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The Urological Aspects of Bilharziasis in Rhodesia

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PART I

Introduction

This study is based on experience gained in investigating and treating urinary bilharzial disease. It includes the examination of pathological material in the *post-mortem* room and laboratory and our studies of the records of 300 European and 100 African patients. The Europeans, who were seen over a 19-year period since 1938, had previously had bilharziasis and were referred for further investigation by doctors in practice. The Africans were studied during the years 1952 to 1953 and from 1955 to 1957 at the Salisbury African Hospital. Any African suspected of having urinary bilharziasis was further investigated, and if a change was noted in the ureter on pyelography, or bladder calcification on the straight X-ray film, the subject was submitted to a full urological study.

Fifteen to twenty years ago the disease was generally regarded as being benign, causing vague ill health or an occasional stricture of the ureter which produced colic. Treatment too was limited largely to the giving of an initial course of sodium antimony tartrate and a subsequent course of injections with each attack of colic. Strictures were usually treated by dilating the ureter regularly every two to three months. Since then the general outlook has changed, and during this time it has become apparent to us, especially from observations in the autopsy room, that the ureteric stricture, although an important lesion, is in fact only one facet of the picture and that great damage frequently occurs in the absence of any ureteric stenosis (Gelfand, 1948; Sayegh, 1950). We therefore persevered with these investigations in the clinical and pathological fields and now consider that we have a much clearer conception of the pathological changes and the results of treatment.

It is now 20 years since we divided our first intramural stricture of the ureter by cystoscopic meatotomy, 16 years since we excised our first strictured lower third of ureter and re-implanted the proximal segment into the bladder at a new site, and three years since we first carried out an ileocystoplasy designed to enlarge the fibrotic and contracted bladder. The results of our observations on these and subsequent cases form part of this paper.

It is clear to us that the disease manifests itself somewhat differently in the two races living in Rhodesia and Nyasaland. The disease has usually been diagnosed in the European while in the acute phase when haematuria presented, but when seen by the urologist the live worms no longer exist and he is faced purely with the results of the fibrotic process. The African is seldom seen until serious complications arise, and when examined he is seen to have both active and the chronic or fibrous disease of long duration.

Many people still regard bilharziasis as a benign disease, not to be feared, and causing little inconvenience apart from occasional slight haematuria and mild general ill health. These ideas are hard to contradict, since the vast majority of people who contract the disease either have no symptoms or only mild ones. The result is that one cannot travel far on the country's main roads without seeing numerous people swimming in the rivers and taking their children to paddle and bathe. The African living in the country has nowhere else to bathe other than in the local stream near which each village is sited. He also has no organised sani-

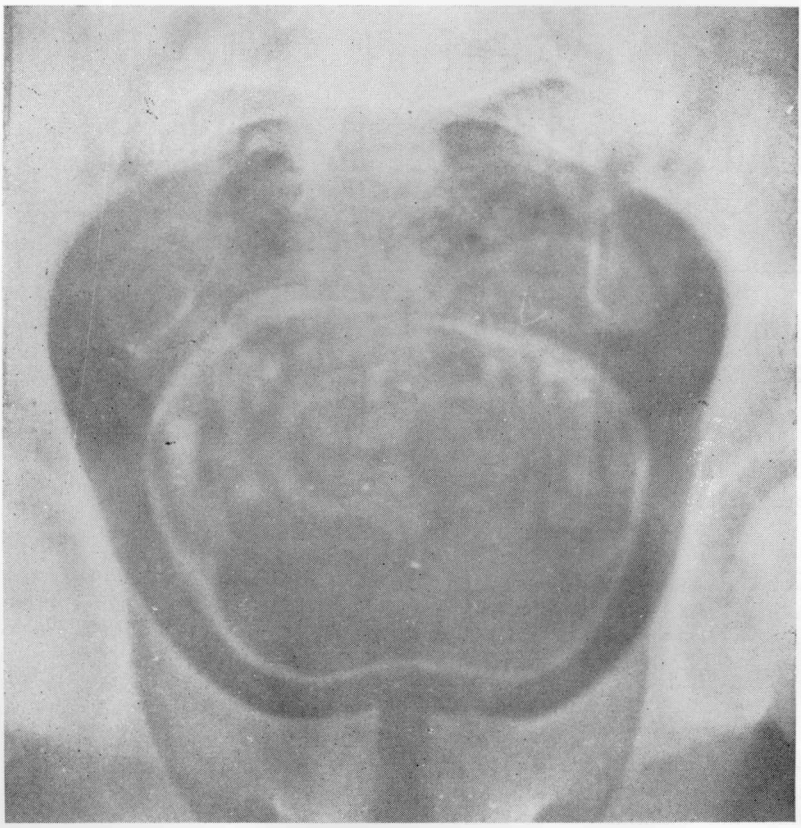

Fig. 1—African aged 15. Gross calcification of bladder and lower portions of ureters. Bladder capacity, 550 ml.

tary arrangements and uses the open veld as a latrine and passes urine freely in the rivers as he bathes or washes.

