Spot-Urine Screening for Primary Hyperoxaluria

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Dear Sir,

In primary hereditary hyperoxaluria (oxalosis), stone formation usually begins in childhood (in 65% of the cases before age 12) and recurs at short intervals. Without treatment, more than 80% of the patients with oxalosis become uremic by 20–30 years of age. Treatment with pyridoxine was reported to reduce endogenous oxalate production as well as stone progression, and to prevent them altogether [1]. Early diagnosis and proper management are therefore essential. The diagnosis of oxalosis rests on the finding of increased urinary oxalate excretion in the 24-hour urine, urinary oxalate excretion > 0.5 mmol/1.73 m²/24 h occurs in primary oxalosis [2]. However, it is sometimes troublesome and inaccurate to collect 24-hour urine volumes in infants and toddlers without bladder catheterization, because they do not void spontaneously. Bladder catheterization should be avoided as much as possible because of the risk of urinary tract infections. We tried to find a more convenient screening method for the diagnosis of primary oxalosis type II.

Urinary oxalate excretion might be influenced by diet, and our study was performed in children eating self-selected diets without fluid restriction or special medications. Children with hypospadias (n = 4, aged 1–4 years), a girl with remission of nephrotic syndrome (aged 9 years) and a boy with post-Henoch-Schönlein purpura (aged 7 years), who had normal renal function and neither gastrointestinal disease nor other serious illness and no family history of urolithiasis, were chosen as normal controls with their parents’ consent. In the children < 5 years old, urine collections were performed by means of bladder catheterization. Urinary oxalate excretion in the controls varied among 0.55 mmol ± 0.16/24 h/1.73 m² when 24-hour urinary oxalate excretion per unit of surface area was con-

\[ y = 0.24 + 4.82x \]

3.0
2.5
2.0
1.5
1.0
0.5
0.0

\[ r = 0.966 \quad (\pi = 26) \]

\[ t < 1 \quad m = 5 \]
Fig. 1. Relationship between urinary oxalate excretion and urinary oxalate/creatinine ratio (r = 0.966). 0 = Normal subjects; • = patients.

No such findings have been reported, as far as we were able to determine. Although Gibbs and Watts [3] conducted a similar study previously, they did not compare the urinary oxalate/creatinine ratio with daily urinary oxalate excretion corrected for body surface area. In our own study, there are normal children whose urinary oxalate/creatinine ratio is < 0.147, corresponding to 1.48 mmol/24 h/1.73 m² (mean ± 2 SD). The normal values we determined were somewhat higher than those reported by Barrat [2] and Morris et al. [4] who found that normal urinary oxalate/creatinine ratios are < 0.086 without any detailed explanation. These differences might be due to differences in the methods used, since urinary oxalate levels obtained by gas chromatography are slightly higher than those obtained by colorimetry in the same urine [5]. Coe and Parks [1] reported that in hereditary oxalosis, patients usually excrete more than 1.7–3.4 mmol/24 h in the absence of renal failure.

Our findings will be convenient and useful in the rapid estimation of urinary oxalate excretion in young children with primary oxalosis, if a reference range in each laboratory is established.

References


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References