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The Official Journal of the Nigerian Association of Nephrology

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Income Distribution and Sources of Funding for Maintenance Haemodialysis of Patients in the University of Port Harcourt Teaching Hospital

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ABSTRACT

End stage renal disease (ESRD) is prevalent in Nigeria, with attendant high morbidity and mortality rates. In Nigeria, there have been reports of low affordability of haemodialysis and dialysis inadequacy. There is however, no formal study of the sources of funding for dialysis in the country. Such studies when replicated across the country will provide an evidence based tool with which to engage Government on the need for a Government driven ESRD program.A prospective direct questionnaire based study of End stage renal disease patients receiving maintenance haemodialysis was conducted at the University of Port Harcourt teaching hospital. Twenty four (24) males and 16 females(M/F=1.4:1) were studied, with mean age of 40.62 $\pm$ 14.9 years, mean e-GFR , 6.53 $\pm$ 1.6 mls/min. and mean duration on dialysis of 5.03 $\pm$ 1.6(3-12) months. The mean annual income of the patients was N1, 147, 172.02 (N60, 000.00 to N3,200,000.00).The estimated annual cost of haemodialysis in Port Harcourt per patient is N2,340,000.00. Sixty (60) percent of the patients earned below one million naira per annum. Only 10 percent of the patients earned over 3 million naira p.a .The annual incomes of 62.5% of the patients were less than fifty percent the annual cost of dialysis. Annual incomes showed positive correlation with the duration on dialysis(r= + 0.14) and number of dialysis sessions received (r= +0.3). Dialysis was funded from family income in 65 percent of the cases. Funding was from extended family members in 17.5% and philanthropic sources in 10% of cases. There was no Government support to any patient or funding through insurance. The annual incomes of the great majority of ESRD patients are less than 50 percent of the annual cost of maintenance haemodialysis and cannot sustain optimal long term haemodialysis. A Government driven ESRD Care program is therefore inevitable in the country if we are to improve access to haemodialysis.

Keywords: *Income distribution, source of funding, maintenance haemodialysis, University of Port Harcourt teaching hospital*

INTRODUCTION

The burden of suffering for the end stage renal disease (ESRD) patient is enormous. It includes the clinical burden of life-long uraemia and its related complications, lowered quality of life, job losses and particularly the huge financial burden of cost of medical care [1,2]. The financial burden for caring for these patients in the United states runs into billions of dollars each year[3,4].

It was in realization of this huge financial burden on the patient and their families and the fact that only very few Americans could foot the bill, that the US Legislature passed the Medicare End Stage Renal Disease (ESRD) programme into law in 1973.

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Ever since, the US government had virtually taken over the financial burden of care from the patients and their families.

In virtually all other developed countries such as Canada, the United Kingdom, and other European countries, similar schemes [6, 7] exist with local variations for the care of ESRD patients.

Nigeria, the sixth major petroleum producing country in the world, with a population of about 140 million people and chronic kidney failure medical admissions rate of 1.6-8 percent [8, 9] has no organized renal care service.

Though, it is common knowledge that the majority of ESRD patients in Nigeria cannot support long term dialysis, there is no factual documentation of this observation. Two previous attempts [10,11] in highlighting this problem did not give details of patients actual incomes relative to average cost of dialysis. Similarly, there has been no previous systematic study of the sources of funds for dialysis by ESRD patients. The availability of such evidence based information would serve as a useful bargaining tool to convince health policy decision makers of the need for Government intervention in ESRD care in Nigeria.

This study is therefore undertaken to determine the income distribution and the sources of funding for maintenance haemodialysis among ESRD patients in the University of Port Harcourt Teaching Hospital.

METHODS

The data for analysis was obtained with aid of a purpose designed semi-structured questionnaire applied to patients undergoing maintenance haemodialysis at the University of Port Harcourt Teaching Hospital during the period of study.

The questionnaire was applied to the patients by direct interview. Medical registrars and house officers on clinical attachments in the haemodialysis unit served as the interviewers, after training by the investigators.

All consecutive ESRD patients undergoing maintenance haemodialysis in our service, who satisfied the inclusion criteria during the period January to June 2009, were included in the study.

Inclusion criteria:

1. Patient must be a confirmed case of end stage renal disease (ESRD) in accordance with the NKF/KDOQI [12] guidelines for diagnosis and staging of CKD.
2. Patient must have been undergoing maintenance dialysis in our centre for an uninterrupted minimum of three months prior to recruitment.

Information obtained with the questionnaire include: the bio-data and basic clinical data of the patient which were obtained from the patients clinical records.

Relevant clinical data obtained include the primary renal diagnosis, the estimated glomerular filtration rate (e-GFR) at first presentation, the duration of the patient on dialysis in months, and the number of dialysis sessions per week attained by the patient.

The data pertaining to the income status and sources of funding for dialysis include the following: educational status, occupation, nature of work, average monthly income, average annual income, family size. Others include sources of funds for payment for dialysis treatment. The options include: personal, family sources, extended family sources, employers, insurance payments, philanthropic organizations, philanthropic individuals and any other sources.

Patients were grouped into six socio-economic classes in accordance with the Registrar General classification of occupations [13] as follows- Class I (top government and corporate executives), Class II (upper middle level executives, professional class), Class III (middle level executives), Class IV(clerical cadre, skilled technicians.), Class IV(semi-skilled artisans), Class VI (unskilled).

Where the patient is a minor or a dependant (e.g child, student or house wife) the income of the bread winner of the family is taken as the income of the patient. For patients who are not on fixed paid incomes, they were requested to give an estimate of how much income they earned in a day, week or a month which was extrapolated to derive the annual income.

In other to determine the relativity between the income of the patients and the annual cost of dialyzing hypothetical ideal patient in the Port Harcourt environs, the annual incomes of the patients was calculated as a percentage of the annual cost of dialysis for an ideal patient dialyzing three times a week in the Port Harcourt and South- south states of Nigeria.

The cost of a one 4hr-session of haemodiaysis in this region, range from ₦15, 000.00 to ₦25, 000.00 (in public and private dialysis facilities). Using the

lowest amount of ₦15,000 .0 per session as a benchmark, the annual cost of haemodialysis for one year comes to N2,340,000.00 a year for an ideal patient dialyzing three times a week.

Data Management

The data were analyzed with Epi- info versions 6.0 statistical package. Quantitative variables are presented as mean ± standard deviation. Student t-test was used to compare quantitative variables with significant levels (p) set at 0.05. Pearson correlation coefficient was used to determine the relationship between dependent variables. Tables are used as appropriate.

RESULTS

A total of forty patients were studied, 24 males and 16 females with a sex ratio of 1.4:1 were studied. Their ages ranged from 14 to 69 years with a mean age of 40.62 ± 14.9 years. Twenty six (65%) were married, 10(25%) were single, 2(5%) were a widow and a widower while 2(5%) were teenagers.

Twenty one (52.5%) had tertiary education, 12(30%) had secondary education only, 6(15. %) had primary education only and 1(2.5%) had no formal education. The family size ranged from 1 to 12, with a mean of 6.1 ± 2.9. The 6-8 family- size groups was more predominant, responsible for 45% of the patients.

The distribution of the primary cause of CKD as obtained from the clinical case records were, chronic glomerulopathy(45%),hypertensive nephropathy(25%),diabetic nephropathy(12.5%), autosomal dominant polycystic kidney disease(5%), nephrotic syndrome(5%),obstructive uropathy(5%) and suspected substance abuse induced nephropathy (2.5%). Their e-GFR ranged from 3.6-10.6 mls/min. with a mean of 6.53 + 1.6 ml/min.

The patients have been on maintenance dialysis for a period ranging from 3 t0 12 months with a mean of 5.03 ± 1.6 months. The mean number of dialysis sessions per week attained was 1.4 + 0.6 [1-3] sessions per week.

Socio Economic and Income Distribution

By socio-economic grouping, they were, Class I - 3(7.5%), Class II - 8(20%), Class –III 8(20.0%), Class IV -9(22.5%), Class V- 8 (20%) and Class VI-4(10%) respectively.

The annual incomes of the patients ranged from 60,000.0 to 4,200,000 naira (428.6-30,000 US-dollars) with a mean of 1,280,292.0 ± 1,147,122.02.(9,144.9us-dollars).

Table 1 shows the annual income distribution of the patients according to socio-economic status. Patients belonging to socio-economic class I earned an average of 4 million naira p.a. while those in the socioeconomic class VI earned an average of 0.37million naira p.a. Table 2 shows the spatial distribution of the annual incomes. Families with annual incomes less than 0.5million naira(27.5%) and 0.5 to 0.99 million naira (32.5%) i.e those earning less than one million naira p.a. constitute 60 percent of the patients. Those earning 1 to 1.49 million

Table 1: Distribution of annual incomes according to socio-economic class

Socio-economic class	Mean annual income	Range. (Million naira)
Class I	4million	3.6-4.2
Class II	2.1m	1.2-3.0m
CLassIII	1.1m	0.6-2.4
Class IV	0.74.m	0.36-1.2m
Class V	0.34m	0.12-0.96m
Class VI	0.37m	0.06-1.0

Table 2: Spatial distribution of family income

Annual income. (million naira)	Number	Percentage
Less than 0.5	11	27.5
0.5-0.99	13	32.5
1.0-1.49	6	15.0
1.5-1.99	2	5.0
2.0-2.49	2	5.0
2.5-2.99	2	5.0
3.0m and above	4	10.0
Total	**40**	**100.0**

constituted 15% of the patients. The three groups above together constitute 75percent of the patients. Thus seventy-five percent of the patients earned less than 1.5 million naira p.a. Only 10 percent of the patients earned 3 million naira p.a. and above.

Of the four patients that earned 3 million naira and above, three worked with major petroleum companies while the remaining one was a permanent secretary in a state government service. Annual incomes showed positive correlation with the duration of the patients on dialysis($r = +0.14$) as well as with the number of dialysis sessions attained per week($r = +0.3$).

Table 3 shows the annual incomes of the patients as a percentage of the annual cost of dialysis in the Port Harcourt area. In 52.5% of the patients their annual incomes are less than 50% the annual cost of dialysis for an ideal patient. Only 15 percent of the patients earned annual incomes either equal or higher than the annual cost of dialysis in the area.

Table 3: Family income as a percentage of *Annual cost of dialysis.for an ideal patient. (3 dialysis sessions per week)

Family income as a percentageof Annual cost of dialysis for ideal patient	Number	Percentage
≤ 25%	12	30.0
26-50	13	32.5
51-75	7	17.5
76-100	2	5.0
>100	6	15.0
	40	**100**

**Calculated Annual cost of dialysis for an ideal patient, based on bench mark of N15,000.0 per dialysis session at three dialysis sessions per week = ₦2,340,000.0.*

Sources of Funding

The sources of funding for dialysis are set out in table 4. Twenty-six patients (65%) paid for their dialysis treatment from personal / immediate family income. Support from extended family sources was obtained in 17.5% of the patients. Thus 82.5 percent of the patient funded their treatment from immediate and extended family sources.

Table 4: Sources of funding for Haemodialysis treatment

Source of funding.	Number	Percentage
Direct family income	26	65.0
Extended family source	7	17.5
Employer support	3	7.5
Philanthropic organization	3	7.5
Philanthropic individual	1	2.5
Insurance payments	Nil	0.0
Government support	Nil	0.0
TOTAL	40	100.0

Four patients (10%) received support from philanthropic sources. One was from a philanthropic individual and three from religious organizations. None of the patients obtained financial support from either insurance payments or from Government.

DISCUSSION

This study provides an objective data that clearly shows the wide gap between the incomes of maintenance dialysis patients and the actual amount required to obtain optimal dialysis. Two previous works [10, 11] in this direction did not study details of patients' income profiles.

Although there was some spread in the distribution of the patients by socio-economic stratification, there is a wide disparity in income distribution (table 1). Sixty percent of the patients do not earn up to 1 million naira p.a. The 10 percent of the patients who earned 3 million naira and above represent the privileged few in the Nigerian society working for big oil conglomerates or top Government officials.

The annual income of 52.5% of the patients was less than fifty percent of the annual cost of dialysis in the region. Only 15 percent of the patients have annual incomes equivalent to or exceeding the annual cost of dialysis.

For those in the low income groups achieving optimal dialysis is simply not feasible. For those in the high income groups, achieving optimal dialysis implies that they have to deploy all their annual

earnings to pay for dialysis at the detriment of medications, erythropoietin, other competing family financial needs, such as feeding, children education, etc. The significance of these findings is that, at the prevailing income levels, virtually none of the patients, including those in the high socio-economic brackets can afford to fund optimal dialysis for long term survival and reasonable quality of life.

This inability to fund dialysis is the dominant factor responsible for the grossly sub-optimal dialysis, as reflected by the mean weekly dialysis frequency of 1.4/week attained by the patients. Dialysis inadequacy is a strong risk factor of poor dialysis outcomes, morbidity and early mortality [14,15].

This situation would most likely explain the earlier observations from dialysis centers across Nigeria of very poor dialysis outcomes. These include high drop out rates, high morbidity rates, poor quality of life and unacceptably high mortality rates of over 80 percent within three months of commencement of maintenance dialysis. [16, 17]

With the exception of very minimal support from philanthropic organization, (mainly religious organizations and one philanthropic individual), patients sourced their funds for dialysis almost entirely from family sources which is grossly inadequate an unsustainable as shown earlier. Significantly no form of government support was available to any of the patients. Similarly none of the patients was covered by medical insurance and this may be because presently the Nigerian National health insurance scheme (NHIS) does not cover for dialysis treatment.

This state of affairs as highlighted above led to the advocacy of early kidney transplantation of ESRD patients in Nigeria, leading to the emergence of a few kidney transplant centers in Nigeria, in the last ten years[18-20]. While this option is reasonable, the presence of an effective, accessible and affordable dialysis service remains critical for ESRD care in any country. ESRD patients requiring transplant will need optimization of dialysis before the transplant. In the event of graft failure, the patients will recourse back to dialysis, while awaiting another transplant. Patients for whom transplant is not feasible will depend on dialysis for survival. Therefore, the imperative for an accessible, affordable and sustainable dialysis system in a country is not in doubt.

Poor dialysis outcomes on account of lack of access was the scenario in the United States of America before 1973 that led to subsequent legislation that enabled the establishment of the Medicare-ESRD program[3,4].

Nigeria as a democratic nation subscribes to the United Nations charter on fundamental human rights. The right to life is fundamental. It provides that no person should be allowed to die of an illness for which treatment is available, on account of inability to pay for such treatment. It is the responsibility of Government to ensure that ESRD patients are given the chance to live in Nigeria as in the developed countries of the world.

A Government driven ESRD program in Nigeria is feasible. Such a program can be articulated and funded through a compulsory contributory Health tax regime, involving Government, (e.g. 0.05% of petroleum and gas revenues can be dedicated for this), the major oil companies and conglomerates, all public servants, and all gainfully employed ratable adults. Of course patients and their families will also make some contributions through capitation payments, once the diagnosis is confirmed and patient registered into the program. In this way the financial burden of care is shared by all.

Such an arrangement will within a short period yield huge amounts of funds as is the experience with the National Health Insurance scheme (NHIS), National pension fund (NPF) and the Education Trust fund (ETF) schemes in Nigeria. It may even be possible to accommodate some other chronic health disorders such as cancer care in the program.

What is important is to put in place mechanisms to ensure long term sustainability by ensuring efficient and effective administration, and regular audit of the process to prevent abuses. In other to actualize this will certainly require the appropriate legislation. The Nigerian association of Nephrology (NAN) and the Federal Ministry of health, should lead the campaign to articulate a professional-private bill to the National assembly for a legislation for the establishment of Renal care program for the country. This will however require a great deal of preparatory work to be equipped with factual data on all aspects of CKD and ESRD in the country.

CONCLUSION

The study has provided evidence based data on the prevailing economic deprivation state among the majority of ESRD patients in Nigeria, and confirmed the fact that ESRD patients in Nigeria (including the high income group) cannot afford to pay for renal replacement therapy.

The findings demonstrates the strong need for a Government driven ESRD care program for the country, as the only way to achieve standard best practices in ESRD care in Nigeria, thereby reducing the prevailing and unacceptably high mortality rates associated with ESRD in Nigeria.

REFERENCES

1. Leaf DE and Goldfarb DS. Interpretation and review of health related quality of life data in chronic kidney disease patients receiving treatment for anemia. Kidney Int 2009; 75:15 -24.

2. Crosby RD, Kolotkin RL and Williams GR. Defining clinical meaningful change in health related quality of life. J Clin Epidemiol 2003; 56:395-407.

3. Xue JL, Ma JZ, Louis TA and Collins AJ. Forecast of the number of patients with end stage renal disease in the United States to the year 2010. J Am Soc Nephrol 2001; 2: 2753-2758.

4. Warwick G. Dialysis Strategies. *In*: Barratt J, Harris K and Topham P. (eds.) Dialysis and Renal Transplantation.(Selected materials from the Oxford Desk Reference Nephrology) 1st ed. Oxford. New York. Oxford University Press 2009: 1-9.

5. Rettig RA. Origins of the medicare disease entitlement. Social security Amendment of 1972. Human Kidney Biomedical politics Washington DC: National Academy Press, 1991: 176-208.

6. Zelmar JL. Economic burden of end stage renal disease (ESRD) in Canada.Kidney Int 2007; 72: 1122-1129.

7. Brunner FP, Brynger H and Chantler C, *et-al*. Combined report on regular dialysis and Transplantation in Europe. Proc Eur Dial Transplant Ass 1979; 6: 4-73.

8. Oyediran ABO and Akinkugbe OO. Chronic renal failure in Nigeria. Trop Geog Med 1970; 22: 41-44.

9. Akinsola W, Odesanmi WO, Ogunbiyi JO and Ladipo GO. Diseases causing chronic renal failure in Nigeria-a prospective study of 100 cases. Afr J med sci 1989; 18(2): 131-137.

10. Unuigbe EI. Funding Renal care in Nigeria critical appraisal. Trop J Nephrol 2006; 1(1): 33-38.

11. Ummate I and Nwankwo EA. Cost analysis of dialysis patients in Maiduguri.(Abstract) Trop J Nephrol 2006; 1(1): 43-44..

12. National Kidney Foundation. K/DOQI Clinical practice Guidelines for chronic kidney disease evaluation, classification and stratification. Am J Kidney Dis 2002; 39(suppl 1): S1-S266.

13. Registrar General 1970 classification office of population census and surveys. Classification of occupations1970. London HMSO, 1970.

14. Hakim RM, Boyer J, Ismail N and Schulman G. Effects of dose of dialysis on morbidity and mortality. Am J Kid Dis 1994; 23: 666-669.

15. Held PJ, Port FK, Wolfe RA, Stannard DC, Carrol CE, Daugrides JT, Grier JW and Hakim RM. The dose of haemodialysis and patient mortality. Kidney Int 1996; 50: 550-556.

16. Arije A. Problems of Haemodialysis in the management of chronic renal failure in Ibadan. Arch Ibadan med 2001; 2(1): 14-15

17. Arogundade FA, Sansui AA and Akinsola A. Epidemiology, Clinical characteristics and outcomes in ESRD Patients in Nigeria; is there a change in trend? Trop J Nephrol 2006; 1(1): 41-42.

18. Bamgboye EL. Haemodialysis: Management problems in developing countries ,with Nigeria as a surrogate. Kidney Int 2003; 63(suppl. 83) :s93-s95.

19. Akinkugbe OO. Renal transplantation in Nigeria the time is now. (Guest editorial). Arch Ibadan med 2001; 2(1)14.

20. Bappa A, Abdu A , Sani MU, ,Alhassan SA and Borodo MM. Three years follow –up of the first renal transplant in Aminu Kano Teaching Hospital: A case report.Trop J Nephrol 2006; 1(1): 29-32.

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Predictors of Kidney Damage in Newly-Diagnosed Hypertensive Nigerians

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ABSTRACT

Systemic hypertension is an important cause of chronic kidney disease. Identifying predictors of hypertensive kidney damage provides clinicians with opportunity for appropriate therapeutic interventions. This will forestall kidney damage and prevent development of chronic kidney disease. The study aims at determining predictors of kidney damage using microscopic haematuria as surrogate marker in newly diagnosed hypertensive patients. A cross sectional study of 138 newly diagnosed hypertensive Nigerian, matched with age and sex controls was conducted. The surrogate marker of kidney damage was microscopic haematuria defined as ≥ 3/hpf, determined by examination of urine sediment under a bright field microscope after application of Sternheimer's stain. Potential predictors of kidney damage evaluated were: age, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP), pulse pressure (PP), and body mass index (BMI). Mean age of the patients was 43.2±9.6years and 76 (55%) were males. SBP correlates positively with kidney damage (r=0.209, p=0.048). Stepwise regression models identified SBP as the best sole predictor of kidney damage (r=0.557, r^2=0.310, adjusted r^2=0.272, df=1, P=0.011), followed by age (r=0.72, r^2=0.505, adjusted r^2=0.447, df=2, P=0.019). Other variables were rejected by the model (probability level of entrance was ≤ 0.05 and removal, 0 ≥ 0.1). The study suggests that systolic blood pressure may predict kidney damage in newly diagnosed hypertensive patients, and it seems to be amplified with increasing age. A longitudinal study with larger population is recommended to confirm this relationship.

Keywords: *Predictors, kidney damage, microscopic haematuria, hypertension, Nigerians*

Conflict of interest: None

INTRODUCTION

Systemic hypertension is a major public health problem and is predicted to remain same in the next decade [1]. It is a well recognized renal and cardiovascular risk, and a leading cause of chronic kidney disease (CKD) [1]. It is also a potent predictor of progression of CKD which has recently attracted attention of the global nephrology community because of its growing incidence and prevalence [2]. The associated morbidities and mortalities and enormous healthcare costs of managing CKD, particularly of the end-stage kidney disease (ESKD) are major concerns. It is for these reasons that preventive measures are widely and vigorously advocated as the most effective strategy to reduce CKD burden. Clinical and epidemiological data have shown that blacks have enhanced susceptibility to developing hypertensive kidney damage. A contributory factor is their propensity for developing intrinsic renal vascular injury facilitated by unique clinical characteristics some of which are largely modifiable or amenable to therapeutic interventions [3].

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Therefore, identifying factors that determine or promote kidney injury early in hypertensive patients would provide opportunity for targeted therapy to prevent initial and further kidney damage thereby reducing the incidence, prevalence and overall impact of CKD/ESKD. One of the veritable tools for early detection of kidney diseases is urine analysis which incorporates microscopy with dipstick tests [4].

The guidelines for the management of hypertension produced by both World Health Organization/ International Society of Hypertension (WHO/ISH) in 1999 and International Forum for Hypertension control and prevention in Africa (IFHA) recommended examination of urinary sediment as a complement to dipstick test in the evaluation of hypertensive patients[5,6]. While dipstick tests are commonly and routinely utilized in hypertensive patients, their urine is rarely examined for .sediments. Indeed literature is extremely sparse on the subject of urinary sediment and hypertension. Microscopic examination of urine is a simple, cheap and cost-effective method of detecting sub-clinical kidney damage. These make it relevant for economically disadvantaged populations who cannot afford the cost of novel markers of kidney damage for majority of their patients. Abnormal urinary sediment manifesting as microscopic haematuria with characteristic dysmorphic features is an evidence of glomerular bleeding in response to varieties of insults[7]. These insults may be initiated by an immunologically mediated disease such as glomerulonephritis or haemodynamically mediated as in systemic hypertension .

Systemic hypertension causes kidney damage predominantly by transmission of the arterial pressure to the glomerular capillaries which overwhelm the intrinsic renal autoregulatory process leading to glomerular bleeding from capillary injury. Haematuria of glomerular origin, like proteinuria, decreased glomerular filtration rate (GFR) or elevated levels of serum urea and creatinine is a marker of kidney damage. There are few studies among Nigerian hypertensive population that assessed factors associated with kidney damage. One of such studies was carried out on treated hypertensive patients [8]. We are not aware of any study that reported predictors of kidney damage using abnormal urinary sediment as the marker in newly diagnosed hypertensive Nigerians, hence this study. The outcome may help to identify target (s) for more aggressive and focused therapy that will protect the kidney from effects of hypertension.

MATERIALS AND METHODS

This is a cross sectional study of one hundred and thirty-eight newly diagnosed adult Nigerian hypertensive patients, who were compared with the same number of age and sex matched apparently healthy non-hypertensive controls from the general population. Ethical clearance was obtained from the ethical and research committee of university of Ilorin Teaching Hospital. Subjects with other conditions associated with urinary sediment formation including urinary tract infection were excluded. The subjects' clinical and biographic data were collated in the outpatient clinic of the hospital while the laboratory components were carried out at the hospitals' chemical laboratory and renal unit laboratory. The surrogate marker of kidney damage was significant microscopic haematuria defined as ≥3/hpf. This was determined by examination of urine sediment under a bright field microscope having centrifuged the urine and application of supravital Sternheimer's stain. Sternheimer's stain is a mixture of Copper-phthalocyanine dye, national fast blue and a xanthene dye (pyronin B) used as an alternative to phase contrast microscope to enhance identification of elements of urinary sediment. The sediment preparation and staining technique were as follows: 10ml of early morning "first void" clean catch urine was collected in a sterile tube and centrifuged for 5minutes. The supernatant was decanted leaving 0.5ml of the sediment. A drop of Sternheimer's stain was added to the 0.5ml sediment and left for 10

Table 1: Age distribution of the subjects

Age (yrs)	Patients		Controls		P value
	n (%)		n (%)		
20-29	11	8	11	8	1.0000
30-39	37	26.8	33	23.9	0.5800
40-49	58	42	63	45.7	0.5442
50-59	25	18	24	17.4	0.8748
60-69	4	2.9	4	2.9	1.0000
70-79	3	2.2	3	2.2	1.0000
Mean age	43.21 (±9.65)		43.19 (±9.55)		0.9862

Table 2: Urinary sediment cells in patients and controls

Cells	(per hpf)	Patients n (%)	Controls n(%)	P value
RBC	Nil	90 (65.2)	119(86.2)	0.0000
	1-2	27(19.6)	14(10.2)	0.0278
	> 2	21(15.2)	5(3.6)	0.0010
WBC	Nil	128(92.8)	125(90.6)	0.5130
	1-2	10(7.2)	13(9.4)	0.5130
	> 2	-	-	-

RBC, red blood cell; WBC, white blood cell

minutes and then examined under the bright-field microscope. Potential predictors of kidney damage evaluated were: age, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP), pulse pressure (PP), and body mass index (BMI). SPSS version 13.0 (SPSS Inc., Chicago, IL, USA) statistical soft ware was used to analyze the data. The strength of association between the variables and kidney damage was determined by correlation statistics while regression methods were used to quantify the association and to predict kidney damage.

