by ova, may be mild, it is true, but not uncommonly the renal pelvis and calyces are much dilated. Patients with this disorder are not infrequently admitted to hospital with an attack of renal colic almost indistinguishable from that of stone. I would estimate that unilateral ureteric disease occurs in about one in 10 young Africans (15-20 years of age) living in endemic regions and that in one in 10 of the latter the effect is bilateral. And if I may hazard a guess (and not a wild one either), one in 10 of those with bilateral obstructive ureteric disease dies from uraemia.

Edington (1957) considers bilharziasis a not unimportant cause of death in Ghana. In 427 autopsies he considered death in nine could be attributed to heart failure from pyelonephritis. Further, he concluded in a separate study that bilharziasis could not be excluded as an aetiological agent of bladder cancer. He also found liver damage secondary to *schistosome* infection in 2 to 3 per cent. of the autopsy material.

Not to be forgotten altogether is the more than occasional calculus found in cases with the more chronic forms of the disease in the bladder. Sometimes these calculi reach quite an enormous size—even up to that of a pigeon's egg or larger. In my experience it is exceptionally rare to find a stone in an African who has not contracted bilharziasis.

In my last 50 cases of severe hypertension admitted under my care during 1965 and 1966, at least 17 had evidence of severe bilharziasis (seven had a calcified bladder and 10 showed ureteric disease with or without hydronephrosis on one or both sides). There may have been more cases, but it was not always possible to investigate the renal tracts or those who were anaemic or very ill. Further, of those 17 cases 11 occurred in men of 40 years and under—an age incidence which is perhaps less than one would expect in hypertension.

Table II

FREQUENCY WITH WHICH SEVERE BILHARZIAL LESIONS WERE FOUND IN 50 CONSECUTIVE AFRICAN CASES WITH SEVERE HYPERTENSION

Number of cases with severe bilharziasis	17
Number of cases without definite evidence of severe bilharziasis	33

Two other important serious effects of the disease we believe are mostly the result of an *S. mansoni* infestation. The first and more common is a severe fibrosis of the liver. Owing to the deposition of ova in the portal tract, granulomatous and fibrous tissue are laid down and this leads to portal hypertension with the risk of haemorrhage and liver failure in the more advanced cases when there is secondary pressure and atrophy of the liver lobules themselves. Although there is general acceptance that bilharzial fibrosis of the liver commonly occurs in Egypt and Brazil, this is doubted in Southern Africa, where many clinicians even wonder whether it exists at all. I personally was doubtful of this condition at first, but I have since found that this disorder is not frequently encountered in the adult over 40 years of age, but is seen in much younger age groups, especially in the juvenile. It is thus a disease of the young, most of whom are carried off by it before they reach the age of 40. I believe bilharzial fibrosis of the liver is not uncommon, certainly in Rhodesia and her neighbouring territories.

In 1964 I seemed to find bilharzial lesions more frequently in needle biopsy of the liver in juveniles affected by cirrhosis than in adult Africans (Gelfand, 1964). Since then I continued with this line of investigation, and although I did not have so many cases in the younger age groups as compared with the adult, there was still this suggestive evidence that cirrhosis in the juvenile is related to bilharziasis, as shown by Table III.

Table III

COMPARISON OF NEEDLE BIOPSY OF THE LIVER IN ADULT AND JUVENILE CASES OF CIRRHOSIS

Number Studied	Positive Bilharzial Biopsy
32 (adults)	3 (7.3%)
11 (juveniles)	4 (36.4%)

I should like to show you what we found in a recent survey in the Muzarabani Reserve, in the Zambesi escarpment, a few weeks ago. I had learnt that many people in this area were heavily infested with *S. mansoni*. We examined about 150 villagers and found nine children with grossly enlarged abdomens, and in all the liver and generally the spleen were clearly enlarged.

We admitted these children to hospital. I do not want to infer that this is the usual finding in any bilharzial region, but I wish to stress that in a heavily infested *S. mansoni* area liver disease is by no means uncommon. Two of the girls in this series also had large masses in the abdomen. In one it cleared with treatment, but bilharzial lesions were found in the liver. In the second the large mass (about the size of a big orange) around the umbilicus was shown to contain bilharzial granular tissue. Largely through Wydell (1958), who worked on an island on Lake Victoria, and Mynors (1957) whilst in the Sudan, our attention had been drawn to these bilharzial masses which may occur in the abdominal cavity or in the bowel. These may become so large or develop in such a site as to cause intestinal blockage. In the last few years we have become more aware of this rather serious complication of the disease.

The serious effects ovideposition may have on the lungs and hearts were brought to the notice of the world from Egypt. As a result of heavy deposits of ova in the small divisions of the pulmonary artery, the flow of blood is blocked. The pressure rises in the pulmonary artery and right heart, strain and failure may ensue. I do not think this condition is as common as was first alleged by Shaw and Ghareeb in 1938, but every now and then we meet it. Interestingly enough, it is commonly seen in association with serious liver fibrosis, so this condition of cor pulmonale seems to occur in relatively young age groups.

Many papers have been contributed on gynaecological disorders due to bilharziasis, but we do not seem to know for certain what effects follow deposition of ova in the tissues of the genital tract. I have seen many ovaries and fallopian tubes grossly damaged by ova, but do not know how much this involvement is responsible for symptoms. It would be tempting, for instance, to attribute a tubal pregnancy to bilharzial granulation tissue in a tube. This seems reasonable. On the other hand, if only a few ova are present in the tube it seems hardly likely that they would disturb its function. In other words, the whole picture of bilharziasis seems to be a matter of the degree of ovideposition in man. Where many eggs are deposited we are likely to find marked pathological effects followed by symptoms in the patient.

When ova are found in certain sites there is no doubt about the association between the patient's symptoms and these eggs. For instance,

haematuria is clearly the result of the lesions in the bladder mucosa. Bilharzial fibrosis in a ureter is accepted as a condition which can give rise to pain. This is because there are few other conditions with which it can be confused; but when it comes to ova in the appendix, fallopian tube or cervix there is invariably some doubt as to whether or not the symptoms could be caused by ova in these sites. When ova are found in a tube in which there has been a tubal pregnancy there is some doubt as to whether this was due to the ova. I think this is mainly because there are other more common causes of ectopic pregnancy, and bilharzial disease is so frequent that we are inclined to regard the passing of ova as coincidental. The same has been said in the past about cirrhosis of the liver and cancer of the bladder.

In conclusion, I would like to repeat that bilharziasis is a serious disease in the African of Central Africa, to whom it is a real handicap, particularly as it is so universal and circumstances are so favourable to heavy infestations. The situation is indeed a grave one—one which threatens the wellbeing of man.

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