Our studies have shown that in a small but significant number of cases serious complications can arise some years after the infestation, and one of the tragedies is that these complications affect comparatively young people. The majority of our patients who were suffering from the late results of this disease were under 25 years of age.

The disease, although it causes chronic pain and ill health in the European, does not cause his death. The African, in whom the disease is more advanced, may die and, as this study illustrates, his lesions may be of a serious nature. Hence bilharziasis should never be dismissed as being merely a benign disease.

The subject will be discussed under the headings of pathology, symptomatology and diagnosis, including radiology, cystoscopic appearances and treatment.

PATHOLOGY

The adult worms inhabit the submucosa and here the female deposits her ova, which escape through the mucosa into the urine by means of their active enzymes. A small amount of bleeding accompanies their escape, especially when the bladder contracts towards the end of micturition. The haematuria in urinary bilharziasis, therefore, is mainly terminal.

Ova of *Schistosoma haematobium* are also found in most tissues in the body, including the rectum and lung, but are not usually found in faeces or sputum. Their enzymes seem specially designed to allow their escape from bladder submucosa. On studying microscopic sections of bladder and rectum one is usually impressed by the amount of fibrous tissue surrounding the ova. They seem to be encased in this site, with little chance of their escape, as foreign bodies. It would appear that miracidia in live ova excrete an enzyme or toxin which causes a local destruction of tissue, permitting the escape of

the ovum. With the death of the miracidia, fibrosis ensues. It is widely believed that dead ova are extruded from the bladder mucosa into the urine as foreign bodies, and therefore the presence of dead ova in the urine cannot be taken to indicate an active infestation with the worms. With this view we disagree. We think that only live ova escape from the tissues of the bladder into the urine, although they may not survive long after their escape and so appear dead when examined. If it were a rule for dead ova to be extruded as foreign bodies, we should expect to see calcified haematobium ova in the stool, since in urinary bilharziasis enormous numbers of ova are deposited in the rectal mucosa, but this is a very rare finding in Rhodesia. We therefore regard the presence of ova in the urine, whether alive or dead, to denote a living worm in the body and to indicate a course of therapy.

Although ova are usually scattered diffusely or collected into small nests in the submucosa,

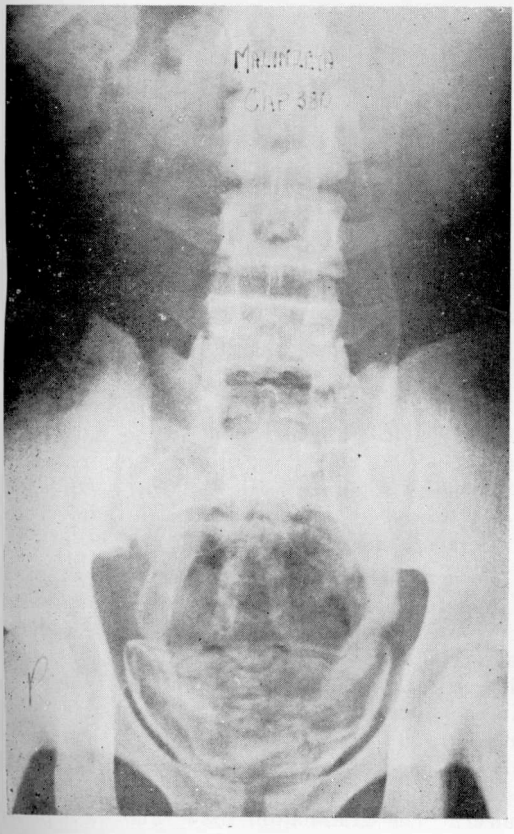

Fig. 2—African aged 32. Gross calcification of bladder, lower portion of right ureter and whole of left ureter. Bladder capacity, 380 ml.

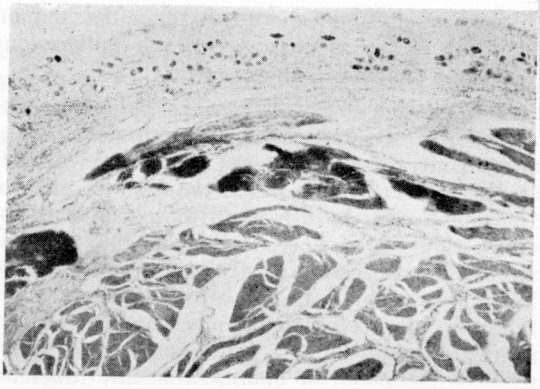

Fig. 3—Section of bladder wall showing deposition of ova in submucosa.

they are also found lying deeper between the muscle bundles in varying numbers and may at times reach the subserous coat and on occasion the peritoneal coat.

