

At the Harare Hospital Staff Round

CASES OF INTEREST RECORDED AND
ARRANGED BY

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Acute Renal Failure in Urinary Schistosomiasis

BY

Mrs. HEATHER DUKES

Death from schistosomiasis of the urinary tract is almost always due to obstructive renal disease. Our experience suggests that this is the commonest cause of death from renal disease in the medical wards of this hospital and accounts for about four deaths per 1,000 admissions.

These patients commonly present in advanced uraemia and it is often difficult to tell whether an acute or chronic process is involved owing to the frequent absence of a previous history of renal disease. They are sometimes referred to the artificial kidney unit with a view to dialysis before diagnosis is possible.

This morning I would like to describe ten patients with this condition who presented with uraemia severe enough in most cases to require dialysis, of whom four subsequently recovered appreciable renal function.

Our scheme of management resembles that of Fox and Parsons (1964), who used pre-operative dialysis in patients with advanced uraemia due to prostatic obstruction. Our patients are dialysed until they are fit for cystoscopy, retrograde pyelograms where possible, renal biopsy and nephrostomy if indicated.

CASE No. 1. ENOS CHITUNHU

This 30-year-old man was admitted on 14th December, 1968, with one week's history of abdominal pain, headache and vomiting and three days' anuria. On examination, he had slight ankle and sacral oedema, his left kidney was palpable, enlarged and tender and his blood pressure was 150/100. His blood urea estimation was 250 mgm./100 ml.

Two days after admission cystoscopy revealed a bilharzial bladder, a left ureteric orifice which was blocked just extramurally, the right orifice being unable to be found.

After four haemo- and one peritoneal dialyses to prepare him for general anaesthetic and sur-

gery, cystoscopy, bilateral nephrostomy and right renal biopsy, together with dilatation of right ureter, were performed 16 days after admission. Both kidneys were hydronephrotic, the left being almost completely destroyed. Post-operatively the right nephrostomy indicated that renal function was recovering by producing large volumes of urine and dropping his blood urea to 90 mgm. per cent. spontaneously. His left nephrostomy never produced much urine, so this kidney was subsequently removed. Three days after his nephrostomies were performed he passed urine urethrally, but this diminished again in the next few weeks.

He was treated with Ambilhar after his left nephrectomy, and intravenous pyelogram showed persisting hydronephrosis and hydroureter.

On 14th February, 1969, two months after admission, an ileal loop was utilised to join the pelvis of the right kidney to the bladder and the ureter was removed (see Fig. 1). Histologically the ureteric strictures were due to calcified bilharzial ova with surrounding fibrosis.

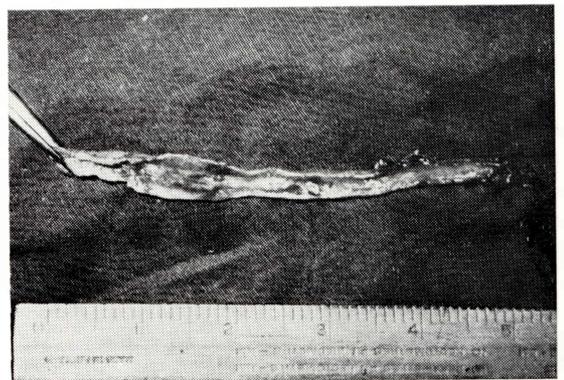
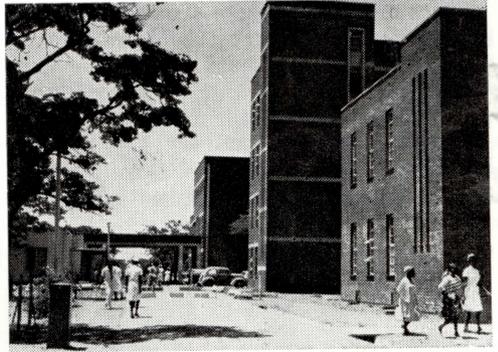


Fig. 1—Ureter with bilharzial stricture and calculus.

His subsequent progress has been very good and his remaining kidney is functioning well (see Fig. 2).

CASE NO. 2. ERIC MUDARA

This 33-year-old man was admitted on 15th January, 1969, in advanced uraemia and very oedematous with congestive heart failure, having

been sick for two or three weeks with haematuria, generalised oedema and diminishing urine output. He was confused, but there had apparently been no renal illness previously. His blood pressure was 150/100, he was very acidotic, his haemoglobin was 36 per cent. and his blood urea 480 mgm. per cent.

