Aspects of renal disease in Zambia

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Chronic renal disease is a problem seen not infrequently on the medical wards of hospitals in Zambia. This paper represents an attempt to study the types seen both clinically and at post-mortem, as have occurred throughout the year 1967 on the medical wards at Lusaka Central Hospital. Both the adult and children's medical wards are included in the study but the surgical wards are excluded.

There have been several other investigations to study renal disease in various parts of Africa.

Hennessey, 1939[1], observed that nephritis was common in Uganda and that patients were often seen at a late stage of the disease.

Davies, 1949[2], reported a post-mortem study of renal disease. Nephritis was common and when a histological diagnosis could be made, glomerulonephritis common and pyelonephritis rare.

Raper, 1953[3], also from autopsy work studied on histological grounds the nephritis lesions found on autopsy over a period of five years at Mulago hospital. By far the most common cause of Bright's disease was chronic pyelonephritis. Amyloidosis was also seen generally confined to the kidneys and usually without a chronic inflammatory cause.

Uys, 1954[4], from South Africa, studied the pathology of renal disease in the Bantu on the Witwatersrand. Out of 3,707 autopsies, 21.6% had renal pathology The maximum age incidence in these cases was 21-40 and the sex incidence was equal. Of these cases 28.7% had renal tuberculosis, usually small miliary lesions, 17.7% pyelonephritis and 8.1% glomerulonephritis. However, when the cases dying from uraemia were considered, glomerulonephritis was seen in 31.2% of cases and pyelonephritis in 17.5%.

Furman, 1955[5], also from South Africa, made a comparison between nephritis in the Bantu and Europeans. The types of nephritis seen were similar although the incidence slightly different.

Gelfand, 1967[6], comments on two common forms of nephritis, one a subacute or nephrotic nephritis with a poor prognosis, and the other chronic nephritis usually with hypertensions and uraemia.

Trowell, 1960[7], comments on the rarity of typical acute glomerulonephritis, but says that most patients present as oedema and proteinuria often of a chronic nature with hypertension and uraemia. Chronic pyelonephritis is also common.

Leather[8] between 1959 and 1961 made several reports on renal disease at Mulago. In 1959 he reported three cases of amyloidosis, none had chronic inflammation or other cause for their amyloid. In 1960 a report was presented of glomerulonephritis as seen on the wards of a medical unit at Mulago. Glomerulonephritis was found to be the most common cause of renal disease in each of the first four decades. The patients were young, eldest, 41 years, there was no sex distribution and the prognosis was poor. In 1961, in a report on hypertension, that secondary to renal disease was common and in these patients glomerulonephritis more common than pyelonephritis.

Allison[9], 1962, describes a retrospective study of renal disease as found in the post-mortem room at Mulago. Chronic pyelonephritis was the most common lesion found both primary and secondary to obstruction. Both sexes were affected and there were a large number of asymptomatic cases.

Hutt and Sodd[10], 1953, also describe a post-mortem study where pyelonephritis was the most common lesion seen.

During 1967 all patients admitted to the medical wards of Lusaka Central Hospital with evidence of chronic renal disease or uraemia were observed and an attempt made to classify the different types seen. This classification has been made on clinical and pathological grounds, the pathological diagnosis results from post-mortem specimens, renal biopsies have not been performed.

A total of 91 cases have been studied. These have been divided into two main groups, those in which an accurate diagnosis could be made by post-mortem study and those in whom the study has been entirely clinical. The first group are classified according to the histological diagnosis and the second group according to the clinical features, rather than by the supposed pathological basis.

The age of the patient was rarely accurately known, so the term adult or child (under 14) has been made. Most adults appeared young.

The results of these investigations will now be described.

POST-MORTEM STUDY

Twenty-eight cases are included in this group; three children—two female and one male; and 25 adults —15 female and 10 male. The frequency of various clinical features can be seen from Table 1.

ABLE 1: CLINICAL FEATURES

	No. of cases		No. of cases
hypertension	14	congestive cardiac failure	9
anaemia	9	"coma"	7
oedema	6	vomiting	4
diabetes	2	pyelonephritis	2
bleeding	1	abortion	1
retinopathy	7		

"Coma" includes all cases admitted after fits or where there was any deterioration of the level of consciousness. Pyelonephritis includes cases admitted with the symptoms of urinary tract infection. Bleeding was usually epistaxis although bleeding from the mouth and gums was observed in some of the patients in the clinical series.