RESULTS

There were seventy-six (55%) males in the patients and eighty (58%) in control group. The age distribution is shown in table 1. Sixty percent of the patients were within 40-59years. Significant proportions of the

Table 3: Correlates of microscopic haematuria in hypertensive patients

Parameters	Correlation coefficient (r)	p value
Age (years)	0.121	0.921
Body mass index (kg/m^2)	0.088	0.409
Systolic blood pressure (mmHg)	0.209	0.048
Diastolic blood pressure (mmHg)	0.156	0.143
Mean arterial blood pressure (mmHg)	0.185	0.080
Pulse pressure (mmHg)	0.186	0.080
Urea (mmol/L)	0.049	0.649
Cr(umol/L)	0.165	0.120
Creatinine clearance (ml/min)	-0.175	0.291

Table 4: Predictors of kidney damage in hypertensive patients

Predictors	r	r^2	adjusted r^2	df	P
1st Model					
Systolic blood pressure	0.557	0.312	0.272	1	0.011
2nd Model					
Systolic blood pressure & Age	0.72	0.505	0.447	2	0.019

P of entrance≤ 0.05; removal ≥0.1

patients are young with about a quarter within 30-39years. Table 2 depicts urinary sediment cells, the erythrocytes and leucocytes. There was a significant difference between the prevalence of microscopic haematuria in patients (15%) and the control group (3.6%) (p=0.001). The results of the correlation of kidney damage with the variables are shown in table 3. Only SBP correlated positively (r=0.209) and significantly with kidney damage (p=0.048). The stepwise regression analysis identified SBP as the best independent predictors of kidney damage in the first model (r^2 = 0.312; p=0.011) while SBP and age were identified in the second model (r^2 =0.505; p=0.001, P=0.019) with entrance level at $p \leq 0.05$, and removal at $p \geq 0.1$ (table 4).

DISCUSSION

Systemic hypertension as a major cause of cardiovascular and chronic kidney disease is associated with significant adverse clinical outcomes, especially in resource poor settings of which sub-Saharan African countries are the most affected. Efforts towards reducing the burden of hypertension and its clinical consequences should aim at early identification of risk markers and risk factors that are potential targets for intervention. Sub-clinical kidney damage in form of abnormal urinary sediment (microscopic haematuria) was detected in 15.2% of patients in this study which is significantly higher than 3.6% reported by Ratto *et al* [9] among the Spanish population. This is in support of the fact that blacks have intrinsic susceptibility to hypertensive kidney injury. Studies in black populations have consistently corroborated this observation by showing hypertension to be a major cause of kidney disease. In Nigeria, Ojogwu [10] had shown that hypertension was the cause of ESKD in 43% of 1980 patients studied prospectively. Similarly, Akinsola *et al* [11] reported that hypertension was second to chronic glomerulonephritis as a cause of Kidney failure. Furthermore, among treated hypertensive patients, CKD measured by estimated GFR, abnormal serum chemistry and urine sediment was observed in 18% of patients [8]. In African American population, hypertension remains the leading cause of CKD. Risk factors that enhance development of CKD/ESKD in hypertensive patients include earlier age of onset of hypertension, long standing severe hypertension, family history and black race [12]. Additional factors reported in African Americans are lower socioeconomic status leading to inadequate health care, illicit drug use and utilization of antihypertensive drugs with less reno-protective effect to treat their BP[3]. Genetic and prenatal programming conferring low birth weight and associated reduced nephron number with consequent development of hypertension and kidney disease has been suggested [13]. In African people who are generally associated with poor socioeconomic status, poor health care services and malnutrition, this idea would be most applicable. The age distribution of the patients shows that significant proportion were young with a quarter less than 40years of age. Blacks have generally been shown to develop hypertension and associated target organ damage at relatively younger age compared with their white counterparts [14]. These suggest that hypertension management guidelines in blacks should recognize these risk indicators and prescribe a more aggressive treatment that would help to attenuate both kidney and cardiovascular damage. The hallmark of this study was the observation that systolic blood pressure correlated positively and significantly with kidney damage. This effect seems to be amplified by increasing age. This observation is in support of other studies that had recognized SBP as a more potent risk factor for development of chronic Kidney disease in hypertensive patients. In a 15year follow-up study of American hypertensive men, systolic pressure was clearly shown to be better than diastolic pressure as a predictor of endstage renal disease, and the risk increases with severity of the BP [15]. The finding was later supported by the work of He *et al* [16].

Among treated hypertensive Nigerians, Ayodele *et al* [8] found a significant association between SBP and target organ damage which include CKD but no association between DBP or pulse pressure and target organ damage. In Framingham study in the preceding four decades, Kannel *et al* had demonstrated that SBP confers a risk for coronary heart disease [17]. Ten years later, he also showed similar relationship of SBP with stroke [18]. More evidences have accumulated in recent times in support of SBP (among other BP components) as the most potent predictor of not only kidney damage but also cardiovascular morbidity and mortality. In a study of 4712 French men, Benetos et al had reported an association of SBP with cardiovascular diseases [19]. The multiple risk factor intervention trial (MRFIT) study on the other hand showed that death rate from coronary artery disease was directly related to the level of SBP in 316,099 middle-aged men during

a 12-year follow-up period [20]. In addition; Systolic Hypertension in the Elderly Study (SHES) documented a significant reduction in stroke by treating isolated systolic hypertension [21]. All the foregoing data are in contrast to earlier practice that assumes DBP to be more important than SBP as a cause of target organ damage in hypertensive patients. This may be responsible for the poor control reported in many studies as focus of therapy and measure of treatment efficacy neglected SBP. Our study also demonstrated an amplification of the predictive power of SBP on kidney damage with increasing age. This is not surprising because older age has long been recognized to be associated with both kidney and cardiovascular diseases, and that isolated systolic hypertension is a common occurrence in elderly people. We did not observe an association of MAP, PP and DBP with kidney damage in this study.

The small sample population and cross sectional design of our study may be contributory factors. However, studies have shown that MAP, PP and DBP are also predictors of cardiovascular disease [22]. Sequel to evidences supporting SBP as a more potent cardiovascular and renal risk, it has been recommended for identification as a primary determinant of outcomes and a useful tool for risk stratification when evaluating patients with hypertension or those suffering for hypertension-related clinical events. It is also recommended that SBP should be a focus of more aggressive treatment. Clinicians involved in hypertension management particularly those practicing in the Sub-Saharan region have therefore been advised to change their attitude towards BP management and treat SBP as a more important component while not undermining DBP[23].

CONCLUSION

This study suggests that SBP may be the best predictor of the damage which seems to be amplified by increasing age. However, the small population sample and the cross sectional design of the study preclude a far reaching conclusions. A longitudinal study with large population size is recommended to confirm this relationship.

REFERENCES

1. Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK and He J. Global burden of hypertension: Analysis of worldwide data. Lancet. 2005; 365: 217–223.
2. El-Nahas M. The global challenge of chronic kidney disease. Kidney Int. 2005; 68: 2918–2929.
3. Agodoa L. Lesssons from chronic renal diseases in African Americans: treatment implications. Ethn Dis. 2003; 13: S118-124.
4. Fogazzi GB, Ponticelli C and Ritz E. The urinary Sediment in the main diseases of the urinary tract. In: The Urinary Sediment, An Integrated View, 2nd Ed. Italy: Masson SPA, 1999: 139-160.
5. Guidelines Subcommittee. 1999 World Health Organization- International Society of Hypertensive.Guidelines for the Management of Hypertension. J Hypertens. 1999; 17: 151-183.
6. Lemogoum D, seedat YK and Mabadeje AFB, *et al*. Recommendation for prevention, diagnosis and management of hypertension and cardiovascular risk factors in sub-Saharan Africa. J Hypertension. 2003; 21: 1993-2000.
7. Fairley K. and Birch D. F. Hematuria: A Simple Method for Identifying Glomerular Bleeding. Kidney Int.1982; 21:105-108.
8. Ayodele OE, Alebiosu CO, Salako BL, Awodein AD and Adigun AD. Target organ damage and associated clinical conditions among Nigerians with hypertension. Cardiovascular J South Afr. 2005;16: 89-93.
9. Ratto E, Campo C and Segura J, *et al*. Prevalence and incidence of urinary sediment alterations hypertensive patients. Am J Hypertens. 2004; 17: 1: S-89.
10. Ojogwu LI. The Pathological basis of end-stage renal disease in Nigerians. W Afr J Med. 1990; 9:193-196.
11. Akinsola W, Odesanmi WO, Ogunniyi JO and Ladipo GO. Disease causing chronic renal failure in Nigeria: A prospective study of 100 cases. Afr J Med med Sci. 1989; 18: 31-37.
12. Anderson S. Mechanisms of nephrosclerosis and and glomerulosclerosis. In: Izzo, JL, and Black HR (Editors) Hypertension Primer, Dallas, American Heart Association, 2003.
13. Brenner BM , Garcia DL and Anderson S. Glomeruli and blood pressure. Less of one,

more the other? Am J Hypertens. 1988; 1: 335-347.

14. Ashaye MO and Giles WH. Hypertension in blacks: A Literature Review. Ethn Dis. 2003; 13: 456-462.

15. Klag MJ, Wheton PK and Randall BL *et al.* Blood pressure and end-stage renal disease in men. N Engl J Med. 1996; 334: 13-18.

16. He J and whelton PK. Elevated systolic blood pressure as a risk factor for cardiovascular and renal disease. J Hypertens. 1999; 2:S7-S13.

17. Kannel WB, Gordon T and Sschwartz MJ. Systolic versus diastolic blood pressure and risk of coronary heart disease: the Framingham Study. Am J Cardiol. 1971; 27: 335-346.

18. Kannel WB, Wolf PA and McGeel DL. Systolic versus diastolic blood pressure, arterial rigidity and risk of stroke: the Framingham Study. JAMA. 1981; 245: 1225-1229.

19. Benetos A, Thomas F, Bean K, Gautiers S, Smulyan A and Guize L. Prognostic value of systolic and diastolic pressure in treated men. 581. Arch Intern Med. 2002; 162: 577-

20. Neaton WD, for the Multiple Risk Intervention Trial Research Group. Serum cholesterol, blood pressure, cigarette smoking and death from coronary heart disease: overall findings and differences by age for 316, 099 white men. Arch Intern Med. 1992; 152: 56-64.

21. SHEP Cooperative Research Group. Prevention of stroke by antihypertensive drug treatment in older persons with isolated systolic hypertension: final result of Systolic Hypertension in the Elderly Program. JAMA. 1991; 265: 3255-3264.

22. Sesso HD, Stampfer MJ and Rosner B *et al*. Systolic and diastolic blood pressure, pulse pressure, and mean arterial pressure as predictors of cardiovascular disease risk in men. Hypertension. 2000; 36: 801-807.

23. Salako BL. Target blood pressure control in Sub-Saharan African: paradigm shift. Postgraduate Doctor Africa. 2004; 26: 10-12.

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Acute Kidney Injury among Burn Patients in a Tertiary Care in Western Nigeria

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ABSTRACT

Acute Kidney Injury among Burn patients have not been widely described especially in developing countries. The current study is aimed at providing information on the pattern of acute kidney injury. This review focuses on burn aetiologies and Acute kidney Injury (AKI) as seen in different types of burns, peculiar features and mortality pattern at a burns facility in a tertiary hospital unit in South Western Nigeria. This is a 5 year audit of all admissions into the burns unit of the Ladoke Akintola University of Technology Teaching Hospital Osogbo, Osun State, Nigeria from May 1st 2004 to May 1st 2009. The case notes of the patients and admission registers of the unit were the sources of information. A total of 147 patients with different types of burns were admitted during the period under review with age range of 1 to 70 years (mean of 28± 4yrs). There were 84 males and 63 females. Majority (133 or 90.4%) of the patients had thermal burns while 7 (4.8%) had electrical burns, 1(0.7%) had chemical burn. Eighteen (12.2%) of the total patients developed acute kidney injury. All the patients with AKI had various forms and degrees of thermal burns (i.e. flame and scald injury). The average length of hospital stay was 18.3 days while sepsis was a major contributory factor to mortality in 15 of the entire patients with burns. Only 9 (50.0%) of the 18 patients survived bringing the mortality among the patients with Acute kidney injury to 50.0%. Mortality is high among patients with acute kidney injury in failure at our burns unit despite intensive care. Adequate conservative care with appropriate early referral of patients remains the cornerstone of burn care. Adequate information on preventive strategies of burns should be vigorously pursued

Keywords: *Acute kidney injury, burn patients, Nigeria*

INTRODUCTION

Management of burn injuries poses lots of challenges. This is more so in developing countries where the few available facilities are either not readily affordable or not functioning optimally [1-3]. Burns injuries have been found to be a major cause of morbidity and mortality in children in southeastern Nigeria [4]. Emergency transport services are also not readily available in most parts of the developing countries especially in sub-Sahara Africa [5-7]. The high cost of managing patients in terms of costs of consumables and prolonged hospitalization in a general surgical

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ward or in the few available specialized units in a low resource setting is a major challenge. These account for the high mortality, usually from sepsis and other major complications such as kidney failure [8,9].

A severe burn is a skin injury accompanied by serious systemic illness, with effects on different organs distant from the site of primary injury. It is known that outcome is often affected by patients' age, percentage of total body surface area burned, pre-existing diseases and associated inhalation injury or other traumatic lesions [1]. Mortality is often from multi organ dysfunction syndrome (MODS), and the lungs are among the organs affected (approximately 100%) followed by the kidneys and the gut in that order[1].

Burn present a challenge to both the resources and skills of the clinicians. With poor facilities and inadequate resuscitation of patients, morbidity and mortality tend to rise. Acute renal failure is one of the major complications of burns (especially major burns) in addition to other changes in the morphological and functional status of the kidneys. These include proteinuria, haematuria, extensive tubular damage, alteration in calcium homeostasis, increased renal blood flow , increased mean kidney mass and also derangements in the fluids and electrolytes balances in the body [1].

Burns injuries may be thermal (flame, scald or contact), chemical burn, seen on exposure to strong acid or alkaline products which occurs most often in industrial settings from assaults in developing countries [10] or electrical injuries. Acute renal failure (a subset of the spectrum of acute kidney injury) is defined as an acute deterioration in renal excretory function, with normal sized kidney, an increased echogenicity on ultrasound scan with a serum urea > 10mmol/l and/or a rise in serum creatinine (Scr) by ^{3} 0.3mg/dl or a percentage increase in Scr of ^{3} 50% from baseline (using the AKIN criteria)[11,12].

Two distinct clinical patterns of acute renal failure (ARF) in burns patients have been described- early ARF, developing a few hours after injury to the first five days and late ARF which develops after five days. The early variety is said to be due to hypovolemia while the late ARF is often as a complication of septicemia and multiple organ dysfunctions [13]. There have been a paradigm shift over the last decade as most patients seen in several burns centres with acute renal failure tend to present with the late variety, most likely as a result of nephrotoxic medications prescribed [13].

The incidence of acute kidney injury in burns patients is said to vary between 0.5% and 30% with mortality as high as 90% -100% [13-15]. Various works have been done on acute renal injury/failure under different settings in Nigeria [16,13] , but none have describe the pattern of acute renal dysfunction in burns patients, with anecdotal reports of figures as high as 100% in our environment. We therefore set out to describe this pattern at the burns unit of the LAUTECH Teaching Hospital, Osogbo, Osun State, Nigeria over a 5 year period. The hospital has 200 beds comprising a purposefully built plastic surgery facility encompassing a 6 bed burns unit, a fully functional hemodialysis unit (which offers only intermittent and maintenance HD) and an intensive care unit.

This retrospective study was informed as a result of the rather high mortality pattern in burns patients with acute kidney failure at our facility and the need to explore ways of limiting this rising profile.

MATERIALS AND METHODS

Review of the burn patients admitted over the period of 1st May 2004 to 1st of May 2009 was done. Information obtained from the case files, unit/ward and hospital records include the biodata, clinical and Laboratory data as well as outcome of the treatment. Inclusion criteria for patients with acute renal dysfunction at our centre was based on the history, urine volume, and serial serum laboratory parameters especially serum urea^3 8 mmol/L and serum creatinine ^{3} 110µmol/L(1.2mg%) or documented oliguria of < 0.5ml/kg/hour for more than 6 hours. It should be noted that while the conventional heamodialysis is the only method of treating renal failure in our centre, most of the patients could not afford this. Septicaemia is defined as a microbiological focus of infection and deterioration of the clinical state evidence by at least one of the following [17]:

(a) Temperature > 39^0c on two or more occasions,
(b) Leucocytosis > 10 x 10^9 /lit,
(c) Positive blood culture.

Multiple organ system failure was defined as the development of abnormalities affecting one

or more organ systems in a critically ill patient [17].

Intravenous Ringer's lactate was used for resuscitation in volumes according to the Parkland formula (4 mL/kg body weight [BW]/TBSA%), with adjustments for individual variations in hemodynamic variables, aiming at least for a mean of 1ml/kg/hour. Excision of the wounds were rarely done due to the fact that patients had to make fund available for this to be done. Wounds were dressed with either silver sulphadiazine cream or honey till wounds healed by epithelialization or eschar separated and wounds grafted.

Data were analysed using the SPSS for windows statistical package Version 15.Continous variables were analysed as mean and standard deviation. Non parametric variables were analysed as percentages.

RESULTS

One hundred and forty seven patients (147) presented with burn injuries during the study period. They consisted of 84 (57.1%) males and 63 (42.9%) females with a mean age of 23.2(±18.8years). Ninety-nine patients (67.3%) presented about an hour after the injuries with the mean number of hours between injuries and presentation at theburn unit being 3.1 (±8.0) hours; a median and modal period of 1.0 hour respectively and variance of 63.6 hours.

Flame was the commonest cause of the injuries in 89 (60.5%) of the patients followed by scald in 44 (29.9%), friction burn in 3 (2.0%), electrical injuries in 7 (4.8%) and chemical in 1 (0.7%).

The mean percentage burn surface was 30.8±24.6%. Ten (6.8%) of the patients had associated suspected inhalation injuries.

Eighteen (12.2%) of the patients developed acute kidney injury and all had various forms and degrees of thermal burns (i.e. flame and scald injury).Table 1

Analysis of the patients with AKI revealed that there were 12 males and 6 females. Their ages ranged between 1.5 years to 62 years, mean of 37 years (±4.5). Ten (55.6%) of the patients had flame burn, 5 (3.1%) had scald injuries, 2 (11.1%) electrical injuries

Table 1. Summary of patients with AKI

N0:	Age	Sex	Aetiology	%TBSA	Inhalation	Urea	creatinine	Organism isolated	Out come
1	55	F	Fl	18	nil	10.6	103	Staph/E.coli	Died
2	7	M	Sc	33	nil	6.4	205	-	Died
3	54	F	Fl	58	nil	9.2	-	-	Survived
4	35	M	Fl	36	nil	3.2	129	—	Survived
5	42	M	Fl	66	nil	11.2	116	-	Died
6	62	F	Fl	98	nil	11.2	—	—	Died
7	36	M	Fl	28	nil	5.5	128	klebsiella	Survived
8	3	M	Sc	51	nil	10	82	—-	died
9	3	M	Sc	53	nil	11.2	62	—-	died
10	9	M	Fl	54	inhalation	22	166	—	died
11	45	M	El	30	nil	9.8	62	—	Survived
12	25	M	Fl	82	inhalation	9	136	-	died
13	25	M	El	15	nil	3.5	145	-	Survived
14	22	F	Fl	64	inhalation	15	102	—	died
15	60	M	TE	96	nil	15	-	klebsiella	Survived
16	55	F	Fl	40	nil	4.2	120	pseudo	Survived
17	1.5	M	Sc	60	nil	16	82	Staph	Survived
18	2.5	F	Sc	15	nil	10.4	125	Staph	Survived

Key: *fl = flame, Sc = scald, El = electrical burns, TE = Toxic Epidermal Necrolysis syndrome, M = male, and F = female,* ***% TBSA*** *= Percentage total burn surface Area.*

and one(5.6%) of the patients had Toxic Epidermal Necrolysis Syndrome (TENS). The body surface area of the Burn injuries ranged between 15 to 96% with a mean of 49.3%. Three (11.1%) of the patients sustained inhalational injuries and died. Patients who had urea of more than 8 mmol/L and or Creatinine of more than 110 were considered as having Acute Kidney Injury and the mean value of urea and creatinine in the patients with AKI were 10.2 and 117.5mmol/L respectively. Six of them had associated wound infections with Staphylococcus aureus alone in 2 patients, Staphylococcus aureus with E.coli in 1 patient, Klebsiella and Pseudomonas infections in 2 other patients

Nine(50.0%) of the patients with AKI died and these included the 3 patients with inhalational injuries.

DISCUSSIONS

Acute renal failure is a common complication seen in patient with burns especially where the initial resuscitation is either not given at all or not adequately given as observed in many patients who presented having been managed in one private hospital or another. Olaitan et al had observed that acute renal failure constituted the greatest (42.8%) cause of death among paediatric age group (21) and 42.1% among adult and children with burns in Enugu, Nigeria [22].

In the profile of the 18 patients with Acute Kidney injury in the current study, most of the patients had various forms of thermal burns and this may suggest that it is likely that the rhabdomyolysis caused by flame injury may have predicted poor survival in this group of patients. The incidence of AKI of 12.2% seen in our unit is comparable to that seen in other centres[23].

The incidence is either relatively low as a result of the aggressive resuscitative measures at presentation, or most of the survivors were those that were earlier managed at another setting before referral.

Five (27.8%) of our patients presented with early renal insufficiency possibly as a result of extent/ degree of burns, late and hypovolemia while 13(72.2%) presented with the late variety likely as a result of complication of treatment (nephrotoxic antibiotics) or part of a multiple organ dysfunction syndrome. This in particular is in agreement with similar studies in done in Germany [13].

None of our patients had any form of renal replacement therapy as majority of them had major burns affecting most part of the body coupled with the non availability of continuous renal replacement therapy (CRRT) at both the burns and intensive care units in the hospital. The mortality rate in this study among the patients with acute kidney injuries is 50.0% compared to the overall mortality of 29.9% among all the patients. Essentially, a major burn is still a major cause of acute kidney injury both in developed and developing countries. This especially in developing countries is often complicated by poverty, ignorance, late referrals and management inadequacies most often at the point of first care. Mortality is high as seen in studies done elsewhere especially in the developing countries. Possible reasons may include de-novo increased insensible fluid loss and subsequent poor fluid management; use of nephrotoxic agents (herbal remedies/antibiotics), underlying chronic renal insufficiency and late referral.

Of course the renal status of most of the patients before presentation was unknown. Efforts should be geared towards preventive mechanisms by way of preventive burn hazards. Better equipping of intensive care facilities and easy recognition of features of acute kidney decompensation are important points to note.

Primary care physicians should be educated on the care of burn patients and to refer the patients as early as possible where this is indicated. The health Insurance scheme which is not enjoyed by many Nigerians will be of assistance in ensuring that patients with serious injuries such as burns are well taken care of. This will ultimately reduce the current spate of acute renal injuries and eventual morbidity and mortality among burn patients.

REFERENCES

1. Mariano F, Gangemi NE and Mauiizio S *et al*. Burns and Acute Renal Failure. Chapter 57. *In:* Critical care Nephrology textbook (eds Claudio Ronco, Bellomo Rinaldo and John Kellum). 1998; 312-318
2. Argent AC. Critical Care in Africa SAJCC July 2009, (25) 1; 4 - 8
3. Oluwatosin OM. Burns in Africa. Afr J Trauma 2004; 2: 20-25.
4. Okoro PE, Igwe PO and Ukachukwu AK. Childhood burns in south eastern Nigeria. Afr J Paediatr Surg 2009; 6: 24-27.

5. Kobusingye OC, Hyder AA and Bishai D. *et al.* Emergency medical services in low and middle income countries, recommendation for action. Bulletin of World Health Organization 2005;83: 626 - 31

6. Adeyemi Doro HO and Sowemimo GOA. Optimal Care for Trauma Victims in Nigeria. Trauma Quarterly 1999; 14: 295 - 300

7. Solagberu BA, Kuranga SA and Adekanye AO. Preventable trauma deaths in a country without emergency medical services. Afr. J. Trauma. 2003; 1; 39 - 44

8. Chrysopoulo MT, Jeschke MG, Dzlewulski P. *et al.* Acute Renal Dysfunction in severely burned adults. J. Trauma 1999, 46: 141 - 144

9. Fitzwater J, Purdue GF and Hunt JL. The risk factors and time course of sepsis and organ dysfunction after burn trauma. J. Trauma 2003, 54: 959 - 966

10. Olaitan P.B and Jiburum B. C. Chemical Injuries from Assaults-An increasing Trend in a Developing country. Indian J Plastic Surgery.2008; 41(1); 20-23.

