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Table 2 shows the clinical signs in malaria parasite positive and negative infants. Only anaemia (haematocrit <33%) showed a significant association with the presence of malaria parasitaemia (P = 0.00035). The other clinical parameters like fever, splenomegaly, and hepatomegaly did not show any significant association with the presence of malaria parasites.

Table 2: A Comparison of Clinical Signs in Malaria Parasite Positive and Negative Infants

CLINICAL SIGNS	MP POSITIVE (n = 178)	MP NEGATIVE (n = 144)	χ^2	P-VA LUE
Fever(at presentation)	102 (57.3%)	72(50.0%)	0.94	0.33
Anaemia (haematocrit<33%)	77 (43.3%)	34(23.6%)	12.75	0.01*
Hepatomegaly	29 (16.3%)	24 (16.7%)	0.0	0.95
Splenomegaly	8 (4.5%)	2 (1.4%)	1.62**	0.1
Hepatosplenomegaly	4(2.2%)	1(0.7%)	0.45**	0.25

*P = 0.05 = statistically significant** Fisher exact results

The sensitivity, specificity, positive predictive value and negative predictive value of the above clinical signs were calculated to investigate their value in the diagnosis of malaria in infancy as shown in table 3. None of the clinical signs showed a high overall sensitivity in the diagnosis of malaria though splenomegaly and hepatomegaly showed high specificities.

Table 3: Sensitivity, Specificity and Predictive Values of Clinical Signs in Diagnosisof Malaria in Infancy by Age Group (In Months)

Variable	Measures	Age 0-=1	>1-=2	>2-=4	>4-=6	>6-=8	>8-=10	>10-=12	All ages
Fever	Sensitivity	63.6	26.7	45.0	51.8	65.8	72.4	57.1	56.7
	Specificity	36.3	75.0	65.0	48.0	36.0	47.8	50.0	49.3
	PPV*	50.0	66.7	56.2	51.8	62.8	63.6	55.5	58.0
	NPV*	50.0	35.3	54.0	48.0	39.1	57.9	51.6	47.9
Anaemia	Sensitivity	18.2	53.3	50.0	40.7	41.5	44.8	45.7	43.2
	Specificity	63.6	100	65.0	60.0	76.0	60.8	90.6	76.4
	PPV	33.3	100	58.8	52.3	73.9	59.1	84.2	69.4
	NPV	43.8	53.3	56.5	45.5	44.2	46.7	60.4	52.1
Hepato-Megaly	Sensitivity	0.0	6.7	15.0	25.9	17.1	17.2	17.1	16.3
	Specificity	100	100	80.0	84.0	76.0	47.8	81.2	83.3
	PPV	0.0	100	42.8	63.6	53.8	29.4	50.0	54.7
	NPV	50.0	36.3	48.5	51.2	35.8	31.4	47.2	44.6
Spleno-Megaly	Sensitivity	0.0	0.0	0.0	7.4	2.4	13.8	2.8	4.5
	Specificity	100	100	100	100	96.0	100	96.8	98.6
	PPV	0.0	0.0	0.0	100	50.0	100	50.0	80.0
	NPV	100	34.8	50.0	50.0	37.5	47.9	47.7	45.5
*H + S	Sensitivity	0.0	0.0	0.0	3.7	4.8	6.9	0.0	2.2
	Specificity	100	100	100	100	96.0	100	100	99.3
	PPV	0.0	0.0	0.0	100	66.6	100	0.0	80.0
	NPV	100	34.8	50.0	49.0	38.1	46.0	47.8	45.1

*PPV: Positive predictive value *H + S (Hepatomegaly + Splenomegaly)
*NPV: Negative predictive value

DISCUSSION

As earlier noted, the clinical presentation of malaria mimics many other disease conditions as seen in the infant. The value of clinical signs such as fever, anaemia, hepatomegaly and splenomegaly in the diagnosis of malaria in infancy were investigated in this study. Fever, hepatomegaly and splenomegaly did not demonstrate any significant association with the presence of malaria parasitaemia and this is as noted in other studies.[7,8,9,10] Only anaemia showed a significant association with the presence of parasitaemia and this finding is corroborated by some other authors.[7,8,11,12] The development of anaemia in malaria parasite positive patients has been adduced to several factors, one of the most important being direct destruction of the red blood cells by the malaria parasites.[13]

