UNIVERSITY OF ILORIN

THE ONE HUNDRED AND SEVENTY-SIXTH INAUGURAL LECTURE

"BEHAVIOUR OF AN AMAZING ORGAN, EVEN AT THE EXPENSE OF ITS SURVIVAL: *NIHIL EST MALI APUD NIGROS*"

By

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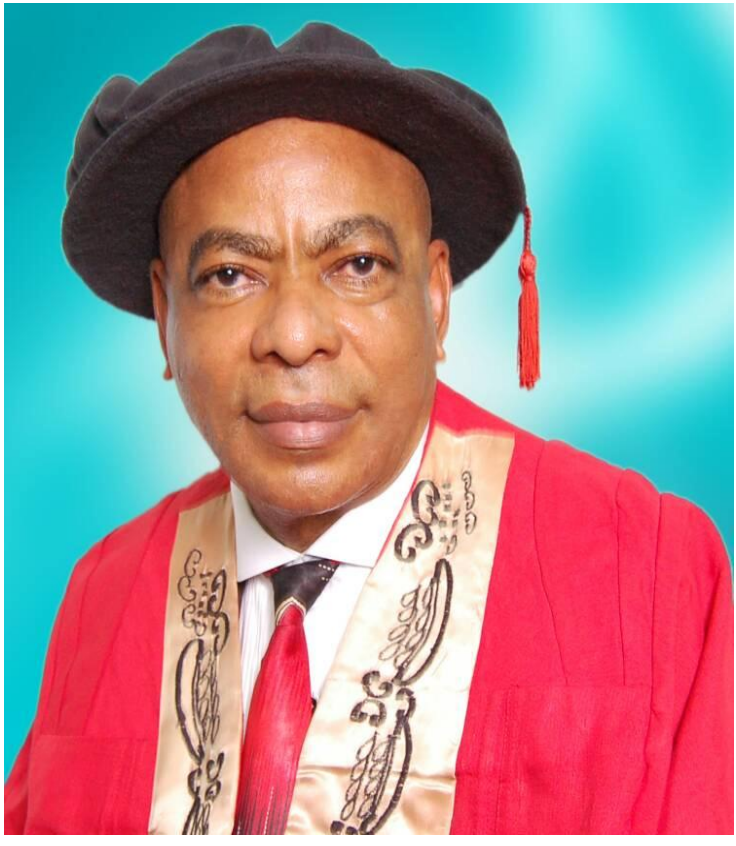

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All other Academic Colleagues,
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My Lords, Spiritual and Temporal,
Distinguished Students of Medicine,
Esteemed Invited Guests, Friends and Relatives,
Great Students of the University of Ilorin (Greatest Unilorites),
Gentlemen of the Print and Electronic Media,
Distinguished Ladies and Gentlemen

1.0 Preamble

I reverence God Almighty for granting me the uncommon grace and wisdom to realise my goals and aspirations in life. Success is predictable as you can only succeed when you replicate what other successful people

have done. You are on the pedestal of success if you set goals and take firm decision to succeed with absolute resolve to pay the price in full. You should resolve to shun all manner of distractions as those who develop obsession for throwing stones at every dog that barks on their way to success never really succeed. Assuredly, the true measure of success is to beam with smiles as you reflect on the past. Truly, as I go down memory lane, I have more than enough reasons to smile and express gratitude to God for making today a reality. I also thank God for Western Civilization and Christianity that abolished an evil practice of killing twins, for I would have been a waste material for the evil forest, soon after birth, as one of a set of twins.

I am grateful to our amiable Vice Chancellor, Prof Sulyman Age Abdulkareem for this wonderful opportunity to showcase my modest contributions to knowledge in the presence of our esteemed audience. This is the 6th in the series of Inaugural lectures from Department of Medicine and One Hundred and Seventy-sixth in the University. The first was "The Normal Electro-Cardiogram (ECG) In Adult Nigerians" delivered by Prof. M. A. Araoye on the 24th Apr, 1986 as the 22nd in the university. The second, "Of Bacterial Pathogens and Diarhoea: Making Visible The Invisible Link" was delivered by Prof. B. J. Bojuwoye on 13th May, 2004 while the third "The Choice is yours but the Burden is Ours" was presented by Prof. Ibrahim Adeola Katibi on 28th March, 2013. The fourth from the department entitled "Towards Better Prevention / Control of Hypertension and Diabetes" was delivered by Prof. E.O. Okoro on 27th February, 2014 while the fifth entitled "Realities of Living with HIV Infection" was presented by Prof. Alakija Kazeem Salami on 26th June, 2014. When I

ruminated on the most appropriate title for my lecture, my thought went through the following: (1) The Sacrifices of One for the Paradise of All: Nothing is Amiss in Blacks, (2) Demystifying the Mysteries of the Black Kidney: Nothing is Amiss in Blacks (3) Travails and Puzzles of an Amazing Organ: Nothing is Amiss in Blacks. Today I feel highly honoured and privileged to deliver the lecture from my Department titled: "Behaviour of an Amazing Organ, Even at the Expense of its Survival: *Nihil Est Mali Apud Nigros"*. It is my candid desire that my thoughts will add value to our existence as we consciously pay attention to the well-being of an amazing and sympathetic organ in this discourse.

2.0 Introduction

Mr. Vice Chancellor Sir, I wish to introduce to you an amazing, compassionate, passionate and versatile organ that God has equipped with certain peculiar qualities to enable it perform uncommon functions which are lacking in most organ systems of humans. This precious organ whose weight is 0.2% to 0.4% of the total body weight has one function that it shares only with the brain (1.7% to 2.0% of the body weight). The brain and the kidneys are the only organs in human body with inherent ability to auto-regulate their vascular perfusion and certain vital functions despite marked disparity in weight. This sympathetic organ is structurally endowed with capillary characteristics that marked it out from other organs. It is so passionate in its protective function of other organs that is often detrimental to its own survival. The organ is known among the Yoruba as "Kidinri", Igbo as "Akiri", Hausa as "Kuda", French as "Un Rein", Latin as "Renes" and "Kidney" in English language.

Mr Vice Chancellor Sir, the behaviour of the kidney is essentially the same among the Black and White populations with specific reference to structure and functions, vulnerability to injury, major aetiological factors and responses to injury. However, disparities exist in healthy and diseased kidneys as it pertains to effects on other organ systems, sensitivity to internal and external environment, mode of clinical presentation, available/accessible diagnostic facilities, awareness and attitude of the population to kidney dysfunction, reaction of the patients and relatives to end stage renal disease, treatment options, preventive measures, perception of outcomes of kidney problems and government attitude to kidney care.

According to the National Bureau of Statistics (NBS,) as at 2016, about 67.1% of the country's total population live below poverty line, using a poverty line of < 1USD/day.. The latest WHO data for Nigeria reveals that average life expectancy at birth for males and females is 53 years and 56 years respectively. The adult literacy rate for males is 58% and 29% for females and about 60% have access to portable water and 30% to good sanitation. It is obvious from the foregoing that renal care is beset with cultural, societal and economic hurdles which must be crossed in a bid to achieve ultimate success.