11. Nissenson AR. Acute Renal Failure. Definition and Pathogenesis. Kidney Int. 1998; 53; 66; S7 – S10

12. Singri N, Ahja SN and Levin ML. Acute Renal Failure. JAMA 2003; 289: 747 - 751

13. Holm C, Horbrand F and Von Donnersmarck GH *et al.* Acute Renal Failure in Severely Burned Patients. Burns 1999; 25: 171 - 178

14. Mustonen KM and Vuola J. Acute renal failure in intensive care burn patients. J. Burn Care Res 2008; 29 (1) 227 - 237

15. Coca SG, Bauling P and Schifftner T *et al.* Contribution of acute kidney injury toward morbidity and mortality in burns; a contemporary analysis. Am J. Kidney Dis 2007, 49: 517 - 523

16. Okunola OO, Arogundade FA and Sanusi AA. Acute Renal Failure in the Intensive Care Unit. Aetiological, predisposing factors and outcome.WAJM 2009;28(4): 240-244.

17. Arogundade FA, Sanusi AA and Okunola OO, *et al.* Acute Renal Failure in Developing Countries: Which factors actually influence survival? Cent. Afr. J. Med. 2007; 53: 34 – 39

18. Shehan H and Remo P. ABC of burns. BMJ 2004; 329: 101-103

19. Mehta RL, Kellum JA and Shah SV *et al:* The Acute Kidney Injury Network. Acute Kidney Injury Network: Report of an Initiative to Improve Outcomes in Acute Kidney Injury. Crit Care 2007; (2); R31

20. Ghani N and Shuraf I. Two years of burns admission at the General Hospital Kuala Lumpur. Journal of Malaysian Medical Association. September, 2003

21. Olaitan PB, Daira MD, Uduezue AO and Ogbonnaya IS. Paediatric Burns: Mortality in a Burn Unit. African Journal of Paediatric Surgery. 2007; 4(2): 82 - 85

22. Olaitan PB and Jiburum BC. Analysis of Burn Mortality in a Burn Centre. Annals of Burn and Fire Disasters. 2006; 19 (2): 59 - 62

23. Thomas MA and Ibels LS. Rhabdomyolysis and Acute Renal Failure. Aust. N.Z.J. Med. 1985; 15; 623 - 628

Chronic Kidney Disease, Mineral Bone Disorder: Review and Appraisal

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ABSTRACT

To review available literature on mineral and bone disorders in patients with chronic kidney disease with the aim of promoting better understanding of the concepts of divalent ions bone disorders . It also aims to highlight new trends in the definition and management, with emphasis on recent treatment strategies focusing on molecular targets. as well as the emerging knowledge of this condition in our local practice. A literature search was done on chronic kidney disease–mineral bone disease, pathogenesis and treatment strategies using the electronic database; MEDLINE, EMBASE, OVID, Google Internet search engines (for local websites and medical journals) and relevant textbooks. Mineral bone disorder is one of the major complications in patients with chronic kidney disease. Normally in people with healthy kidneys, normal serum levels of phosphorus and calcium are maintained through the interaction of parathyroid hormones(PTH) and 1,25 $(OH)_2D_3$ (calcitriol), the active metabolite of vitamin D_3. However in Chronic Kidney Disease, this interaction is deranged . This could result in various types of bone diseases viz high turnover bone disease (osteitis fibrosa cystica) , low bone turnover disease or a mixed variety. The clinical features could be asymptomatic initially, but could later manifest as myopathy and bone pains with increased risk of fractures and metastatic calcification of soft tissues including lungs, kidneys and joints. Cardiovascular complications are among the common causes of morbidity and mortality.

In Nigeria, It was formerly thought to be rare due to the tropical diet and also the fact that due to lack of renal replacement therapy, patients with CKD did not live long enough to develop mineral bone disease. However, with more centers offering renal replacement therapy and more patients on long term haemodialytic support and the attendant better chances of survival, cases of CKD-MBD are increasingly being seen in our practice. Mineral bone disorders are a major complication of chronic kidney disease and the incidence with its attendant complications is increasing globally. Early recognition and detection and aggressive treatment with prescribed diets and medications must be pursued.

Keywords: *Mineral bone disease, chronic kidney disease*

INTRODUCTION

Chronic Kidney Disease (CKD) – Mineral Bone Disorder (MBD) is defined as a systemic disorder of mineral and bone metabolism due to CKD manifested by one or a combination of the following:

(i) abnormalities of calcium, phosphorus, PTH or vitamin D metabolism,

(ii) abnormalities of bone turnover, mineralization, volume, linear growth or strength and / or

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(iii) vascular or other soft tissue calcification.

It also encompasses renal osteodystrophy which is described as an alteration in bone morphology in patients with CKD[1].

The link between renal failure and bone disorders was first described by Lucas in 1883 when he reported an association between albuminuria and late rickets [2]. Sixty years later, the term renal osteodystrophy was coined to describe an interplay between vitamin D, parathormone and divalent ions in patients with chronic renal failure[3]. In the 70s, and 80s, osteomalacia was identified as a major cause of mineral bone disease especially the presence of aluminium in dialysis water and its use as phosphate binders[4]. Over the last two decades, there have been a renewed interest in the knowledge of metabolic bone disorders especially the identification of calcium sensing receptors (CaSR) and their cloning[5], changing bone patterns, the identification of newer vitamin D (third generation) analogues and vitamin D receptors[6] and lately the implications of mineral metabolism in cardiovascular disorders[7].

This new interest is also due to the increasing incidence especially in post renal transplantation patients and the consequent newer guidelines by associations such as NKF/DOQI and the British Renal Association (by way of dietary regime guide and medications)[1, 8, 9].

Normal Homeostasis of Calcium, Phosphorus, Vitamin D and Parathormone Calcium Homeostasis

The main sources of calcium in the diet include; milk, cheese, eggs, meats, peas, dried fruits and nuts. Calcium is absorbed actively from jejunum and passively from the ileum. Intake of vitamin D, acidic PH and presence of proteins in the food favour absorption.

The total body content of calcium is 1200g in an adult. This is present in the skeleton, teeth, plasma and in all tissues of the total body calcium. Ninety eight percent of the total body calcium is in bones as calcium phosphate (hydroxyapatite) held in a protein matrix (osteoid). In the plasma, 50% of the circulating calcium is in ionized form i.e. free calcium, 40% is protein bound and 10% is complexed with citrate and phosphate ions. Calcium gives the strength and rigidity to the skeleton. In the plasma, calcium is present as the free ions, albumin bound form and as complexes which are diffusible. In health, serum calcium level ranges from 9.0 – 10.5mg% (2.25-2.6mmol/L). Note that a decrease in serum albumin of 1g/dl is usually associated with a decrease of 0.8mg/dl in total calcium concentration. Calcium and phosphorus levels also vary inversely with each other and the Calcium **x** Phosphate product is almost constant around $40mg^2/dl^2$

Corrected serum calcium is calculated as; measured serum calcium + (0.8 x (4 - serum albumin in g/dl)). Calcium regulation involves three tissues, namely the bone, kidney and intestine. It also involves three hormones, PTH, calcitonin and activated vitamin D.

Phosphorus Homeostasis

The total body content of phosphorus is 800-900g and most of it in the body is present in the bones and teeth. In all tissues, this element is present intracellularly as phosphates. In the plasma, it occurs as inorganic phosphates at a concentration of 2.8 – 4.5mg/dl (0.81-1.45mmol/l). Around 88% of phosphorus is in ionic form and the rest is present in the protein bound form. Phosphorus is contained abundantly in eggs, cereals, meat and diary products. Normal diet supplies 800 – 1400mg/day while absorption from the gut is passive with 60 – 80% absorbed. The serum level is controlled mainly by the renal excretory mechanism. Around 85% of filtered phosphate is absorbed at the proximal tubule and only 10 – 15% is lost in urine. Negative phosphate balance is usually caused by the abnormalities of renal clearance of 1, 25 $(OH)_2$ D_3 which promotes absorption.

Apart from its role in stimulating the parathyroid gland and thus contributing to hyperparathyroidism, hyperphosphatemia (serum phosphates above 6.5mg/dl) represents an independent risk factor for death in patients treated with HD and death from cardiovascular causes account for the excess mortality [10]. A strong relationship has been found between cardiac deaths and factors that favour metastatic calcification (i.e hyperphosphatemia and increased ca **x** phosphate product)[11]. Serum phosphate has been found to stimulate the phenotypic transformation of vascular smooth muscle cells into osteoblasts capable of producing a 'promineralisation milieu'. In this circumstance, the supersaturation of extracellular calcium and phosphorus tend to accelerate the development of medial wall calcification which is associated with an increase in

arterial stiffness , left ventricular size and all cause mortality in patients on haemodialysis[10, 11].

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Homeostasis of Parathyroid Hormone

Parathyroid hormone (parathormone) is secreted by the chief cells of the parathyroid gland which are situated normally posterior to the thyroid gland. It is a single chain polypeptide having 84 amino acids encoded by a gene on the short arm of chromosome 11. It is secreted as a pre-prohormone with 115 amino acid residues and is further converted into a prohormone containing 90 amino acid residues. The active hormone contains 84 amino acid residues.

In circulation, PTH is a mixture of polypeptide chains of different biological activity. The secretion of PTH is controlled by several factors. Circulating ionized calcium exerts the major control of PTH secretion and release. A fall in ionized serum calcium stimulates PTH secretion while a rise in serum phosphate stimulates PTH secretion indirectly by decreasing calcium levels detected by specific calcium sensing receptors on the membranes of the parathyroid cells. PTH can be estimated by radioimmunoassay (RIA). Parathyroid hormone levels estimated by this method may not reflect true biological activity since a part of the estimated hormone may not be biologically active .It has a very short half life (2-4 minute) and the normal range of the intact PTH assay is 10-65pg/ml while the recommended target range for CKD stages 3, 4 and 5 are 35-70pg/ml, 70- 110pg/ml and 150-300pg/ml respectively(fig 1).

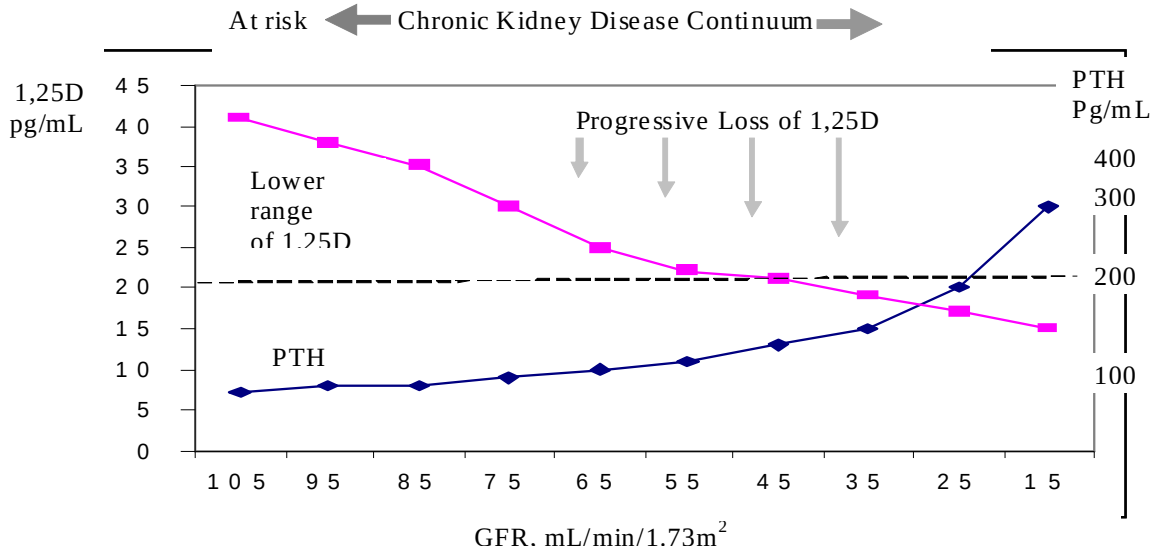
The ultimate effect of PTH is to conserve body calcium and increase its level in extracellular fluid. It exerts its functions in several ways and most of these are receptor mediated. These include increased distal tubular reabsorption of calcium, increased absorption of calcium from the intestine, increased renal excretion of phosphate and hydroxylproline resulting in decreased plasma phosphate. It also increases calcium resorption from bone, and raises serum calcium.

There are three molecular targets regulating parathyroid gland function. These are (i) the G protein-coupled calcium-sensing receptor (CaSR) which in conjunction with calcium is the major regulator of PTH transcription, secretion, and parathyroid gland hyperplasia; (ii) Vitamin D receptor (VDR) located in the parathyroid glands which is acted upon by calcitriol to suppress PTH transcription only and (iii) extracellular phosphate sensors which have direct effects on parathyroid production through the regulation of PTH message stability.

Homeostasis of Vitamin D

Vitamin D is obtained from the diet or produced in the skin as cholecalciferol (vitamin D_3) by sunlight photoactivation of 7-dehydrocholesterol. The latter is the primary source of vitamin D metabolites. Vitamin D_3 is transported to the liver via a Vitamin D-binding-protein (DBP) where it is converted by microsomal and mitochondrial hydroxylases to 25-

Figure 1 showing relationship of GFR to the PTH



Above illustration culled and modified from Nephrol Dial Transplant; 1996 (Suppl 3): 22-28.(with permission).

OHD_3 the major circulating form of Vitamin D. This is further transported via DBP to the kidney where it is converted to either the active vitamin D metabolite 1, 25 $(OH)_2D_3$. or to the less active form 24, 25 $(OH)_2D_3$. The enzymes responsible for these conversions are the cytochrome P_{450}-dependent 25-hydroxy 1α and 24 hydroxylases respectively. They are located in the proximal tubule and are responsive to PTH and to plasma phosphate concentration. Production of 1,25 $(OH)_2D_3$ is favoured by hypocalcemia, high PTH and low phosphate and its metabolism is tightly regulated and mediated via changes in serum calcium, phosphate and parathyroid hormone (PTH). 1, 25 $(OH)_2D_3$ has a variety of biological actions in the kidneys, bones and in the gut. It stimulates absorption of calcium and phosphate from the gastrointestinal tract via increase in the production of a specific calcium binding protein by duodenal and ileal cells where they increase active calcium transport. Phosphate absorption is also stimulated by 1, 25 $(OH)_2D_3$ This occurs in the duodenum and is independent of the Vitamin D induced effect on calcium absorption.

In the bone, 1, 25 $(OH)_2D_3$ is effective in promoting mineralization of osteoid, while it also has a role with PTH in promoting bone remodeling via resorption and remineralization i.e. facilitates deposition of calcium in bone. In healthy subjects, total vitamin D level is 35.0 ± 3.4mg/ml, while $25(0H)D_3$ and 1,25 $(OH)_2D_3$ are 28.5±2.0mg/ml and 35.0 ± 3 pg/ml respectively. In chronic kidney disease, circulating levels of $1,25(OH)D_3$ are low.(fig.2).

Pathogenesis of Mineral Bone Disorder in Chronic Kidney Disease

Normally the parathyroid hormones in conjunction with other phosphaturic hormones like phosphatonins and its derivatives like fibroblast growth factor 23 (FGF23) have an innate phosphaturic action by decreasing activity of the sodium phosphate cotransporter in the proximal renal tubule and facilitating the excretion of phosphates. However as early as stage II chronic kidney disease, when the GFR reduces, this phosphaturic action is blunted and the serum phosphate begins to rise (fig 2).

Vitamin D_3 is also reduced in chronic kidney disease owing to reduced 1α hydroxylation of vitamin D which occurs as a result of the reduced renal mass. Reduced vit D_3 leads to lower intestinal calcium absorption and ultimately low serum calcium. The low serum calcium stimulates parathyroid hormone synthesis.

Also as a result of this low serum calcium, the increased parathyroid hormone tries to normalize the serum calcium levels by acting on the bone by promoting phosphate excretion, hence a new steady state is achieved at the expense of hyperparathyroidism .A major characteristic of secondary hyperparathyroidism is increased parathyroid cell proliferation, as CKD progresses, this increase in cell proliferation results in diffuse parathyroid gland hyperplasia. Both hypocalcemia and hyperphosphatemia stimulate parathyroid gland proliferation and hyperplasia which also result in a decline in calcium sensing receptors CaR expression (a characteristic of actively proliferating cells of the parathyroid gland). (fig 2).

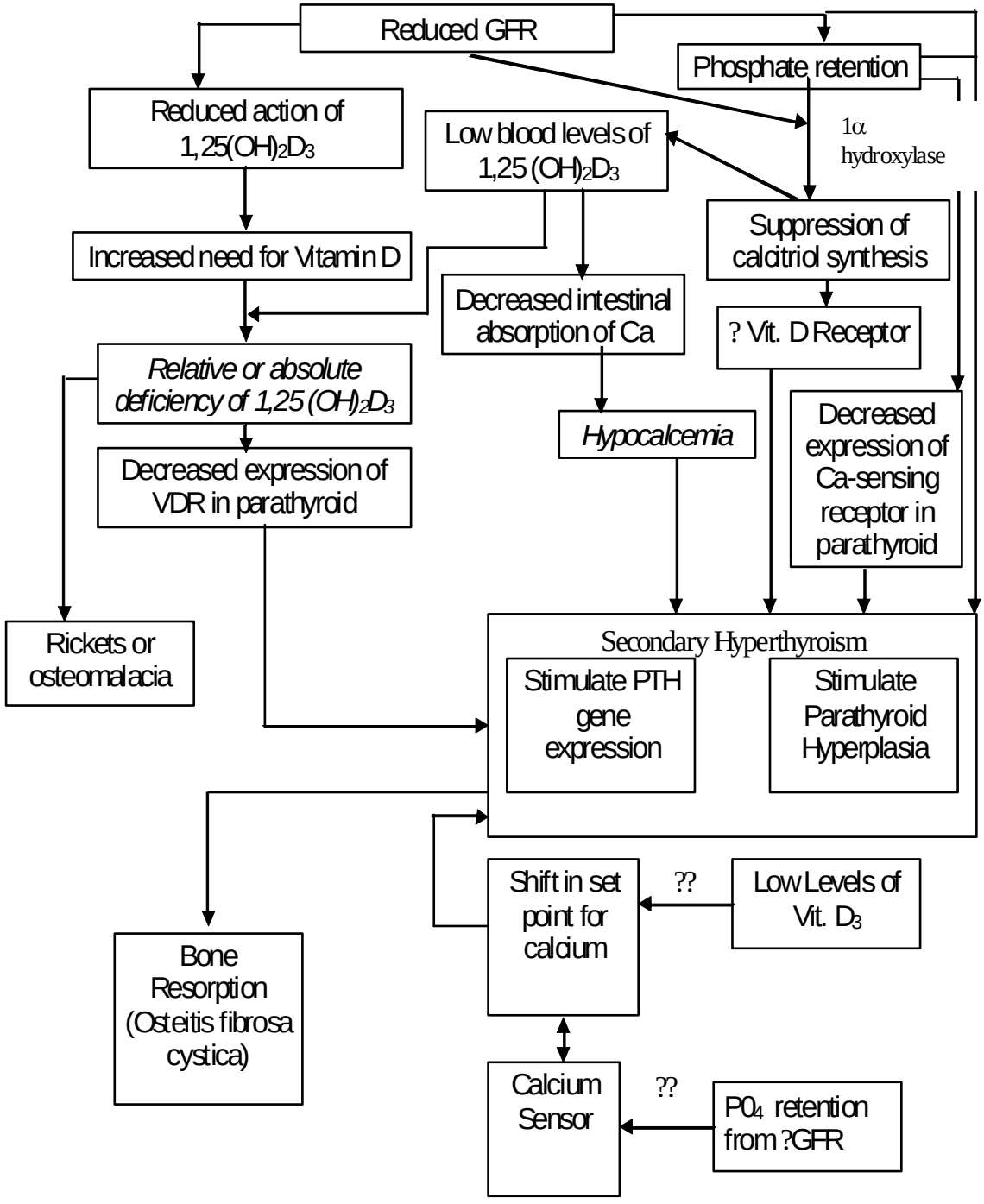
In summary, hyperparathyroidism in chronic kidney disease is as a result of a number of reasons viz; hyperphosphatemia resulting from reduced phosphate excretion which inactivates 1α hydroxylase leading to low vitamin D_3 synthesis; and low vitamin D_3 which reduces intestinal absorption of calcium. With the elevated phosphates, the serum calcium level goes down to achieve a constant solubility product. Low vitamin D_3 leads to reduced vitamin D_3 expression on the parathyroid, with the ameliorating effect on the normal inhibition of PTH secretion. Low serum calcium also inactivates Calcium sensing receptors on the parathyroid leading to enhanced PTH secretion, and when prolonged leads to hyperplasia. Elevated phosphates also stimulate PTH gene expression directly as a result of the increased parathyroid hormone.

Histological Classification of Bone Disease Associated with Chronic Kidney Disease

Bone disease associated with CKD has traditionally been classified according to specific histological types being due to different degree of bone turn over and impaired mineralization at the matrix. The high bone turnover type is osteitis fibrosa cystica while the low bone turnover types include osteomalacia and adynamic bone disease. It could also be a mixed variety i.e combination of high and low bone turnover [1].

I-PTH levels above 300pg/ml are associated with the high turn over type while the low turn over variety is suggestive in those with less than 150pg/ml

Pathogenesis of Abnormalities in Mineral Metabolism and Bone Disease in CKD



Modified from Pathogenesis of Abnormalities in mineral Metabolism and Bone Disease in CKD. From NKF/DOQI guideline, Handbook on bone metabolism and disease in chronic kidney Disease(CKD Stages 3-4)

Fig. 2: Showing the relationship between serum calcium, phosphates, vitamin D and hyperparathyroidism

[1]. Other markers of differentiating them include bone specific alkaline phosphatase levels, serum tartrate- resistant acid phosphatase-5b, osteocalcin and osteoprotegerin amongst others[12].

Clinicopathological Features

(i) High bone turnover disease-; this is typified by osteitis fibrosa cystica. It is caused by excess parathyroid hormone and it is characterized by increased bone turnover as evident by excessive proliferation and increase in size of the osteoclasts and osteoblasts, there is increased number of resorption lacunae with scalloped trabeculae. There is increased in the bone formation rate (BFR) with peritrabeculae fibrosis. The mineralization lag time is the mean time interval between osteoid deposition and its mineralization.

(ii) Low bone turnover disease; this could either be osteomalacia or adynamic bone disease [14]. Osteomalacia; this is caused by the accumulation of aluminium or other metals (strontium, iron) or diefficiency of 25 (OH) cholecalciferol or phosphate depletion. There is reduced bone turnover i.e. reduced osteoblastic / osteoclastic activity with increased mineralization lag time of more than 100 days relative to the normal time of 35days. The bone volume is low to medium. Aplastic/ Adynamic bone disease; this is caused by aluminium deposition. It may also be caused by parathyroid suppression and other factors such as deficiency of bone growth factors or increased suppression of bone remodeling. It may be associated with increased or reduced aluminium.It is seen more frequently in patients on long term peritoneal dialysis, diabetes mellitus and older patients. Histology shows more of osteoid tissues [14, 15].

(iii) Mixed disease; This is caused by a combination of secondary hyperparathyroidism and aluminium deposition or in some instances, the cause is unknown. The bone volume is normal; the turnover is increase, while the mineralization lag time is abnormal.

(iv) Aluminium related bone disease (ARBD); this may be caused by reduced renal excretion of aluminium, intake of aluminium salts as phosphate binders or dialysate aluminium concentration above 2-3mg/litre. There is extensive accumulation of aluminium at the mineralization front and this is diagnostic if it covers more than 15% of the trabecular surface and bone formation rate is reduced to less than the lower limit of normal (< 220mm^2/mm^2/day). Other features include reduced osteoblasts and increased osteoid volume [16]. Aluminium related bone disease could however be seen in any of the following:

(a.) Vitamin D resistant osteomalacia;

(b.) Specific adynamic bone disease; this is caused by excessive suppression of parathormone by 1, 25 dihydroxy cholecalciferol.

(c.) Mixed bone disease; this is characterized by a defect in matrix synthesis caused by PTH suppression and aluminium toxicity.

Clinical features of ARBD include severe diffuse bone pains, muscle weakness (especially in the upper limb), spontaneous fracture, neurological syndromes or microcystic anemia.

Diagnosis of ARBD is by detection of increased amount of aluminium in the serum > 50ug/ litre, slightly elevated calcium (which may increase with vitamin D dosing) and a normal serum alkaline phosphatase.

Early recognition of ARBD requires a desferrioxamine (DFO) test in combination with a serum iPTH measurement. DFO is a chelating compound that will liberate aluminium from the body stores, resulting in the formation of Aluminium –DFO complex (Aluminioxamine) to enter the bloodcompartment. A low dose of 5mg/kg is administered and aluminium estimation is done before and forty eight hours after the DFO challenge with a DC plasma emission spectrophotometer. An increase in serum aluminium after the DFO administration suggests the presence of ARBD. It is a useful test to differentiate patients with ARBD from those with an increased risk of aluminium toxicity and aluminium overload [16].

ARBD is prevented by avoiding aluminium in high risk patients (diabetics and children), avoiding concomitant administration of citrates in patients on

aluminium and reducing aluminium in dialysis solutions to less than 2-3ug/lit.

CKD MBD in Nigeria

Renal bone disease a few decades ago was initially thought to be rare in Nigeria and in the tropics for several reasons. The nature of the diet in the tropics was said to be protective partly due to limited intake of diary products; consumption of vegetables which contain phytates which mop up phosphates, food processing habits which encourage the use of large amount of water which is often decanted intermittently during cooking and by this the amount of phosphates is reduced; exposure to abundant sunlight which is thought to enhance the availability of vitamin D; and most of the patients do not live long enough for full establishment of the clinical symptoms of renal bone disease partly due to rapid progression for the disorder to manifest and lack of renal replacement therapy.