Nine days after admission, and after four haemodialyses, his urea remained high and it was decided that his urinary investigations should be performed under local anaesthetic. Cystoscopy proved impossible as the patient was unco-operative, but bilateral nephrostomies were performed, together with renal biopsies, which showed hydro-nephrotic changes.

The response from the left nephrostomy was immediate, but the right side required replacement of the catheter. He needed two post-operative dialyses to support his remaining renal function, but his urea now is approximately 150 mgm. per cent.

The patients are summarised in the following table:

Of the ten patients, four recovered: Enos, Eric, Jack and Charles (who recovered appreciable renal function, but died of complications after his second operation).

Our reason for presenting these cases is to show that advanced obstructive bilharzial renal disease is recoverable in a considerable proportion of cases, provided that diagnosis is made early, supportive dialytic therapy is instituted where needed and early surgical relief of obstruction is performed. The greatest single complication is infection and this must be rigorously combated.

Professor Gelfand: I would like to congratulate Mrs. Dukes on this work, as I think what she has shown today is very important. I am quite satisfied that these patients develop acute renal failure from the chronic disease that they had

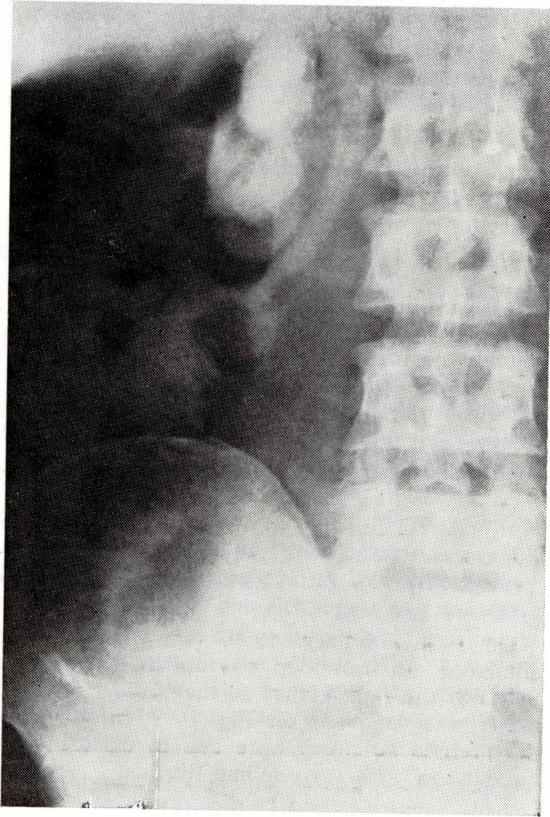


Fig. 2—I.V.P. showing ileal loop.

Table 1

Case	Age	Oliguric	No. of Dialyses	B.P.	Complications	Outcome
Esnath	28	Yes	1	240/140	Abortion	Died
Daniel	21	?	2	200/130	Pericarditis	Died
Tafanana	13	Yes	0	130/90	Terminal on admission	Died
Matthias	16	No	0	140/80	Infected nephrostomy	Died
Mairos	27	?	0	170/90	Terminal on admission	Died
Jack	40	Yes	1	120/80	Urinary calculi	Recovered
Ananias	30	No	0	160/110	Infected after instrumentation	Died
Charles	30	Yes	1	130/80	Infected post-operatively, but re-covered good renal function	Died
Enos	30	Yes	5	150/100	Infected left nephrostomy	Alive
Eric	33	Yes	6	150/100	Infected right nephrostomy	Alive

before. The lesson that she illustrates so well is that one should persevere even when the blood urea is so high and the patient is uraemic, and I have seen some recover from this degree of uraemia. One small point I would like to take Mrs. Dukes up on in the first sentence. You said patients usually died from obstructive disease, but in fact a great many die from carcinoma of the bladder and liver disease.

Mrs. Dukes: I said "in urinary schistosomiasis."

Professor Gelfand: Be that as it may, we do not know whether it is aetiological or not. I showed a case this morning with schistosomiasis in the pulmonary system and what we actually mean is that it is an important cause, but not the only cause. Probably schistosomiasis is the major cause, but I think that schistosomiasis has a far greater connotation than in the urinary system alone.

Mr. Gordon: In a communication from Dr. Pilbeam, pathologist in Blantyre in 1960, mention was made of the fact that schistosomiasis might be the basis of a good many conditions outside the urinary system, such as carcinoma of the lungs. He felt it might be the basis of a good many conditions which had as yet received scant attention. He said it was such a dreadful disease and so widespread that it deserved far greater clinical and pathological study.