The overall sensitivity of clinical signs in this study for the diagnosis of malaria in infancy was not high and this is in keeping with the demonstrated lack of association between malaria parasitaemia and the clinical signs noted above. Thus only hepato-splenomegaly showed a relatively high degree of specificity ranging from 83.3%-99.3% for hepatomegaly and splenomegaly respectively. This high degree of specificity noted implies that hepatomegaly and splenomegaly are non-specific as signs in distinguishing malaria from other infections. There are other disease conditions that can give rise to enlargement of the liver and spleen in infancy such as sepsis, cytomegalovirus infection, toxoplasmosis. In consequence, elements of clinical algorithms which incorporate fever =38^{o}C and splenomegaly,[14,15] as significant predictors of *P. Falciparum* parasitaemia were not found to be useful in this study. They have been found to have increasing significance only in children above one year of age and in those with malaria parasitaemia>10,000 parasites/µl[7]Generally, parasite counts/densities in infants have been found to be low with 41-49% of parasite positive infants having a parasite density of less than 1000/l.[16]
In this study, the highest parasite count was 5,600 parasites/µl and therefore further corroborates the fact that the outlined parameters are not useful in malaria diagnosis when the parasite count is less than 10,000 parasites/µl.

In summary, the use of clinical algorithms which incorporate certain clinical signs such as fever, splenomegaly and hepatomegaly have not been found to be useful as an aid in diagnosing malaria in infancy. In this study, even though anaemia was found to have a significant association with the presence of malaria parasitaemia, it was not found to have a high overall sensitivity as a clinical sign which could be used as an aid in diagnosing malaria in infancy. However, a high index of suspicion for malaria should be exercised in any ill infant who presents with anaemia in the presence of non-specific symptoms and signs. Such an infant should be given the benefit of the doubt and treated for malaria while being investigated to exclude other possible diagnoses.

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often, no treatment is necessary.[13] It is however paramount to determine the underlying etiology and treat accordingly. Correction of blood sugar, treatment of underlying infection and management of the structural lesion remain the mainstay of therapy.[13,20] Patients with hemiballismus secondary to acute ischaemic stroke will require evaluation and treatment.[21] In patients with hyperosmolar hyperglycaemic state, correction of blood sugar and longtime diabetic control is optimal for good recovery.[20]

Medical treatment in hemiballismus has a dual role. Control of these violent movements helps in the prevention and reduction of injuries in the severe cases. Similarly in pronged cases of ballismus, the use of medications is of paramount importance to prevent exhaustion. In 1976, Klawans et al reported 11 patients who developed hemiballismus and had a marked response to neuroleptic therapy.[6] A similar observation that Levodopa worsened chorea led to the hypothesis that the dopamine system was involved in ballism. Early therapy with dopamine receptor antagonists was noted to be successful in these patients.[21] Neuroleptics therefore have been the mainstay of drug therapy for hemiballismus in the past three decades. Of all the neuroleptics, Haloperidol has been the most successful.[12] Other drugs like Chlorpromazine, Primozide and Diazepam have been tried. More recently, newer atypical neuroleptics (Olanzapine[22] and Clozapine[23]) and Dopamine depleting agents (Tetrabenazine[24]) have been reported to be of benefit. Drug- induced Parkinsonism is a noted complication of the dopamine antagonist or depleting agents .[21]

All our patients had similar management which was mainly supportive. This included rehydration with normal saline, correction of blood sugar for those with hyperglycaemia and Haloperidol 5 mg daily. Some of the patients had to be sedated with Diazepam in the Normal Saline infusion. Supportive care to avoid injuries was also given including providing a safe environment. The movements reduced and patients were discharged home well on antihypertensive drugs, hypoglycaemic agents and Statins as applicable. All the patients were placed on Aspirin for secondary prevention of stroke. Only two of the patients came for follow up. Both are well with no residual abnormal movements.

Prognosis

The natural history of hemiballismus varies depending on the etiology.[5] Most patients who develop acute hemiballismus however, have a favorable prognosis irrespective of the cause.[6] In a 2010 observational study of 15 patients with hemiballismus, 8 patients(53%) had a rapid resolution of the movements without treatment and over a period of 17 months, 11patients (73%) had full resolution.[25] Most of these eleven patients had full resolution of movements within two months.[25] In patients developing hemiballismus secondary to hyperosmolar hyperglycaemic state, the clinical course is favourable with the normalization of blood sugar.[5] In our series, all the patients had a remarkable reduction of the movements before discharge. The only two persons who continued follow up had full resolution within one month.