With the passage of time the ova become calcified. When there is a heavy deposition of calcified ova in the bladder wall, this can be seen radiologically as either a thin line or occasionally as plaques or islands of greater or lesser density within the bladder outline (Fig. 1). The calcification may also be seen extending up the ureter, usually involving only the segment nearest to the bladder; but when more extensive, along the whole length of the ureter as far as or even into the renal pelvis (Fig. 2).

The small seed-like swelling or tubercle, composed of granulation tissue, is the earliest lesion seen in the bladder. It is pale greyish-white in colour, firm to the touch and its base is surrounded by a thin line of pinkness due to capillary congestion at this site.

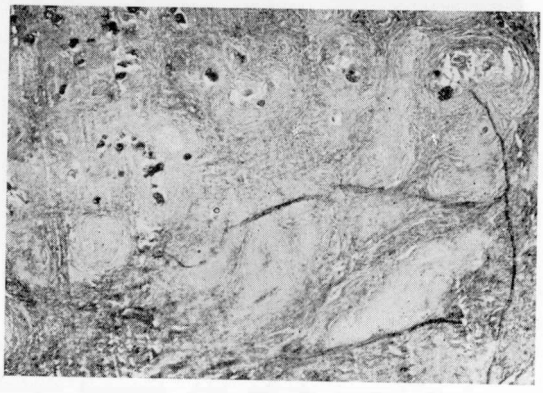

Fig. 4—Section of bladder wall showing ovideposition in muscular layers, with extensive fibrosis destroying and displacing the muscle.

Soon after, and consequent upon the amount of fibrosis, the mucosa assumes a pale colour with loss of the capillary vessels.

The tubercles, which may be few or many, are mostly seen on the base of the bladder in the vicinity of the ureteric orifices and on the fundus, although no area of the bladder mucosa is exempt. In the European the tubercle is the usual sign of the disease, but in the African, although tubercles are frequently found, the sandy patch which denotes a long-standing infestation is more typical. Sandy patches are less commonly seen in the European. The sandy patch is composed of innumerable tiny tubercles which have coalesced, or massive deposition of ova in the submucosa which have become encased in fibrous tissue (Fig. 3). The size of the patch varies from 0.5 cm. in diameter up to a diffuse yellow discolouration of the whole bladder mucosa. There is thus considerable individual

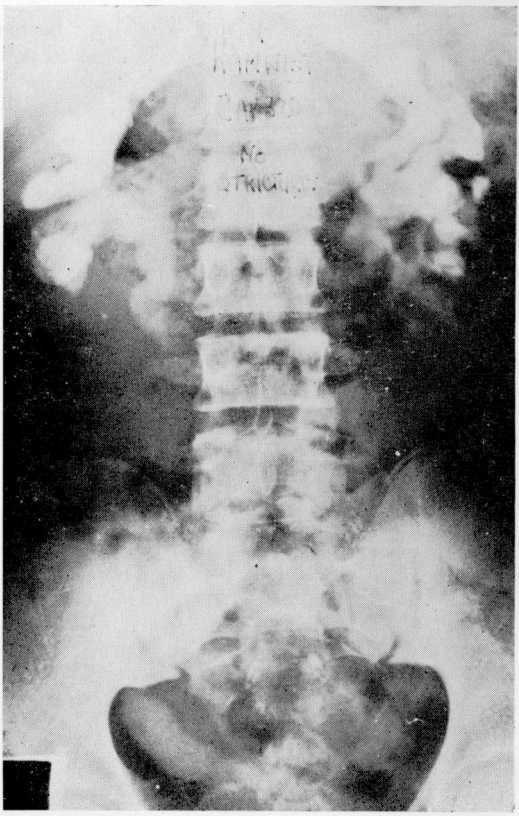

Fig. 5—African aged 22. Intravenous pyelogram showing gross hydro-ureter and hydronephrosis due to fibrosed bladder with decreased capacity (310 ml.) and high intravesical pressure. No ureteric stenosis. Treated satisfactorily by ileocystoplasty.