Mr Vice Chancellor Sir, the burden and enormity of kidney disease of varied etiologies are more prevalent in Sub-Saharan Africa, including Nigeria than in developed nations because of what is considered to be a double jeopardy. While the developed nations contend with non-communicable diseases that affect the kidneys, most Black nations battle with both communicable and non-

communicable diseases of the kidney. Studies have unequivocally shown that status of renal diseases that culminate in end stage renal disease is essentially the same in Blacks and Whites. The current prevalence of CKD is similar among Blacks and Whites in contrast with earlier studies that documented 3.8-fold racial disparity in which Blacks were more prone to CKD (USRD: 2004 Annual Report). The observed disparity is attributable to differences in the rate of progression of CKD and overall survival in advanced CKD. The Whites die earlier from cardiovascular events in contrast with Blacks while Blacks progress fasters to ESRD (Clase CM et al, 2002)

3.0 Normal structure and function of the kidney

The kidneys are two bean-shaped organs, anatomically located in the retroperitoneal space at the level of the lower thoracic and upper lumber vertebrate (T12-L3). Each weighs about 150gms and measures approximately 10cm and 12.5cm in bipolar length by ultrasound and IVU respectively.

The kidney is a highly vascularised organ per gram of tissue and about 25% of cardiac output passes through the kidneys. Renal arteries and veins are derived from abdominal aorta and inferior vena cava respectively. The renal poles are relatively avascular except in about 30% population with aberrant (accessory) renal arteries.

The functional unit of the kidney is called the nephron which consists of the glomerulus, tubules and collecting ducts. Each kidney has about 1 million nephrons distributed in cortical and medullary region. The glomerulus consists of afferent arteriole, capillary network, bowman's capsule and efferent arterioles while the glomerular tuft is

made up of three or more aggregates of capillaries in which each capillary basement membrane do not completely encircle the capillary wall resulting in summersaulting of one basement membrane to the other. The tubular structure consists of proximal tubule, descending tubules, the Henle's loop, ascending tubules and the collecting ducts. The hairpin capillary loops (vasa recta) and the tubules are arranged in a fashion that allows the direction of fluid flow within them to be in counter current manner. This characteristic arrangement is what equips the kidney with ability to either dilute or concentrate substances in blood circulation.

Figure 1: Structure of the Kidneys (Gross)

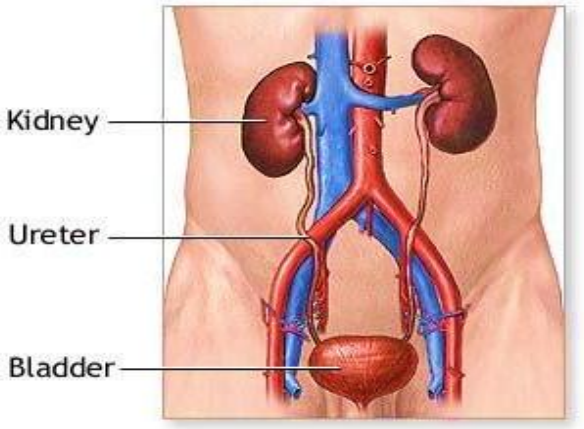

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Figure 2: The Nephron

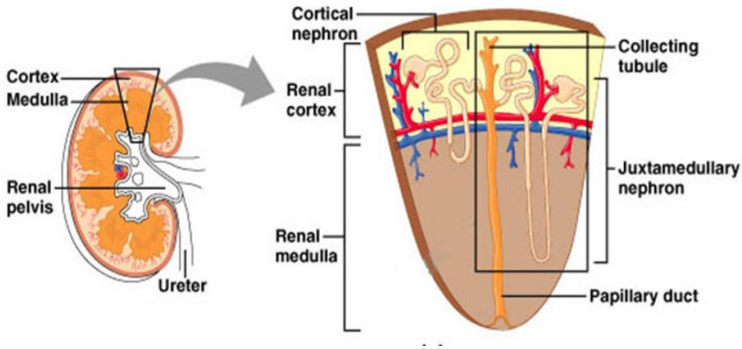

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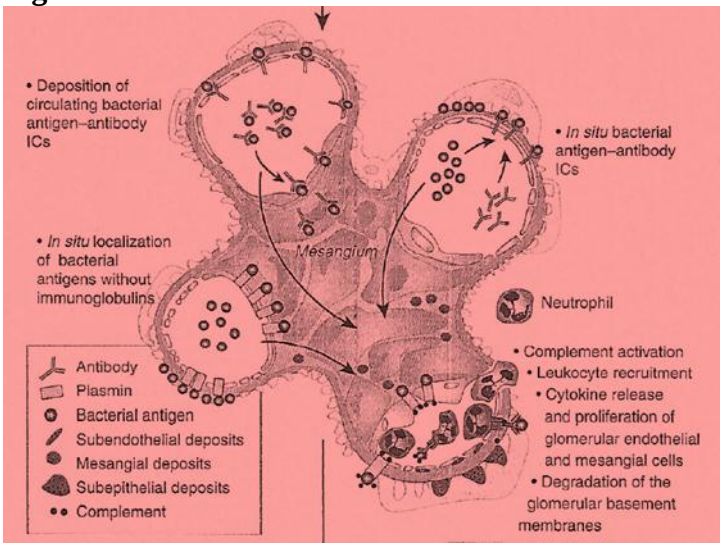
Figure 3: Structure of the Glomerular tuft



(Nasr SH, et al, 2013)

3.1 Functions of the Kidney

1. Excretion of waste products of metabolism.
2. Maintenance of water and electrolyte balance.
3. Maintenance of acid-base balance
4. Metabolism of certain drugs and toxins into active or inactive metabolites.
5. Synthesis of some hormones/compounds:

a) Rennin production **(b)** Erythropoietin **(c)** Vitamin D

4.0 Reasons for kidney vulnerability to injury

1. It is highly vascularised with the highest perfusion per gram of tissue that varies from 5cc/g/min in outer cortex to 0.25cc/g/min in the inner medulla.
2. The high-blood flow rate (25% of cardiac output) ensures enormous contact between kidney tissue and injurious agent.
3. It possesses the largest endothelial cell surface by weight of any other organ in the body which exposes it to damage by immune complexes.
4. Renal concentrating mechanisms raise concentration of toxins and drugs in the medulla to higher levels than anywhere else in the body.
5. The mechanism of concentrating urine exposes the tubular epithelium to higher concentration of some potential toxic compounds.
6. There is increased tendency of ascending infections to gain access to the renal parenchyma from lower urinary tract in the setting of obstruction/stasis.