CONCLUSION

Hemiballismus may not be as rare as previously thought. There is need for a high index of suspicion. The sudden surge may also be a reflection of the paradigm shift from communicable diseases to non- communicable diseases seen in Africa. Possible association of environmental and genetic factors is of considerable importance. The prognosis is good.

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patients were from the Ibibio ethnic group in the South South, Nigeria residing in Akwa Ibom state. Considering the rarity of this disorder, this surge seen in persons of a particular ethnic group residing in the same environment raises the question of possible environmental or genetic predilection.

The uncontrollable flinging movements started suddenly in all our patients. They all presented within the first week of the abnormal movements. There were no other complaints. No history of fever, seizures, loss of consciousness or slurred speech, blurred vision or headaches. No associated weakness of one side of the body, deviation of the mouth to one side or speech deficits. There was no history of weight loss or parasthesias.

All the patients had risk factors for stroke (nonmodifiable like older age, black race, male sex or postmenopausal women) and at least one modifiable risk factor for stroke. These were hypertension, Diabetes mellitus, obesity and dyslipidaemias. These remain the commonest risk factors for stroke in the Nigerian practice.[7] Some had more than one modifiable risk factor. Five (83.33%) out of the six patients were persons with known diagnosis of hypertension. The admitting blood pressure however was markedly elevated in all the six patients. Two had a previous diagnosis of Diabetes Mellitus with associated obesity. These two presented with severe hyperglycaemia and one was managed for hyperosmolar hyperglycaemic state. Brain imaging was not available in the hospital as at the time these patients presented. This was one great challenge in this observation. The hospital however purchased a CT scan machine shortly. Other investigations done were mainly base line investigations and those to identify the risk factors.

Localization/Pathophysiology

As far back as 1927 when it was first described, it was also recognized that patients developing acute onset hemiballismus/ hemichorea often had damage to the contralateral basal ganglia or thalamus typically acute ischaemic or haemorrhagic stroke. The corpus luysii e.g. subthalamic nucleus (STN) was identified early as the vital structure involved in the pathophysiology of hemiballismus[8]. Martin proposed naming the condition the 'syndrome of the body of Luys.[3] Further studies noted that lesions outside the sub thalamic nucleus can also cause hemiballismus. Some of these were found in experimental animal studies with rhesus monkeys.[9] More recently, surgical literature has provided additional insight to the localization of hemiballismus based on experience with stereotactic operations (thalamotomy and pallidotomy) for Parkinson disease in the 1960s.[10] Other structures implicated in hemiballismus are the lateral thalamic nucleus and rarely the globuspallidus.[11]

Etiology

Vascular and structural lesions within the contralateral STN and the basal ganglia remain the commonest causes of hemiballismus[1]. Stroke is by far the most common etiology[1] even though hemiballismus is seen in only 0.45% of stroke patients. Two studies noted 11 of the 21 patients with stroke in one and 18 of 25 in another.[11,12] In our series, all the patients had risk factors for stroke. Another commonly recognized etiology noted is hyperosmolar hyperglycaemic state. This is found mostly in South East Asia but there has been a report of six cases elsewhere in Nigeria.[12] Two of our patients had severe hyperglycaemia. Other more rare causes from case reports include encephalitis, vasculitis, central nervous system (CNS) Lupus, cerebral toxoplasmosis with AIDS, neurocystercosis, mass lesions (neoplasms and cysts) and multiple sclerosis. These are mostly from case reports.[12,14-16] Even more rarely, drugs have been implicated.[17,18] Traumatic brain injury as a cause of hemiballismus has been recently reported.[19]

Diagnosis

Diagnosis is characteristically clinical since the abnormal movements are usually dramatic. Characteristic CT scan and MRI findings are seen in almost all cases. Contralateral basal ganglia hyperdensities are common on head CT scan signifying haemorrhage.[19,20] Brain MRI shows T1 hyperintensities and T2 hypointensity in the contralateral basal ganglia.[20] We made a clinical diagnosis in all our patients based on the history and characteristic violent flinging movements. The hospital had not acquired a CT scan as at the period of this study. This absence of recent modalities of brain imaging was a major challenge in this report.

