PRESENTATION AND CLINICAL COURSE OF END STAGE RENAL FAILURE IN GHANA - A PRELIMINARY PROSPECTIVE STUDY

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SUMMARY

Thirty-four patients with end stage renal failure seen over a 1-year period were included in this study. All patients presented with severe hypertension, moderate anaemia and raised blood urea and creatinine levels, findings which in the tropical setting usually suggested the diagnosis of end stage renal failure.

INTRODUCTION

Clinical and pathologic findings suggest that end stage renal failure is a common cause of hypertension, morbidity and death among young adults and the middle aged in this country. The incidence, prevalence and aetiologic factors remain conjectural, others[1] have found chronic glomerulonephritis common in children and suggested that 30% of these may contribute to the cause of death in end stage renal failure under the age of 15 years in their post mortem material.

Kovi[2] on his pathological study of postmortem kidneys in Ghanaians found that interstitial disease of the kidney was the most frequent lesion and that at least one third of these cases were due to urinary schistosomiasis.

The purpose of this prospective study on end stage renal failure was to ascertain the clinical criteria on which the diagnosis can be firmly based in our tropical environment.

PATIENTS AND METHODS

Physicians in the medical unit of our teaching hospital were asked to refer patients who were found to be hypertensive, anaemic and with elevated blood urea and creatinine levels. Some of them presented as acute uremic emergencies.

Investigations that were requested included full blood count and film comment, blood cultures, plain abdominal and chest X'rays, electrocardiogram, urine examination, mid stream urine culture and creatinine clearance. The kidneys could not be visualized on intravenous pyelography in any of the patients on whom the test was done.

RESULTS

There were 17 males and 17 females, the average age was 38.9 $\pm$ 12.5 (range 21 to 72).

The blood pressure profile, hemoglobin, blood urea and creatinine levels as well as findings at

autopsy are shown in Table III and IV. The average duration of hospital stay from day of admission to day of death was 11 days (range 2 - 26 days) for both males and females.

One female patient was put on chronic intermittent haemodialysis. This patient was admitted moribund but improved gradually whilst on regular bi-weekly 6-hour dialysis. She died 8 months later from haemorrhage when her fistula developed into an aneurysm and ruptured.

One male patient was put on a programme of intermittent peritoneal dialysis. Initially he had a

TABLE I

HYPERTENSION IN CHRONIC RENAL FAILURE

References	Patients with Hypertension
(a) Thomson, Waterhouse, McDonald & Friedmann 1967[13]	21 of 21
(b) Shupak, Sullivan & Lee 1967[14]	26 of 26
(c) Curtis, Eastwood, Smith, Storey Verrbust, de Wardmer, Wing 1969[15]	25 of 25
(d) Wilkinson, Scott, Uldall, Kerr 1970[16]	31 of 45
(e) This Study	34 of 34

TABLE II

RESULTS AND FEATURES OF PATIENTS DIAGNOSED AS END STAGE RENAL DISEASE

	Males	Females
Number of Cases	17	17
Lost to follow-up	3	1
Death in hospital	9	11
Death at home	2	2
Follow-up at Clinic	2	2
Discharged on request	0	1
Number who had post mortem	9	11