However, due to the increasing availability of renal replacement therapy in the country and improved management strategies, the lifespan of our end stage kidney disease patients is prolonging and the problem of renal bone disease may become obvious.

A few local studies done in Nigerians have been reported and these were mainly radiological and clinico-pathological studies[17, 18,19]. Limitations were the lack of parathyroid hormone estimation and vitamin D assay in these studies coupled with limited bone histology samples and reports.

Odenigbo *et al* in 2001 reported on the radiological prevalence of renal osteodystrophy in 90 patients studied and concluded that osteitis fibrosa cystica was the commonest form and this was more prevalent in female patients [17]. This finding was also confirmed in another work done in Nigeria which also showed that osteitis fibrosa cystica was common among the patients seen. The histology of the bone biopsies done in 10 patients were confirmatory. Hypocalcemia was further seen in 71% and hyperphosphatemia in 79% [18].

Sanusi *et al* in a prospective study of 40 patients in 2009 reported the increased prevalence of mineral and bone disorders especially the low turnover variety and it was said to be common in males. Hypocalcemia was present in 59.3%, hyperphosphatemia in 75%, low vitamin D was found in 83.3%. 18% of the patients had hyperparathyroidism while 42.9% of the patients had increased alkaline phosphatase. Majority of the patients wereasymptomatic[19]. Radiological features of osteitis fibrosa were seen in 23%, unlike 3.35% and 2.2% reported in the other two studies. In all these studies, there was paucity of clinical features of bone disease in the patients.

Clinical Manifestations of Bone Diseases Associated with CKD

Most patients with CKD and mildly elevated circulating PTH are asymptomatic. When present, the clinical features of bone disease may manifest with musculoskeletal features which include fractures**,** tendon rupture, bone pains and weakness around the low back, hips and legs aggravated by weight bearing, bone deformities, muscle pain/weakness**,** periarticular pains and hip fractures. The latter is clinically significant and it is high among stage 5 CKD patients. It is particularly associated with an increased risk for death.

Extraskeletal manifestation is mainly pruritus which is seen in advanced chronic kidney disease especially patients on HD, and it is possibly related to the deposition of calcium and phosphorus in the skin.

Cardiovascular Complications

This accounts for half of all deaths in haemodialysis patients [7, 8, 20]. Calcium is deposited on heart valves especially the mitral and aortic valves and in the myocardium causing arrhythmias, left ventricular dysfunctions, aortic and mitral stenosis, ischemia, congestive cardiac failure and death. An association has also been described between left ventricular hypertrophy and parathyroid hormone levels in CKD patients with secondary hyperparathyroidism. Hypertrophied growth of cardiomyocytes and smooth muscles cells due to activation of cardiomyocyte protein kinnase have been reported.[21].

Coronary artery and vascular calcifications also occur frequently in majority of patients on renal replacement therapy and this is probably due to excessive use of calcium containing phosphate binders and vitamin D analogues. Coronary artery calcification is detected by non-invasive electron-beam computed tomography (EBCT) [20]

Calciphylaxis/ calcific uremic arteriolopathy(CUA). This is a vascular disorder characterized by systemic medial calcification of the small arterioles and resulting in ischaemic necrosis of skin and soft tissues. It is often complicated by

capillary thrombosis and recanalisation and it is seen primarily in CKD stage 5 and occurs in 1-4% of maintenance haemodialysis patients [22]. Although less frequently seen, it may be a feature of the cardiovascular complication of Chronic Kidney Disease-MBD. Calcification could be of proximal or distal types. Proximal calcification presents with painful lesions over the thigh, abdomen and buttocks and it is seen commonly in pre-dialytic CKD patients. Risk factors include; White race, female gender, morbid obesity and poor nutrition. Distal calcification is seen in the lower extremity especially in patients on long term HD with hyperparathyroidism and high Calcium x Phosphate product (mg^2/dl^2). It is more commonly found at local trauma areas of injections especially insulin, heparin and iron dextran infusions [22, 23].

Patients manifest with non healing ulceration of the skin and gangrene resistant to medical therapy and this often leads to amputation and death. There is high mortality due to uncontrollable sepsis[23].

Diagnosis is by a general evaluation of the clinical features clinical ,skin biopsy or three phase technetium 99m methylene diphosphate bone scintigraphy. This reveals calcifications in subcutaneous nodules or non-ulcerating lesions in viable tissues.

Specific management of CUA includes stoppage of oral calcium and usage of non-calcium containing binders, control serum phosphates to <6mg/dl, wound debridement and parathyroidectomy(if PTH levels are above 600pg/ml).

Mechanism of Calcification

Under the influence of uraemia and hyperphosphataemia ,the smooth muscle gene expression is down regulated in preference for osteoblastic differentiation**.** Promoters of vascular calcification like bone morphometric protein-4 (BMP-4) and osteopontin expressions are also increased, while low levels of inhibitors of vascular calcification like 2-Herman Schmid glycoprotein, Fetuin-A and matrix –GLA protein have been demonstrated in uremic patients[7, 23]. Fetuin A is a serum glycol-protein that binds calcium and phosphorus in circulation and forms "calciprotein particles" in order to clear the circulation of excess calcium and phosphorus . It is a major systemic and circulating inhibitor of calcification synthesized in the liver. Matrix glycoprotein A is synthesized by vascular smooth muscle and chondrocytes and may be responsible for inhibiting calcification of arteries and cartilage as has been demonstrated experimentally[23].

Amyloidosis

This is a musculoskeletal abnormality seen in patients on maintenance heamodialysis for more than 7 years[23]. It is not related to disordered calcium and phosphate homeostasis but there is osteoarticular B_2-microglobulin amyloid deposition. It manifests as erosive cystic changes, carpal tunnel syndrome and as a destructive spondyloathropathy in the cervical and lumbar spine ultimately leading to spinal instability and neurologic compression and pathological fractures . There is no effective treatment except kidney transplant. Prevention is through the use of high flux dialysers such as polysulfone or polyacrylonitrile (AN69) that enhance β_2M excretion [23].

Diagnosis of CKD – MBD

1. *Biochemical parameters*

PTH levels; The normal range of the intact PTH assay is 10-65pg/ml in normal individuals. While the recommended target range, (using the K/DOQI guideline) for serum iPTH for stage 3 is 35-70pg/ml, stage 4 , 70-110pg/ml and stage 5, 150-300pg/ml [8].

Biochemical serum markers of bone turnover; These includes Total alkaline phosphatase, osteocalcin, procollagen -1-propertide and parathormone which are all elevated in high turn over bone disease.

2. *Imaging*

There is increased osteoblastic activity with associated increased trabeculae bone volume which accounts for the sclerotic changes i.e. rugger jersey spine pattern seen on the X ray . Other features seen on spine and hand roetgenogram include; subperiosteal erosions which are best seen at the distal end of the phalanges, erosion of the distal extremities of the clavicles and at the sacroiliac joints. Mottled appearance are seen on the skull while expansile lytic lesions (Brown tumors) can be seen in severe osteitis fibrosis . Pseudofractures show up as wide radiolucent bands perpendicular to the bone long axis(e.g looser's zone) as seen in osteomalacia.

3. *Bone biopsy*

The gold standard for diagnosing renal osteodystrophy is by Jamshidi needle aided trans illiac bone biopsy after double labeling with two doses of tetracycline two weeks apart, prior to biopsy[24]. The KDIGO recommends that bone biopsies in patients with CKD should be further characterized bydetermining the bone turnover (histomorphometric), mineralization and volume as it provides information on type and severity of MBD[1, 23].

MANAGEMENT OF MBD – CKD

The goals of management are to maintaining blood levels of Calcium and Phosphorus to as close to normal; to prevent/ treat abnormally elevated levels of parathyroid hormone early; to prevent parathyroid hyperplasia; to prevent extra skeletal calcification and to avoid the over-suppression of PTH secretion by vitamin D_3.[23]

The various options for treating 2^O HPTH and hyperphosphatemia include; dietary phosphorus restriction, calcium and non-calcium based phosphate binders, calcitriol or other active Vitamin D analogues, calcimimetics and parathyroidectomy.

Controlling Serum Phosphorus

Foods such as diary products, nuts, certain vegetables, chocolates and colas should be restricted for patients with CKD in especially for those in stages 4 and 5 because they contain high phosphate levels.KDOQI guidelines recommends that dietary phosphorus intake be restricted to 800 – 1000mg especially if serum phosphate concentrations is >4.6mg/dl at stage 3 and 4 of CKD and > 5.5mg/dl(1.78mmol/L) in patients with CKD stage 5. Serum phosphates concentrations should also be monitored every month after the start of dietary phosphate restrictions.

Phosphate Binders

They combine with dietary phosphates in the gastrointestinal tract to form an insoluble complex that is excreted in the stool. Indications include; elevated phosphate concentration despite compliance with dietary phosphate restrictions and elevated blood PTH concentrations after dietary phosphorus restrictions.

TYPES

This could either be aluminium containing binders, calcium based binders, non calcium based binders or non calcium, non aluminium based binders

Aluminium Based Binders.

This forms insoluble and non absorbable aluminium phosphates which precipitate in the intestinal lumen. They are effective binders which are eliminated in the kidney and there is gradual tissue accumulation of absorbed Aluminium with resultant toxicity. They were previously used in the last 3 decades to treat renal osteodystrophy but went out of favour due to the side effects..These manifest majorly in the bone, skeletal muscle and the CNS, leading to low bone turn over osteomalacia and adynamic bone disease, refractory microcytic anemia, myopathies and dementia [25]. These binders are known to have comparable better phosphate control without any increase in the serum calcium levels.

K/DOQI guidelines recommend the use of Aluminium binders only in patients with serum phosphorus>7.0mg/dl(2.26mmol/l), i.e. one course short term therapy for less than 4 weeks only and to be replaced thereafter by other phosphate binders.

Calcium Based Phosphate Binders (Calcium carbonate/calcium acetate)

These include calcium carbonate, calcium acetate, calcium citrate and calcium ketoglutarate and they are often recommended as an initial therapy. Calcium carbonate has been widely used, although calcium acetate is a more effective phosphate binder as it dissolves in both acid and alkaline medium [26]. They are most effective when taken with meals as it binds dietary phosphorus and therefore leaves less free calcium available for absorption. There is an increased risk of hypercalcaemia and metastatic calcifications which are associated with cardiovascular mortality [10, 11, 27]. To avoid the above complications, the K/DOQI guidelines suggest that the total dose of elemental calcium (including dietary sources) should not exceed 2000mg.

Non calcium Based, Non aluminium Based Phosphorus Binders

Sevelamer Hydrochloride; This is a cross linked poly-allylamine hydrochloride exchange resin which binds phosphorus and releases chloride. They are moderate phosphorus binders[24]. Dosage is 800-1600mg with

major meals to a maximum of 4800mg/day.They reduce serum phosphates and calcium –phosphorus product with a potency almost equivalent to that observed with calcium acetate therapy but with less risk of hypercalcemia They also attenuate the progression of coronary calcification(a predictor of all cause mortality) as compared to calcium acetate in both the *Prevalent [treat to goal trials][28]* and *Incident [renagel in new dialysis]* trials[29,30].

They lower lipids and uric acid levels, reduce inflammation and oxidative stress, increases fetuin A levels and ultimately improving bone health. They are not selective for phosphorus ions only, as it can bind other negatively charged ions such as chloride and bicarbonates. Major side effect is reduction in serum bicarbonate level.

Lanthanum Carbonate

This is a relatively effective and practical non aluminium/non- calcium based phosphate binder. They are trivalent metallic cations with ability to chelate dietary phosphates. They bind phosphate effectively across the physiological pH range of the upper GIT hence has low systemic absorption. They also reduce PTH concentrations and calcium-phosphate product in CKD patients[31, 32]. They have no detrimental effects on calcium/vitamin D metabolism and are well tolerated however, commonly reported adverse effects are nausea,peripheral oedema and myalgia. The dose is 250-500mg (maximum of 1500mg) and effects are seen within 3 weeks of treatment.

Vitamin D

These could be classified as Vitamin D precursor, active vitamin D compounds and Vitamin D analogues ,. as first, second and third generation compounds or as nutritional vitamin D (ergocalciferol and cholecalciferol), vitamin D receptor activators (calcitriol, alphacalcidiol, doxercalciferol) and D – mimetics (paricalcitol, maxacalcitol).

Ergocalciferol (Vitamin D_2) is a vitamin D precursor. It requires hydroxylation within the liver to calcifediol and a second hydroxylation within the kidney to form active vitamin D compound. It is of limited value in the latter stages of Chronic Kidney Disease. Dose is 400 – 50,000I.U. orally or intravenously.

Calcitriol (1,25 dihydroxyvitamin D) is a first generation active Vitamin D. It does not require any further activation and has been shown to effectively suppress parathyroid hormone secretion. The dose is between 0.25 – 5mcg, orally daily or thrice a week.

Doxercalciferol(1α- dihydroxy vitamin D_2) is a second generation vitamin D analog, like all second generation analogues, they have a side chain modification of the vitamin D molecule to minimize calcium and phosphorus absorption, but still requires activation and conversion to its active form of 1α-25 dihydroxyvitamin D_2 in the liver. Dose is 5–20mcg (orally) or 2 – 8mcg (IV), thrice weekly[33].

Paricalcitol (19-nor- 1,25- dihydroxy vitamin D_2) is a third generation Vitamin D analog with a high affinity for the Vitamin D receptor. The chemical modification to the vitamin D ring structure makes paricalcitol have less calcaemic and phosphatemic effect than calcitriol. Dose is 1 – 4mcg thrice daily orally or 2.5 -15mcg intravenously thrice weekly[34, 35]. It could suppress PTH to dangerous levels.

Generally Vitamin D sterols reduce the transcription of the parathyroid hormone gene and hormone synthesis over a period of several days.

Calcimimetics (Cinacalcet HCl, AMG-073)

This is a novel approach to treating secondary hyperparathyroidism without raising serum calcium or using active Vitamin. D analogues. These agents mimick the effects of blood ionised calcium on the parathyroid. They also stimulate caR (calcium sensing receptors) found in the parathyroid and C thyroid glands as well as renal tubular cells, they reduce PTH secretion and also control hyperplasia[36]. Activation of this receptor by calcimimetics increases intracellular calcium concentration, which causes rapid reduction in PTH secretion(within a few hours after administration), serum phosphorus levels, and the calcium **x** Phosphorus product, which remain suppressed for up to 3 years[37]. Oral dose is 30-180mg once daily.

Parathyroid Intervention Therapy

This could either be surgical or medical. Surgical treatment is via parathyroidectomy while the medical treatment is by percutaneous direct injection therapy. Indications for parathyroidectomy include; persistently elevated intact PTH (<800pg/ml) which is associated with hypercalcemia and/or hyperphosphatemia despite medical management, mass on imaging > 0.5-1g, calciphylaxis (with increased iPTH), severe bone pains/ fractures in the presence of elevated intact PTH level and severe pruritus refractory to medical

management[33]. It could either be via subtotal parathyroidectomy or total parathyroidectomy in which there is no re-implantation of parathyroid tissue in the forearm as done in metastatic calcification while re-implantation is often performed in some other instances particularly to avoid hyperparathyroidism especially after renal transplantation[38,39].

Percutaneous ethanol injection therapy

This is an alternative procedure to surgical parathyroidectomy. Side effect is mainly recurrent laryngeal nerve injury. However this have been replaced by calcitriol preparation for percutaneous injection into parathyroid gland or percutaneous calcitriol analogue injection therapy (22-oxacalcitriol) which has shown suppressive effects on PTH level as well as reduction in the size of the enlarged glands [23].

REFERENCES

1. Moe S, Drueke T and Cunningham J *et al:* Definition, evaluation and classification of renal osteodystrophy: a position statement from kidney disease: Improving Global Outcomes (K/DIGO) Kidney Int. 2006; 69: 1945-1953.
2. Lucas RC: Form of late rickets associated with albuminuria, rickets of adolescents with albuminuria, rickets of adolescents lancet 1883:1; 993 - 1015.
3. Liu SH and Chu HI: Studies of calcium and phosphorus metabolism with special reference to pathogenesis and effects of dihydrotachysterol (ATIO) and iron. Medicine 1943; 22; 103-161.
4. Ward MK, Feest TG and Ellis HA *et al:* Osteomalacic dialysis osteodystrophy: Evidence for a water-borne aetiological agent, probably aluminium. Lancet 1978;1: 841-845.
5. Brown EM, Gamba G and Riccardi D *et al:* cloning and characterization of an extracellular Ca2+ sensing receptor from bovine parathyroid. Nature 1993; 366; 575 – 580.
6. Christakos S, Dhawan P and Liu Y *et al:* New insights into the mechanism of Vitamin D action. J cell biochem 2003; 88: 695 - 705.
7. Hruska KA, Mathew S and Lund RJ *et al:* The pathogenesis of vascular calcification in the chronic kidney disease mineral bone disorder. The link between the bone and the vasculature. Seminars in Nephrology. 2009; 29; 2: 156-165.
8. National Kidney Foundation. K/DOQI. Clinical practical guidelines for bone metabolism and disease in chronic kidney disease. Am J. Kidney Dis 2003; 42: 51 - 201.
9. Renal Association and Royal College of Physicians. Treatment of Patients with Renal Failure: Recommended Standards and Audit Measures. 3rd edn. London: 2002.
10. Kesterbaum B, Sampson JN and Rudser KD .Serum phosphate levels and mortality risk among people with chronic kidney disease. JASN 2005; 16(2): 520-528.
11. Block GA, Klassen SS and Lazarus JM. Mineral metabolism, mortality and morbidity in maintenance haemodialysis. J. Am .Soc.Nephrol.2004;15(8): 2208-2218.
12. Martin KJ, Olgard K and Coburn JW et al. Diagnosis, assessment and treatment of bone turnover abnormalities in renal osteodystrophy. Am J Kidney Dis.; 43(3); 55865.
13. Llach F and Fernandez E,. Overview of renal bone disease : causes of treatment failure, clinical observations, the changing pattern of bone lesions, and future therapeutic approach. Kidney International, 2003; 64; (Suppl 87): S113-S119.
14. Couttenye MM, D'haese PC, Verschoren WJ et al. low bone turnover in patients with renal failure. Kidney International ; 1996; 56:S70 -S76.
15. Sherrard DJ, hercz G and Segre G. The a plastic form of renal osteodystrophy. Nephrol Dial Transplant.1996;11(suppl 3) : 29-31.
16. D'Haese PC, Couttenye MM and De broe ME. Diagnosis and treatment of aluminium related bone disease. Nephrol Dial Transplant. 1996; 11(suppl 3): 74-79.
17. Odenigbo UC, Ijoma CK, Ulasi I, Udeh AC and Ibeh CC. The prevalence of radiological markers of renal osteodystrophy in patients with chronic renal failure in Enugu. Niger J. Clin Pract. 2006; 9: 147 – 152.

18. Onyemekehia R. Renal osteodystrophy in Benin. A dissertation submitted to the National Postgraduate Medical College of Nigeria. Faculty of Internal Medicine, November, 2004.

19. Sanusi .AA, Arogundade FA, Oginni A and Akinsola A. The prevalence and pattern of Renal Bone Disease in End Stage Renal Disease patients in Ile-Ife, Nigeria. West Afr J med. 2010;29(2): 75-80.

20. Ketteler M, Gross ML and Ritz E: Calcification and cardiovascular problems in renal failure. Kidney Int. 2005; 94: S120 – 127.

21. De Fransisco AL. Secondary hyperparathyroidism: Review of the disease and its treatment. Clin Ther 2004; 26 (12): 1976 – 1993.

22. Liach F. The evolving Clinical Features of Calciphylaxis. Kidney Int. Suppl 2003 Jun (85); S122 - 124.

23. El-Kishawi and El-Nahas. Renal Osteodystrophy: Review of the Disease and its Treatment. Saudi J Kid Dis Transplant. 2006; 17 (3): 373 - 382.

24. Pecovnik Balon B and Bren A. Bone histomorphometry is still the golden standard for diagnosing renal osteodystrophy. Clin Nephrol 2000; 54 (6): 463 – 469.

25. Saluski IB, Foley J and Nelson P *et al*. Aluminium accumulation during treatment with aluminium hydroxide and dialysis in children and young adults with chronic renal disease.N Engl J Med 1991;324(8): 527-31.

26. Qunibi WY and Nolan CR.Treatment of hyperphosphatemia in patients with chronic kidney disease on maintenance hemodialysis: Result of the CARE study.kidney Int. 2004; 90(suppl): S33-S38.

27. Saluski IB and Goodman WG; Cardiovascular calcification in end stage renal disease.Nephrol Dial Transplant 2002: 17: 336- 339.

28. Chertow GM, Burke SK and Raggi P. Sevelamer attenuates the progression of coronary and aortic calcification in hemodialysis patients. Am J Kidney dis. 1999; 33: 694 – 701.

29. Chertow GM, Burke SK and Lazarus JM *et al*. Poly-allyamine hydrochloride (Renagel): A non calcemic phosphate binder for the treatment of hyperphosphatemia in chronic renal failure. Am J Kidney dis. 1997, 29 (1): 66 - 71.

30. Block GA, Spiegel DM and Ehrlich J *et al*. Effects of sevelamer and calcium on coronary artery calcification in patients new to hemodialysis. Kidney Int. 2005; 68: 1815 - 1824.

31. Chiang SS, Chen JB and Yang WC: Lanthanum Carbonate (Fosrenol) efficacy and tolerability in the treatment of hyperphosphatemic patients with end-stage renal disease. Clinical nephrology. 2005; 63 (6): 461 – 470.

32. Malluche HH, Siami GA and Swanepoel C *et al:* Improvements in renal osteodystrophy in patients treated with lanthanum carbonate for two years. Clin. Nephrol. 2008; 70: 284 – 295.

33. Friedman EA: Consequences and management of hyperphosphatemia in patients with renal insufficiency. kidney international 2005; (67): S1-S7.

34. Akizawa T, Shiizaki K and Hatamura I *et al:* New Strategies for the Treatment of Secondary Hyperparathyroidsm. Am J Kidney Dis 2003; 41(Suppl 1): S100 - 3.

35. Slatopolsky E, Finch J and Brown A: New Vitamin D Analogs. Kid Int. 2003; 63; 283 – S287.

36. Goodman WG, Hlakik GA and Turner SA *et al:* The Calcimimetic Agent AMG 073 lowers plasma parathyroid hormone levels in hemodialysis patients with secondary hyperparathyroidism. JASN 2002; 13 (4): 1017 -1024.

37. Urena Torres P: Clinical Experience with Cinacalcet HCL. NDT 2004; 19 (5); 27 - 33.

38. De Fransisco AM, Ellis HA and Owen JP *et al:* Parathyroidectomy in Chronic Renal Failure. Q J Med 1985; 55: 289-315.

39. Wahab MA and Kanhal F. Calcification after parathyroidectomy in chronic renal failure. Saudi Journal Kid Dis Transplant. 2008; 19(5) : 854-860.

Psychiatric Manifestation in a Child with Uraemia: A Case Report

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INTRODUCTION

Adequate renal function is required for normal mental function. Hence deterioration in renal function could result in neuropsychiatric effects ranging from subtle, non-specific changes to gross distinct abnormalities. [1] When there is a severe reduction in glomerular filtration rate (GFR) either acutely or chronically, uraemic encephalopathy occurs [2]. The rate at which uraemic encephalopathy develops is proportional to the rate at which GFR falls. Acute renal failure precipitate a rapid and florid failure of mentation, whereas in chronic renal failure, it slowly evolves. [3] All aspects of psychic can be affected including cognition, psychomotor activity and personality. [4] However, the overt personality disorder manifesting in neuropsychiatric disorder are not often seen. The few that have been reported have been in adult. We therefore report a 15 year old child who presented with features suggestive of nephrotic syndrome which later progressed to acute renal failure and subsequently developed neuropsychiatric symptoms in the course of the uraemia.

Keywords: *Uraemia, mania, African child*

CASE REPORT

A 15 year old Nigerian boy was referred from a secondary health facility with 3 month history of recurrent body swelling. There was initial diurnal variation but became constant throughout the day two weeks to presentation. There was no reduction in urine output and no haematuria. There was preceding history of scorpion sting twice three days before onset of the most recent swelling. No history of using mercury containing soap, preceding sore throat or skin rash. He was taken to the referring hospital for the first two episodes only to recur again for the third time.

Examination revealed anasarca, he was not pale or jaundiced. The blood pressure was 130/ 84mmHg. There was massive ascites with no renal angle tenderness. Urinalysis revealed proteinuria of 4+ and blood was negative. Serum cholesterol was 9.5 mol/l, serum protein was 49g/l, serum albumin 13g/l, urea 8.2mmol/l, creatinine 110µmol/l potassium 3.1 mmol/l, sodium 133mmol/l, calcium 1.5 mmol/l, phosphate 2.1mmol/l, PCV -23%, 24hour urinary protein 2.29g/day. Abdominal ultrasound revealed that both kidneys were normal in size and outline with slight increase in echo and moderate calyceal fullness suggestive of grade II parenchyma disease.

An assessment of nephrotic syndrome was made and he was commenced on parenteral frusemide, hydrochlorothiazide and spironolactone. The edema resolved only to recur again with similar fluctuation in urine output between 0.6-1.9ml/kg/hr during the first 40 days of admission.

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Oral prednisolone (60mg/m^2) was commenced on the 27th day on admission when oedema had subsided significantly. It was stopped on the 30th day on admission when a resurgence of oedema was noticed again accompanied with further reduction in urine output.