5.0 Kidney dysfunction

5.1 Acute Kidney Injury (AKI) or Acute Kidney Attack(AKA) connotes sudden rise in serum Creatinine(50%) from base line value and/or fall in GFR by 25% and/or decrease in urine output below 0.5 mls/kg/hour from 6 hours to 7days in previously normal kidneys. Current concepts of AKI diagnosis is based mainly on increase in serum creatinine. This classifies AKI according to Risk, Injury, Failure, Loss of function, and End-Stage Renal Disease (RIFLE) or AKI Network (AKIN) consensus criteria.

Table 1: Stages of Acute Kidney Injury (RIFLE criteria)

Stage	**GFR % Loss**	**Creatinine Level**	**Urine Output**
R	25%	1.5x increased	<0.5ml/kg/hr for 6 hrs
I	50%	2x increased	<0.5ml/kg/hr for 12 hrs
F	75%	3x increased	<0.3ml/kg/hr for 24 hrs
L	100%	Complete loss	Complete loss ≥ 1 month
E	ESRD	Persistent loss	Persistent loss ≥ 3 months

(Roy AK et al, 2013)

Table 2: Stages of Acute Kidney Injury (AKIN criteria)

Stage	Change in serum creatinine	Urine output
1	Increase ≥ 0.3 mg per dL (26.52 μmol per L) or ≥ 1.5- to twofold from baseline	< 0.5 mL per kg per hour for more than six hours
2	Increase > two- to threefold from baseline	< 0.5 mL per kg per hour for more than 12 hours
3	Increase > threefold from baseline or ≥ 4.0 mg per dL (353.60 μmol per L) with an acute rise of at least 0.5 mg per dL (44.20 μmol per L)	< 0.3 mL per kg per hour for 24 hours or anuria for 12 hours or RRT required

(Roy AK et al, 2013)

5.3 Chronic Kidney Disease (CKD) is defined as structural and/or functional abnormalities of the kidney for more than 3 months, with/without a decrease in glomerular filtration rate (GFR) or decline in GFR to less than 60ml/min/1.73m^2 for more than 3 months.

5.4 Classification of CKD

Table: CKD is classified into different stages of increasing severity using GFR as a reference.

Stage	Glomerular Filtration Rate (GFR)
Stage I	≥90mls/min
Stage II	60 – 89mls/min
Stage IIIa	45 – 59mls/min
Stage IIIb	30 – 44mls/min
Stage IV	15 – 29mls/min
Stage V	<15mls/min

(Roy AK et al, 2013)

5.5 Aetiology of AKI

Extracellular volume loss (Gastroenteritis, burns and urinary losses), Intravascular volume loss/redistribution (sepsis, hemorrhage and hypoalbuminaemia), Decreased cardiac output (Heart failure, cardiac tamponade and cardiac surgery) Vasomotor nephropathy (shock, trauma, sepsis & hypoxia), Nephrotoxins-antibiotics, analgesics, contrast media, heavy metal poisoning and high protein diet. Intratubular obstruction (myeloma, urate crystals, stones and rhabdomyolysis)

5.6 Aetiology of CKD

Congenital - polycystic kidney disease, Alport's syndrome, hypoplastic kidneys, Acquired - CGN, systemic HT, DM, CPN, CIN, Multisystemic disease (Amyloidosis, myeloma kidneys, SCD), Obstructive uropathy, Heavy metal poisoning, chronic analgesic abuse, CTD (Lupus Nephritis, PAN).

Epidemiology of ESRD is well investigated in Whites in contrast to Blacks, where there is no reliable data due to lack of national registries. Aetiology of ESRD in sub-Saharan Africa are hypertension, chronic glomerulonephritis and diabetes mellitus, while in developed world, the order is diabetes mellitus, hypertension and chronic interstitial nephritis (Naiker S, 2003, Chijioke A, et al, 2003). Huge proportions of ERSD in sub-Saharan Africa are of unknown etiology (30%, as opposed to 11% in whites) because of late referral, inadequate diagnostic facilities and paucity of manpower.

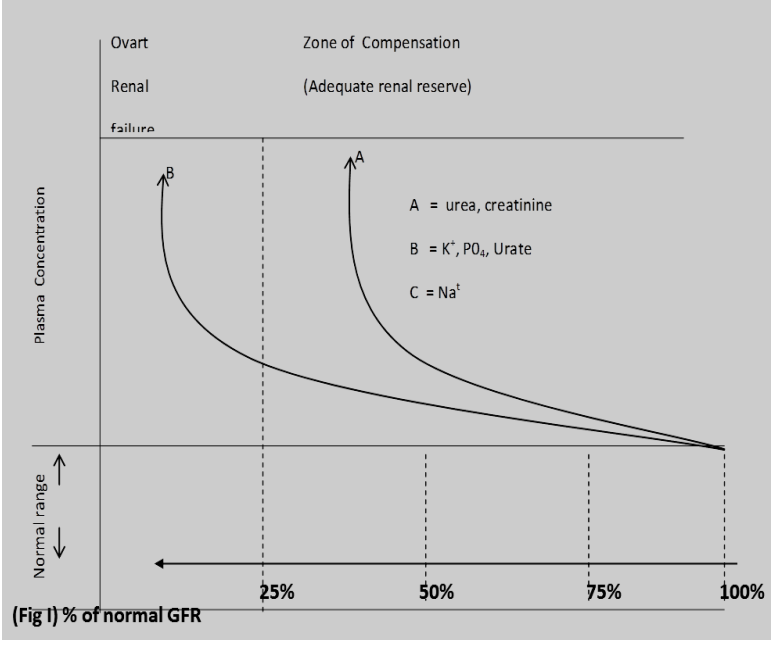
6.0 Pathophysiology of kidney failure

Renal insult by any of the aetiological agents results in reduction in the number of functioning nephrons and compensatory hyperfunction of the remaining nephrons. This leads to glomerular hyperfiltration and progressive damage of the surviving nephrons through glomerulosclerosis with subsequent secondary tubular atrophy. Therefore, nephron loss, glomerular sclerosis and tubular atrophy are usual findings in human end stage kidneys, irrespective of the primary lesion. The glomerular capillary thrombosis, mesangial cell injury and lipoprotein deposition within the mesangium are contributory factors in the pathophysiology of kidney failure.

Once CKD is established (CFR < 15mls/min), it tends to progress to ESRD irrespective of any form of therapeutic intervention (Levey AS et al, 2011). Therapy may slow down but does not stop the progression of chronic renal disease because of the presence of both reversible and irreversible factors implicated in the disease process which include systemic hypertension, UTI, Heart failure, high

protein diet, hyperphosphataemia, angiotensin 11 and dyslipidaemia that are potentially reversible(Epstein FH. 1998). The implicated irreversible factors include; epithelial growth factor, platelet derived growth factor, fibroblast growth factor, insulin like growth factor and nitric oxide. The renal functional reserve is remarkable in the sense that the kidney would have lost 50% of its glomerular functional mass before clinical and biochemical evidence of renal dysfunction.