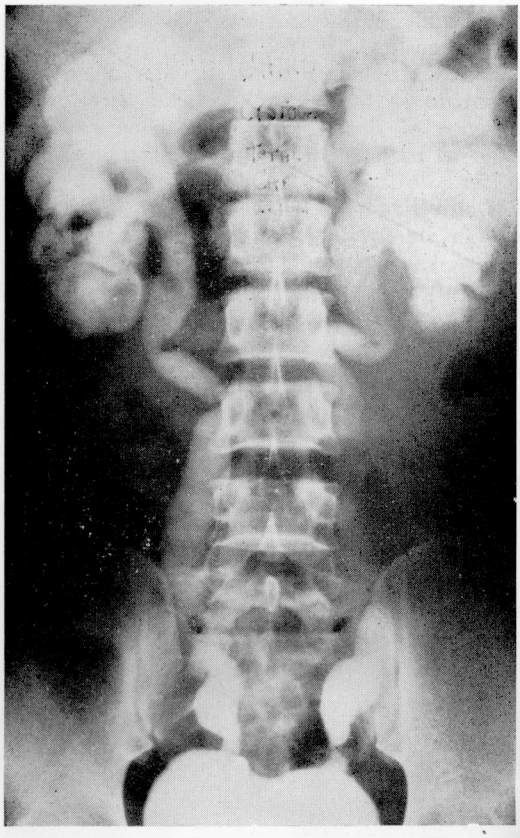

Fig. 6—African aged 23. Immediate cystogram showing reflux, gross hydro-ureter and hydronephrosis, due to fibrosed bladder with decreased capacity and high intravesical pressure. Total capacity of bladder, ureters, pelves and calices, 240 ml. Died in uraemia following bladder drainage.

variation in the size, shape and position of sandy patches. The affected mucosa too can assume different tints of yellow, orange and grey. This interesting colour variation of the patch is said to be caused by the deposition of urinary crystals in varying amounts.

A more advanced stage is the area of granulation tissue, which is of a bright red or deeply congested colour and usually present in a bladder alongside a sandy patch and is liable to bleed. It is most often single, but there may be several in the same bladder. The lesion may be sessile, measuring from 0.5 cm. to 1.5 cm. in diameter, or its base may be below the surface of the surrounding mucosa and form a granulating ulcer. The granulation tissue may also present in the form of a pedunculated tumour, which is usually solitary, but not necessarily so. It is mostly small, about 0.5 cm. in length and half

this in breadth, fleshy and solid-looking and without the shiny mucosa of the neoplastic papilloma. It may become avascular and calcified and form a "hanging" calculus. The tumour may be referred to as either a bilharzial papilloma or a bilharzioma.

A still rarer lesion is the small cyst of only a couple of millimetres in diameter, found in the bladder and ureter, and similar to the cysts seen in cystitis and ureteritis cystica of non-bilharzial origin.

Differences in the African and European Bladder

Because the disease in the African is far more advanced and of longer standing, sandy patches, bilharzial papillomata and cysts can be expected in turn, whereas in the European bladder pallor of the mucosa and tubercles only are more likely to be encountered.

When the fibrosis extends into and replaces the muscular layer (Fig. 4), an extensive rigidity of the bladder, which interferes seriously with its capacity to expand, follows. This has previously been described by Gelfand (1948) and Sayegh (1950). This decreased capacity leads to an increased intravesical pressure, with consequent increase in pressure in the ureters and pelves, unless it is possible for the individual to empty the bladder frequently, before the pressure is allowed to rise. Parous females seldom suffer obstructive uropathy from this cause, as the external sphincter fails under the strain of increased intravesical pressure and allows of stress incontinence with frequent trickling urination.

With marked fibrosis the bladder capacity is reduced to approximately 60-120 ml., and the pressure, which is greatly increased, is transmitted back to the ureters and pelves, causing great dilatation and destruction of renal tissue. This is one of the important causes of uraemia in the African and one of the most common ways in which urinary bilharziasis can cause death (Figs. 5 and 6). This gross hydronephrosis leading to death frequently occurs in the absence of stenosis—a point Sayegh (1950) did not make clear.

On the other hand, the bladder wall may become thinner and atrophic, probably because the blood supply has been greatly interfered with. It is not uncommon, therefore, to meet cases with even a greater capacity than normal. Such a bladder when full may easily be torn in relatively mild injuries delivered to the hypogastrium (Fraser Ross, 1949).

Another effect of the fibrosis in the bladder described by Makar (1955) in Egypt is contraction of the bladder neck and internal urinary meatus, causing urinary obstruction, in a similar manner to non-bilharzial bladder neck contracture. In our experience it is rare in the African and we have not met it in the European.

Although at autopsy the bladder mucosa may appear normal, digestion of the viscus in caustic potash may reveal ova (Gelfand, 1949). It was therefore pointed out that while cystoscopy may not be enough to rule out the disease in all cases, a vesical biopsy may help in those cases where the mucosa appeared to be normal. This was also suggested by Sayegh in 1950.

(To be continued. References will be given at the end of the series.)