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Nephrotic Syndrome in Port Harcourt — Clinical Presentation and Response to Steroids

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SUMMARY

Eke FU. Nephrotic Syndrome in Port Harcourt – Clinical Presentation and Response to Steroids. *Nigerian Journal of Paediatrics,* 1990; 17:59. Thirty children who presented at the University of Port Harcourt Teaching Hospital with nephrotic syndome were studied retrospectively. The peak incidence was between the ages of 3 and 5 years. All showed heavy urinary protein excretion ($>40mg/m^2/hr$), hypoalbuminaemia ($<25g/l$) and hypercholesterolaemia ($>6mMol/l$). Plasmodium falciparum was isolated from the blood of 2 patients (6.7%). Seven patients who had no evidence of sickle cell anaemia, hepatitis, hypertension or impaired renal function were treated with prednisolone $60mg/m^2/day$ and went into remission in 10 – 30 days after starting therapy, ($p = 0.001$). Renal biopsies, performed in 3 frequent relapsers showed hypercellularity of mesangial cells with normal glomerular capillary walls, on light microscopy. In conclusion, steroid sensitive nephrotic syndrome is not uncommon in the Rivers State of Nigeria and although the aetiology is unknown, it appears worthwhile treating such patients with steroids prior to a renal biopsy.

INTRODUCTION

NEPHROTIC Syndrome in children has been widely studied in developed countries, where the majority are secondary to minimal change lesions in the glomerular basement membrane,[1–5] and respond to steroids.[6] Studies done in Western[7] and Northern Nigeria[8] and in East Africa[9] have shown a high prevalence of quartan malarial nephrotic syndrome in children. The majority of childhood nephrotics in these areas present after the age of 5 years and do not respond to steroids. To date there are no published data on nephrotic syndrome in children from around the Rivers States (Eastern Nigeria). A retrospective study was therefore undertaken of children admitted with Nephrotic Syndrome into the University of Port Harcourt Teaching Hospital since its inception in 1984. The aim of the study was to review the mode of presentation among children with Nephrotic Syndrome in and around Port Harcourt and their possible response to steroid therapy.

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PATIENTS AND METHODS

Thirty patients aged $0 \leqslant 15$ years were studied. The criteria for inclusion into the study consisted of generalized body swelling or marked facial and leg oedema; heavy proteinuria (>40mg/m^2/hr) and hypoalbuminaemia (<25g/l). Patients with a history suggestive of acute glomerulonephritis such as a sudden onset of coke-coloured urine or with haematuria accompained by moderate proteinuria (10–40mg/m^2/hr) and a predominance of granular casts on urine light microscopy were excluded from the study. Three patients previously treated with prednisolone were also admitted into the study.

All the patients had a urinalysis (dipstick) and urine microscopy performed on admission as well as a 24-hour urine or a timed overnight urine collected and quantitated for protein. Most but not all the patients had daily urinalysis with a Multistix throughout their period of hospitalization. Blood was taken for malaria parasites, haemoglobin electrophoresis, urea and electrolytes, creatinine, serum albumin and cholesterol. Three frequent relapsers had renal biopsies performed.

Seven patients who did not have sickle cell anaemia, hypertension or persistently elevated serum creatinine, were given prednisolone 60mg/m^2/day until the urine was free of protein for three consecutive days and then 40mg/m^2/day as a single dose three days a week for one week.

Chi-squared test with Yate's correction was used to test significance of data and the method of least squares for linear regression.

RESULSTS

The age range of the patients is shown in Figure 1 where a peak is noted at the age of 3–5 years. There was no significant sex difference with a male to female ratio of 2: 1.8. The presenting complaints are outlined in Table I and the complications in Table II. All the patients had massive proteinuria (>40mg/m^2/hr), hypoalbuminaemia and hypercholesterolaemia (mean values ± 1 standard deviation were 17.2 ± 4.4g/l and 10.29 ± 3.2mmol/l, respectively). Normal values are 25–45g/l and 2.6–6.0mmol/l respectively. There was no significant correlation between age and serum albumin or serum albumin and cholesterol ($r = 0.30$ and 0.26; $p > 0.05$ and

TABLE I

Presenting Complaints (N = 30) in Childhood Nephrotic Syndrome

COMPLAINT	NO.	%
Body Swelling	30	100
Oliguria	10	33.3
Fever	9	30
Pain in the Limbs	5	16.6
Abdominal Pain	3	10
Frequency of micturition	1	3.3

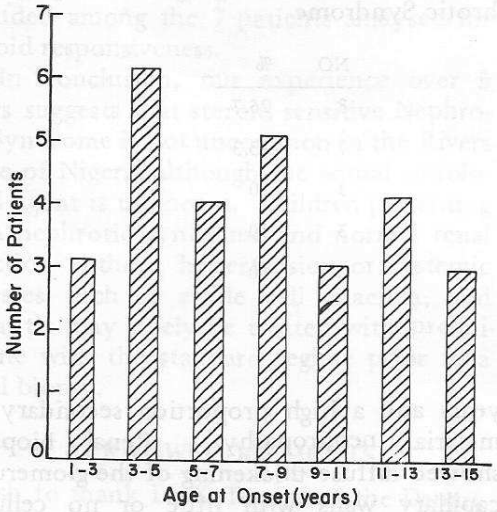

Figure 1: The age range of the 30 patients at the onset of Nephrotic Syndrome.

