Proteinuric renal disease in children in South-Western Uganda

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Sir,

Proteinuric renal disease is common in Africa in both children and adults: in the 1960s it accounted for 2–3% of medical admissions.[1] The condition is more severe than nephrotic syndrome in developed countries, incurring high mortality and typically being resistant to corticosteroids or antimitotic agents. Admission rates with nephrotic syndrome are higher during periods of intense malaria transmission, and an association has been reported with *Plasmodium malariae* infection.[2,3] Most literature on this subject pre-dates the epidemic of human immunodeficiency virus (HIV) infection in sub-Saharan Africa. HIV nephropathy is increasingly recognized in the developed world,[4] but renal manifestations of HIV have not been studied in Africa.

Thirty-six children (20 males, 16 females, age range 1.5–14 years, median 6 years) were admitted to Mbarara University Hospital with proteinuric

renal disease in the first 11 months of 1999: approximately 160/million population/year, or eight times the UK incidence of childhood nephrotic syndrome. The most common clinical diagnosis was acute nephritic syndrome (impaired renal function and/or hypertension and/or active urine sediment, 18 cases, 50%); 'pure' nephrotic syndrome (isolated proteinuria, normal blood pressure, normal serum creatinine) was rare (five cases, 14%), but mixed pictures were common (13 cases, 36%). Median serum albumin was 15.5 g/dl, median serum creatinine was 129 µmol/l. Malaria data were collected in a separate study by Médicine Sans Frontieres: 307/541 children (57%) were *Plasmodium*-positive; 94% of isolates were *P. falciparum*, *P. malariae* being rare (5%). The seroprevalence of HIV and hepatitis B were assessed by anonymous analysis of 77 consecutive paediatric in-patients: 16/77 (20%) were HIV-positive, but only 2/77 (3%) had antibodies to hepatitis B. Of the 36 children with proteinuric renal disease, only one had proven HIV infection, and none had evidence of hepatitis B. Overall outcome for these 36 children is shown in Table 1. Treatments included diuretics, anti-hypertensives, and dietary modifications whenever possible (increasing protein intake, removing salt). Eleven children received corticosteroids; two subsequently also received cyclophosphamide. Mean duration of hospital admission was 11 days (range 1–90). There was some improvement in 69%. Six children died (17% mortality). 'Pure' nephrotic syndrome carried the best prognosis (all improved, none lost to follow-up, no deaths), but this was the least common subtype, accounting for only 14% of cases. In the group with acute nephritic presentation, overall mortality was 28%, and may have been even higher if additional deaths occurred among children lost to follow-up.

The assumption that *P. malariae* is the major cause of proteinuric renal disease in Africa has dominated literature on this subject for 40 years, but seems unlikely to be the explanation for the high rate of acute renal disease we observed in Mbarara, SW Uganda. In Mbarara in 1999, only 5% of malarial infections amongst children were due to *P. malariae*. Nor did we find any evidence that HIV infection is an important cause: only one proteinuric child had proven HIV infection and the mean age of affected children (6.7 years) is against a role for HIV, since vertically transmitted HIV infections are usually manifest at a younger age. The explanation we favour is that the majority of these patients have infection-related glomerulonephritis (GN). The most typical form of this entity is acute proliferative GN complicating streptococcal infection. Each of these possible explanations can only be adequately tested by prospective studies: we have embarked on such a study in Mbarara. Intriguingly, our preliminary findings in a small number of renal biopsies do not show the typical lesion of post-streptococcal GN, but instead an eosinophil-rich mesangiocapillary GN picture (Peat *et al.*, unpublished observations) which may represent GN complicating parasitic infections.

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Table 1 Overall outcome in 36 children admitted to Toto Ward, Mbarara with proteinuria between 1 January and 1 December 1999

Clinical syndrome	Number (%)	Age range (years) [mean]	Pred.	Improved	Lost to follow-up	Died (%)
Nephritic	18 (50 %)	1.5–12 [6.2]	1/18	10	3	5 (28 %)
Mixed	13 (36 %)	3–14 [6.5]	6/13	10	2	1 (8 %)
Nephrotic	5 (14 %)	2.5–10 [7.3]	4/5	5	0	0 (0 %)

Pred., treatment with prednisolone.

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