Management

Hemiballismus resolves with time, and

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Outcome of Paediatric Renal Diseases in University of Uyo Teaching Hospital, Uyo, Nigeria

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ABSTRACT

Paediatric renal diseases constitute a very important non-communicable disease entity with high mortality or long term sequelae in survivors. End Stage Renal Disease is usually the end of the spectrum and its management is out of the financial reach of many Nigerians. Care of the chronically ill child has far-reaching effects on the family. It is essential to describe the outcome of this disease entity in our community so as to know what interventions could improve it. The objective was to determine the response to treatment of various childhood renal diseases in terms of number of discharges, follow-up or mortalities. A prospective study of consecutive children aged 17years and younger admitted with renal diseases at the paediatric nephrology unit of the University of Uyo Teaching Hospital, Nigeria, from January 2003 to December 2012. Information entered into the renal register included the patients biodata, clinical findings, results of investigations, diagnoses, treatment modalities and outcome of management. The data was analysed using SPSS version 17. Renal diseases constituted 3.1% of the 5,275 paediatric admissions over the study period. Overall mortality rate was 11.7%. Nephrotic syndrome was the most prevalent diagnosis and contributed the largest mortality (52.6%) mainly from complications of CKD, followed by Acute Kidney Injury (26.3%). A total of 38.5% of the patients were discharged against medical advice due predominantly to financial constraints. The mortality rate was high, and financial constraints necessitated many discharges against medical advice. We recommend Government's support for renal replacement therapy for children.

***Keywords: Children, Chronic Kidney Disease, Nephrotic Syndrome, Nigeria, Outcome, Renal Diseases*.**

INTRODUCTION

Paediatric renal diseases constitute important causes of morbidity and mortality[1]. Our national reports of the contributions of these diseases to paediatric admissions in teaching hospitals have ranged from 2.9% in Jos,[2] to 4.5% in Benin-City.[3] Elzouki in Libya, Africa reported a prevalence of 3.0%.[4] Simple entities like urinary tract infections can be managed with good short term outcome even in resource poor countries. The more resource-demanding disorders like severe acute kidney injury (AKI) usually pose management challenges.[5] In settings where human and technological advances are inadequate for effective management, paediatric renal mortalities have been high.[6] Case fatality rates from AKI were as high as 42.6% and 42.9% from south-western[5] and mid-western[3] Nigeria respectively in comparison with a good immediate outcome of 95% reported by Hiu-Sickle *et al*[7], in American paediatric patients who required intensive-unit's care for AKI. Survivors of AKI may progress to chronic kidney disease thereby placing heavy physical and psychological burdens, not only on the affected children but also on their care-givers, with attendant long-lasting consequences.[8-10] Askenazi *et al*[11] documented that 40-50% of children who survived AKI showed signs of renal insufficiency after 3-5 years follow up.

The association between low socio-economic status, chronic illnesses, and their interplay on family dynamics has been documented.[8-10] The financial demands of management of paediatric chronic kidney diseases is usually beyond the reach of families with low incomes especially in the settings where effective health insurance programmes are lacking. In resource-poor countries with a constellation of negligible public health care funding, rudimentary technological advancements and poor health seeking behaviours, the outcome of management of these diseases is worth documenting.

We set out to document the outcome of management of the paediatric renal diseases in a newly established tertiary health facility in a developing country over a ten year period. It is hoped that our findings will contribute to the information and knowledge needed to improve the care of our children.

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the sudden flinging of the arm. The right arm was involved in the four females while the left was involved in the two men. All of them had some involvement of the ipsilateral lower limb but to a much lesser extent. There was no associated weakness of the limbs or face seizures, difficulty in speech or loss of consciousness. There was no preceding headache, vomiting or vertigo. There was no history suggestive of a transient ischaemic attack or a previous stroke in any of the patients. No other neurological deficits were found on examination rather they were all agitated and fatigued from the uncontrollable movements.

Five of the patients were persons with previous diagnosis of hypertension but all had markedly elevated blood pressure on presentation ranging from 160/110 to 230/140. Two of the patients were persons with a previous diagnosis of diabetes mellitus and both presented with markedly elevated blood sugar. One was managed for hyperosmolar hyperglycaemic state. Two of the patients had morbid obesity with BMI of 37.5 and 53 Kg/mm.[2] Three of the patients had dyslipidaemias. The ancillary investigations like full blood count, electrolytes urea and creatinine done were normal for four of the patients. There were however deranged electrolytes in the two who had hyperglycaemia. Chest X-ray showed features of hypertensive heart disease in all the patients. Brain CT scan was not carried out since the hospital did not have a CT scan as at the period of this observation. The hospital however has a CT scan now.

All the patients were admitted in the medical wards of the hospital. Management was mostly supportive care to avoid injuries. The patients were rehydrated. Medications and infusions given were Mannitol, Normal saline and Haloperidol. The two who had hyperglycaemic emergencies had rehydration with normal saline and soluble Insulin for blood sugar control. Some of the more restive patients also had diazepam. Aspirin, Vitamins C and E were also given to all the patients.

