

Body mass index modulates postural proteinuria

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Dear Sir,

In contrast to constant proteinuria, postural proteinuria has been generally regarded as a benign condition [1–4]. However, anecdotal observations suggest that postural proteinuria might sometimes reflect incipient kidney disease [5, 6]. To further address this controversy we recently reassessed 15 subjects with fixed and reproducible postural proteinuria two or more years after the initial diagnosis.

After breakfast and after starting an appropriate water load, the subjects came to the clinic. They had been advised to bring along a portion of the first morning urine (“recumbent”) for determination of the total protein-to-creatinine ratio (reference value: ≤ 30 mg/mmol) [7]. The second morning urine sample was collected for urinary sediment analysis and blood was taken for determination of creatinine. Urinary total protein-to-creatinine ratio was also determined with the subjects in a nonrecumbent

position for ≥ 3 h. Finally height, body weight, and sitting blood pressure (reference: systolic and diastolic blood pressure < 90 th centile for gender, age, and height in subjects less than 18 years of age and $< 140/90$ mmHg in the remaining subjects) were recorded and a complete physical examination performed. Body mass index was calculated as the body weight in kilograms divided by squared height in meters [8, 9] and the glomerular filtration rate (reference value: ≥ 90 ml/[min. 1.73 m²]) estimated from height and circulating creatinine using the “height to creatinine equations” [10]. Since body mass index (in kg/m²) varies with age, this parameter was also plotted on the body mass index for age and gender charts and expressed as centile adjusted for age and gender [8].

Descriptive statistics are presented as numbers for qualitative data, and as median and interquartile range (which extends from the value at the 25th to that at the 75th centile and includes half of the data points) for quantitative data. χ^2 -test (qualitative data) and Mann-Whitney-Wilcoxon test for independent samples (quantitative data) were used for analysis. Statistical significance was set at the 0.05 level.

In the recently reevaluated subjects, whose age ranged from 10 to 24 years, the diagnosis of postural proteinuria had been made 2.3–15 years earlier. Physical examination, blood pressure, glomerular filtration rate, urinalysis, and recumbent urinary total protein-to-creatinine ratio were normal in the subjects. Nonrecumbent urinary total protein-to-creatinine ratio

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was normal in eight and pathologically increased in the remaining seven subjects. Subjects with and without persisting postural proteinuria did not differ with respect to age at diagnosis, time after diagnosis, current age, renal function, blood pressure or recumbent proteinuria (Table 1). Both absolute body mass index (15.6 [14.9–18.9] kg/m² versus 22.0 [21.1–23.5] kg/m²; $P < 0.03$) and body mass index adjusted for age and gender (19 [8–41] centile versus 47 [36–53] centile; $P < 0.05$) were significantly lower in subjects with persisting postural proteinuria as compared with those without postural proteinuria (Fig. 1).

The present observations in subjects with fixed and reproducible postural proteinuria confirm that this form of proteinuria does not reflect incipient kidney disease [5, 6], support the assumption that it is often transient [1–4], and indicate that antiproteinuric management with drugs blocking the renin–angiotensin II–aldosterone system is unnecessary [11]. Finally our data indicate that the body mass index, a key index for relating a person's body weight to their height and therefore of relative body fat content, modulates the resolution respectively the persistence of postural proteinuria.

Following not mutually exclusive mechanisms might underly postural proteinuria [1–4]: (a) since proteinuria increases with assumption of upright posture in normal subjects even though it remains normal, postural proteinuria might be an exaggerated

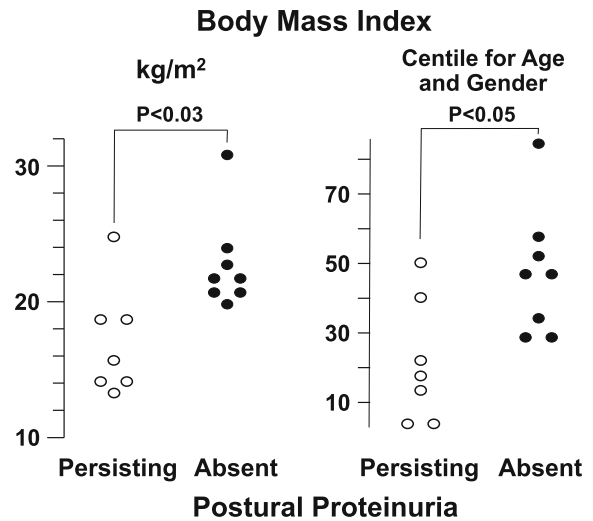


Fig. 1 Body mass index (kg/m²) and body mass index adjusted for age and gender (*centiles*) in 15 subjects aged from 10 to 24 years with past history of fixed and reproducible postural proteinuria

normal response; (b) subtle glomerular lesions might require a superimposed hemodynamic abnormality to lead to postural proteinuria; and (c) increased angiotensin II or norepinephrine release when upright in susceptible subjects. More recently it has been suggested (d) that postural proteinuria often results from the compression of the left renal vein between the superior mesenteric artery and the abdominal

Table 1 Clinical and laboratory data in 15 subjects with postural proteinuria reevaluated 2.3–15 years (median 5.1 years) after the diagnosis. The results are given either as median (with interquartile range between brackets) or as relative frequency

	Postural proteinuria Persisting	Postural proteinuria Absent	Significance
N	7	8	
Gender, females:males	5:2	5:3	Not significant
Age at diagnosis, years	10 [7.7–11]	13 [11–16]	Not significant
Time after diagnosis, years	5.3 [2.8–11]	5.0 [2.5–6.2]	Not significant
Current age, years	17 [13–20]	19 [16–20]	Not significant
Glomerular filtration rate <90 ml/[min. 1.73 m ²]	0/7	0/8	Not significant
Arterial hypertension	0/7	0/8	Not significant
Pathological hematuria	0/7	0/8	No significant
Urinary total protein-to-creatinine ratio			
Recumbent, mg/mmol	12 [10–16]	8 [7–10]	Not significant
Nonrecumbent, mg/mmol	67 [42–98]	12 [10–16]	*

* Subjects with and without persisting postural proteinuria were not compared with respect to this parameter, a statistically significant difference being predictable

aorta [12, 13]. The present observations support the assumption that the space between the aorta and the superior mesenteric artery is modulated by the body mass index. Imaging studies are required to confirm this hypothesis.

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