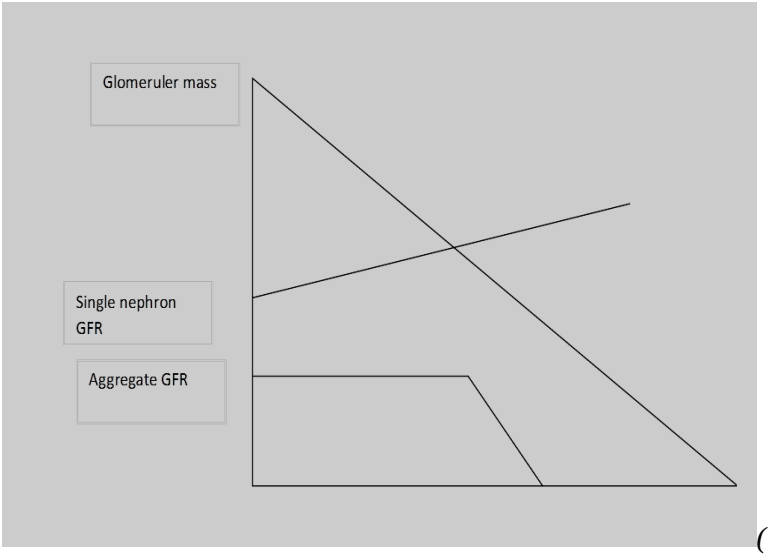
Figure 4: Relationship of GFR to serum biochemistry



(Lakhmir S. C, et al, 2016)

Kidney response to progressive loss of glomerular mass is increase in single nephron GFR and normal aggregate GFR until the glomerular mass in reduced to 50% of normal before reduction in aggregate GFR. The single nephron GFR however continues to increase till late in the disease process when virtually all the glomeruli are sclerosed.

Figure 5: Relationship of glomerular mass with nephron function



(Lakhmir S. C, et al, 2016)

7.0 Sensitivity to internal and external environment

The kidney is a perfect sensor that regulates the interplay between internal and external environment of an individual with intent to maintain electrolyte and water balance. It performs this function by its inherent ability to either concentrate or dilute solutes and solvents as they

circulate through the kidneys. The intake of plenty of water or water deprivation usually results in passage of dilute or concentrated urine subject to cold or hot season. Adequate knowledge about this regulatory function will enable us to know when to increase our fluid intake or decrease it, as very dilute urine will appear colourless while the concentrated urine will vary from deep amber to deep brown.

Therefore, our attention should be drawn to a possible kidney dysfunction when normal or increased intake of fluid leads to reduction in urine output, body swelling and/or weight gain. A recurrent facial and/ or body swelling should raise strong suspicion of kidney dysfunction regardless of the pattern of urine output as it may be reduced, normal or increased in proven cases of kidney failure. Water intake should be encouraged especially in tropical environment in comparison to temperate countries as insensible loss through the skin is enormous in hot weather with increased risk of dehydration. Adults with healthy kidneys in tropical settings are advised to take at least 4 to 5L of water in order to maintain healthy kidneys and other organ systems depending on the prevailing weather condition.

8.0 Mode of presentation of kidney dysfunction

The protean manifestations of kidney dysfunction often pass unrecognised. It is usually difficult to differentiate between reversible AKI, acute – on – chronic from terminal or end stage renal failure in majority of patients at first presentation as virtually all are characterised by oliguria. The clinical manifestations of renal diseases tend to lag behind the disease process in majority of cases

because of the inherent functional renal reserve and glomerulo –tubular balance. The earliest symptom is usually nocturia followed by malaise and easy fatigability. As CKD advances, organ system complications of ureamia begin to manifest and are itemised systematically for clarity.

Hypertension develops in more than 80% of patients with advanced CKD and virtually all ESRD patients have elevated BP. Its absence may indicate hypovolaemia or salt wasting kidney disease (medullary spongy kidney). The control of BP may be difficult even with combination of drugs and dialysis.

9.0 Availability and accessibility to diagnostic facilities

Diagnosis of renal failure (AKI and CKD) could be glaringly obvious in certain situations but extremely difficult in others especially among Blacks in resource poor settings. High index of clinical suspicion, updated knowledge, clinical skill, availability and use of relevant investigation facilities could be very helpful even where there are no nephrologists. Serum creatinine is currently a marker of kidney dysfunction because it takes about 48 to 72 hours to rise above baseline after AKI. The more sensitive markers for detecting early kidney injury within 2 to 6 hours after the event include neutrophil galactinase associated lipocalin (NGAL), kidney injury molecule(KIM) and cystatin C. Urine output alone is not a sensitive index for detecting loss of kidney function because patients with AKI, CKD or ESRD can present with oliguria, normal urine output and/or polyuria.

Renal imaging in the diagnosis of renal failure is increasingly being utilised. Renal ultrasound is a commonly

requested investigation because it is free from radiation and does not require contrast material for the procedure. It provides valuable information about kidney size, outline, echotexture and corticomedullary differentiation. Intravenous urogram (IVU) and contrast computed tomography (CT) are usually avoided because of the risk of radio-contrast nephropathy. Micturating cystourethrography and retrograde pyelography carry substantial risk of urinary tract infection (UTI), hence prior antibiotic coverage is usually advised. Retrograde pyelography is contraindicated in polycystic kidney disease because of risk of cyst infection. The kidney size may be normal or increased in a setting of ESRD. Kidney size may be normal or increased in certain genetic or infiltrative kidney diseases including polycystic kidney disease, sickle cell nephropathy, diabetic nephropathy, HIVAN, amyloid disease of the kidney, multiple myeloma, sarcoidosis, hypernephroma, renal tuberculosis and hydronephrosis.

Figure 6: Biopty Gun renal biopsy by the Lecturer with Resident Doctors

Figure 7: Renal Biopsy, using Tru-Cut needle

Renal biopsy is only done in CKD when kidney size is normal or near normal. Important indications for renal biopsy in patient with AKI include; AKI of unknown etiology, suspicion of glomerulonephritis, delayed recovery of AKI after 6 weeks of dialysis with no more recurrent insults, and systemic disease or allergic interstitial nephritis as cause of AKI, which may provide justification for lifesaving therapy.

10.0 Challenges in diagnosis of kidney dysfunction among Blacks

The prerequisites for accurate diagnosis includes: knowledge of the definition, various possible causes, presentations, complications, detailed history with high index of suspicion, thorough physical examination, appropriate investigation and interpretation. Despite prognostic relevance of classification and criteria using

serum creatinine, the diagnosis of AKI is delayed for 48 to 72 hrs. The diagnosis of AKI with novel renal biomarkers of tubular injury like NGAL is now possible within 2-6 hours of injury.