The uncontrollable movements were controlled with Haloperidol 5mg. The frequency and speed of the movements decreased within 3 days in all the patients. They were all discharged with residual but markedly improved condition with antihypertensives, hypoglycaemic drugs and statins as indicated. Aspirin was commenced for secondary prevention. They were counseled on lifestyle modification. Only two patients came back for follow-up. Both are stable with no residual limb movements.

DISCUSSION AND REVIEW OF LITERATURE

History

Hemiballismus was first reported in 1927 by James Purdon Martin[3] when he described a patient who suddenly developed prominent flinging movements of his right arm and leg. He noted that these types of movement characterized by high amplitude arrhythmic motions especially at the shoulder and hip were not often seen in chorea.[3] The original report used terms like flinging, swinging, throwing and tossing about to describe the violent movements which are still seen in these patients. He initially termed the disorder 'hemichorea' but most neurologists have accepted the correct terminology for this condition as hemiballismus. Further observation of these movements revealed that some patients developed violent ballistic movements while others developed movements which were of lower amplitude, less frequent, distal and more fluidy[4]. These reduced movements were suitably termed hemichorea. In due time, it was noticed that hemiballismus and hemichorea were related and represented different degrees of severity.[4] It is noteworthy that the hemiballistic movements actually become hemichorea as the patient recovers. For this reason, most authors use hemiballismus and hemichorea interchangeably.

Epidemiology

Hemiballismus is a rare movement disorder[5]. It is 500 times rarer than Parkinson's disease[1]. Klawans et al reported 11 cases seen over a nine year period.[6] We describe six patients who presented with these classical characteristic movements on one side of the body within an observation period of just three years. In this case series, there were two males and four females. None of the patients had ballism (bilateral variety). Hemiballismus is usually seen in persons older than 60 years. The age range was 55-68 years in our series with only one person who was younger than 60 years. There was no young patient. There is no known preference for sex usually but we saw more females than men with a ratio of 2:1 and all the four women were postmenopausal. Most of the patients were of lower socio-economic class being farmers and artisans. One was a full time house wife. All the

Case Report

Hemiballismus: A Case Series in Uyo, Southern Nigeria and a Review of Available Literature

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ABSTRACT

Hemiballismus is a dramatic movement disorder that is typically acute in onset. It usually involves one side of the body affecting the proximal limb more than the distal. The commonest recognized etiology remains cerebrovascular diseases. Very few cases have been reported in Africa. We describe six cases seen in our hospital with their presentation, management and eventual outcome. The participants were six patients admitted and treated in our practice from January 2010-December 2012. The folders of those who had been discharged were reviewed and reported as follows. Six patients presented with sudden uncontrollable flinging movements. There were two males and four females. The age range was 55-68 years. All the patients were from the Ibibio ethnic group in the Southern Nigeria. The patients presented within the first week of the abnormal movements. They all had modifiable and non-modifiable risk factors for stroke. All the patients had supportive management and had remarkable improvement. Hemiballismus may not be as rare as previously thought. This sudden surge may be part of the transition from communicable to non-communicable diseases noted in sub Saharan Africa. There may also be an associated environmental factor and/or genetic predilection given that all the patients come from the same ethnic group and reside in the same environment. Patients do well on supportive care.

Keywords: Hemiballismus, risk factors, Nigeria, hypertension, hyperglycaemia

INTRODUCTION

Hemiballismus, also called hemiballism is a rare dramatic movement disorder that is typically acute in onset. It is defined as repetitive but constantly varying, large amplitude involuntary movements mostly involving the proximal part of the limbs.[1] The activity is almost ceaseless and movements are often complex and combined[2]. It is classically characterized by involuntary movement of the extremities which are often violent. The term hemiballismus refers to violent flailing movements observed on one side of the body while ballismus or ballism refers to the rarer bilateral variant. Some cases may include the facial muscles.[2] The movements like other abnormal movements usually increase in activity and reduce during relaxation and disappear during sleep. The commonest cause is a lesion of the contralateral subthalamic nucleus.[3] This is usually from hypertensive lacunar stroke. Occasionally, it may develop sub acutely or chronically from other lesions. Few cases have been reported in Africa. We report six cases seen in our hospital between January 2010 and December 2012 with their presentation and management.