In only three of the cases had there been a previous admission to hospital with renal disease, and in a further case was there a history of "swelling of the body" many years ago. For the remaining 24 cases, this was their only admission to hospital and no past history was available.

Post-mortem findings are shown in Table II. This shows that the majority of cases had a mixed pathology, and these have been further broken down as shown in Table III, to show the incidence of each histological feature.

TABLE II: HISTOLOGY

	No. of cases
Chronic pyelonephritis	7
Chronic glomerulonephritis	8
Amyloidosis	2
Acute pyelonephritis	1
Diabetic glomerulosclerosis	1
Diabetic glomerulosclerosis with chronic pyelonephritis	1
Chronic pyelonephritis with acute focal pyelonephritis and malignant nephrosclerosis	1
Chronic pyelonephritis superimposed on chronic glomerulonephritis	2
Acute tubular necrosis	1
Polycystic kidney with malignant nephrosclerosis	1
Malignant nephrosclerosis with chronic pyelonephritis	1
Malignant nephrosclerosis	1
Acute pyelonephritis or chronic glomerulonephritis	1

TABLE III: HISTOLOGY

	No. of cases
Chronic pyelonephritis	12
Chronic glomerulonephritis	11
Acute pyelonephritis	3
Malignant nephrosclerosis	4
Amyloidosis	2
Diabetic glomerulosclerosis	2
Polycystic kidney	1
Acute tubular necrosis	1

The sex incidence for the most common lesions seen (Table II Nos. 1 and 2) was almost equal. Chronic pyelonephritis—four male and three female and chronic glomerulonephritis—five female and three male.

The clinical features are similar in both cases of chronic glomerulonephritis and chronic pyelonephritis except when there was a history suggestive of the nephrotic syndrome, when the pathology was usually one of glomerulonephritis.

The cause of death in the majority of these patients was uraemia. Two patients in whom the blood urea was normal, died, one following a cerebral haemorrhage and hypertension when histology of the kidneys showed malignant nephrosclerosis, and the other, a patient who died from peritonitis and the nephrotic syndrome in whom histology showed amyloidosis. Four patients died, with a high blood urea but not of a sufficiently high level to die from uraemia, one had the nephrotic syndrome one diabetes and hypertension, one congestive cardiac failure, and in the other, the cause of death was not definite but may have been hyperkalaemia.

CLINICAL SERIES

This group includes patients discharged from hospital and those who died but without post-mortem examinations. Sixty-three patients are included, classified as follows:—

1. The Nephrotic Syndrome.
2. The Nephrotic Syndrome with urea retention.
3. "Similar Syndrome".
4. Uraemia.
5. Miscellaneous.

1. The Nephrotic Syndrome

For inclusion in this group, the following criteria had to be fulfilled — oedema, heavy proteinuria, hypoproteinaemia and hypercholesterolaemia. No urea retention was present. Sixteen cases are included—six children, three male and three female, and ten adults, six female and four male. Seven of the cases also showed hypertension and two patients gave a past history, suggestive of renal disease.

The following methods of treatment were used:

(a) Bed rest—all cases.

(b) Diuretics: These were used in 11 cases. Seven of these had a good reponse (three of these had granular casts in the urine) and four showed either little or poorly maintained reponse (all had granular casts in the urine). In only five cases were the symptoms mild enough to withold diuretics: of these, one patient had malaria treated with chloroquine but with no effect on the proteinuria. One patient had a course of steroids with no effect, and one patient appeared to have a complete remission but there was no long-term follow-up.

(c) Steroids: These were used in four cases. One responded to steroids alone, two responded to

steroids and diuretics, and in one there was no response.

(d) In one case control of the hypertension was attempted but this had to be abandoned due to a rising blood urea.

Only two patients attended for further follow-up—one a child who remains well and the other an adult who remains asymptomatic but with heavy proteinuria.

2. Nephrotic Syndrome with Urea Retension

The criteria for inclusion in this group are as in the nephrotic syndrome but urea retention is present. Eleven cases are included, three children—all female, and eight adults—two male and six female. Six cases showed hypertension and the blood ureas varied between 50mg.% and 120mg.% at the first estimation. Only one patient had a previous admission to hospital. In all cases the urine showed heavy proteinuria and in eight cases granular casts were present.