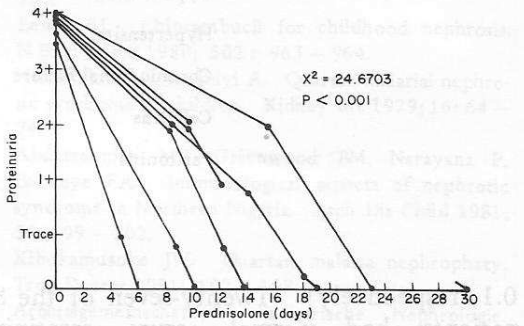

Figure 2: Fall in proteinuria with Prednisolone therapy. The broken line (2nd from the top) depicts a patient who absconded but continued on steroid therapy till he emerged on day 30. The second broken line depicts a patient who died from vigorous diuretic therapy.

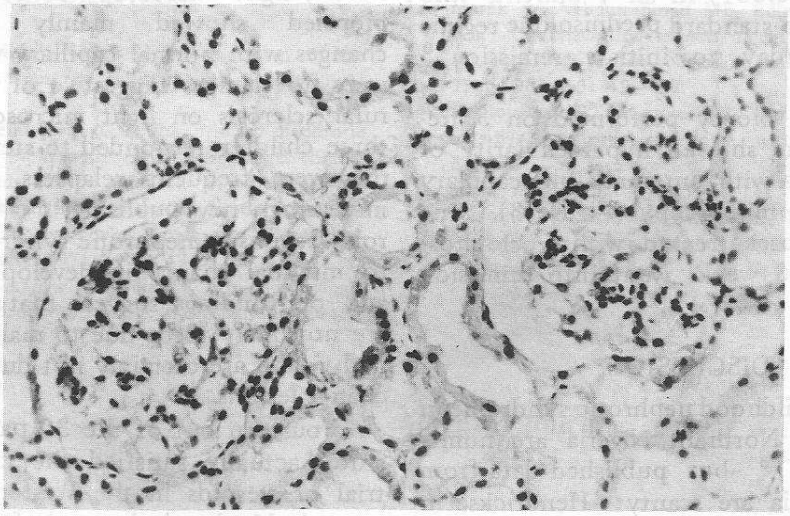

Figure 3: High power light microscopy of a renal biopsy showing mesangial proliferation of the glomeruli, normal tubules and normal capillary walls.

TABLE II

Complications Of Nephrotic Syndrome

	NO.	%
Hypotension	8	26.7
Hypertension	7	23.3
Chronic Renal Failure	3	10
Cellulitis	3	10
Peritonitis	3	10

0.1, respectively). Twenty-seven of the 30 patients had normal serum creatinine (<100uMol/l). Plasmodium falciparum was isolated from the blood in 2 patients and Hepatitis B surface antigen in 1 of the 6 patients in whom it was measured.

Urinary protein excretion fell from 4 + to 2 + (using the Multistix) within 16 days of commencement of steroids and more significantly, it fell to 0 within one month ($p = 0.001$) in all 7 patients who were given the standard prednisolone regime of $60mg/m^2$/day to initiate remission[10] (Figure 2).

The renal biopsy performed on 3 frequent relapsers showed hypercellularity of mesangial cells with unremarkable capillary walls on light microscopy (Figure 3). One frequent relapser treated with cyclophosphamide for 6 weeks went into remission for 5 months.

DISCUSSION

Studies on childhood nephrotic syndrome in Western and Northern Nigeria are numerous[7, 8, 11–13] but published data from Eastern Nigeria are scanty: Hendrickse in 1979[7] and Abdurrahman[8] in 1981 studied children with nephrotic syndrome and found a peak incidence after the age of 5 years and a high proportion secondary to malarial nephropathy. Renal biopsies showed diffuse thickening of the glomerular capillary walls with little or no cellular proliferation and such children did not respond to steroids.

In this study the peak incidence was slightly lower at between 3 – 5 years, and although electron microscopy and immunofluorescence were not available at our centre, light microscopy in the 3 patients biopsied showed mainly proliferative changes with normal capillary walls. There were no changes suggestive of focal glomerular sclerosis on light microscopy and all three children responded to steroids though they were frequent relapsers. There are increasingly new published[14, 15] data of steroid responsive nephrotic syndrome not due to minimal change in developed countries and our findings suggest that our 3 cases are not likely to be due to malarial nephropathy, and are certainly not due to minimal change lesions.

Fourteen out of the 30 patients in this study actually satisfied the criteria for a trial of steroids in the Western World i.e. normal biochemical renal function tests results and no hypertension or associated underlying disease, but 7 were found to have

been treated with Prednisolone in a dose of 0.1mg/kg to 1mg/kg, and so were not included among the 7 patients analysed for steroid responsiveness.

In conclusion, our experience over 5 years suggests that steroid sensitive Nephrotic Syndrome is not uncommon in the Rivers State of Nigeria although the actual aetiological agent is unknown. Children presenting with nephrotic syndrome and normal renal function without hypertension or systemic illnesses such as sickle cell anaemia, and hepatitis may safely be treated with prednisolone with the standard regime prior to a renal biopsy.

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