On the 35th day, the blood pressure was noticed to be rising and blood pressure of 120/100 mmHg was recorded. On the same day, he started to talk to self, talk excessively and sing loudly on the ward. His sleep became grossly inadequate, was aggressive and hyperactive and had to be restrained physically. There was no episode of convulsion or loss of consciousness. He had no past episode of mental illness and there was no family history of mental illness.

Mental state examination revealed a boy who was conscious and alert, restless and uncooperative. He had irritable mood and affect, was fully oriented to time, place and person but attention and concentration was poor. His uncooperative attitude made it difficult to ascertain presence of perceptual abnormality but his behaviour was suggestive of someone having visual hallucination. There was marked pressure of speech and it was difficult to interrupt his conversation. He occasionally had flight of ideas which made it difficult to know what he was saying. Low dose haloperidol (antipsychotic drug) was added to his medication, to which he initially responded positively.

Serum creatinine rose to 247µmol/l and urea was 11.9 mmol/l, potassium 2.3 mmol/l and sodium 131mmol/l. There was also recurrence of oedema requiring plasma transfusion. Blood pressure became 140/100mmHg. Hypertensive/uraemic encephalopathy were suspected. He was therefore commenced on captopril. Subsequently talkativeness, aggressive behavior and insomnia continued to worsen in spite of haloperidol, benzhexol and diazepam. The urine output declined to as low as 0.3ml/kg/hr on the 46th day (about 10 days after commencement of irrational talk). Meanwhile, proteinuria remained between 3 and 4+ since admission. In view of the declining urinary output and refractory oedema despite the administration of diuretics and plasma transfusion, renal replacement therapy was commenced on the 50th day. There was subsequent improvement in urine output to as high as 4.1 ml/kg /hr on the 60th day after 4 sessions of dialysis. However, some oedema still remained and the aggressive behaviour persisted. Proteinuria reduced to 2+ and creatinine reduced to 121µmol/l. The patient was discharged home on the 68th day, when they could no longer afford further dialysis on captopril, nifedipine, diuretic and antipsychotic drugs. On follow up about three months after discharge, he was withdrawn and sluggish in responding to questions. Oedema had reduced but with some residual leg oedema up to the distal third of both legs. Blood pressure was 130/90mmHg. At the 5th month post discharge all the psychotic symptoms had disappeared and he was no longer withdrawn, responded normally to questions and oedema had fully subsided. Proteinuria was 2+ and BP was 120/70mmHg. All the diuretics were stopped and he was placed on only lisinopril 10mg daily. Serum creatinine was 42 µmol/l, urea 4.2 mmol/l, potassium was 4.2 mmol/l, and sodium was 137 mmol/l. Serum protein was 57g/l, albumin was 28 g/l. He remains stable on anti-psychotic medication and still being followed up in the psychiatric outpatient clinic.

DISCUSSIONS

The clinical feature of this case did satisfy the criteria for mania due to a general medical condition (uremia) or secondary mania. [6] Uraemic encephalopathy was suspected, but the absence of confusion and disorientation made it unlikely. [7] Furthermore, the psychiatric symptoms predated the commencement of dialysis thereby ruling out dialysis disequilibrium syndrome. For similar reasons, dialysis dementia which is a known cause of secondary mania was also out of consideration. [8] The possibility that the commencement of steroid could have triggered the psychiatric symptom was also not likely because it had been discontinued 5days before the manifestation of those symptoms and it was administered at the standard dose for just three days after which it was discontinued due to resurgence of oedema. A familial link could also not be established because of absent family and previous history of psychosis in this child. There was no family separation as the father and mother were together providing support for him. The extended family also provided ready financial support for the dialysis sessions received.

Manic manifestations found in this patient was in keeping with the report of other workers even though those reports were in adults who had all commenced dialysis. [8 - 11] Our patient had not

commnenced dialysis before the symptoms were manifested and he is the first child known to us with that manifestation.

There are several aetiological factors that should be considered in the pathogenesis of this organic manic syndrome. Since the disorder was precipitated by renal failure due to a glomerulopathy, it has been suggested that the metabolic derangement of uraemia include a factor present within the serum that reduces the activity of the sodium-potassium pump within neuronal membranes leading indirectly to increased neurotransmitter release and the production of mania [9]

The aetiology has also been linked to the reduced clearance of neurotoxins such as ammonia, middle molecular weight toxins (which are not effectively removed by haemodialysis (HD), but better removed by peritoneal dialysis) and porphyrins. Psychiatric disturbances are also strongly correlated with hyperparathyroidism which is a common occurrence in renal failure. It is mediated in part by hyperphosphataemia that attends renal failure and decreased degradation of parathormone [12- 13] The incidence of psychiatric disturbance in patients with primary hyperparathyroidism has been estimated to range from 3-67% depending on the type of findings which can range from mild apathy to personality changes to florid psychosis[14]In secondary hyperparathyroidism, the psychiatric manifestation is qualitatively similar. [15]

As found by other workers neuropsychiatric manifestations of uraemia are possible and plausible in view of the uraemic toxin which could be an irritant to the brain, hence the uraemia is likely to be the trigger for the manic illness in our patient. What was however surprising to us was the failure of the neuropsychiatric manifestation to resolve after the correction of the uraemia with the series of dialysis session. It may be possible that the dialysis compounded the problem. It may also be possible that dialysis does not completely clear all the neurotoxin since many molecules are involved [16] and some of these molecules are susceptible to differential clearance depending on the mode of dialysis with peritoneal dialysis (PD) generally been regarded as more effective. Indeed the type of dialysis used affects the neuropsychiatric presentation. Given the apparent susceptibility of the middle molecules to clearance by PD, it remains an important potential cause of psychiatric illness. Another possibility is the fact that it will take time for the after effect of the toxin to wear out like it occurs in tetanus. In fact the psychiatric symptoms in our patient continued unabated after the few sessions of dialysis he had and needed continuous antipsychotic therapy until it fully subsided about 5 months after discharge. He is still been managed by the Psychiatrist and on antipsychotic medication.

The serum level of creatinine in our patient though elevated did not seem significantly high enough to produce severe problem such as a neuropsychiatric manifestation. This raises the question of at what level of uraemia should one anticipate neuropsychiatric manifestation.

Finally, uraemia is a stressor, needing multiplicity of interventions some of which have attendant side effects and could run a chronic course which is enough to cause a psychological disturbance. The totality of the effect of uraemia is therefore capable of producing neuropsychologic imbalance which could cause neuropsychiatric manifestation which may have been the case in our patient. We recommend an early neuropsychologic evaluation for all children with uraemia.

REFERENCES

1. Brown TM and Brown RLS. Neuropsychiatric consequences of renal failure. Psychosomatics 1995; 36(3): 244-253.
2. Teschan P and Arieff A. Uremic and dialysis encephalopathies in cerebral energy metabolism and metabolic encepaphalopathy, edited by McCondlers D, New York, plenum 1985.
3. Locke S, Merill J and Tyler H. Neurological complications of acute uremia. Arch Intern Med 1961; 108: 75-86.
4. Raskin N and Fishman R. Neurological disorders in renal failure (first of two parts).New Engl J. Med 1976; 294:143-148.
5. American Psychiatric Association Diagnostic and Statistical Manual of Mental disorders 4th edition (DSM-1V). Washington D C : APA, 1994.
6. Krauthammer C and Klerman GL. Secondarymania.Manicsyndromes associated with antecedent physical illness

or drugs. Archives of General Psychiatry 1978; 35: 1333-1339.

7. Marshall JR. Neuropsychiatric aspects of renal failure. J Clin Psych 1979; 40: 81-85.

8. Jack RA, Rivers-Bulkeley NT and Rabin PL. Secondary mania as a presentation of progressive dialysis encephalopathy. J Nervous and Mental Disease 1983; 171: 193-195.

9. El-mallakh RS, Shrader SA and Widger E. Mania as a manifestation of end stage renal disease. J Nervous and Mental Disease1987; 175:243-245.

10. Cooper AJ.Hypomanic psychosis precipitated by haemodialysis. Comprehensive Psychiatry 1967; 8: 168-174.

11. Thomas CS, Neale TJ. Organic manic syndrome associated with advanced uraemia due to polycystic kidney disease. Br J Psychiatr 1991;158: 119-121.

12. Rodriguez M, Felsenfeld A and Llach F. Calcemic response to parathyroid hormone in renal failure: role of calcitrol and the effect of parathroidectomy. Kidney Int 1991; 40: 1063-1068.

13. Klahr S and Slatopolsky E. Toxicity of parathyroid hormone in uremia. Annual Review of Medicine 1986; 37: 71-78.

14. Mallet L, Bilezkian J and Health D et al.Primary hyperparathyroidism: clinical and biochemical features. Medicine 1974; 53: 127-146.

15. Fraser CL, Arieff AI. Nervous system complications in uremia. J Clin Psychol 1982; 38(3): 490-496.

16. Gebril M, Weinkove C, Ead R, McDonald K and Morton R. Plasma porphyrins in chronic renal failure. Nephron 1990; 55: 159-163.

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ABS-OR-1001

THE EVALUATION OF LEAD EXPOSURE, BLOOD PRESSURE AND RENAL FUNCTION INDICES IN NIGERIANS WITH OCCUPATIONAL LEAD EXPOSURE

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Background: Occupational and environmental lead exposure is acknowledged to result in renal function impairment and hypertension.

Aims and objectives: The objective of this study was to evaluate the association between lead exposure, blood pressure and renal function in lead exposed workers in Port Harcourt Nigeria.

Materials and methods: A prospective cross sectional comparative study of 190 adult subjects with occupational lead exposure and 80 matched controls was performed in Port Harcourt. Blood lead was used as the biomarker of lead exposure. Renal function indices, systolic and diastolic blood pressures and other demographic and lifestyle variables were also measured.

Results: Occupationally lead exposed subjects had higher mean blood lead 50.37±24.58 ug/dl, than controls 41.40±26.85 ug/dl (p= 0.008). There was a significant difference in the mean systolic blood pressure of subjects and controls 118.49 (14.67) mmHg vs. 113.62 (11.31) mmHg (p = 0.008). No difference was observed in the mean diastolic blood pressure of subjects and controls 74.64 (10.98) mmHg vs. 73.10 (7.47) mmHg (p= .285). A higher proportion of subjects had systolic and diastolic pressure > 140mmHg and >90mmHg compared to controls 9.47% vs. 1.25% and 10.51% vs. 2.54%, with (p = .016), RR=1.38<1.21<RR<1.58> and (p = .028), RR=1.33<1.13<RR<1.55> respectively. The mean values of serum urea, serum creatinine and serum uric acid were significantly higher in study subjects compared to controls 3.06±0.81 mmol/L vs. 2.7±0.84 mmol/L (p = 0.002), 87.2±14.30 umol/L vs. 80.68±14.70 umol/L (p = 0.001) and 271.93±91.18 umol/L vs. 231.1(62.70) umol/L (p = 0.000) respectively. Creatinine clearance was significantly lower in study subjects compared to controls 98.86±21.26 ml/min/1.72m^2 vs.108.18±25.16 ml/min/1.72m^2 (p = 0.002). There was no significant difference in urine albumin excretion. Blood lead correlated positively only with blood urea [r = .031, r2 = .017, p = .031] and negatively [r = -.144, r2 = .021, p = .018] with serum phosphate. With simple linear regression analysis blood lead level was significantly and positively associated with blood urea [y=.0043x + 2.76, r2 = .017, p = .031] and significantly negatively associated with serum phosphate [y=.007x + 1.42, r2 = .021, p = .018]. The duration of occupation correlated negatively with GFR [-.126, p =.038] and positively SBP [.256, p = .000]. In linear regression modelling the duration of occupation was associated with GFR, SBP and DBP, this effect was modified only by age after adjustment.

Conclusion: The results of this study indicate a significantly higher risk of renal function impairment and elevated blood pressure among lead exposed workers in compared to controls. Longer duration of exposure was significantly associated with renal function impairment and elevated blood pressure, with age as a

predictor of this association. Serum urea was the most sensitive index of lead induced renal function impairment in this study with low serum phosphate as a significant predictor of lead induced increase in serum urea levels. It is advocated that more attention be given to the role of environmental and occupational nephrotoxins like lead in CKD prevention in Nigeria.

Keywords: LEAD EXPOSURE; BLOOD PRESSURE, RENAL FUNCTION INDICES

ABS-OR-1002

THE ASSOCIATION OF URIC ACID AND RENAL FUNCTION INDICES AMONG LEAD EXPOSED WORKERS IN PORT HARCOURT NIGERIA

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Background: The presence of hyperuriceamia and renal function impairment, especially in the absence urate stone formation is strongly suggestive of lead nephropathy.

Aims and objectives: To determine the relationship between serum uric acid and renal function indices in lead exposed workers.

Materials and methods: A cross sectional comparative study of 190 adult subjects with occupational lead exposure and 80 matched controls was performed in Port Harcourt, South-south Nigeria. Blood lead was used as the biomarker of lead exposure. Serum uric acid and other renal function indices measured.

Results: Occupationally lead exposed subjects had higher mean blood lead 50.37±24.58 ug/dl, than controls 41.40±26.85 ug/dl (p= 0.008). The mean values of serum urea, serum creatinine and serum uric acid were significantly higher in study subjects compared to controls 3.06±0.81 mmol/L vs. 2.7±0.84 mmol/L (p = 0.002), 87.2±14.30 umol/L vs. 80.68±14.70 umol/L (p = 0.001) and 271.93±91.18 umol/L vs. 231.1(62.70) umol/L (p = 0.000) respectively. Creatinine clearance was significantly lower in study subjects compared to controls 98.86±21.26 ml/min/1.72m^2 vs.108.18±25.16 ml/min/1.72m^2 (p = 0.002). Serum uric acid correlated positively with serum creatinine [r=.134, p=028] and negatively with GFR = [r=-.151, p=.013]. In linear regression modelling uric acid was associated with reducing GFR [r= .151, r2 = 023, B= -.151, p = .013] and increasing serum creatinine [r= .134, r2 = .018, B= .134, p = .028] .Blood lead was not associated with uric acid in single model and after adjusting for other study variables.

Conclusion: The results imply that subjects with occupational lead exposure comprise a susceptible population for hyperuricaemia and renal impairment. The association of increased uric acid with renal function impairment in this study supports the role of hyperuriceamia as a consequence and pathogenetic mechanism of renal impairment in lead exposed subjects.

Keywords: OCCUPATIONAL LEAD EXPOSURE; HYPERURICAEMIA; RENAL FUNCTION IMPAIRMENT.

ABS-OR -1003

PREVALENCE OF TOXIC NEPHROPATHY IN AN OIL PRODUCING COMMUNITY IN DELTA STATE, NIGERIA

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Introduction: Toxic nephropathy is a common cause of end stage renal disease that arises as a result of exposure to nephrotoxic chemicals, drugs, heavy metals and hydrocarbons.
Objective: To document the prevalence of Toxic Nephropathy particularly due to hydrocarbon from crude oil and natural gas.

Methods: 64 subjects from a rural community with about 10 oil wells and natural gas plant were recruited for the study. Following a general physical examination, blood and urine were collected for fasting blood sugar, urea, creatinine, serum electrolytes and urinalysis.
Results: 6 (9.4%) of the subjects had elevated serum urea and creatinine while 23 (36%) had significant proteinuria. 5 (3%) had elevated fasting blood and only 2 of the 5(40%) of these subjects had glycosuria. 7 (11%) had elevated blood pressure. None of the hypertensive or Diabetic subjects had proteinuria or elevated serum urea and creatinine.

Discussion: Long-term hydrocarbon exposure may predispose individuals to the development of several different types of renal diseases. The community where this study was done has many oil wells and a gas plant with previous oil spillages and gas explosion hence the population has over time been exposed to hydrocarbon. From our study, none of the hypertensive or diabetic group had proteinuria or elevated serum urea and creatinine. This may suggest that there may be other causes other than the common medical cause of chronic renal failure. Also a significant proportion of the population though had normal serum creatinine, had proteinuria. This corroborates other studies which reported increased proteinuria but normal serum creatinine in subjects exposed to petroleum based mineral oil.

Conclusion: In conclusion, toxic nephropathy is associated with long-term hydrocarbon exposure and may be prevalent in oil producing communities. However further studies is required to elucidate this.

ABS-OR-1004

AKI IN ADULT NIGERIANS: A SINGLE CENTRE EXPERIENCE

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Background: Acute Kidney Injury (AKI) is an abrupt and sustained decrease in Glomerular Filtration Rate (GFR), urine output or both. It is associated with significant morbidity and mortality among hospitalised patients. Despite the enormity of the problem, data is lacking regarding the epidemiology of AKI in developing countries.

Aims and objectives: To determine the incidence of AKI in UBTH and to study the aetiological pattern, clinical presentation, complications and outcome of the disease; and to determine the predictor of poor outcome among cases studied.

Materials and Method: The case records of all case ok AKI admitted into the medical wards from August 2007- September 2009 were reviewed. Information on the sociodemographic data, clinical features, laboratory findings and management modalities were obtained.

Results: There were 2431 Medical admissions during the study period and of this there were 33 cases of AKI with an incidence rate of 1.36%. 25 case records were reviewed in detail.Patients were aged 18-95 years with a mean age of 37.4±18.6 years and were made up of 12(48%) males.
The commonest presenting features were fever (72%), leg swelling (56%), vomiting (52%), oliguria (52%) and facial swelling (48%). 45% were hypertensive , 20% confused , 12% unconscious and 8% had a pericardial rub. Twenty (80%) patients had proteinuria and 22 (88%) were anaemic. The commonest cause of AKI was Sepsis (40%). Twelve 12 (48%) patients had haemodialysis. Mortality rate was 36%. Lack of dialysis was associated with poor outcome.

Conclusion: AKI is a treatable cause of mortality. Late presentation and lack of access to dialysis due to financial constraints are major difficulties encountered.

ABS-OR-1005

PATTERNS OF LEFT VENTRICULAR HYPERTROPHY AND GEOMETRY IN NEWLY DIAGNOSED HYPERTENSIVE ADULTS IN NORTHERN NIGERIANS

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Background: In hypertensives, left ventricular hypertrophy predicts increased cardiovascular morbidity and mortality. Adding to this burden is abnormality of left ventricular (LV) geometry. Knowledge of the left ventricular geometric patterns in our newly diagnosed hypertensives may have some prognostic significance.

Methods: One hundred (100) newly diagnosed hypertensives (61males and 39 females) and 78 normotensives (46 males and 32 females) were recruited for the study. All were clinically evaluated and an echocardiographic examination performed.

Results: Mean ages for the study subjects and controls were 51.40 ±11.60 and 51.50 ±11.50 years respectively (P = 0.47). Only 24% of the hypertensives had normal geometry with 76% being abnormal. Normal geometry was found in 63% of the controls with 37% being abnormal. Statistical significance was noticed when the geometric patterns of the hypertensive and controls were compared (p value < 0.001).

Conclusion: This study showed that only 24% of our hypertensives had normal LV geometric pattern at diagnosis while over 35 percent of the controls had abnormal geometry. Early diagnosis and aggressive treatment to control hypertension should be taken with all seriousness.

Keywords: HYPERTENSION, LEFT VENTRICULAR HYPERTROPHY, GEOMETRY, ADULTS, NIGERIANS

ABS-OR-1006

PATTERN OF ACUTE RENAL FAILURE IN ILORIN: A REVISIT

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Objective: Acute renal failure is a common cause of morbidity and mortality in Nigeria. The early detection and prompt treatment of acute insults to the kidneys may prevent renal failure which is capital intensive in its management. Majority of Nigerians cannot afford the cost of renal replacement therapy which is available but expensive. There is no renal replacement therapy subsidy in Nigeria and the current NHIS is blank on renal replacement therapy. These underscore the need for some form of preventive nephrology in order to reduce and possibly avoid renal failure. In line with the foregoing, a 19 year (January 1990- December 2008) retrospective appraisal of causes, management and outcome of acute renal failure was undertaken. This study is a revisit as a similar study was carried out about a decade ago in our centre.

Methods: All the patients that met the criteria for ARF and presented primarily to the nephrology unit or were referred to the unit from other departments of UITH for intervention were studied. Data was analysed using SPSS version 16.

Results: A total of 113 patients (52males and 61 females), age range between 3-69 years with mean of 28.3 were reviewed. About 80.5% of the patients were less than 40 years of age with male to female ratio of 1:1.2 and mean ages of 27.3 and 29.1 years respectively. Eight six (76.1%) patients were oliguric at presentation. Unusual weakness, altered sensorium, vomiting and hiccups were presents in 92%, 86.7%, 37.1% and 27.4% respectively. Severe anaemia that necessitated blood transfusion was present in 48 cases (42.4%). The main aetiological factors were septicaemia (36.2%), severe gastroenteritis (22.1%), AGN (9.7%), Drug induced (7%) and ante/post partum haemorrhage (6.1%). Obstructive uropathy, septic abortion, acute pyelonephritis, intravascular haemolysis and holy green water constituted 5.3%, 4.4%, 3.5%, 2.6% and 0.8% respectively while 1.7% were unknown. Sixty three patients were managed conservatively with 62% mortality while 33 and 9 patients had haemodialysis and peritoneal dialysis with mortality rates of 15% and 67% respectively. The important prognostic factors identified were extremes of age, severe infection, late presentation, delayed intervention therapy and underlying/concurrent medical illness. Major factors that influenced mode of therapy were severity of ARF and financial constraints. Haemodialysis seems to be the preferred method of renal replacement therapy as it was associated with better outcome.

Conclusion: There is an improvement in mortality from ARF in the last decade as a previous study in our centre revealed mortality rate of 31% and 64% among haemodialysed and conservatively managed patients. Majority of the patients were below 40 years of age and the leading aetiological factors still remain preventable and treatable conditions. Haemodialysis remains the most cost effective modality of treatment for severe ARF in our environment.

ABS-OR-1007

ACUTE KIDNEY INJURY: A REVIEW OF THE CAUSES, SEVERITY AND OUTCOME IN ILE-IFE, NIGERIA

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Background: Acute kidney Injury (AKI), defined as an abrupt deterioration in kidney function is associated with high mortality even in developed countries. A new staging was recently introduced by the AKI network.

Aims and Objectives:

1. To assess the magnitude, causes, severity and outcome of AKI in our setting
2. To determine the relationship between the stage (severity) and mortality

Patients and Methods: We retrospectively reviewed case records of patients treated for AKI over a 2 – year period (2008-2009). Patients with evidence of CKD were excluded. Information on socio-demographic data, precipitating factors, investigations, treatment and outcome were collated and analyzed.

Results: Thirty-two (32) patients treated within the period met the inclusion criteria. The age ranged from 15 to 75years with a mean (± SD) of 38.5 (± 15.54). More females were affected with a percentage of 65.6% and a M:F ratio of 1:1.9. The major precipitating factors identified were sepsis, hypovolaemia and nephrotoxins. Twenty six (83.9%) presented with oliguria and 5(16.1%) were non-oliguric, 29(93.5%) of the patients presented in AKI stage 3 while 2(6.5%) in stage 2. The biochemical parameters at presentation revealed serum urea, creatinine , sodium, potassium and bicarbonate of 30.3mmol/l , 768.24µmol/l, 130.13mmol/l, 4.5mmol/l and 19.17mmol/l respectively. 19 patients (61.3%) survived while 12(38.7%) died. 90.6% of the patients had Haemodialysis. Age of patients was found to significantly influence outcome ($p = 0.025$) while AKI staging, gender, number of sessions of Haemodialysis did not influence outcome.

Conclusion: Oliguric AKI (very severe) remained the commonest presentation with attendant high mortality. Age of patients above 60 years predicts poor outcome.

ABS-OR-1008

SICKLE CELL NEPHROPATHY IN CHILDREN SEEN AT UITH ILORIN

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Introduction: Sickle cell nephropathy (SCN) is an anticipated complication in children with sickle cell disease especially in the second decade of life but it is seldom detected early except they present with clinical features such as haematuria, enuresis or oedema. The spectrum of sickle cell nephropathy varies from hyposthenuria to nephrotic syndrome. It is known to occur in both sexes and varies in severity.

Objective: To review the cases of sickle cell nephropathy seen over the last 14 years (1995-2009) at the Paediatric Nephrology clinic of UITH.

Methodology: The five cases of SCN seen during the period were analyzed for age, sex, and renal manifestations.

Results: The age range of the children was 9-15years with a mean of 11years. Four of the five patients were females, with one male. Three of the four females presented with features suggestive of nephrotic syndrome while the other one had gross haematuria which resolved within 24 hours. The only male had enuresis. The NS in one of the patients progressed to end stage renal disease requiring renal replacement therapy

Conclusion: Children with sickle cell disease should be screened for renal complications especially from the late first decade of life. This will help in the early detection of renal disorder that could lead to chronic kidney disease. It is also suspected that the severe forms of SCN such as NS may have a predilection for the female gender. A more extensive study is needed to test the veracity of this observation.

ABS-OR-1009

CHRONIC KIDNEY DISEASE:PATTERN AND PREDICTORS OF PROGRESSION IN CONSERVATIVELY TREATED PATIENTS

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Background: Chronic Kidney Disease (CKD) is a progressive disease with majority terminating in end-stage kidney disease with associated significant morbidity and mortality except renal replacement therapy is instituted optimally. Pattern and predictors of CKD progression in conservatively managed patients who were on regular treatment and long-term follow-up have not been adequately described.

Aim: To determine the rate of progression of treated Pre-renal replacement therapy CKD as measured by annual decline in eGFR and to identify the determinants of the progression.