The factors that may suggest chronicity of kidney failure include: duration of symptoms for months and years, nocturia with hyposthenuria, very high urea and creatinine, anaemia of chronic disorder, bone disease, sexual dysfunction, pruritus, neurological complication, evidence of long standing hypertension, small kidneys on renal imaging, carbamylated haemoglobin, creatol and endothelin-1. Histological diagnosis is often limited to light microscopy with hematoxylin & eosin staining in resource poor settings. Immunofluorescence and electron microscopy facilities are lacking in most centres including Nigeria.

11.0 Awareness and attitude to kidney dysfunction and ESRD

There is overwhelming evidence that Blacks have the greatest burden of unrecognized and untreated chronic kidney disease. The poor are more prone to kidney diseases due to lack of access to goods and services, information about preventive measures, adequate nutrition and health (Li S et al, 2004). Despite similar prevalence rates of early stages of chronic kidney disease in Whites and Blacks, ESRD are about four times higher among Blacks with poverty further increasing the disparity (Volkova N et al, 2008*).* Poverty related conditions like infectious diseases secondary to poor sanitation, unsafe water supply, environmental pollutants and high concentrations of disease transmitting vectors continue to promote development of chronic kidney disease in Blacks. The exposure to

agrochemicals, dehydration, and consumption of contaminated water and use of traditional herbal remedies are frequently associated with chronic kidney disease in Blacks (Almaguer M et al. 2014, Ulasi II et al, 2010).

11.0 Treatment Options in Kidney Failure

11.1 Conservative Treatment: include strict fluid and electrolyte balance, restriction of protein, fat, salt and phosphate intake, control of blood sugar, hypertension, dyslipidaemia, active infection and anaemia.

11.2 Renal Replacement Therapy (RRT): This entails the substitution of renal function in situation of severe renal failure which has failed to respond to conservative measures. The therapy could be temporary as in acute renal failure or permanent in cases of end stage renal disease.

11.3 Dialysis

The basic principle of dialysis is the ability of crystalloids to diffuse down a concentration gradient through a membrane separating two solutions.

Types of Dialysis: (a) **Haemodialysis** [Hospital based, Home choice, Satellite station] (b) **Peritoneal dialysis** [CAPD, Intermittent (Home choice), CCPD, Automatic PD (NTPD and DTPD), Acute PD]

11.4.1.1 Haemodialysis (HD) is a form of extra corporal blood purification process that takes advantage of the properties of a semi-permeable membrane. It utilizes artificial membranes. The decision to utilise HD depends on rate of urea and creatinine turnover, availability of staff and facilities, safety of heparin, hemodynamic stability, risk of

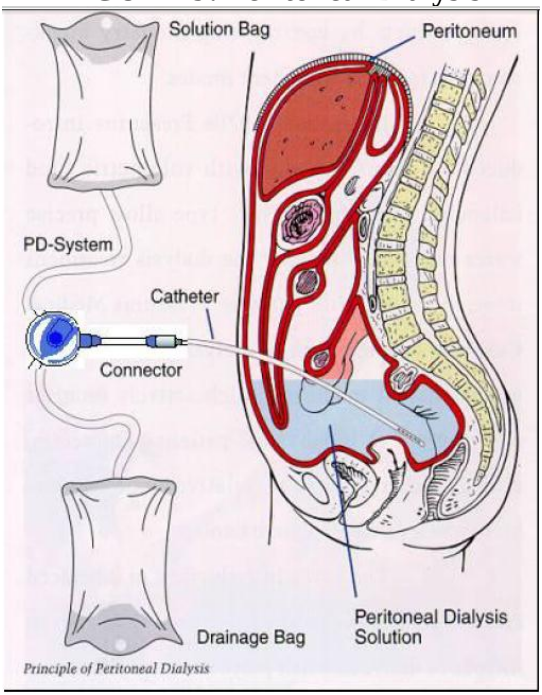
venous and pulmonary complications, and danger of placing a catheter in the peritoneum.

Complication of HD include hypotension, dialysis dysequilibrium syndrome (DDS), first use syndrome, anaemia, infections (HIV, HBV), aluminum dementia, plasticizer toxicity, bleeding, air embolism, vascular access complications and arrhythmias.

FIGURE 8: Fresenius machine *(left)* and Gambro machine *(right)*

11.4.1.2 Peritoneal Dialysis (PD) is a blood purification process that takes advantage of the semi-permeable properties of the peritoneal membrane. It entails the insertion of a temporary or permanent catheter and exchange of solutes between the capillary ramification of the peritoneal membrane and the dialysis fluid instilled into the peritoneal cavity.

FIGURE 9: Peritoneal Dialysis



(www.nephcure.org)

CAPD is the preferred mode of PD because waste removal occur on 24hrs basis, no machine is required, patient movement is unrestricted, diet is more liberal, it is more suitable for the very young, elderly, diabetic, heart disease and vascular problems, it is the preferred method for HIV positive patients. CAPD however bears some demerits such as peritonitis, tunnel and exit site infections, weight gain, frequent interruption of activities to exchange bags, and splinting of the diaphragm which may lead to atelectasis.

Hemodialysis is the most popular form of renal replacement therapy worldwide as 80% of patients on

hemodialysis are in the developed world with less than 15% in sub-Saharan Africa. Ironically, 85% of patients that merit hemodialysis in sub-Saharan Africa can hardly afford it.

FIGURE 10: Automated Peritoneal dialysis Machine

Renal replacement therapy is capital intensive and healthcare expenditures in most Black nations do not support dialysis, because funding is patient-driven, resulting in out-of-pocket expenditure. Seventy per cent of people in Sub-Saharan Africa live below poverty line (1 USD per day). Despite the foregoing limitations, the demand for hemodialysis continues to increase leading to expansion of existing facilities and establishment of new ones. Unfortunately the government neglect has resulted in setting up of hemodialysis units by people with inadequate or low training in dialysis therapy, purely driven by financial consideration.

Paucity of quality control mechanism permits these units to get away with unacceptable compromises like measures to prevent transmission of infection, poor water quality standards and non-adherence to standards while reusing dialysers and blood tubings. The excuses often given for such fraudulent practices are that it allows for cost cutting and provision of cheap therapy. These unfortunately lead to unacceptably high rates of infection, vascular access complication, septicemia and premature death. The appropriate approach include pre-dialysis education, adequate information about dialysis modalities and freedom of making choices, timely creation of vascular access or implantation of peritoneal dialysis catheter and initiation of dialysis, adherence to quality standards and provision of holistic care to patients.