Treatment was similar to the previous group. Only one patient responded to bed rest alone, the blood urea falling and the severity of the proteinuria.

Diuretics were used in 10 cases, five responding well (three with casts in the urine) and five showing little response (four with casts in the urine). Steroids were not used.

Only four cases attended for further review, one remaining well with only a trace of protein in the urine and three gradually deteriorating with a rising blood urea.

Two patients died, one from uraemia and one with a gradually deteriorating general condition.

3. Similar Syndrome

These patients presented with similar features to the nephrotic syndrome but the proteinuria was decreasing and only one case had hypercholesterolaemia. Hypoproteinaemia was present.

Four cases are included, all male—two children and two adults. Two patients showed hypertension and one child had scabies. One patient gave a past history suggestive of the nephrotic syndrome. These patients had urea retention and the urine in all cases contained protein, and in three—granular casts.

Treatment was as for the previous groups. Diuretics gave a variable response: one patient responded well to steroids and the scabies was treated with benzyl benzoate.

There was no long-term follow-up.

4. Uraemia

Patients who presented with the clinical features of uraemia and those in whom the uraemia was an incidental finding are included here. Eighteen cases are included; 17 adults—seven male and ten female; and one child—a female.

TABLE IV: CLINICAL FEATURES

	Cases		Cases
Hypertension	**10**	**Malaise**	**7**
Anaemia	**6**	**Vomiting**	**5**
Dysuria	**3**	**Uraemic frost**	**3**
Bleeding	**2**	**Oedema**	**2**
Bilharzia	**2**	**Pericarditis**	**1**
Headache	**1**	**"Coma"**	**1**
Malaria	**1**	**Cough**	**1**
Haematuria	**1**	**Retinopathy**	**3**

The clinical features were as shown in Table IV. Blood ureas estimated at the first visit ranged between 80 and 535mg.%. The urine in all cases showed protein, in 10 cases a few pus cells, eight cases granular casts and three cases schistosoma haematobium. Several specimens contained organisms, no signficance was attached to these as the specimens were not clean.

The following methods of treatment were used:

(a) Broad spectrum antibiotics were given to treat any acute infection precipitating acute or chronic renal failure. One case had a good response to this regime.

(b) The bilharzia was treated with ambilhar in two patients, in one there was a fall in the blood urea but in the other three was no change.

(c) One patient with epistaxis required blood transsion.

(d) The remainder were treated according to the degree of uraemia, symptom-free patients were given no treatment, whilst the others were given a low-protein diet and high fluid intake if the urine out-put was adequate. One patient had a good response to this regime.

(e) The hypertension was not treated. This was usually mild.

The prognosis was poor. Four gradually deteriorated and died, and the remainder showed no change whilst in hospital.

5. Miscellaneous

Fourteen cases are included in this group, divided into five sub-groups.

(a) **Hypertension.** These patients presented with hypertension and there was evidence of renal disease. It was not possible to say whether the renal disease was the primary lesion or whether hypertension has lead to renal failure. Eight cases are included here—all adults, seven female and one male. One had a normal blood urea initially but the remainder had levels between 100 and 200mg%. The urine contained protein and granular casts.

In three patients an attempt was made to to control the hypertension; in two the blood urea rose (one of these was the patient with the normal level initially) and in one patient there was no change. In two patients the cardiac failure only was treated. In these and the remaining patients no attempt was made to control the hypertension, either because of unwillingness to remain in hospital or a rising blood urea.

(b) **Diabetes Mellitus.** An insulin requiring male diabetic patient showed granular casts in the urine and a blood urea of 41mg%.

(c) **Children.** Two children, both female were seen: one with oedema hypertension and grossly infected urine responding well to antibiotics and diuretics. The other presented with the picture of acute nephritis, vomiting, puffy face, hypertension, smoky urine and a blood urea of 108mg. %. She responded well clinically and biochemically to bed rest.

(d) **Acute or Chronic Renal Failure.** This is given as the provisional diagnosis in two adult female patients. They both presented clinically as chronic renal failure, but in both there was a slow and substantial fall in the blood urea. In neither case was there any oliguric episode, and the only treatment given was an adequate fluid intake with a low protein diet. In one patient the blood urea fell from 250mg% to 13mg% and in the other from 302mg.% to 21mg% In both cases the blood urea took 2-3 months to fall.