Mr. Thompson: Is there any evidence of direct attack on the kidney itself by the schistosomiasis?

Dr. Tim Ashworth: Very rarely one may find schistosome eggs within the renal substance, but I feel that this is merely an incidental finding.

Dr. W. Buchanan: It is indeed rarely that eggs are found within the renal substance.

Professor Mynors: I have seen one patient with a calcified bladder, calcified ureters and a calcified kidney, and this was in the Sudan. I have severe doubts about schistosomiasis being the causal agent of the carcinoma of the bladder. In the Sudan we saw a great many cases of urinary schistosomiasis, but very few cases of carcinoma of the bladder. However, in Egypt there are many cases of carcinoma of the bladder.

Dr. David Dukes: There is doubt about the part played by schistosomiasis in carcinoma of the bladder, but there is a very good statistical relationship, as shown by Professor Gelfand, that this is in fact probably so. In most cases of carcinoma of the bladder the presence of severe schistosomiasis may be demonstrated. I think there is a relation between schistosomiasis and carcinoma of the bladder, but it may not be the

sole factor in its production and there may be many factors in the production of the carcinoma.

Mr. Gordon: Without a doubt Dr. Dukes is right. In my own view there is no single cause of any carcinoma, but the neoplasm results from the multiplicity of factors. If one considers such well-known factors as smoking and carcinoma of the lungs, the incidence varies greatly between communities of similar smoking habits indicating that there must be other factors in the production of the tumour.

Dr. J. Forbes: For how long can a kidney be completely obstructed and yet be able to resume function when the obstruction is removed? We had a lady at the end of last year rather similar to this who had a stone impacted at the lower end of the ureter and it was known to have been at the site for the best part of the year. She presented as an acute renal failure. At nephrostomy a great deal of thick jelly-like material was removed from the left renal pelvis. She was then dialysed, and once her condition improved the stone was removed from the right ureter and immediately it was obvious that the kidney commenced to function. This was despite many months of obstruction. One has had the feeling that once the stone has been there for a number of months intervention is worthless as the kidney function would be lost, but I think this disproves that contention, and I wonder if there is any time limit.

Professor Mynors: I do not think anybody does know how long the kidney can resist obstruction, but provided there are nephrons present there is no reason why function should not be restored. Take an example anywhere within the body with a similar situation: providing that pressure has not destroyed the tissues, there is no reason why function should not be restored. Even in the lungs it was thought that after four years of collapse there was no point in endeavouring to restore alveolar function, but in many cases with chronic empyaema function has been restored after extremely long periods.

Mr. Thompson: With acute obstruction there may be a pressure head which destroys the tissues.

Dr. J. Forbes: In the case under discussion, of course, the obstruction would be of a more gradual onset. Tying a ureter is a sudden obstruction.

Professor Mynors: In the sudden acute obstruction, conditions are such that the kidneys would probably be less damaged—there would be no pre-existing infection. It is said that it produces a limited hydronephrosis which may stop func-

tioning quite quickly, therefore the tissue is less likely to be permanently damaged.

Professor Gelfand: Many of these severe hydro-nephrotic pictures may improve dramatically with anti-bilharzial treatment once you have the urine flowing. Would it not be wise to give him a course of Miracil which would inhibit the disease and the granulation tissue? In many of these patients you see a reasonable improvement. We have shown this to be possible and have obtained a remarkable reduction of obstruction by giving them an anti-bilharzial drug when both the oedema and granulation tissue may respond.

Mrs. Dukes: This may be so in young people, but in the older age group there is a fibrotic process with severe stricture formation and I do not think that these would respond to anti-bilharzia therapy.

Professor Gelfand: If you read the report from West Africa you would find that it does. The cases there responded dramatically to anti-bilharzial treatment.

Professor Mynors: Were they all children?

Professor Gelfand: No, there were adults as well. I think that even what does look like fibrosis may respond to anti-bilharzial treatment. This happens in syphilis where an apparent fibrosis vanishes with arsenical compound.

Mrs. Dukes: Enos did in fact have Ambilhar which did not improve his urinary output.

Mr. Thompson: Is there any reason why the stricture is in the lower part of the ureter? Is this intra-mural?

Mrs. Dukes: You will find strictures anywhere, but the most common site is the lower end of the ureter for one to two inches just outside the bladder wall.