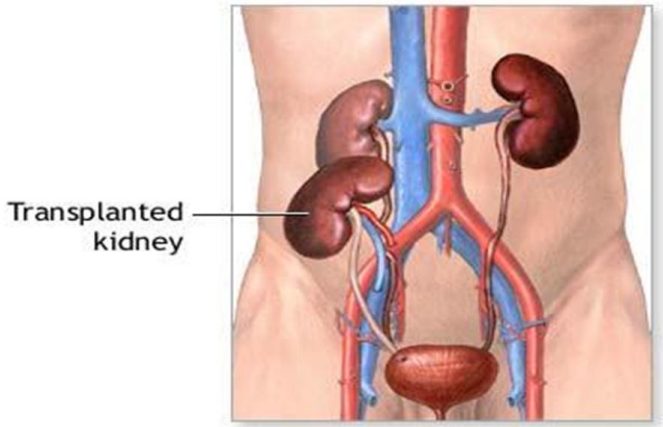
11.4.2 Kidney Transplantation

In adults, the graft is placed extraperitoneally in the contra lateral iliac fossa with the renal artery anastomosed end to end to the recipient's hypogastric (internal iliac) artery or end to side to the common iliac artery. The renal vein is anastomosed to the iliac vein in adults. The donor ureters are inserted by creating submucosal tunnel in the recipient's urinary bladder. Well perfused viable kidneys usually produce urine immediately especially in simultaneous method of transplantation. Our lone case of kidney transplantation had delayed function because sequential method was used due to paucity of man power. The native kidneys are usually left in-situ except in very huge kidneys (PKD) or the presence of recalcitrant hypertension and chronic renal infection.

11.4.2.2 Immunosuppression

All kidney transplant recipients require life-long immunosuppression to prevent alloimmune rejection response (Gallagha M et al, 2009, Kramer BK et al, 2005). The main purposes are to prevent acute and chronic rejection, minimise drug toxicity and infection rate, reduce risk of secondary malignancy and achieve highest possible patient and graft survival rate. The immunosuppressive agents are of two categories; namely anti-rejection induction drugs and maintenance immunotherapy. There is usually no consensus as to the best protocol and each Centre uses varied combinations that are slightly different. Induction immunotherapy usually consist of monoclonal anti-sera (OKT3) or polyclonal anti-sera (ALG, ATG). Maintenance phase drugs may include prednisolone, azathioprine, mycophenolate morfetil, cyclosporine A, tacrolimus, sirolimus and belatacept (Yakupoglu YK et al,2003).

Figure 11: Transplant Recipient



(www.adamimages.com)

12.0 Challenges in treatment of kidney failure among Blacks

There is lower graft survival rate in Blacks than Whites and the adduced contributory factors include heightened immune responsiveness, lack of Human Leukocyte Antigen (HLA) matching, high error rate in HLA serologic identity and ***irregular medication from poor finances*** (Li S et al, 2004. Sukhuja V et al, 2003, Shieppati A et al, 2005)**.**

Majority of CKD patients in Ilorin suffer unnecessarily and die prematurely because of payment out of pocket, poor infrastructure and inadequate equipment. The outlook for patients with severe acute kidney injury requiring dialysis is very good as majority of them will recover complete renal function after two to three sessions of HD. Inadequate infrastructural facilities and limited number of dialysis machine have resulted in long waiting list for dialysis therapy and rescheduling of patients for admission in university of Ilorin teaching hospital, Ilorin. We had only two functioning haemodialysis machines, out of six machines in our renal care centre at the time of writing this communication and we are able to take four out of twenty-seven CKD patients (15%) per day for the therapy.

In India and Pakistan, less than 10% of ESRD patients receive any kind of renal replacement therapy, while the vast majority stops treatment because of cost constraints within three months (Sukhuja V et al, 2003). Unfortunately, most patients in early stages of AKI and CKD are asymptomatic and therefore escape detection or are undertreated in Blacks.

The annual cost of hemodialysis per patient in the USA is about 52,000 dollars while the cost of transplant plus maintenance per year is 18,500 dollars. Although renal transplant is the cheapest option, only about 5% of ESRD have transplant in India and Pakistan *(United States Renal Data System, 2004).* In sub-Saharan Africa, economic and manpower factors dictate a conservative approach to therapy in most cases as majority of ESRD patients perish for lack of funds. *(Naicker S et al, 2003)*. Major constraints to renal replacement therapy in Blacks include; restriction of hemodialysis to urban centres, absence of government funding/subsidy, frequent breakdown of old/obsolete machines, inadequate maintenance and technical support, shortage of spare parts and consumables and frequent power outage (Chijioke A et al, 2003.Li S et al,2004, Garcia-Garcia G et al,2005). These foregoing inadequacies contribute to medical tourism and capital flight abroad.

Challenges in transplantation include protection from rejection as immune system poses the largest stumbling block to success between a donor and recipient who are not genetically identical. Regarding transplants in Blacks, living donor provides 95% to 100% of donations as compared to 1% to 20% in Whites. Religious beliefs in many Sub-Saharan countries regard organ retrieval as mutilation of the body, resulting in low donation rates.

The generally low cadaveric donation rates result in renal commerce in many Black nations where kidneys can be purchased from $1000 to $3000 depending on the purchasing ability. Appropriate and optimum immunosuppression is hardly possible in Blacks. The main constraints are non-availability of all the useful drugs and high cost. Many of these Black nations resort to generic

drugs instead of the expensive branded types with increased rates of rejection episodes.

As economic stringency is the main Achilles heel to the initiation of renal replacement therapy, I advocate that the government should provide 40% of the total cost of an individual dialysis session or renal transplantation while the rest is derived from patient's relatives and NGOs. This has been successfully executed and sustained in Pakistan and India, because of the ***inbuilt transparency, public audit and accountability*** (Rizvi SAH, et al, 1998. Sukhuja V, et al, 2003). Renal transplant rates is low in Blacks because of a combination of inadequate infrastructure, geographical remoteness, lack of legislation governing brain death, constraints imposed by religion, culture, social status and commercial incentives that favour dialysis. *(*Garcia-Garcia G, et al, 2005*).*

The prognosis of patients who present late with CKD is abysmally poor in Blacks. Renal replacement therapy is generally available among Whites but a very select few in Blacks. This harsh reality can be reversed by early detection and intervention in some countries where little or no treatment for ESRD is available. In developing countries like Nigeria, people do not regularly seek medical advice due to lack of awareness about renal disease, belief of being apparently healthy in the absence of symptoms. This often results in too late a referral, if any or more commonly unnecessary suffering and premature death.

14.0 My contributions to knowledge in renal medicine

Mr Vice Chancellor Sir, in the course of my career as Professor of Medicine and consultant nephrologist, I carried out several research works on human kidneys. In

relation to diseased kidneys, I came across some disease entities involving the kidneys that were alleged to be rare in Blacks. These disease entities were often published as case reports because of the perceived rarity in Blacks. I developed special interest as some of these diseases like polycystic kidney disease; sickle cell nephropathy; diabetic nephropathy are heritable conditions. I found it difficult to comprehend why there should be racial disparities in diseases that are either genetic or familial in nature. The foregoing observations prompted me to focus my research on validation or otherwise of the racial disparities. A select few of my research exploits are presented as follows:

Hypertension is very prevalent among chronic kidney disease patients with increased risk of cardiovascular death and progression to end stage renal disease. Resistant hypertension which is the use of three or more drugs including a diuretic at maximal tolerated doses in order to achieve control (>130/80 mmHg) is more common among chronic kidney disease patients than general population. We used the current benchmark of resistant hypertension to evaluate control of BP among our chronic kidney disease patients, and found that only 18% of them adhered to treatment or achieved the treatment target with >80% having resistant hypertension. The drug regimen that achieved goal included a combination of ARBs, ACEIs, CCBs and diuretics. We identified ignorance, poverty and poor compliance as risk factors for resistant hypertension which underscore need for health education with emphasis on renal check-up and compliance on drugs (Makusidi et al, 2011).