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METHODS

This is a consecutive case series on patients with hemiballismus seen and managed in the medical wards of University of Uyo Teaching Hospital Uyo, Southern Nigeria. The observation period was from January 2010 to December 2012. Diagnosis was made clinically in all the cases.

Case Reports

We report six cases of patients who presented with sudden flinging of the arm. There were two males and four females. The age range was 55-68 years. Actually the 55 year old man was the only person that was younger than 60 years. All the patients were of Ibibio ethnicity and reside in Uyo Southern, Nigeria. One of the patients had an occasional contract job in Enugu still in Southern Nigeria. They were all Christians but of different denominations. All the patients were of low educational status. They all had blue collar jobs (farming, artisans, and petty trade).

The patients all presented within the first week of

METHODOLOGY

This was a longitudinal prospective study of paediatric patients admitted into the paediatric renal unit of the University of Uyo Teaching Hospital (UUTH), over a ten-year period (January 2003 to December 2012). The renal unit serves as the only tertiary paediatric renal centre in Akwa Ibom State, and caters for children with significant renal diseases within the state and neighbouring states like Cross River, Abia and Imo states. The state has a population of 3.9million people majority of who are civil servants[12]. A few others are involved in business with small-scale business being more common. The hospital has a haemodialysis unit which started four years after the paediatric renal unit. There are 5 haemodialysis machines which serve both adult and paediatric patients.

Children aged 17 years and below, who presented with clinical features suggestive of renal diseases were admitted into the unit for investigations and treatment and their data entered into the renal register. Children with uncomplicated urinary tract infections (UTI) were usually managed on out-patient basis and only admitted if they had associated complications like toxaemia, or persistent vomiting with dehydration. Discharged patients were followed up in the Children's Outpatient Clinic and readmitted as indicated. Admission into the paediatrics renal unit is mainly via the Children's Outpatient Clinic and Children's Emergency Unit (CHEU).

Data entered into our renal register includedage, sex, presenting complaints, duration of symptoms prior to presentation, occupation and educational level of parents or care-givers (from which their social class was determined using Oyedeji's criteria[13]), important clinical examination findings, results of investigations, and the initial diagnosis were also recorded. Data was put on SSPS data page.

Nephrotic syndrome was diagnosed with massive proteinuria of = 3+ on dipstick urinalysis, spot urine protein: creatinine ratio of >2.0, and serum albumin of <2.5g/dl. Acute glomerulonephritis was diagnosed with clinical features of peripheral oedema, with or without coke-coloured urine, oliguria, hypertension and likely past history of a throat or skin infection in the preceding month. Laboratory evidence of significant haematuria of =2+ on dipstick urinalysis was also included. Positive urine cultures confirmed urinary tract infections. Imaging studies such as micturiting cystourethrogram were used to diagnose posterior urethral valves and abdominal ultrasonogram for nephroblastoma.

Renal biopsy for histology is yet to be done in our developing renal centre.

The outcome of each patient's management was also documented. Discharged patients were given regular appointments for follow-up in the specialist paediatrics renal clinic. Patients who had HIV/AIDS were co-managed with infectious diseases specialist team.

Treatment modalities comprised antimicrobials for urinary tract infections and sepsis, oral steroids with or without cyclophosphamide for steroid resistant and steroid-dependent nephrotic syndrome cases, and immunomodulators like levamisole. Diuretics, anti-hypertensive agents and dietherapy were also included as required. Haemo- and peritoneal- dialyses were done when indicated and when feasible.

Prior to the commencement of haemodialysis in our centre in 2007, eligible patients were usually referred to Obafemi Awolowo University Teaching Hospital, Ile-Ife to access haemodialysis. With the initiation of our dialysis unit, some eligible older children had haemodialysis depending on the extent of financial capability.

Statistical Analysis

Data was analysed using SPSS package version 17. Statistical mean ± SD were compared using Student *t* test, chi-square was carried out to compare proportions for statistical analysis. Values of $P < 0.05$ were regarded as statistically significant.

RESULTS

Five thousand two hundred and fifty-seven (5,257) children were admitted during the study period of which 162 (3.1%) had renal diseases. One hundred and fifty-six of them had single episode of admission while 6 were admitted twice.

Table 1 shows that there were 93 (57.4%) males and 69 (42.6%) females (male: female ratio of 1.3:1). Children below 5 years of age contributed the largest percentage of 38.3% while those less than 15years were least represented with a frequency of 11.1%. Only 2.5% of our patients belonged to the high social class 1. Majority (67.9%) of the patients belonged to the lower social classes 1V and V.