Dear Sir,

Fractional excretion of uric acid (FEUA) has been described as being derived from 4 different mechanisms: glomerular filtration, presecretory reabsorption, tubular secretion and postsecretory reabsorption [1]. Fractional excretion of uric acid in man ranges between 10 and 16% of the amount filtered [2]. This percentage is much higher during the neonatal period (34.6%) [3], and diminishes significantly along the first year of life, reaching values similar to those of the adult by the end of the first decade [4]:

The different components of uric acid tubular management can be determined by the pyrazinamide and sulfinpyrazone tests, and the normal adult values have already been established [5].

Hyperuricemia has been reported to be due to different etiologies which include both abnormalities in the purine metabolism and a familiar type of tubulointerstitial nephropathy, which is commonly associated with gout.

We have detected 2 patients with hyperuricemia, but without hyperuricosuria and with diminished FEUA, and have submitted them to the pyrazinamide and sulfinpyrazone test. Both of them were males, aged 13 and 14 years, and the cause of their admission to hospital was the existence of macroscopic hematuria, accompanied by nephritic colic in 1 case. They showed normal coagulation, blood pressure and renal function (measured by blood urea and creatinine concentrations as well as creatinine clearance), with high levels of serum uric acid (10.2 and 7.8 mg/dl, respectively), uricosuria within the normal range (310 and 600 mg/l.73 mVday), and decreased FEUA (3.3 and 4.68%, respectively).

In table I, the values of uricosuria, blood uric acid and FEUA of both patients are compared to the same parameters of 8 normal children, aged between 11 and 14 years.

Table I. Analytical findings

Parameters (mean ± SD) taken from 8 normal children.

Table II. Tubular phases of urate excretion

Presecretory Tubular Postsecretory
Parameters (mean ± SD) taken from 10 normal adults [5].
Both patients, following a normal diet and without any medication, were submitted to the pyrazinamide and sulfinpyrazone test, according to what has been previously described in the literature [6]. The results are shown in table II, and compared to those reported for adults [5]. In our 2 patients, we found a normal presecretory reabsorption as well as tubular secretion, with an increase in postsecretory reabsorption. This last fact seems to be the only cause that justifies the small FEUA with normal uricosuria in the 2 patients, despite their hyperuricemia.

It has been stated that there would be a normal tubular management of uric acid in gout and, in some cases, a decrease in tubular secretion caused by hyperuricemia [7]. In both of our patients, another possible renal cause of hyperuricemia can be suspected: an increase in postsecretory reabsorption, which should be considered in gouty patients.

References