Autosomal dominant polycystic kidney disease is the most common hereditary renal disease with extra-renal

manifestations and very important cause of ESRD among Whites, but thought to be rare in Blacks. This emanated from earlier studies in tropical environment that documented rarity of polycystic kidney disease as a cause of chronic kidney disease. (Gabow PA, 1993; Akinsola W, et al, 1989) In a related study of cystic kidney disease, we found that 5% of the cases were autosomal dominant polycystic kidney disease, while simple cysts accounted for 38%. The wide spread access and use of imaging techniques in recent times have brought to the fore that it is paucity of diagnostic facilities that must have led to the erroneous impression that autosomal dominant polycystic kidney disease was rare in Blacks (Chijioke et al, 2010).

Acute kidney injury is a common mode of presentation of kidney disease in our environment. Majority of the cases that come to the knowledge of the Nephrologist are usually in a bad state requiring renal replacement therapy. Majority of them die due to late presentation, severity of the AKI at presentation, delay in intervention including those that border on poor finances, and prohibitive cost beyond the reach of most Nigerians (Chijioke, 2003). The irony of it is that major causes of AKI are volume responsive preventable conditions and yet they die from uremia. In the developed world, it is criminal to allow a patient with AKI to die from uremia.

Although ESRD is found among all races and in all parts of the world, there is this lingering notion that the type of disease burden and causative factors differ from one race to the other, even in the same race, in different or same locations. This prompted our study of end stage renal disease patients in Guy's Hospital London and UITH Nigeria in their national environment. We found that the

spectrum of renal disease among Blacks in Nigeria and Whites in London were generally the same, but the prevalence, natural history and major causative factors did vary. It was also noted that the predominant age bracket were the second to third decade in Blacks in contrast to the fifth and sixth decades in Whites. Equally disturbing is the diabetic nephropathy which was thought to be rare in Blacks that ranked third to kidney infection and hypertension in our study (Chijioke, et al, 2003) .This was not surprising as many Blacks in urban settings have adopted Westernised lifestyle (smoking, alcohol) and fast food with little or no exercise.

We studied causes and prognosis of acute kidney injury taking cognisance of the fact that high morbidity and mortality is associated with it in our environment. We found that majority were multifactorial in a setting of infection, gastroenteritis, and obstetric causes, involving young and middle aged adults with late presentation. Also noteworthy is the high prevalence of acute kidney injury due to exogenous nephrotoxins, mainly NSAIDs, herbal remedies and holy spiritual water (Chijioke et al, 2007). The high mortality was in a setting of overwhelming infection and severe azotemia which ironically were caused by volume responsive and potentially preventable conditions. There is need to intensify awareness programmes on early detection of acute kidney injury and prompt treatment.

End stage renal disease is a major cause of unnecessary suffering and premature death especially among Blacks and the most viable option for its management is renal transplantation. We looked at factors that may influence attitude to kidney donation for transplant and found that the major constraints were fear of surgical

pain and death, belief in life after death and uncertainty of the donor outcome (Chijioke et al, 2010). Public awareness programmes are paramount in overcoming these constraints with involvement of traditional heads and religious leaders; emphasising that a person can survive with one kidney and die naturally from an unrelated illness.

In recognition of the fact that lipid disorders are risk factors for cardiovascular disease and progression of CKD to ESRD of varied etiologies, we looked at lipid profile among chronic kidney disease patients and found that dyslipidemia is very common among our dialysis naïve chronic kidney disease patients with mean lipid of 5.4 ± 4.5 mmol/L in the patients compared to controls with a value of 1.7± 0.2 mmol/L. Majority of these patients died from cardiovascular disease before reaching end stage renal disease (Chijioke, et al, 2011). This underscores the need to assess serum lipid level in these patients as prompt treatment will prevent cardiovascular events and retard progression of CKD to ESRD.

HIV is an important cause of kidney disease in Sub-Saharan Africa and there is paucity of studies on the burden of CKD among HIV patients in our setting. We carried out cross sectional study among newly diagnosed HIV patients compared with controls. We found out that 48% of HIV positive patients in contrast to 16% of controls had associated kidney disease (Dada SA, et al, 2015). In view of the findings, we strongly recommend screening and early intervention for chronic kidney disease as part of management protocol of HIV positive patients.

In cognisance of the earlier notion that African kidneys are resistant to tuberculosis, we set out to look at renal tuberculosis among active pulmonary tuberculosis,

patients in Ilorin making use of urine AAFB and renal histology. We found that prevalence of renal tuberculosis among pulmonary tuberculosis was 14%. Most cases of pulmonary tuberculosis screened for renal tuberculosis outside Nigeria found prevalence rate of 6% to 10%. Our study showed that renal tuberculosis, among pulmonary tuberculosis patients is even commoner in Blacks than the White population (Chijioke, et al, 1997). The diagnosis though difficult, a high index of clinical suspicion with combined diagnostic tools are definitely rewarding. As a follow up study, I reported two cases of proven renal tuberculosis in which the patient presented with recurrent loin pain for two and five years duration respectively having been variously managed as cases of bacterial chronic kidney infection because of the general impression that renal tuberculosis is rare in Blacks (Chijioke, 2001). The conclusion is that it is the lack of awareness and difficulties in diagnosis of renal tuberculosis with varied mode of presentation, self-medication and inadequate diagnostic facilities combine to explain the erroneous assumption that renal tuberculosis is rare in Blacks.

Figure 15: Screening for markers of CKD at Offa in Kwara State

The magnitude of CKD is difficult to evaluate in a community because the early stages of the disease are largely asymptomatic. This is because of the inherent adequate compensation for progressive loss of kidney function making people oblivious of the fact that they have sick kidneys until it is advanced or they are rushed to the hospital for salvage hemodialysis. It is in recognition of the foregoing that we carried out community-based free study in our quest to unravel the presence of markers of CKD. We found significant urinary abnormalities including blood, pus and protein (i.e hematuria, pyuria and protein) in 15% to 18% of cases, while obesity (BMI ≥30Kg/m2) and hypertension (BP ≥ 140/90mmHg) accounted for 3.6% and 4.6% respectively (Chijioke A, et al, 2008). A periodic clinical evaluation and simple screening test is advocated in

communities to forestall late presentation of patients with CKD, the management of which is capital intensive.

Nephron number is set at birth but as individuals gain weight, single nephron GFR increases to keep pace with metabolic demands. Therefore, individuals born with fewer nephrons run the highest risk of glomerular hypertrophy and nephrosclerosis if they eventually become obese.