TABLE III CLINICAL AND LABORATORY DATA OF MALE CASES

NAME	AGE	DIAGNOSIS	BP	Hb gm/dl	Urea mmol/L	Creatinine mg/dl	Outcome
RB	38	ESRD and Severe Hypertension	240/180	8.6	29.0	10.0	PM LVH, contracted kidneys, thin cortex and adherent capsule 80gm each.
HKA	42	CRF and Hypertension	180/110	7.6	20.0	4.5	On follow-up.
FA	42	ESRD and Hypertension	250/150	6.9	38.0	?	PM massive LVH, granular contracted kidneys.
AH	26	CRF and Hypertension	220/160	6.8	22.0	124.0	Lost to follow-up.
SW	35	CRF and Hypertension	210/140	8.0	28.6	14.2	Lost to follow-up.
DA	32	ESRD and Hypertension	150/100	11.4	16.9	6.4	Died at home.
NK	21	ESRD and Hypertension	230/160	5.2	30.1	17.1	PM scarred contracted kidneys.
RS	25	ESRD and Hypertension	200/140	8.0	31.5	15.0	Lost to follow-up.
OA	52	ESRD and Hypertension	170/110	7.9	30.6	14.2	PM LVH scarred kidneys 60gm each.
EAM	52	ESRD	165/120	5.2	35.2	14.0	PM contracted kidneys 80gm each.
FO	35	CRF and Severe Hypertension	220/130	4.2	30.0	11.0	PM end stage kidneys 70gm each
KK	40	CRF and Hypertension	230/125	5.6	42.9	15.0	Lost to follow-up.
SF	65	CRF and Hypertension	210/110	10.8	21.9	7.3	On follow-up
SJ	48	Hypertension, CRF Chronic glomerulonephritis	220/150	9.2	20.6	?	On a follow-up clinic
WP	37	LVF, CRF, Hypertension	260/160	7.4	20.7	18.6	Died, relatives refused PM.
AB	35	Nephrotic syndrome, ESRD	240/130	10.0	28.0	10.0	PM, LVH, scarred kidneys, 40gm each
YA	42	ESRD and Hypertension	200/100	6.7	38.0	15.0	PM, LVH,, pericarditis, contracted kidneys. 50gm each with adherent sub capsular scars.

ESRD - End Stage Renal Disease
CRF - Chronic Renal Failure
PM - Post Mortem
LVH - Left Ventricular Hypertrophy
BP - Blood Pressure
LVF - Left Ventricular Failure

Tenchkoff catheter inserted into the peritoneum but this failed and he died from overwhelming abdominal sepsis.

DISCUSSION

The disease affected young and middle aged adults who invariably presented with severe hypertension and severe anaemia. Most of the patients presented as preterminal or terminal uraemic emergencies and had probably had asymptomatic renal disease for months or years which had suddenly become worse (acute on chronic) or given rise to uraemic symptons because of intercurrent renal insult[3]. It is difficult to estimate from the literature the prevalence of hypertension in chronic renal failure. Table I illustrates some of the quoted series. Hypertension was a universal finding in all our patients. We were unable to establish whether the hypertention was related to salt and water retention or related to high levels of renin activity or increased aldosterone levels. Hypertension is often assumed to be an important cause of renal failure, but it is clear from many reports that there is considerable variation in the development of kidney disease in patients with high blood pressure. Perera[4] studied the natural history of hypertension, finding that the expected life of a hypertensive was almost 20 years from the onset of elevated blood pressure, and that only in the last five years was there clinical evidence of organ impairment including renal disease. In patients presenting with terminal renal failure Moorhead et al[5] found that about 80% were hypertensive and a quarter of these had malignant hypertension. In our own uncompleted histological study out of 20 patients dying from chronic renal failure the causes were hypertensive renal damage (10 patients, two with vesical schistosomiasis), polyarteritis (3 patients), and one each of focal segmental glomerulonephritis, AA amyloid, mensangio-capillary glomerulonephritis, polycystic disease of the kidneys, chronic pyelonephritis, diabetic nephropathy and unexplained renal failure. At this stage of our study it seems that in our setting hypertensive renal damage is probably the commonest cause of end stage renal disease. In a histological study Kincaid-Smith et al[6] found 40% of 124 patients with malignant hypertension had arteriolar nephrosclerosis, 21% had pyelonephritis, and 15% had glomerulonephritis. The last figure is surprising in view of general claims that glomerulonephritis is the commonest cause of terminal renal failure. In this respect the Australian patients of Kincaid-Smith resemble our unpublished Ghanaian patients.

It has been said that in temperate climates the presence of anaemia and cutaneous pigmentation point towards chronic renal disease[7] and that in the tropics anaemia has too many causes and uraemic pigmentation is undetectable. All the patients studied were hypertensive of moderate degree, were anaemic and had raised serum creatinine and urea levels. Postmortem consistently demonstrated small scarred end stage kidneys. It is our impression that nail and mucous membrane pallor and puffy face have more significance in Africans than pigmentation.