(e) **Congestive Cardiac Failure.** A female child aged eight died with severe congestive cardiac failure and a blood urea of 232mg.%.

DISCUSSION

Various limitations of a survey of this kind must first be pointed out. The survey was confined to the medical wards and therefore renal disease secondary to obstruction is not included, being seen mainly on the surgical wards. The pressure on beds and early discharges from hospital limits the post mortem section, as chronic patients are often discharged to die at home, and the clinical section as there will be some asymptomatic cases perhaps missed. There may also be some cases not included because of inadequacy of records. All these limitations will, of course, make the incidence of chronic renal disease and uraemia higher than it has been described here.

The post-mortem series show chronic pyelonephritis and chronic glomerulonephritis to be the most common histological lesions seen, and to be almost equal to incidence. This would appear to show a higher incidence of glomerulonephritis than earlier recorded, but this can be explained by the fact that previous surveys were general autopsy studies whilst this was selective—only post-mortems from patients dying from renal disease in the medical wards are included. This would be confirmed by the evidence from South Africa where post-mortem lesions of pyelonephritis were more common than those of glomerulonephritis, but when cases dying from uraemia were considered, glomerulonephritis was found to be more common than pyelonephritis.

In Great Britain, Platt and Davson 1949 made a clinical and pathological study of renal disease. They found Type I glomerulonephritis, Type II, glomerulonephritis and pyelonephritis to be of almost equal incidence. From this survey, Type I glomerulonephritis would appear to be much more rare as a cause of death, but the equal incidence of pyelonephritis and Type II glomerulonephritis would seem to be similar.

More recently both in Great Britain and the U.S.A. surveys have shown that infection of the kidney is being recorded with increased frequency, and various explanations for this are suggested; either chronic nephritis is being less often identified as a cause of death, or a true change may be taking place in the natural history of the nephritides. If a change in terminology is the answer, then this must be taken into account in the comparison between present histology and that of some years ago.

Comparison of the sex incidence is difficult owing to the greater willingness of the male population to attend hospital as compared with the females. Although accurate figures are not available, the admissions for 1967 to the male medical wards were substantially higher than to the female medical ward. From this series chronic pyelonephritis and chronic glomerulonephritis have been shown to be almost equal in incidence. This would indicate that the diseases are in fact more common in females than males. When the complete figures for the survey are considered, 73 adult patients were seen—48 female and 25 male.

A total of 31 adult patients died—approximately 13% of deaths on the adult medical wards showing chronic renal disease to be an important cause of death.

The two cases of amyloidosis are similar to those described from East Africa, the amyloid deposits being confined to the kidneys, and they appeared to be cases of primary amyloidosis, there being no chronic disease elsewhere.

To continue the study of chronic pyelonephritis—a short survey was carried out on all patients admitted to the female medical ward between June and October, 1967. These patients all had a random specimen of urine examined and the presence of a significant number of pus cells was taken as evidence suggestive of a urinary tract infection. No notice was taken of the presence of organisms as the specimens were not clean. Thirty-seven cases were seen; of these 54% had symptoms of urinary tract infection, 16% were asymptomatic, 25.5% had fever alone and 5.5% had hypertension. This shows the high percentage of probable urinary tract infections, although the majority of these could not be investigated further or the response to treatment assessed owing to failure to attend for follow up. However, perhaps more pressure on patients with urinary tract infections to attend for follow up may lead to a diminution in the figure representing those dying from chronic pyelonephritis.

The clinical series have been classified on purely clinical grounds, but it is likely that those patients in groups 1, 2 and 3 would histologically have chronic glomerulonephritis, whilst patients in Group 4 have to be classified as chronic renal failure of unknown aetiology. In all these groups, hypertension is common and retinopathy not unusual.

The subacute or nephrotic nephritis as reported by Gelfand is definitely seen and is common. Typical acute glomerulonephritis would appear rare as reported by Trowell. The results would agree with those of Leather,

glomerulonephritis being a common cause of renal disease.

Bilharzia of the urinary tract, although this is undoubtedly seen commonly in Zambia, the obstructive lesion from bilharzia was not seen in the post-mortem series.

SUMMARY

A survey of renal disease as occurring on the medical wards at Lusaka Central Hospital during 1967 has been described. The results show that renal disease is common, and that chronic glomerulonephritis and chronic pyelonephritis are the most common lesions seen.

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