Sirs.

Steroid-resistant nephrotic syndrome (SRNS) is believed to be associated with a high risk of developing chronic renal failure (CRF). The underlying histopathology usually affects the course of the disease and the response to treatment [1]. Focal segmental glomerulosclerosis (FSGS) was reported as the main cause of SRNS in Western countries [2] as well as in many other parts of the world [3]. However, in black South Africans, membranous nephropathy associated with hepatitis B infection was reported as a main cause of SRNS [4], and minimal change nephrotic syndrome [minimal change disease (MCD)] was reported as the main cause in Kuwaiti children [5].

In this study we report on the pattern of the histopathology of SRNS in children coming to King Abdul-Aziz University Hospital (KAUH).

We reviewed the notes of all children with primary nephrotic syndrome that was steroid resistant who had come to the pediatric renal unit at KAUH between 2002
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and 2007. Only patients who had undergone renal biopsy were included in the study.
The histopathology slides were reviewed by two renal pathologists independently. Patients with congenital nephrotic syndrome, lupus or sickle cell disease, were excluded from the study.
Thirty-six children fulfilled the inclusion criteria: 25 girls and 11 boys, with a female (F) to male (M) ratio of 2.3:1. Fifty percent (18 children) were Saudi and the other 50% were from various racial backgrounds (nine Asians, four Arabs, two Africans and three from the Far East).
Their mean age at presentation ± standard deviation (SD) was 4.3±3.0 (range 1-12) years. Their mean serum albumin level at presentation was 15.6±7.1 g/l. and all had 4+ proteinuria. Five children had high creatinine concentrations at presentation. while the mean serum
creatinine level was 50.4±45.6 jmol/l. Three children had low levels of complements at presentation, and none had positive test results for hepatitis surface antigen or antinuclear antibody (ANA). Renal histopathology was compatible with focal segmental glomerulosclerosis (FSGS) in 39% (14 children), immunoglobulin (Tg)M nephropathy in 28% (ten children), mesangio-proliferative glomerulonephritis (MesPGN) in 17% (six children), MCD in 8% (three children), complement Clq nephropathy (ClqNP) in 8% (three children) and IgA nephropathy in one patient (3%).

The incidence and frequency of various histological subtypes of steroid-resistant nephirotic syndrome in children in the Kingdom of Saudi Arabia (KSA) do not seem to differ from those observed in Western and other countries. Therefore, similar diagnostic and therapeutic strategies for SRNS can be applied to our patients in KSA. Genetics studies have to be done in the future.

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