The causes of renal failure in our patients will be fully determined when our work is completed. One male patient was known to have nephrotic syndrome of long standing before he was later found to have preterminal renal failure which led to his demise. It is possible that some of these cases may represent tropical (quartan malarial) nephropathy[8,9] which is characterised by steriod resistance and may progress to chronic renal failure[7]. Kovi[2] on his pathological study of postmortem kidneys found that 8 cases out of 31 of those

with interstitial renal disease also had left ventricular hypertrophy and elevated blood pressure recorded in their notes.

Glomerulonephritis accounts for the largest number of patients who present with renal failure in Europe. The Registration Committee of the European Dialysis and Transplantation Association reported in 1973[11] that of a total of 18,750 patients reported to the Registry with renal failure, 56% had glomerulonephrisis, 25% pyelonephritis, 3% polycystic disease and the remainder a combination of renal vascular disease, congenital abnormalities and other disorders.

Also in North America the causes of chronic renal failure according at Wineman et al[12] are glomerulomephritis 41.6%, cardiovascular disease and hypertension 13.5%, other urinary tract disease 10.5%, unknown 8.4%, congenital abnormalities 7.6%, diabetes 7.2% and kidney infection 6.1%.

The contribution of glomerulonephritis to our renal failure was surprisingly small and may reflect the pattern of hospitalization. It seems, therefore, that the contribution of glomerulonephritis to end stage renal failure has regional variation.

REFERENCES

1. Awunor-Renner and Smith : The kidney in Parry E. H. O. Editor. Principles of Medicine in Africa, 1984 893-929.

2. Kovi J. : The Pathology of Renal Disease in Ghana : Ghana Med. J. 1964; 3: 36 - 38

3. Oliver Wrong : Management of the Acute Uremic Emergency. Brit. Med. Bull. 1971, 27: 97 - 102

4. Perera G.A. : Hypertensive vascular disease : description and natural history. Journal of Chronic Disease 1955 1 : 32-42.

5. Moorhead J.F., Baillod R.A., Hopewell J.A. : Home dialysis. Proceedings of the 4th International Congress of Nephrology, 1969 3: 131 - 140.

6. Kincaid-Smith P., McMichael J., Murphy E.A. : The Clinical cause and pathology of hypertension with papilloedema (malignant hypertension). Quarterly J. of Med 1958 27, 117-153.

7. Hutt M.S.R., Wing A.J. : Renal Failure in the Tropics. Brit. Med. Bull. 1971, 27: 122 - 127.

8. Edington G.M. & Mainwaring A.R. : Nephropathies in Africa. In the Kidney ed. Mootofi F.K.R., Smith D.E., p. 488. Baltimore : the Williams Wilts. 1966.

9. Kibukamusoke J.W., Hutt M.S.R., Wilks N.E. : The nephrotic syndrome in Uganda and its association with quartan malaria. Quart. J. Med. 1967 36 : 393 - 408.

10. Kibukamusoke J.W., : Nephrotic Syndrome and Chronic Renal Disease in the Tropics. Brit. Med. J. 1968 2, 33.

11. Garland H.J., Brunner F.Y.V., Del in H., Harlen H., Parsons F. M., Scharer K. (1973) : Combined Report on Regular Dialysis and Transplantation in Europe III. Proceedings of the European Dialysis and Transplant Association, 10. pp XVII - LVII. London & Pitmans medicals.

12. Wineman R. J. : End Stage Renal Disease : Dialysis Transplantation 1978 7 : 1034.

13. Thompson G. E. Waterhouse K., McDonald H.P., Friedman E.A. : Hemodialysis for chronic renal failure. Clinical observations. Arch. Intern. Med. 1967. 120; 153 - 167.

14. Shupak E., Sullivan J.F., Lee D.U. : Chronic Hemodialysis in "Unselected Patients". Ann of Intern. Med. 1967 67 : 708-717.

15. Curtis J.R., Eastwood J.B., Smith E.K.W., Storey J.M., Verroust P.J., Wardener H.E., de Wing A.J., Wolfson E.M. Maintenance Hemodialysis Quart. J. Med. 1969 38 : 49-89.

16. Wilkinson R., Scott D. F., Uldall P. R., Herr D. N. S., Swinney J. : Plasma Renin and exchangeable sodium in the hypertension of chronic renal failure. Quart. J. Med. 1970. 39 : 377-394.