Method: This is a retrospective cohort study of three hundred and ten CKD patients who were managed conservatively for minimum of $4^1/_2$ years at the CKD clinic of Sheffield Kidney Institute in UK. Data were obtained from the computerized proton database of the hospital. Patients with early stage 3 CKD (GFR of 40-60ml/min) who have not received kidney transplant or commenced dialysis were recruited for the study. Initial and Mean-follow up data were obtained for systolic blood pressure (SBP), diastolic blood pressure (DBP), Mean arterial pressure (MAP), eGFR, 24hour urinary protein (24hp), haemoglobin (Hb), serum albumin (ALB), bicarbonate (HCO3), parathyroid hormone (PTH), phosphate (P04) and calcium Phosphate product (CAP). CKD progression was defined as mean eGFR decline of e"2ml/min/year. SPSS version 13 was used to analyze the data. Variables with non-parametric distribution were log-transformed and the results used in the analysis. Correlation and regression models were used to determine predictors of progression of CKD.

Results: The mean age of the patient was 58.59±14.69years and 191(61.6%) were males. Mean duration of follow-up was 7.49±1.80years. Median annual decline in eGFR was 1.62ml/m/year (range: -2.3 to 6.8ml/m/year), showing progressors (≥2ml/m/year) in 132(42.6%) cases, non progressors (0 to 2ml/m/year) in 150(48.4%) cases and improvers (-2.3 – 0ml/m/year) in 28 (9%) cases. HCO3, ALB and Hb correlated negatively with progression ($r = -0.366$, $p = 0.000$; $r = -0.233$, $p = 0.000$; and $r = -0.289$, $p = 0.000$ respectively) while 24hp, PTH, PO4 and CAP correlated positively with progression ($r = 0.320$, $p = 0.000$; $r = 0.391$, $p = 0.000$; $r = 0.392$, $p = 0.000$; $r = 0.267$, $p = 0.000$). Age; SBP, DBP and MAP did not correlate with progression. Stepwise linear regression showed that P04 was the best independent predictor of progression ($r = 0.573$, $r^2 = 0.322$, P= 0.000) followed by PO4 and HCO3($r = -0.470$, $r^2 = 0.401$, P(PO4) = 0.000, P(HCO3) = 0.000). PO4, HCO3 and PTH were the next predictors accepted by the model ($r = 0.376$, $r^2 = 0.422$, P(PO4) = 0.000, P(HCO3) = 0.002 and P(PTH) = 0.046). 24hp > 0.5g also predicts progression but in small number of patients ($r = 0.237$, $r^2 = 0.056$, P= 0.005).

Conclusion: Significant proportion of CKD is progressive even with treatment although kidney function may improve in small number of patients. With adequate control of blood pressure and proteinuria which are potent predictors of CKD progression, hyperphosphatemia, acidosis and hyperparathyroidism play significant role in determining further progression of CKD.

ABS-OR-1010

CHARACTERISTICS OF PATIENTS ATTENDING DIALYSIS UNIT OF AHMADU BELLO UNIVERSITY TEACHING HOSPITAL, ZARIA

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Background: Ahmadu Bello University Zaria has been offering care for renal patients during the last 3 decades and up to the 1990's its catchment area covered the entire much of the northern Nigeria with a population of over 50 million people. However, haemodialysis services only became available late 2005.

Methodology: This is retrospective study of over a three-year period from january 2006 to january 2009. A total number of 120 patients were studied. Statistical Package for Social Sciences (SPSS) computer version 15.0 was used to analyze the data.

Results: There were 80 males (66.7%) and 40 females (33.3%). Age ranged between 15 to 75 years with mean age of 40.32 15.23. The indication for dialysis among these patients were as follows: chronic glomerulonephritis 56 (46.7%), with mean age of 28.40 ; hypertension 40 (33.3%), mean age 51.13 8.20; acute renal failure 9 (7.5%) with mean age 31.80; obstructive uropathy 8 (6.7%) with mean age 66.63; diabetic nephropathy 5 (4.2%), with mean age 57.60; others 2 (1.7%),with mean age 48.0 The mortality was observed in 48 (40%), while 61 (50.8%), were lost to follow up, 11(9.2%) were discharged. Access used were femoral 99 (82.5%), arterio-venous fistula 14(11.7%), and jugular 7(5.8%). Only two (1.7%) of the patients were transplanted.

Conclusion: Chronic glomerulonephritis is the commonest cause of ESRD and indication for dialysis ,followed by hypertension. Obstructive uropathy is the 3rd cause of ESRD in Zaria. The latter is wholly preventable by early urological intervention and closer collaboration between nephrologists and urologists in our centre. Majority of our patient absconded because of prohibitive cost of dialysis therapy.

ABS-OR-1011

THE BURDEN OF CHRONIC RENAL FAILURE AMONG MEDICAL ADMISSIONS IN AHMADU BELLO UNIVERSITY TEACHING HOSPITAL SHIKA, ZARIA

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Introduction: Chronic kidney disease (CKD) is an important public health issue globally as its assuming an epidemic proportion. The actual burden of CKD in Africa and Nigeria in particular is largely conjectural. CKD is associated with increased cardiovascular morbidity and mortality.

Aims and objectives: The study was carried out to determine the relative contribution CKD to medical admissions and determine causes of CKD in the new site of Ahmadu Bello University Teaching Hospital Zaria.

Methodology: This a retrospective study over a 3 year period between january 2006 and December 2008.Admissions case notes were studied to establish the hospital prevalence of CKD in Zaria.

Results: A total number of 3281 patients were admitted into the Medical ward in the period under review. Chronic Renal failure accounted for 112 (3.41%) of total admission. Only 109 cases were available for review. There were 57 males (52.3%), while females were 52 (47.7%). Their ages ranges between 12 to 80years with mean age of 41.89 Chronic glomerulonephritis accounted for 49.5% (54) of the cases, Hypertension 35.8% (39), Diabetic Nephropathy 8.3% (9), Obstructive Uropathy 5.5% (6), and others 0.9% (1).

Conclusion: CKD is a minor contributor to medical admissions in Zaria. Chronic glomerulonephritis remains the most important cause of chronic renal failure in our hospital, while hypertension ranked second and Diabetes mellitus is the 3rd aetiological entity.

ABS-OR-1012

PROTEINURIA AMONG MEDICAL ADMISSIONS IN BENIN CITY: A STUDY OF 100 CONSECUTIVE CASES.

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Background: Proteinuria is a risk factor for cardiovascular disease and mortality. Persistent proteinuria is diagnostic of chronic kidney disease(CKD) regardless of GFR level. Interventions that reduce proteinuria such as Blood pressure control can retard the progression of kidney disease.

Aims and objectives: To determine the prevalence of proteinuria among medical admissions in University of Benin Teaching Hospital (UBTH) and the relationship between proteinuria and some risk factors studied.

Materials and Method: 100 consecutive medical patients admitted through the UBTH emergency unit over a 30 day period were studied. Information on their sociodemographic and health status as well as their laboratory investigations and final diagnosis were obtained. Data were analysed using the SPSS version 15.

Results: Of the 100 patients studied 46% were males giving a sex ratio of 1:1.2(M:F). Mean age was 47.5±15.9yr. Majority were Binis(54%). Prevalence of proteinuria was 65%. Proteinuria was seen in 66.6% of hypertensives, 58.6% of diabetics,68% of patients who had CKD, 67.4% of febrile patients and 75% of HIV seropostive patients. There was no statistically significant association between proteinuria and DM, HTN or RVD. 12% of the 100 patients had CKD while only 2% had AKI.

Conclusion: Urinalysis is recommended for all medical cases at initial contact, however only persistent proteinuria predicts renal damage. Proteinuria was more prevalent among diabetics, hypertensives, renal disease and HIV patients but a substantial proportion of individuals with proteinuria did not have these conditions.

ABS-OR-1013

QUALITY AND PATTERN OF SLEEP IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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Introduction: Sleep problems are common in many chronic medical illnesses. However, no indigenous study has examined the quality and pattern of sleep in Nigerian patients with chronic kidney disease.

Aim: To determine the prevalence of 'poor sleep' in patients with Chronic Kidney Disease (CKD) in Benin City and to examine its association with some clinical and laboratory parameters in these patients

Methods: This was a cross-sectional analytical study of patients presenting consecutively in the outpatient department and the casualty unit of the University of Benin Teaching Hospital. Quality of sleep was measured in these patients using the Pittsburg Sleep Quality Index (PSQI) which is a scale that examines seven sleep domains (sleep latency, sleep disturbance, habitual sleep efficiency, subjective sleep quality, daytime sleep disturbance, sleep duration, and use of sleep medicine). The scores of these patients were compared with those of healthy control subjects which were selected from the general population.

Results: A total of 65 CKD patients (52M, 13F) completed the study. The mean age of subjects with CKD was 50.28+/-17.9 years. Glomerulonephritis (33.9%), Hypertension (23.1%) and Diabetic Nephropathy(23%) were the principal aetiologies of CKD amongst the patients. A total of 46 (70.8%) CKD patients were found to be poor sleepers(PSQI score > 5) with a mean score of 9.28 ± 6.17 compared to control subjects with a mean PSQI score of 4.45 ± 2.83 and only 37% being poor sleepers. Three(4.62%) of the CKD patients had impairment in all seven sleep domains, 5(7.69%) had no impairment in any of the domains while impairment was found to exist in varying severity in the different sleep domains in the other CKD patients. There was no significant difference in the total PSQI score between patients with CGN, hypertensive nephrosclerosis or diabetic nephropathy, the three principal aetiologies of CKD in these patients. Blood Pressure was found to be a determinant of quality of sleep, as there was a significant statistical difference in the mean scores between patients with and without controlled BP, with the former having a higher PSQI score and thus worse sleep quality. PCV was strongly inversely correlated with total PSQI score as well as number of sleep domains affected (p=0.003 each). Estimated GFR was also significantly inversely correlated with number of sleep domains affected as well as total PSQI score.(p=0.000 and p=0.006 respectively)

Conclusion: From the results of this study, it is reasonable to conclude that a very large percentage of CK D patients suffer from poor sleep and that optimizing renal function, increasing haematocrit level as well as ensuring blood pressure control to target goals are measures likely to positively impact on the quality of sleep in these patients.

ABS-OR-1014

BASELINE DEMOGRAPHIC AND LIFESTYLE CHARACTERISTICS OF THE KHDC PREVENTION PROGRAMME PARTICIPANTS AT MAIDUGURI

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Background: The non-communicable diseases such as chronic kidney disease, hypertension, diabetes and cardiovascular disorders account for the majority of global deaths. At the level of the general population in developing countries these diseases are largely undetected, with low awareness and treatment rates. This Vascular disease detection and Intervention Programme in Northeast Nigeria (VADIPIN) is an ongoing International Society of Nephrology (ISN) supported KHDC prevention programme.

Methods: Our community based study commenced in April 2008 and involved the general population of adults 18 years and older who resided in 6 clusters locations in Maiduguri. Following the ethical approval from the relevant authorities at Maiduguri, we embarked on public enlightenment campaigns using the local TV stations. All consenting adults were requested to fast and to assemble at the designated mobile clinics

located in the vicinity of their residences. Participants filled out or were interviewed using the questionnaire that enquired into their demographic, lifestyle, personal and family medical histories.

Results: One thousand and forty-three individuals (f = 469) aged 18 to 79 (mean 36.8) years participated in the surveys. 99% of the study participants were Nigerians of whom the majority hailed from the ethnic groups of the Northeast, Nigeria. Seventy-four percent of the population had at least some primary school education. 92% of respondents had a minimum of one serving of fruits/vegetables per week. Dietary salt restriction was reported by 4% while 11.9% actually believed that their salt intake was greater than average. 93% of respondents had never smoked while 87.6 reported abstinence from alcohol. 73% reported that they had physical exercises for up to three days per week while 11% used complexion cream preparations. A minority (8%) reported that they avoided the use of energy drinks but 18% resorted to local herb for treatment of different ailments.

Conclusion: The study population was physically active but had a low awareness of the benefit of salt restriction to the general population. Alcohol and smoking habits were uncommon but the use of local herbs and complexion creams by significant number of individuals maybe issues of public health importance.

ABS-OR-1015

KNOWLEDGE, AWARENESS AND PREVALENCE OF THE RISK FACTORS OF CHRONIC KIDNEY DISEASE IN A COMMUNITY IN SOUTH WEST NIGERIA

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Background: Chronic kidney disease is a global health problem. The prevalence in developed, economically emerging and developing countries has been on the rise and in fact more than the rate at which population growth rises. Chronic kidney disease, from some hospital-based studies in sub-Saharan Africa (SSA), has epidemiological characteristics that are strikingly different from those observed in other regions. Despite the menace of chronic kidney disease in SSA and especially in Nigeria, the most populated country in Africa, there is paucity of data on community studies.

Aims: The aim of this study was to determine the knowledge, awareness and prevalence of chronic kidney disease and its associated risk factors in a community in South West Nigeria.

Methods: A total of 468 participants were randomly recruited for the study using multi-stage technique. Structured questionnaires were used to extract information on socio-demographic characteristics and information on their knowledge of kidney disease with biophysical measurements done. Participants were screened for the presence of macroalbuminuria and microalbuminuria. Serum creatinine were measured with Glomerular Filtration Rate estimated using Modified Diet in Renal Disease equation. Prevalence of the risk factors for CKD, level of knowledge and the association between the risk factors and CKD were determined.

Results: Four hundred and fifty four participants were included in this study. The mean age $\pm$ SD of the participants were 45.8 $\pm$ 19.0 years. Among the participants, 20.4% of the participants 0.6% gave history of diabetes but 3.7% were diagnosed to have diabetes. The overall prevalence of albuminuria was 12.7%. The prevalence of macroalbuminuria and micralbuminuria was 8.9% each with higher value among females. The prevalence of chronic kidney disease with GFR < 60 ml/min/1.73m^2 was 12.3%. With logistic regression, increasing age (OR =0.919, 95% CI = 0.878-0.961) and female gender were predictive of chronic kidney

disease (OR = 4.87, 95 % CI=1.336-17.736) using GFR < 60 ml/min/1.73m^2. With albuminuria, systolic blood pressure (OR=1.038, 95% CI=1.006-1.071), diabetes mellitus (OR=15.764, 95% CI=1.247 – 199.238) were predictive of chronic kidney disease. Only 35.5 % of the participants had heard of chronic kidney disease before the time of the study. Less than 30% had good knowledge of chronic kidney disease.

Conclusion: Chronic kidney disease is prevalent in our communities. The risk factors were also prevalent and effort should be made to introduce policies and strategies to reduce the burden of chronic kidney disease and its associated risk factors in the community. There is need to also increase the awareness of chronic kidney disease.

ABS-OR-1016

SAFETY AND COST EFFECTIVENESS OF RENAL BIOPSY AS A DAY PROCEDURE: ZARIA EXPERIENCE

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Background: Renal biopsy is an important and intergral part of management of most glomerular diseases. In fact is the 'gold standard' for making such diagnosis. Renal biopsy involves patients being admitted after the procedure. This study was set out to evaluate and examine the safety of the procedure as day-case.

Methodology: We Examined the records of patient who have undergone renal biopsy in our centre , over a 3 year period. (January 2006 to Jan. 2009). A total of 30 patients were studied. The indications for the procedure were Nephrotic Syndrome 18 (60.0%),HIV with signifant Proteinuria 10 (33.3%) and unexplained proteinuria in apparently healthy adults 2 (6.7%).There were 21 males (70.0%) and 9 females (30.0%) and their ages ranges between 12 and 55 years, with mean age of 29.97 11.30. All had pre- biopsy evaluation that included PT,KCCT, FBC and Platelets, urine m/c/s and renal ultra sound scans. All patients were rested supine for 3 to 4hrs after biopsy, and had no visible haematuria or abnormal vital signs. Only one (3.33%) of the patients complained of loin pain a week after the procedure. A repeat renal ultrasound and renal function did not revealed any abnormality. Pain was controlled with simple analgesia.

Conclusion: Renal biopsy is quite safe as a day case procedure. Its also cost effective as it obviates the need for admission especially in our hospital that is constrained by availability of bed space.

ABS-OR-1017

HISTOPATHOLOGIC CHARACTERISTICS OF PATIENTS PRESENTING WITH NEPHROTIC SYNDROME IN THE GUINEA SAVANNAH BELT OF NIGERIA

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Background: In the last 3 decades, since Awounor -Renner reported on the histological pattern of glomerulonephritis in children with nephrotic syndrome in zaria, no similar report has emerged from this part

of the country. The aim of this study was therefore to evaluate the histological pattern of adults presenting with nephrotic syndrome in this centre.

Methodology and Results: Twenty adult Nigerians who presented with nephrotic syndrome were studied. They consisted of 13 males (65.0%) and 7 females (35.5%). Age ranged between 12 to 55 years with mean age of 29.97All had percutaneous blind renal biopsy, after ultra sound mapping and determination of kidney depth from skin surface prior to the procedure. All had baseline investigations done which included, evaluation of renal function (urea, electrolytes and creatinine), full blood count platelets count, and clotting profile, urine culture, and renal ultra sound. The kidney tissue obtained was fixed in 10% formaldehyde, paraffin-embedded and stained with haematoxyllin and eosin, periodic acid schiff, congo red, methenamine silver blue and viewed with light microscopy. The Histological characteristics were as follows: Minimal change 8 (40.0%), Membranous glomerulopathy 6 (30.0%), Focal segmental glomerulosclerosis 4 (20.0%) and Membranoproliferative glomerulonephritis 2 (10.0%).

Limitations: Lack of immunofluorescent and electron microscopic techniques were significant constraints for reliable tissue diagnosis

Conclusion: This study illustrated that the most common histologic pattern in adults this environment is minimal change disease (MCD). This finding was somewhat suprising as it might reflect artefact from sampling error where focal lesions may be missed.

ABS-OR-1018

A REVISIT OF THE PATTERN OF RENAL DISEASE IN THE UNIVERSITY COLLEGE HOSPITAL, IBADAN OVER A ONE YEAR PERIOD FROM NOVEMBER 2008-2009

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Objectives: To highlight patterns of renal disease in the renal unit of UCH.

Materials and Method: This is a retrospective study. The ward admission register of the renal wards was used for collation of patients from November 1st, 2008-November 1st, 2009. The diagnosis were reviewed vis a vis their clinical and biochemical data and data analyzed by statistical means.

Results: A total of 256 patients were seen within the period under study (2008-2009) 126 were males (49.2%) of the total patients seen. 130 were females (50.8%). In the year under study, chronic kidney disease was the commonest disease presentation, accounting for 86% and the commonest aetiology was chronic glomerulopathy (60%).The mean age of presentation was 48.2 for males and 47.68yrs for females. Others causes include hypertensive nephrosclerosis (7.8%), Diabetes mellitus(6%), and retroviral infection(5.8%). Other renal conditions seen were acute renal failure (10%), urinary tract infection (2%), obstructive uropathy(1.5%) and others like urolithiasis, polycystic kidney disease accounted for the remaining negligible percentage. In other related studies done in sub-sahara Africa (1994), glomerulonephritis was recorded as the commonest cause of ESRD (1771 people-52.1%) and hypertension in (1549 people-45.6%) of patients by the South Africa dialysis and transplant registry, comparable with our findings. In a ten-year study of 368 patients with chronic renal failure in Nigeria, the etiology of renal failure was undetermined in 62%. Of the remaining patients whose etiology was ascertained, hypertension accounted for 61%, diabetes mellitus for 11% and chronic glomerulonephritis for 5.9%. Chronic glomerulonephritis and hypertension are principal causes of CRF in tropical Africa and East Africa, together with diabetes mellitus and obstructive uropathy.

Conclusion: CKD still remains the commonest presenting disease in the renal unit with chronic glomerulonephritis being the commonest cause, the average age of presentation being 47.94yrs. Retroviral infection is rapidly emerging contributor to renal disease. There is a need for regular screening of the populace to reduce the burden of CKD.

ABS-OR-1019

PREVALENCE OF HYPERTENSION, DIABETES AND PROTEINURIA: 2009 WORLD KIDNEY DAY SCREENING PROGRAM AT OSOGBO, OSUN STATE, NIGERIA

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Background: Chronic kidney failure (CKF) which represents the end of the continuum of chronic kidney disease (CKD) is a devastating medical illness with dire social and economic consequences for the patients, their families and the country. CKD is asymptomatic in early stages which lead to delay in recognition. Reports from Nigeria showed that hypertension, glomerulonephritis and diabetes are the leading causes of CKD. Early detection of these diseases is possible with measurement of blood pressure, blood glucose and urine examination, which in turn will allow early initiation of appropriate medical therapies and educational strategies with likely positive impact on kidney outcome.

Aims and objectives: To determine the prevalence of diabetes, hypertension and proteinuria among participants of the 2009 World Kidney Day Screening Program (WKDSP).

Materials and Methods: A cross sectional study involving 586 participants (360 males, 226 females) was conducted in 4 centres as part of 2009 WKDSP. Each participant completed a simple questionnaire and underwent blood pressure (BP) measurement, blood glucose determination and urinalysis.

Results: Ninety six (16.4%) and 11 (1.9%) participants were known before screening to have hypertension and diabetes respectively. Fifty six (9.6%) and 11 (1.9%) participants were newly diagnosed to have hypertension and diabetes respectively. Forty one (42.7%) participants with known hypertension were uncontrolled. Fourteen (2.4%) of the participants had proteinuria e”1+. Participants with proteinuria had significantly higher systolic and diastolic BPs.

Conclusion: This study showed a high prevalence of undetected and uncontrolled hypertension among participants. There is a need to put in place public health policies to stem the scourge of hypertension, diabetes and CKD.

ABS-OR-1020

A RETROSPECTIVE STUDY OF THE PATTERN OF CLINICAL PRESENTATION AND OUTCOME OF LUPUS NEPHRITIS IN ADULT NIGERIANS

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Background: SLE, a multisystem autoimmune disease with predominant female and racial predilection is uncommon in our setting though the incidence is increasing. Lupus Nephritis is common and is recognised to be a major predictor of mortality.

Aim & Objectives: 1. To assess clinical characteristics and renal histopathology
2. To assess response to steroid and/or immunosuppressive therapy.
3. To determine (if any) factors that portend worse outcome.

Materials and Methods: We retrospectively studied 23 patients that manifested e"4 ACR criteria for diagnosis of SLE. Socio-demographic data, clinical and laboratory parameters were collated. All patients had combination of ACEI, diuretics and steroids. Induction remission therapy was done using I.V. methyl prednisolone 500mg daily for three days and maintenance therapy with prednisolone at the dose of 1mg/kg/ day. 15 of the patient were thereafter managed with immunosuppressive.

Results: The age ranged between 15 and 63yrs (mean± S.D.; 31.7±1.28yrs). The common clinical findings at the time of diagnosis included body swelling (88%), frothy urine (80%), facial rash (84%), joint pain (84%) and anaemia (92%), 70% had massive proteinuria with mean(±SD) of 3.74(±1.37)g. Antinuclear antibodies and anti-double stranded DNA antibodies were detected in 40%. Of the 9 patients that had renal biopsy done 4 had membranous GN (stage V). Fifteen patients developed renal failure, 13(56.2%) had HD while CAPD and renal transplantation were offered to 1(4.3%) and 2(8.7%) respectively. Eight (32%) patients had sustained remission while others had between 1-3 relapses during the follow up period. The mean duration of survival after diagnosis was 20±1.56months.

Conclusion: The incidence of Lupus nephritis is increasing it significantly contributes to morbidity and mortality.

ABS-OR-1021

PATIENT LEVEL COST OF DIALYSIS TREATMENT IN NIGERIA

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Background: The cost of treating individuals with kidney failure is prohibitive in all the countries of the world. In the developing countries besides the issue of availability, the affordability of renal replacement therapy may be the most important factor that determines survival of patients with ESRD.

Methods: In this study we sought to determine the costs of dialysis treatment in the different centres from all parts of Nigeria according to their practices in the year 2008. We employed the use of questionnaires which were filled out by the Nigerian nephrologists and dialysis nurses in attendance at the Abuja 2009 AFRAN-NAN conference.

Results: Responses came from 21 (two private and 19 public) dialysis centres situated in six of the 6 geopolitical zones of Nigeria. The centres had between one and 19 years with a mean of 5.4 years of operational experience. The prevalent dialysis populations in the centre summed up to 401 (mean = 23) whereas 276 (mean = 19) patients initiated treated in the previous year. In 17 of the centres the majority of the incident patients were lost to follow up in the first month. The mean cost per session of dialysis in the public hospitals was =N=15,210 ($100) and about 2x more expensive in the private centres. In all but one centre (where some persons were insured) all the patients settled dialysis fees by out of pocket payments. Continuation of dialysis treatment for patients with ESRD beyond the first year was possible in a minority of cases in the majority of the centres.

Conclusion: Taken at the face value the cost of dialysis in Nigeria is cheaper than in the Western industrialized countries. If however the purchasing power parity and modes of financing are put into consideration, the cost of dialysis care in Nigeria is largely unaffordable and unsustainable for the majority of ESRD patients.

ABS-OR-1022

TWO YEARS OF HAEMODIALYSIS IN UYO: A DESCRIPTIVE STUDY

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Background: The epidemiologic transition appears to be gaining a foothold in Nigeria with an increase in the number of individuals presenting in our centres with chronic non-communicable diseases. In January 2008, the Akwa Ibom State government set up a dialysis centre to cater for patients who require haemodialysis in the state and its environs. It is important to review the baseline sociodemographic and clinical characteristics of our patients and assess the predictors of mortality.

Methodology: This is a retrospective analysis of data collected over two years on patients seen at the dialysis unit at the University of Uyo Teaching Hospital, Uyo. Descriptive analysis of the characteristics of the patients was performed using Chi-square for categorical variables and the Wilcoxon rank sum test for continuous variables that were not normally distributed. A logistic regression model was built to determine factors that predict mortality in our patients.