Obesity exerts extra burden on the nephron which promotes progression of CKD. Obese patients have higher risk of developing proteinuria and CKD after unilateral nephrectomy than lean individuals. This supports the crucial role of hemodynamic factors in CKD-Obesity relationship (Prage M, et al, 2000). In metabolic syndrome, the risk of CKD increases as the number of components increase. Mechanisms contributory to renal injury from dyslipidemia as a component of metabolic syndrome include 1. Toxicity to glomerular capillary endothelial cells. 2. Accumulation of lipoproteins in the glomerular mesengium which stimulates matrix production and glomeruloscelrosis. (Prasad GV, 2014; Cases A, et al, 2005)

In our study of obesity as a risk factor for CKD, we found that a sizeable number of obese patients had associated diabetes mellitus (51% versus 35%), and hypertension (53% versus 34%) in contrast to lean persons. The prevalence of CKD among obese patients in our study was 35% and 20% in lean persons. Obesity was also found to be an independent risk factor for CKD after correcting for diabetes and hypertension. An elevation in waist-hip ratio and high artherogenic index were strongly associated with chronic kidney disease in our study (Afolabi AO, Part 11 Dissertation for Fellowship Award, 2017).

I am the pioneer leader of the first case of Living Donor Kidney transplantation performed on 10th September, 2012 in our Renal Care Centre with Technical support from OAUTHC renal transplant team. The transplant recipient was presented as poster in 2013 World Congress of Nephrology (WCN) in Hong Kong and at the Satellite meeting in Aberdeen, Hong Kong. Some patients and Donors are currently being worked up for the procedure.

15.0 Prevention of kidney dysfunction

Health spending in Blacks is between 0.8% and 4% of Gross National Product in contrast to 10% to 15% in Whites. Therefore, the care of kidney health in Blacks must contend against about 60% of the population living below poverty line (<1 dollar/day), poor literacy (58% males and 29% females), less access to portable water and basic sanitation. Cultural and societal constraints combine with economic obstacles to translate into poor renal care in Blacks.

The collaboration of doctors, nurses, patients and volunteers under one coordinating centre is vital to ensure reliable screening and best use of limited resources to tackle CKD. A major impediment is that screening for CKD offer little returns in terms of visibility and image for doctors and especially politicians and decision makers. Unfortunately, high income countries focus more on kidney repair as their mindset is that selling drugs and dialysis machines are lucrative business in comparison to preventive medicine. According to data provided by Moeller S, et al, 2002, about 1.4 million people are on renal replacement therapy, of which more than 80% reside in Europe, North America and Japan *(*Moeller S et al, 2001; Sukhuja V et al, 2003*).*

Adequate knowledge about the function of the kidney will enable us avoid activities/habits that tend to damage the kidneys. The needful include; maintaining normal Body Mass Index(BMI), regular exercise, avoidance of high protein and fatty meals, avoidance of refined sugars and fast foods ,encouragement of low salt and low phosphate diet, avoidance of bleaching creams/soap, avoidance of herbal remedies, adequate treatment of infections, avoidance of over-the-counter drugs or abuse of analgesics, adequate control of hypertension and diabetes, encouragement of screening of asymptomatic individuals for markers of kidney damage and risk factors of kidney disease.

Challenges in preventive measures

There is high rate of under-diagnosis and under-treatment of renal disease because of low index of clinical suspicion. Majority of patients in the early stages of AKI and CKD escape recognition by the general practitioners because of paucity of symptoms and signs. Many patients in the later symptomatic stages of the disease present late to the nephrologists or not at all because of ignorance and abject poverty. The culture of routine medical check-up for urinary anomalies, blood pressure, blood sugar, lipid profile and complete blood count for evidence of infection is lacking in many Black nations. The patronage of herbalists, spiritualists and consumption of unhealthy diet contribute to late presentation that culminates in emergency RRT.

The top priority for controlling CKD, which is to ensure secure and sustainable access to low-cost antihypertensive drugs, blood sugar and cholesterol lowering agents, avoidance of herbal remedies, high salt intake and NSAIDS are lacking in Black nations. Treatment

of kidney failure especially ESRD constitutes an enormous economic burden within health systems and is grossly inadequate in low income nations (Uchino S, et al, 2005; Li S, et al, 2004). This underscores the need for preventive strategies. A comprehensive team-based multidisciplinary intervention including lifestyle changes and pharmacological agents are cost effective measure to reduce CVD and CKD (Shieppati A, et al, 2005). The challenge faced by Blacks with kidney disease are enormous, as patients, care givers and investigators struggle to measure the high disease burden, delivery of high cost RRT in the face of limited resources, implementation of preventive strategies and difficult ethnic dilemma.

16.0 Recommendations

Mr Vice Chancellor Sir, my plea is that we should not allow anybody to die, especially AKI patients, because he/she cannot afford to live. In the world of Nephrology, it is criminal to allow acute kidney injury patients to die from uremia.

> ***"of all the forms of inequality, injustice to health is the most shocking and inhumane" ---(- Marthin Luther King)***

Therefore, my major recommendations include the following:

1) Compulsive intake of 3 to 4L or 4 to 6L of water per day in Temperate and Tropical settings respectively is strongly recommended as a ***panacea*** for prevention of kidney diseases and treatment of some established kidney diseases in adults.
2) In a bid to prevent CKD, failure to check Urine and Creatinine status 4 times and/or two times in a year

for persons with and without family history of kidney disease is a lost opportunity for early diagnosis and prompt treatment.

3) Avoidance of herbal remedies, exposure to agrochemicals, dehydration and consumption of contaminated water, use of NSAIDs and judicious adjustment of MUST USE drugs, timely referral to nephrologist and multidisciplinary follow-ups for all patients with CKD should be vigorously pursued.
4) As economic stringency is the main Achilles heel to the initiation of RRT, I advocate that the government should provide 50% of the total cost of each session of dialysis or renal transplantation, while the rest is derived from patient relatives and NGOs. This has been successfully executed and sustained in Pakistan and India, because of the ***inbuilt transparency, public audit and accountability***
5) ***My candid plea is that the Federal Government of Nigeria should swing to action and legislate on meaningful renal subsidy for our ever increasing End Stage Renal Disease patients in which about 80% are below 40 years of age***
6) The solutions to renal problems are simple: alleviate poverty, educate the general public and expand renal care programs in public sector hospitals where commerce is unlikely to play a major role.
    - In the days of *Sir Robert Hutchinson (1871-1960),* he stated and I quote " ***...****the ghost of deceased patients that haunt us, do not ask why we did not employ the latest fad of clinical*

*investigations. But they ask "**Why did you not test my Urine?"…..."***

- In the present day practice, I make bold to say, ***"**…**...**the ghost of deceased patients that haunt us, do not ask why we did not employ the latest state of the art clinical investigations. But they ask "**Why did you not check my Urine and Creatinine status?"…..."***

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