Results: 96 patients were dialyzed in the 2 year period, 54 (56.8%) and 42 (43.2%) were male and female respectively. The median creatinine level at presentation was 1180µmol/L . The mean age at presentation was 44.9 years and median Packed Cell volume at first dialysis was 23%. 29 (30.85%) had chronic glomerulonephritis, 14 (13.82%) came in acute renal failure, 15 (16%) had ESRD due to diabetic nephropathy. Only about 25% of the patients had at least 70% compliance to scheduled dialysis and the median number of sessions before death was 3 per patient.

Conclusion: There is great need to intensify public health enlightenment on renal health issues and to encourage government subsidy on the cost of dialysis in Nigeria.

ABS-OR-1023

THE ASSESSMENT OF HEMODIALYSIS ADEQUACY AMONG ESRD PATIENTS IN ILORIN USING UREA REDUCTION RATIO

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Introduction: Urea Reduction Ratio (URR) as a method of measuring adequate dialysis that correlate with patient outcome was first popularized by Lowrie and Lew in 1991. URR is a measure of adequacy of delivered dose of dialysis expressed as a percentage reduction in blood urea level after a session of dialysis. It is mathematically related to Kt/V and both can be derived from each other with some amount of precision by various equations or a normogram. Although, Kt/V is recommended as the best measure of dialysis adequacy, URR is the most utilized because of its simplicity and both methods have similar predictive power in terms of patient outcome. A URR of 65% which corresponds with Kt/V of 1.2 is the minimum acceptable dose in the standard thrice weekly hemodialysis. Unlike in Europe and America, there is no unified data system for recording and analyzing URR from various dialysis units in tropical developing countries. In Nigeria, there is paucity of data on adequacy of hemodialysis. The few available reports showed that inadequate dialysis is common and patients survival is very poor. A one year review of patients on maintenance hemodialysis at UITH, Ilorin was evaluated to determine the adequacy of delivered dose of dialysis and the outcome.

Methods: All cases that met the criteria for ESRD and had hemodialysis between October 2008 and November 2009 were retrieved. Patients that had regular 4 hourly session of dialysis for at least twice a week in two consecutive months were included in the study. Data was analyzed using SPSS version 16.
Results: Twelve out of 33 patients (36%) with ESRD met the inclusion criteria. The mean age of the patients was 48.25 ± 17.85 with male to female ratio of 2:1. Majority were retired civil servants (33.3%), followed by serving civil servants (25.0%) and students (16.7%). The etiological factors of ESRD were Hypertensive nephrosclerosis (41.7%), CGN (33.3%), Diabetic nephropathy (25.0%), chronic allograft dysfunction (8.3%). None of the patients was able to sustain thrice weekly hemodialysis sessions. Mean pre-dialysis and post dialysis urea were 25.29 ± 11.87 mmol/l and 14.78 ± 8.10mmol/l respectively. Mean URR was 41.83 ± 16.30% and overall mortality was 66.7%. The factors that appeared to have contributed to inadequate dialysis and poor outcome were late presentation, uremic bleeding, septicemia, repeated blood transfusion and inability to sustain recommended thrice weekly haemodialysis due to poor finances.

Conclusion: Our study showed that inadequate hemodialysis is common and is associated with high mortality rate. Major contributory factors to poor outcome were ignorance, late presentation and poor socioeconomic status of these patients. There is need to intensify awareness programs on early diagnosis of CKD. We recommend incorporation of renal replacement therapy subsidy into the current National Health Insurance Scheme of the Federal Government.

ABS-OR-1024

A TEN YEAR REVIEW OF INTRA-DIALYSIS COMPLICATIONS AS SEEN AT RENAL CARE CENTRE ILORIN

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Introduction: In Nigeria, haemodialysis (HD) is the most readily available dialytic therapy. It remains the most viable management modality for our uraemic syndrome patients who either present in acute renal failure or chronic renal failure. Peritoneal dialysis could have been the best option in the tropics including Nigeria as it is neither machine nor power dependent. However, most centres utilized HD due to scarcity and high cost of peritoneal dialysis fluids with associated high rate of peritonitis. Despite the in-built safety measures of HD machines, it is still associated with complications, some of which may be life threatening. We therefore undertook a study of the pattern of intra-dialysis complications and their outcome in our centre in the last decade. The factors that contributed to these complications and measures taken to ameliorate them were identified.

Method: All case notes of patients who had dialysis in our centre between December 1999 and November 2009, with diagnosis of either ARF or CRF were retrieved and analysed. The socio- demographic, clinical diagnosis, intra- dialysis complications and the outcome were obtained for analysis using SPSS version16.

Results: One hundred and fifty three patients were managed with 802 sessions of HD. Their ages ranged between 10 and 75 years with male to female ratio was 1.2:1. Haemodialysis sessions were bicarbonate based and blood flow rate of 200- 350 mls per minute, using hollow fibre dialyzer via femoral/jugular cannulations. Majority of the cases were chronic renal failure (70.5%). One hundred and ninety seven sessions were associated with various complications (24.6%). Hypotension was the commonest (40%) of the intra- dialysis complication followed by hypertension (29%), convulsions (8%), muscle cramps (6%), back pain (5%), pruritus (4%), vomiting (3%), chest pain (3%), disequilibrium syndrome (1%) and dialysis membrane rupture (1%). There were no significant differences in intra- dialysis complication pattern between cases of ARF and those with CRF. The overall mortality from intra- dialysis complications was 3%.

Conclusion: Intra- dialysis complications are common with majority due to hypotension. Most of the identified complications are preventable and treatable conditions as the mortality rate was low. There is need to be proactive in identifying potential precipitating factors and prompt treatment of complications in order to avoid unnecessary discontinuation of the procedure and/or patient death.

ABS-OR-1025

HEPATITIS C- INDUCED KIDNEY AND LIVER DISEASEE AMELIORATED BY INTERFERON AND RIBAVIRIN THERAPY: CASE REPORT

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Background: Asymptomic hepatitis C infection is common is haemodialysis population. We report a Nigerian woman whose liver and kidney injuries abrogated after antiviral therapy. And hepatitis C -associated liver and kidney co-infection is an important cause of increased morbidity and mortality in patients with chronic liver and kidney diseases.

Case report: N.S is a 28 years old lady who presented to our renal unit via the general outpatient department (GOPD) on account of newly diagnosed hypertension and proteinuria. She had minimal urinary symptoms. No history of exposure to nephrotoxic drugs. She offered no past history of jaundice, blood transfusion or indiscriminate exposure to sharps objects. She had no personal history of diabetes. She described a history suggestive of Alport's syndrome in two of her siblings who died from chronic kidney disease. Mother is type 2 diabetic. Her medications in the preceeding 6 months prior to presentation include ramipril and hydrochlorthiazide. Physical examination was unremarkable, blood pressure being 126/84mm Hg. Results of blood tests were as follows: urea, electrolytes and creatinine were entirely normal; .corrected serum calcium-2.66mmmol/l , phosphate 0.52mmo/luric acid-320µmol/l , creatinine clearance – 104ml/min/1.73m^2,24 hrs. Urine protein- 1.2gm/day. HBsAg –negative, HCV antibodies- reactive, Platelets 373 x10^9/L,PCV - 33%,WBC- 6.2 x 10 ^{9}, serum proteins -65g/l, albumin 49g/l, globulin -16g/l, AST – 5 i.u/l, ALT- 26i.u/l, AST : ALT ratio < 1,AlP -25 i.u/l. Abdominal ultrasound scan showed normal sized kidneys with good cortico-medullary differentiation. Retroviral screening was negative, clotting profile including PT -20s, (control =15s) KCCT-48s (control=32s). Light microscopy findings of kidney biopsy was suggestive of mesangiocapillary glomerulonephritis, while liver biopsy was consistent with chronic persistent hepatitis. She was placed on subcutaneous pegylated interferon 180µg weekly and ribavirin 400mg twice a day for 24wks. Renal and liver biopsies performed 6 months after therapy showed complete resolution of the respective lesions.

Conclusion: It's imperative to fully evaluate the liver and kidney in patients positive for Hepatitis C antibodies presenting with glomerular disease as antiviral agents may exert dramatic benefit.

ABS-OR-1026

HAEMODIALYSIS THERAPY IN ACUTE RENAL FAILURE ILORIN EXPERIENCE

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Introduction: Haemodialysis is most utilized form of renal replacement therapy in our environment both for acute and chronic renal failure. This is largely because of high infection rate associated with peritoneal dialysis and unaffordable cost of renal transplantation. A 17 year review of haemodialysis therapy in acute renal failure at UITH Ilorin was evaluated to determine its cost effectiveness in view of the ever rising cost of this mode of therapy.

Methods: All cases that met the criteria for ARF and who had haemodialysis between November 1992 to October 2009 were retrieved and analysed using SPSS version 16.

Results: There were 45 patients (19 males and 26 females), with male to female ratio of 1:1.4. The age range was between 10-69 years while 76% of cases are less than 40 years of age. Majority were traders followed by students. The aetiological factors were septicaemia (35.5%), AGN (15.5%), septic abortion and herbal remedies (11.1% each), ante/post-partum haemorrhage and severe gastroenteritis (6.6% each), drug induced (4.4%), eclampsia (2.2%) and unknown (6.6%).The duration of illness before dialysis range between 1-30 days with 80% of cases less than 14 days. The waiting time before commencing dialysis range between 1-9 days with majority less than a week (91.1%). The range of blood transfusion was between 1-6 units with 65% of cases receiving 1-3 unit of blood. Sessions of dialysis ranged between 1-6 with 89% of cases having less or equal to 3 sessions before recovery. The duration of hospitalization was less than 3 weeks in 76% of cases. Hypotension was the commonest (44.4%) intra-dialysis complication followed by twitching (15.5%), dialysis membrane rupture (6.6%), muscle cramps, back pain (4.4% each), chest pain, weakness and headache (2.2% each).The outcome was favourable with 6 deaths constituting 13.3% of the cases.

Conclusion: Acute renal failure is a common cause of morbidity and mortality in our environment. Majority of the patients are less than 40 years of age with slight female preponderance. The implicated aetiological agents still remain preventable and treatable infectious causes, majority of which presented in a setting of septicaemic illness. Duration of hospital stay, waiting time before dialysis and total duration of illness appear to have positive influence on the outcome as majority were less than two weeks.We recommend at least 3 sessions of haemodialysis in ARF patients as majority of our patients did well after 3 sessions.

ABS-OR-1027

EFFICACY AND COST EFFECTIVENESS OF DIALYSER RE-USE: A METHOD OF REDUCING THE COST OF DIALYSIS IN A RESOURCE POOR COMMUNITY

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Background: The cost of dialysis is exorbitant and relatively unaffordable to the majority of needy patients.Re-use haemodialysis may offer some relieve.

Aims and objectives: 1.To assess cost effectiveness of dialyser re-use in our patients on maintenance haemodialysis.
2.To determine the adequacy of HD delivered to the patients.

Methodology: We study a total of 14 patients that had a total of 42 sessions. Their socio-demographic data, predialysis and postdialysis results were collated and analyzed. Dialysis adequacy was assessed using urea reduction ratio (URR). Data was analyzed using SPSS package version 14.

Results: Their ages ranged between 18 and 67 years, with median of 45 years. First and second dialyser use constituted 33.3% each while the third use was in 31%. Only one patient had fourth session (2.4) %. Mean URR achieved during first use, second and third dialyser use are 60.6%, 58% and 65% respectively. 50% achieved adequate dialysis on first use while 42.8% achieved adequate dialysis on second and third re-use. No statistical significant difference between first, second and third dialyser re-use. The cost of a session of first session of dialysis was 17,200 while the costs of each re-use ranges between 6,500 and 10,000.

Conclusion: Dialyser re-use is an effective cost saving treatment modality particularly in underserved communities.

ABS-OR-1028

A CLINICAL PROFILE OF COMPLICATIONS IN ADULTS WITH THE NEPHROTIC SYNDROME UNTREATED WITH STEROIDS OR CYTOTOXIC AGENTS

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Background: The Nephrotic syndrome continues to be one of the common modes of presentation of CKD in Nigeria accounting for 2 – 4 % of hospital admissions. A higher morbidity and mortality has been noted in those who present with complications early. Male gender, hypertension, impaired plasma creatinine and creatinine clearance in patients with this syndrome have been associated with progression to chronic renal failure and death. This study set out to determine the clinical profile of patients with the nephrotic syndrome attending the renal clinic and the complications encountered during a one year follow up.

Method: Fifty two (52) adult patients with clinical features of idiopathic nephrotic syndrome being managed with diuretics and lipid lowering agents alone were followed up for one year in the renal clinic, University College Hospital, Ibadan. Urine samples for urinalysis, urine microscopy, culture, sensitivity, 24 hour urinary protein estimations, creatinine clearance and blood samples for serum electrolytes, urea, creatinine, lipids, albumin and total protein were also taken both at the onset and end of the follow up period. Periodic blood pressure and weight measurements were done and other investigations were requested based on the suspected complications.

Result: 77.8% of the patients were between the ages of 18 and 30. More males (55.6%) were seen. All had generalized edema, 96% had nephrotic range proteinuria, 55.6% microscopic hematuria and 37% leucocyturia only 7.4% of which had a growth of pathogenic organisms. The commonest complications were refractory edema (68.5%), renal failure (60.4%), anemia (58.7%), and hypertension (29.6%). Other encountered complications were infections (UTI and chest) 5.7%, spontaneous bacterial peritonitis (5.6%), arrhythmias (5.6%) and DVT (3.8%).The majority had financial constraints (85.2%) .

Conclusion: The nephrotic syndrome remains a problem in our practice, presenting mainly in young males in the prime of life. Most of them present late and develop complications early and have financial constraints which make the management difficult and the outcome poor. We recommend that management of CKD should be included under the NHIS scheme.

ABS-OR-1029

NEPHROTIC SYNDROME IN NIGERIAN CHILDREN: CLINICAL AND GLOMERULAR CHARACTERISTICS

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Aims and objectives: This study determined the incidence and prevalence of childhood nephrotic syndrome (CNS), pre-treatment glomerular pathology, renal and patient outcome in Nigerian children with idiopathic CNS (ICNS).

Materials and methods: A non-randomized 9-year (June 2000 and June 2008) prospective study of CNS was conducted. Pre-treatment percutaneous renal biopsy was performed in all patients after obtaining an informed consent. The patients were followed-up for varied time length of at least six months. Patients recruited up till June 30, were followed till December 31, 2008. The Kaplan-Meier and log-rank statistics were used for comparative analysis.

Results: CNS accounted for 1.26% of paediatric admissions. The overall CNS incidence was 0.64/100000/ year (prevalence, 5.1/100000). Median CNS onset age was 7.1 (2.5–14.0) years. Male: female ratio was 1.7. Fifty-four of 78 (69.2%) nephrotic children had ICNS (incidence, 0.44/100000/year; prevalence, 3.53/ 100000) while 24 (30.8%) had secondary CNS. Thirty-nine of 54 (72.2%) ICNS patients were 5 years old and above. 18 of 54 were hypertensive (33.3%) with a mean blood pressure of 124.4 ± 12 / 87.5 ± 13 mmHg. 25/54 (46.3%) had reduced eGFR (59.2 ± 14.9 mL/min/1.73m^2) and 17/54 (31.5%) had microhaematuria. The glomerular lesions in ICNS were membranoproliferative glomerulonephritis (MPGN, 44.4%), focal segmental glomerulosclerosis (FSGS, 25.9%), minimal change disease (MCD, 18.5%), mesangial proliferative glomerulonephritis (7.4%) and membranous nephropathy (3.7%). Overall ICNS cumulative complete remission (CR) rate 4 to 8 weeks post prednisolone treatment was 49.6%. Twenty-two of 25 with CR were early steroid responders while 3 were late responders. Median time to CR was 12.0 (3.0 – 46.0) days. Thirty relapses occurred; median time to first relapse was 11.0 months. Cumulative five-year relapse-free rate was 26.6%. Five-year renal survival was 16.1%. All patients with CR were followed-up for 6 – 93 (median, 22.0) months.

Conclusions: Prevalence of non-MCD was very high with significant resistance to prednisolone; poor renal survival was due to high frequency of MPGN and FSGS. Pre-treatment renal biopsy is advocated in our kind of patients so that steroid-sparing agents can be started early.

ABS-OR-1030

MICROALBUMINURIA IN HAART-NAÏVE HIV PATIENTS IN NIGERIA

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Background: HIV is an important cause of chronic kidney disease (CKD) in sub-Saharan Africa, the cost of management of which is highly prohibitive in most countries in the region. This observation demands the necessity of screening tools to detect early kidney involvement in HIV for prompt intervention. Microalbuminuria is a marker of cardiovascular and CKD; and early detection provides opportunity for

targeted therapy to delay or prevent development of ESKD. Studies on microalbuminuria in HIV in Nigeria are sparse, so its magnitude and utility in this patient population is not well described, hence this study.

Methods: This is cross-sectional study of consecutively recruited HAART –naïve HIV-seropositive patients seen at HIV clinic of University of Ilorin Teaching Hospital. Demographic data of the patients were collated at the clinic and blood samples were analyzed for CD4+ count, electrolytes, pack cell volume (PCV), urea and creatinine. Sport morning urine samples were collected for microalbuminuria determination by HemoCue point of care analyzer. SPSS version 16 (SPSS Inc, Chicago, IL, USA) was used to analyze the data. Correlation statistics were used to determine strength of association between severity of HIV (as indicated by CD4+ count and PCV) and microalbuminuria.

Results: 72patients out of 102 recruited so far have enough data for analysis. The mean age was 39±10years with 33(45%) males and 39(54%) females. Fifty (69%) patients had AIDs (CD4+ count <200cells/uL) while microalbuminuria defined as urine albumin estimation of >30mg/L was present in 51(70.8%) of patients. Median CD4+ count and microalbuminuria were 94(6-729) cells/uL and 60(10 ->150)mg/L respectively; while the mean PCV was 25±7. Microalbuminuria correlates negatively and significantly with CD4+ count ($r = -0.246$, $p = 0.039$) and PCV ($r = -0.348$, $p = 0.003$). CD4+ also correlates positively and significantly with PCV ($r = 0.466$, $p < 0.001$).

Conclusions: Microalbuminuria is very common in HAART-naïve HIV patients in Nigeria and seems to be worse as the disease progresses. A larger sample size and prospective study is required to confirm this relationship. Screening for microalbuminuria and treatment is however recommended to reduce the burden of HIV-related CKD in sub-Saharan Africa.

ABS-OR-1031

PREGNANCY AND CHRONIC KIDNEY DISEASE(CKD): CHALLENGES AND OUTCOME

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Background: Pregnancy is associated with various physiological and haemodynamic changes resulting from various hormonal interplay. This increases the functional burden on the kidney. The ability of the kidney to cope with this heightened functions determine the outcome of the pregnancy. Thus pregnancy in kidney with impaired function poses great challenges as the increased functional demand worsens the innate kidney function, and the impaired kidney function has adverse effect on the outcome of the pregnancy. We report a case of pregnancy in a patient with chronic kidney disease.

Objective: To highlight the challenges and outcome of pregnancy in a patient with chronic kidney disease.

Case Report: A 24 years old youth corper, referred from a private hospital on account of pregnancy in a background chronic kidney disease. She presented at the referral hospital 2 years earlier with CKD stage 3 secondary to chronic glomerulonephritis but was neither compliant to medication or regular follow up. At presention she was 14 weeks gestation, hypertensive, had proteinuria and azotaemia. The challenges, possible complications and outcome were explained to the patient and spouse. She developed eclampsia at 22 weeks gestation and blood pressure was very difficult to control despite using maximum doses of antihypertensive

with deteriorating kidney function. She had intra uterine fetal death at 24 weeks. Two weeks later blood pressure control and renal function improved remarkably.

Conclusion: Pregnancy in patients with CKD is associated with lots of challenges and outcome is not good.

ABS-OR-1032

LAPAROSCOPIC DYE-INDUCED ACUTE RENAL FAILURE: CASE REPORT

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Background: Acute renal failure is an important cause of morbidity and mortality in our emergency and ICU settings. Despite plethora of data on acute renal failure associated with radiocontrast agents, only scant data are available pertaining to renal failure after exposure to laparoscopic dyes in gynaecologic setting. Therefore, we report a Nigerian lady who developed acute renal failure after evaluation for infertility using laparoscopy and dye test.

Case report: S.S is a 26 years old nurse assistant. She presented with 6 weeks history of generalized body weakness, anorexia, vomiting, abdominal pain, 2 weeks history of facial swelling and diminution in urine output. She had a laparascopy and dye test for primary infertility 5 days prior to development of the aforementioned symptoms. She had been on fertility medications (clomiphene) for 2 months. There was no history suggestive of abuse of non steroidal antiinflammatory drugs, or use of mercury containing creams or soaps. No history of consumption of herbal remedies. No past history of body swelling. Other relevant inquiries including for hypertension, diabetes, kidney disease and family history were not contributory about family history. Physical examination revealed a young lady, dyspnoeic, pale, anicteric, with anasarca. BP-130/80mmHg, normal heart sounds, with no pericardial rub, Aside basal crackles on chest auscultation the rest of the systems were unremarkable. Results of blood tests were as follows: packed cell volume-25%, total white blood cell-$8.8x10^9$L, Erythrocyte sedimentation rate- 140mm/hour, urea -29.5mmol/l, creatinine-742 µmol/l; electrolytes were within normal limits. Urine showed trace protein on dipstick, and bland sediment on microscopy. Both kidneys showed normal bipolar dimensions on ultrasound with normal corticomedillary differentiation. eGFR-9.03ml/min/1.73m^2, with protein excretion -1.3g/24hrs. 12-lead electrocardiogram revealed normal findings. She made a remarkable recovery after 6 sessions of HD, and was subsequently discharged. She has remained well.

Conclusions: We recommend inquiry of history of dye exposure during gynaecological evaluation in women with kidney disease

ABS-OR-1033

SOLID ORGAN D NATION IN NIGERIA: HOW ALTRUISTIC?

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Background: Organ transplantation has a dual arm of recipient and donor. Organ donation is most limiting factor determining the rate of organ transplantation in most countries. There are various guidelines and laws guiding organ donation. Altruistic donation without attachment to material or monetary gains is a widely accepted condition for acceptance of a donor for transplantation. In Nigeria there is no formal guideline or

law on organ donation and transplantation. This study is to highlight how altruistic is organ donation among kidney transplant patients in Nigeria.

Materials and Method: The clinical details of end stage kidney disease patients who has had or being prepared for kidney transplantation presenting from 1st January2009 to 31st December 2009 were documented. The data obtained were analysed.

Results: A total of eleven patients presented during the period, 73% were males, age range of 24 to 75 years, and 36% were businessmen. Duration of renal disease varies between 3 to 38 months before going for transplantation, financial constraint caused the delay for transplantation in 55% of patients, only 36% of patients sponsored their transplant, various organizations sponsored transplantation in 45% of patients. Only 18% of recipient had relationship with the donor, the others had financial commitment with the recipient paying between 1 and 1.8 million naira to the donor. There were middle men in 64% of cases. Seventy three percent of the transplant was done in India, with only one patient (9.1%) transplanted in Nigeria.

Conclusion: Poverty is the leading factor causing delay in kidney transplant in Nigeria and kidney donations are not altruistic in majority of cases. This could be due to high level of poverty in the country.

ABS-OR-1034

RENAL ANGIOMYOLIPOMA MASQUERADING AS GIANT RENAL ABSCESS- A CASE REPORT

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Introduction: This report describes an unusual presentation of renal angiomyolipoma in a 51yr old man who presented with left-sided flank pain and high-grade fever of the same duration. Clinical evaluation was in keeping with left-sided pyonephrosis and the diagnosis was confirmed by Abdominal CT. He subsequently had nephrectomy on the affected side and microscopic pathology of the nephrectomy specimen showed typical features of angiomyolipoma.

He has since made an uneventful recovery and is being followed up at the outpatient clinic.

Discussion: Angiomyolipoma of the kidney is the most common benign neoplasm of the kidney. Its clinical manifestation varies with the size of the tumour. Its clinical presentation as a giant renal abscess, though rare, can be explained by the biologic behaviour of the tumour. In evaluating renal abscesses one should be aware of underlying local causes that may predispose to abscess formation.

Keywords: RENAL ANGIOMYOLIPOMA, RENAL ABSCESS, BENIGN NEOPLASMS

ABS-OR-1035

PATTERN OF UROLOGICAL ABNORMALITIES SEEN IN CHILDREN AT THE UITH ILORIN

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Introduction: There is increasing diagnosis of urological abnormalities in recent times because of the presence of hi-tech equipments such has ultrasound and CT scan. Hitherto, they were missed largely because IVU is not part of the routine medical test done during pre-school entrance screening except they were indicated. Furthermore, UTI which could have raised the suspicion of some anomalies are either missed or has low incidence around there.

Objective: To describe or determine the pattern of urological anomalies seen in children at the UITH.

Methodology: A review of urological anomalies seen from 1995 to 2009 was carried out using the renal register kept at the nephrology division of the UITH.

Result: A total of 21 cases were seen during the period under review. The leading urological disorders encountered were PUV in 6 children. Others include uretero-pelvic junction obstruction-4, polycystic kidney-3, All the rest which include right ectopic kidney, Bilateral vesico–ureteral reflux, multicystic kidney, right renal cyst, left pelvic kidney, bladder diverticulum, bilateral ureterocoele and dysplastic kdney occurred in one child each. It amounted to about one case seen per year. 2 deaths were recorded among the patients with PUV.

Conclusion: There is still remarkable number of cases of urological anomalies occurring in children. Despite consanguinity among some tribes around here, few cases of urological anomalies of genetic origin were seen. The outcome in all of them has been satisfactory with few deaths recorded. Routine antenatal screening for urological anomalies should be stepped up

ABS-OR-1036

CORRELATION OF ANTHROPOMETRIC MEASUREMENT WITH RENAL SIZES IN CHILDREN

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Background: Renal dimensions are important for diagnosis and prognosis of nephropathies. They are dependent on anthropometry.

Objective: To determine the renal sizes in children sonographically and to correlate these measurements with anthropometric measurements.

Methodology: This is a prospective renal ultrasonography on patients attending our out -patient clinic between June and August 2009. Weight and height were done using standard methods while ultrasound measurement was done using Siemens ultrasonography machine. Longitudinal ,transverse and AP measurements were taken and recorded to nearest millimeters. Surface area(SA) was calculated using the

formula and BMI using Weight in kg/ height in meters 2. Renal volume was also calculated. P values </= 0.05 was regarded as significant.

Results:A total of 100 subject were enrolled into the study 48% were female while 52% were males. In male infants the left kidney bipolar length, AP and transverse parameters were significantly larger than the right measurements(P=0.02,0.000,0.005 respectively) The left kidney measurements in the male preschool and school were bigger however not significantly so(p=0.19) The mean right kidney in the adolescents was 67.0SD7.04 and the left kidney 70.5SD6.85 (p= 0.085) .The left kidney volume was significantly larger than the right kidney (p=0.000)

Conclusion: It was concluded that kidney sizes increases early in infancy as well as during adolescent and that age and weight are strong determinants of kidney volume.

ABS-OR-1037

MANAGEMENT CHALLENGES IN A SMALL CHILD WITH ACUTE RENAL FAILURE OF UNKNOWN CAUSE

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Background: Acute renal failure can be a severe life threatening condition especially in small children

Objective: To highlight management challenges encountered while managing the case.

Methodology: A case report on A.B who presented with severe acute renal failure.

Results: AB was a 3 year old girl with rickets, recurrent oedema and hypertension which culminated into acute renal failure. She had three sessions of haemodialysis after which patient had remarkable recovery, though materials had to be sourced from a very far distance. There were diagnostic and treatment challenges but dialysis presented a special challenge as there were no appropriate dialysers, material for access. There were problems with making an accurate diagnosis, limited response to treatment and eventually developed acute renal failure (ARF) necessitating dialysis. Haemodialysis presented a special challenge because of lack of material for vascular access, appropriate dialysers. The report of how these challenges were overcome is presented.

Conclusion: Facilities for paediatric haemodialysis of children of all ages should be made available in all our tertiary institutions.

ABS-PO-2001

OCCURENCE OF RHEUMATIC HEART DISEASE AND ACUTE GLOMERULONEPHRITIS

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Introduction: Rheumatic heart disease and acute glomerulonephritis rarely co-exist. Only few occurrences have been reported in the literature with none among blacks of African descent.

Case Report: We report a 13 year old girl from South West Nigeria who presented in our hospital with 3 month history of cough, orthopnea and breathlessness both on exertion and at rest and three weeks history of bilateral pitting oedema up to the sacrum from the foot. T here was previous history of leg swelling some months prior to this presentation which resolved without hospitalization. There was associated raised jugular venous pressure and pan systolic murmur radiating to the axilla with pulmonary accentuation. Blood pressure of 170/120mmHg at admission. Proteinuria was 3+ at admission however serum protein albumin and triglyceride were within normal range. Serum creatinine was on the upward trend from 315umol/l at admission. Erythrocyte sedimentation rate was 45mm/hr. Urine output was between 0.3-1.1mls/kg/hr and the oedema never really subsided. Echocardiography suggested rheumatic heart disease and mitral incompetence.

Conclusion: In view of the presence of cardiac symptom and increased erythrocyte sedimentation rate with oedema, hypertension, azotaemia and previous history of body swelling, a suspicion of RHD and AGN which may have progressed to CGN is suspected. These remain rare combinations with few reports in the literature and in Blacks living in Africa.

ABS-PO-2002

PSYCHIATRIC MANIFESTATION IN A CHILD WITH URAEMIA: A CASE REPORT

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Introduction: Uraemia manifest in various ways including central nervous system manifestations. Common CNS manifestations include drowsiness, slurred speech, memory loss, seizures and coma. Psychiatric manifestations are rare but could present as acute confusional state.

Case report: We report a12year old boy who first presented in our clinic with anasarca. Examination revealed generalized oedema, massive proteinuria, hypoproteinaemia, hyperlipidaemia. A diagnosis of nephrotic syndrome was made. He was admitted, commenced on diuretics and later had steroids. However, the oedema wanes and recurs until oedema no longer resolve with diuretic alone necessitating plasma transfusion to which the patient responded for a while only to become severely oedematous again. On the 35th day, he began to talk excessively and irrationally. He was also talking and singing to self loudly on the ward. He was aggressive, restless and insomniac. Analysis of his speech indicated that he was talkative and was also talking out of context. His behavior was suggestive of someone having visual hallucination but there was no evidence of delusion. He had no family history of psychiatric illness and has never had any episode of

psychiatric illness. Low dose haloperidol (antipsychotic drug) was added to his medication by the psychiatrist, to which he responded positively. The creatinine was found to be on the upward trend. He had about 4 sessions of dialysis during which the oedema will resolve only to resurge again. At a point they could no longer afford dialysis anymore and he had to be placed on diuretics, antihypertensive and steroid and discharged to both the psychiatric clinic and the nephrology clinic. All the psychiatric symptoms have resolved, oedema has subsided and serum creatinine has normalized.

Conclusion: In view of the absent family and previous history of psychiatric illness in this child, we are of the opinion that psychiatric manifestation is as a result of uraemia.

ABS-PO-2003

CONGENITAL MESOBLASTIC NEPHROMA- CASE REPORT

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O.A is a 12 day old male neonate who was referred to OOUTH on the 21st of September 2007 with a provisional diagnosis of intestinal obstruction but was found to have renal mass at surgery and histopathology reported congenital nephroblastoma.

ABS-PO-2004

UNDIAGNOSED HYPERTENSION AND PROTEINURIA IN A MARKET POPULATION: RESULTS OF WKD 2009 SCREENING EXERCISE

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Background: Hypertension and kidney diseases are common in our urban and rural communities but majority of affected individuals do not know. We conducted free medical examination and screening during WKD 2009 which happens to coincide with one of the market days in Odo-Ogbe market, Ile-Ife, Nigeria to be able to detect possible magnitude of undiagnosed hypertension and possibly kidney disease.

Aim and Objectives: To find out the percentage of the participants with undiagnosed hypertension or proteinuria. We also try to ascertain the anthropometric correlates of both in the studied population.

Materials and Methods: Participants were taken through a brief medical history and had their socio-demographic data and anthropometric data taken. Blood pressure was assessed using mercury sphygmomanometer on the left arm using a standard cuff size, with the patients in sitting position while urinalysis was done with the aid of diptick. Weights were taken using bathroom scale while the heights were recorded using stadiometer. Data was analysed using SPSS package version 16.

Results: A total of 286 participants aged 13 – 90 years (Mean ± SD; 49.53 ± 15.65yrs) were screened. There was female preponderance with 278 (90.2%) being females. Systolic and diastolic blood pressures

ranged between 90-220 mmHg and 50-120mmHg respectively, 108 (37.7%) of participants had hypertension out of which only 20 (6.7%) were previously diagnosed. Sixty nine (24.1%) of participants had stage 1 hypertension while 39 (13.6%) had stage 2 (JNC VII). The BMI ranged between 15.6 and 46.6 Kg/m^2 (Mean ± SD; 26.76 ± 5.28 Kg/m^2). The 59.2% of participants had BMI above 25Kg/m^2. Eighty five participants (29.7%) had proteinuria while only 13 (4.5%) had glycosuria. A significantly higher percentage of participants with stage 2 hypertension had proteinuria compared with stage 1 or non hypertensives ($p<0.0001$). Similarly, a significantly higher percentage of participants with hypertension had glycosuria compared with non hypertensives ($p=0.009$). There was a good correlation between age and BMI($r=0.171, p=0.004$) as well as Systolic ($r=0.378, p<0.0001$) and diastolic blood pressures ($r=0.197, p=0.001$). The correlations were sustained even after correcting for age.

Conclusion: A high percentage of the studied population (31%) had undiagnosed hypertension and proteinuria (29.7%) while BMI significantly correlated with blood pressures. Community screening for these disabling non communicable diseases and lifestyle modifications should be encouraged.

ABS-PO-2005

THE PREVALENCE AND PATTERN OF UNDIAGNOSED HYPERTENSION AMONGST FAMILY PRACTICE POPULATION IN ILE-IFE, NIGERIA

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Background: The prevalence of hypertension and its complications amongst the black race is high and constitute a significant burden of the health problems. We set out to determine the prevalence of hypertension and its correlates amongst our family practice population.

Methods: All new patients attending the family practice clinic (general outpatient department)over a periods of six months were screened for hypertension. The diagnostic criterion used was as in the JNC VII or WHO guidelines. Those found to be hypertensive were further investigated. Their anthropometric parameters were taken and their serum chemistry, fasting blood sugar and lipid profile were determined. In addition their abdominal ultrasound scan was done to assess structural abnormalities of abdominal viscera particularly the kidneys.

Results: Out of a total of 1106 patients that were screened, 216 (19.6%) were found to be hypertensive. The age of the respondent s ranged between 17-82 years (Mean 57.53 ± 13.02) and majority of them were females 61.4%. Mean systolic , Diastolic Blood Pressure , Serum Creatinine and Urea were 172.9 (±19.2) mmHg, 95.42 (±14.72)mmHg, 94.50 (±42.13) µmol/L, 5.94(±11.62) mmol/L. The Mean total cholesterol, Triglyceride and HDL were 4.39(±0.99) mmol/L, 1.07(±0.66) mmol /L, 1.20(±0.61) mmol /L. 58% of the patients were either overweight or obese while 40% had glomerular filtration rate (GFR) less than 60 mls / min. Age of the patient significantly correlated with Systolic and Diastolic blood pressures with $p=0.001$ and $p=0.000$ respectively. Triglyceride also significantly correlated with BMI, waist-hip ratio $p=0.035$ and 0.007 respectively. GFR negatively correlated with age and systolic blood pressure.

Conclusion: A high proportion of our patients have CKD. Regular community screening and Preventive programme mounted at the primary and secondary care levels would assist in retarding progression.

ABS-PO-2006

INCOME DISTRIBUTION AND SOURCES OF FUNDING FOR MAINTENANCE HAEMODIALYSIS OF PATIENTS IN THE UNIVERSITY OF PORT HARCOURT TEACHING HOSPITAL

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Background: End stage renal disease (ESRD) is prevalent in Nigeria, with attendant high morbidity and mortality rates. In Nigeria, the evidence for the inability of ESRD patients to pay for their treatment has mostly been empiric. Similarly there has been no formal study of the sources of funding for dialysis in the country. Such studies when replicated across the country will provide an evidence based tool to engage Government on the need for a Government driven ESRD program.

Methods: A prospective direct questionnaire based study of End stage renal disease patients receiving maintenance haemodialysis was conducted at the University of Port Harcourt teaching hospital.

Results: Twenty four (24) males and 16 females(M/F=1.4:1) were studied, with mean age of 40.62 $\pm$ 14.9 years, mean e-GFR , 6.53 $\pm$ 1.6 mls/min. and mean duration on dialysis of 5.03 $\pm$ 1.6(3-12) months.The mean annual income of the patients was N1, 147, 172.02 (N60, 000.00 to N3,200,000.00).The estimated annual cost of haemodialysis in Port Harcourt per patient is N2,340,000.00. Sixty (60) percent of the patients earned below one million naira per annum. Only 10 percent of the patients earned over 3 million naira p.a .The annual incomes of 62.5% of the patients were less than fifty percent the annual cost of dialysis.
Annual incomes showed positive correlation with the duration on dialysis(r= + 0.14) and number of dialysis sessions received (r= +0.3). Dialysis was funded from family income in 65 percent of the cases. Funding was from extended family members in 17.5% and philanthropic sources in 10% of cases. There was no Government support to any patient or funding through insurance.

Conclusions: The annual incomes of the great majority of ESRD patients are less than 50 percent of the annual cost of maintenance haemodialysis and cannot sustain optimal long term haemodialysis. A Government driven ESRD Care program is therefore inevitable in the country if we are to improve access to haemodialysis.

Keywords: INCOME DISTRIBUTION, SOURCE OF FUNDING, MAINTENANCE HAEMODIALYSIS, UNIVERSITY OF PORT HARCOURT TEACHING HOSPITAL

ABS-PO-2007

CREATING AWARENESS AND COMPARATIVE SCREENING FOR DIABETES IN RURAL AND URBAN COMMUNITIES IN OGUN STATE, NIGERIA

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Background/Aim: The 21st century has the most diabetogenic environment in human history. In 2007, there were 246 million people with diabetes in the world, but by 2025, that number is estimated to reach 380 million. Diabetes is now the major cause of end stage kidney failure throughout the world in both developed and emerging nations. The focus on diabetic kidney disease for World Kidney Day 2010 brings awareness of the magnitude of the problem and ramifications for global health for people with diabetes and kidney disease. Diabetes mellitus is the most common endocrine disorder in Nigeria. The role of community

participation in the prevention of diabetes and hypertension cannot be overemphasized. This informed the study, with the aim of creating awareness at the grassroot community level, emphasising preventive measures.

Methods: In 2007, a diabetes awareness campaign with free blood glucose screening, aimed at preventing diabetes was conducted within rural Isara community and urban Sagamu community in Remo division in Ogun state, Nigeria. Diabetes was defined as fasting blood glucose > 126mg/dl and random blood glucose > 200mg/dl. Hypertension was defined as blood pressure measurements > 140/90 mmHg. Obesity was also assessed using Body Mass Index and the waist – hip ratio. Data was analyzed using SPSS software version 13.

Results: In rural community of Isara, two hundred and forty respondents (18-80years) were screened during the campaign for diabetes and hypertension. The mean age, Body Mass Index and Waist-Hip ratio were 53.9+15.7years, 25.9+4.8 Kg/m2 and 0.91+0.08 respectively. The mean random blood glucose was 102.9 + 25.5 mg/dl. The mean systolic blood pressure was 134.2+24.8 mmHg, while the mean diastolic blood pressure was 78.7+14.4mmHg. In the urban Sagamu community, a total of 340 respondents were screened. The mean age, Body Mass Index and Waist-Hip ratio were 47.7+15.4years, 28.8+6.3 Kg/m2 and 0.99+0.1 respectively. The mean fasting blood glucose was 95.2+32.9 mg/dl. The mean systolic blood pressure was 128.8+18.6 mmHg, while the mean diastolic blood pressure was 82.1+12.4mmHg.

Conclusions: Our findings suggest that overweight and obesity are becoming a public health burden in the urban Nigerian community. Creating awareness on diabetes and hypertension and instituting lifestyle modification measures to curb non-communicable disease and obesity are of paramount importance.

ABS-PO-2008

PREVALENCE OF ANAEMIA AND OTHER HAEMATOLOGIC DERANGEMENTS IN END STAGE RENAL DISEASE PATIENTS IN THE UNIVERSITY OF PORT HARCOURT TEACHING HOSPITAL

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Background: Anaemia and other haematologic derangements are common in patients with chronic kidney disease, especially end stage renal disease. Anaemia is an independent risk factor of cardiovascular morbidity and mortality in CKD. The prevalence of anaemia and other haematologic derangements in the population of ESRD patients before commencement of maintenance dialysis at the University of Port Harcourt Teaching hospital (UPTH) is not known.

Objective of study: To determine the prevalence of anaemia and other haematologic derangements in dialysis naive end stage renal disease patients in the University of Port Harcourt Teaching Hospital.

Methods: A retrospective analysis of the haematologic indices of ESRD patients at the UPTH from January to December 2007 was conducted.

Results: Fifty males and 20 females (M/F= 2.5:1) were studied, with mean age of 44 $\pm$ 17.0(18-85) years and mean e-GFR of 7.1 $\pm$ 2.1(3.5-10.8) mls/min. Mean haematocrit was 22.8 $\pm$ 3.1(10-38) percent, with mean haemoglobin concentration of 8.8$\pm$3.1(3.3-16) g/dl. Others were mean ESR, 93.1 + 45.1(7-136)mm/hr, mean peripheral total leukocyte count 7,533.5 $\pm$ 3,949.6(2,499-18,800)/mm^3 and a mean platelet count of 145,000 $\pm$ 66,605 (60,000-400,000)./mm^3 . Anemia was the dominant haematologic abnormality occurring in

66 (94.3%) patients. Moderate to severe anaemia occurred in 58 (82.9%) of the patients. Twelve patients (17%) had leukocytosis, 2 (2.9%) had leucopenia and there were no abnormalities in platelet count. The e-GFR of the patients showed positive correlation with haematocrit (r= +0.2) and haemoglobin concentration (r= +0.1) respectively, while serum urea and creatinine showed negative correlation with haematocrit (r = -0.2) and haemoglobin concentration (r = -0.2) respectively.

Conclusions: Anaemia was the dominant haematologic abnormality in dialysis naïve ESRD patients in the University of Port Harcourt teaching hospital. Both haematocrit and haemoglobin levels showed positive correlation with e-GFR. There is need for more attention to be paid to the correction of anaemia in our patients.

Keywords: HAEMATOLOGIC ABNORMALITIES, END STAGE KIDNEY FAILURE, UNIVERSITY OF PORT HARCOURT TEACHING HOSPITAL

ABS-PO-2009

PREVALENCE OF RISK FACTORS FOR CHRONIC KIDNEY DISEASE IN A RURAL ADULT POPULATION IN RIVERS STATE

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Background: The prevalence of chronic kidney disease (CKD) is on the increase globally with attendant heavy disease burden and high morbidity and mortality especially in the resource poor countries. Due to the magnitude of CKD burden and the high cost of care, especially for end stage kidney disease, preventive measures are increasingly being explored. Early detection of modifiable risk factors of CKD in population groups and early intervention is the strategy to possibly prevent and reduce the incidence and prevalence of CKD in the population. We undertook a survey to determine the prevalence of some risk factors of CKD and identify the at-risk individuals.

Method: Body mass index (BMI), dip-stick urine protein and urine glucose, random blood glucose and blood pressures were measured in adult subjects of Barako, a rural community in the Gokana Local Government area of Rivers state during a one day Rotary eye camp exercise.

Results: Out of the 154 subjects that responded, 152 satisfied the inclusion criteria and were studied. They had a mean age of 48.9 $\pm$ 14.8(18-85) years and M:F ratio of 1:1.4. The mean body mass index (BMI) was 25.8 $\pm$ 4.8 (11.1-40 .9) kg/m^2. Forty-nine subjects (34.8%) were pre-obese while 13.5% were obese. Proteinuria was seen in 29.7% while none of the subjects had glycosuria. The mean random blood glucose was 6.6 $\pm$1.4(4.2-9.8) mmol/l. Four subjects (5%) were previously diagnosed diabetics. The mean systolic blood pressure was 129.9 $\pm$ 21.6(100-220) mmHg, mean diastolic blood pressure was 70.9 + 13.1(50-110) mmHg and the prevalence of hypertension was 27.9%. BMI showed positive correlation with proteinura (r = +0.2), while both systolic and diastolic blood pressures showed weak positive correlations with proteinuria (r = +0.02 and r = +0.06 respectively).

Conclusions: The study shows, that the evaluated risk factors of CKD, obesity, hypertension, diabetes and proteinuria are common in this rural community of Rivers State. The subjects of the community are at risk of CKD and there is therefore need for intervention.

Key words: CHRONIC KIDNEY DISEASE (CKD), RISK FACTORS, RURAL COMMUNITY, RIVERS STATE

ABS-PO-2010

SCREENING FOR CHRONIC KIDNEY DISEASE IN OIL PRODUCING COMMUNITY IN RIVERS STATE

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Background: Petroleum product had been associated with acute and chronic kidney disease. Nigeria is a major oil producing and exporting country. The degree of impact of these products in kidney function of communities in Nigeria where oil is being explored is not known.
Aims and objectives: To determine the frequency of chronic kidney disease in an oil producing community.

Subjects and Methods: This is a pilot study.Tthe study location was Ido in Asari Toru Local Government Area of Rivers State. All subjects aged 18 years and above were screened. Their height, weight, blood pressure were recorded. Their urine was collected for dipstick urinalysis, blood was collected for electrolyte, serum urea, serum creatinine and lipid profile. The body mass index was calculated using height and weight, the glomerular filtration rate was calculated using Cockcroft and Gault fomular. The data was analysed using SPSS vs 13..

Results: A total of 99 subjects were screened, 64% were female, less than 15% were aged 30 years and below while16.7% were above 60 years. The mean GFR was 80.2+33.3ml|min, 32.6% had GFR less than 60ml|min, and 2.2% had GFR less than 30ml|min. the GFR had a significant negative correlation with family history of diabetes mellitus (-.35, p - 0.016) and total cholesterol(-2.2, p – 0.045), positive correlation with BMI(0.24, p – 0.018). 36.5% had proteinuria, 29.8% 1+, 5.7% 2+, 1% 3+.

Conclusion: The rate of chronic kidney disease is high in oil producing community, and exposure to crude oil is a likely contributory factor.

ABS-PO-2011

DIALYSIS TREATMENT FOR ACUTE KIDNEY INJURY IN ADULT PATIENTS AT THE UNIVERSITY OF PORT HARCOURT TEACHING HOSPITAL: PREVALENCE AND CLINICAL CHARACTERISTICS

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Background: Acute kidney injury (AKI) in adults is a common and frequent cause of hospital morbidity and mortality especially in the developing countries. A proportion of AKI patients require renal replacement therapy (RRT) in the course of their management. Information on the hospital prevalence and the clinical attributes of such adult AKI patients treated by dialysis in Nigeria is sparse.

Objective: To determine the prevalence, epidemiologic and clinical characteristics of adult patients who received RRT for AKI in our hospital.

Methods: A retrospective analysis of the clinical data of all non-intensive care unit (non-ICU) adult AKI patients treated with RRT in the form of intermittent haemodialysis during an interrupted six year period (1997-1999 and 2007-2009) at the University of Port Harcourt teaching hospital was conducted.

Results: During the periods understudy a total of 6151 medical admissions were recorded, of which 614 (9.9%) were chronic kidney failure and 121 (1.9%) AKI patients. Of the 121 cases of AKI, 62 (51.2%) received intermittent haemodialysis. Thus the prevalence of dialysis treated adult AKI patients was 1.0%. They constitute 8.4% of kidney failure and 51.2% of AKI patients. They comprised 34 males and 28 females (M/F = 1.2:1) with a mean age of 41.3±18.5 (13-83) years. The clinical settings for AKI for these dialysis treated patients were medical 44 (70.9%), surgical 15 (24.2%) and pregnancy related 3 (4.8%). The indications for dialysis in the patients were severe azotaemia at presentation or rapidly rising azotaemia 39 (48.4%), uraemic encephalopathy 20 (32.3%), severe metabolic acidosis (serum bicarbonate d" 15 mmol/L) 19 (30.7%), acute pulmonary oedema 3 (4.8%) and severe hyperkalaemia (plasma potassium e" 6.5mmol/L) 3 (4.8%).
Conclusion: AKI patients who received dialysis constitute about fifty percent of our AKI patients and a great majority present in the failure grade of RIFLE criteria.

ABS-PO-2012

CASE FATALITY AMONG PATIENTS WITH CHRONIC KIDNEY DISEASE IN UNTH ENUGU .

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Background: Chronic kidney disease (CKD) incidence is increasing in our environment. Most of the patients present late so mortality is very high.

Aims and objectives: To determine the death rate and causes of death among patients with CKD in an urban tertiary hospital in 2006.

Materials and methods: The medical records of in-patients with CKD admitted into the medical wards of University of Nigeria Teaching Hospital, Enugu between January to December 2006 were reviewed. From the available data, we calculated the case fatality for the period of study.

Results: During the period, 2610 admissions were made into the medical wards. Of these 55(2.1%) were due to CKD. Of the CKD admissions, the male female ratio was 34(61.8%): 21(38.2%). Thirty seven of the CKD patients died giving a case fatality of 67.3%. Of 20 patients that died from CKD, uraemic encephalopathy and congestive cardiac failure were the commonest causes of death accounting for 55% and 25% respectively. Other causes of death were cerebrovascular accident, anaemic heart failure, hypertensive encephalopathy and post dialysis hypoglycaemia, each accounting for 5%.

Conclusion: CKD is an important cause of hospital admission. The case fatality is very high most of which are preventable. There is need to institute a free dialysis programme in government owned hospitals.

ABS-PO-2013

PREVALENCE OF CHRONIC KIDNEY DISEASE IN A RURAL NORTHERN NIGERIAN SETTLEMENT

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Background: Chronic kidney disease (CKD) has been increasing globally. However, there is limited data about the population prevalence of CKD in developing countries.

Aims and objectives: The aim of this study was to investigate the prevalence of CKD in adults in a rural settlement in Northern Nigeria.

Materials and Methods: A crossectional study was carried out. Using a multistage stratified random sampling, 480 adults were recruited into the study. Relevant demographic and clinical data were obtained using a questionnaire. Urine and blood Samples were taken for relevant investigations. Results were analysed using SPSS for windows.

Results: Overall, CKD (defined based on K/DOQI definition) was found in117 (26%) of the study population. The prevalence of various stages of CKD was 20% for stage 1, 3.6% for stage 2, 0.7 % for stage 3, 0.9% for stage 4 and 0.7% for stage 5.

Conclusions: CKD, especially the early stages, is common in the study population, providing a good opportunity for community based prevention strategy.

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