Population Based Data on Urinary Excretion of Various Metabolites in Children of North Western Region of Pakistan

Pages with reference to book, From 241 To 242

Nabeel Y. Khalil (Institute of Biochemistry, University of Balochistan, Quetta,)
Sher Ahmad (Department of Urology, Bolan Medical College, Quetta.)
Masha Khan (Department of Urology, Bolan Medical College, Quetta.)
S. Atif Ali,Syeda Ishrat Bukhari,M. Masoom Yasinzai (Institute of Biochemistry, University of Balochistan, Quetta.)
Qurban Ali (Department of Urology, VRI, Brewery Road, Quetta.)

Abstract

Population based data on urinary excretion of various metabolites of pathological importance, Calcium, Magnesium, Sodium, Potassium, Oxalates, Citrates, Phosphates, Uric acid and urea have been collected from around three hundred children of the Quetta valley. The body weight was in the range of 11-50 kg and the age was in between 4-16 years. The urine excretion average was 987.5±452.5 ml per 24 hours. There was 11.5% incidence of hypercalciuria, 8.5% incidence of hyperuricosuria, 2.0% hyperphosphaturia, 2.5% hypomagnesuria, 3.5% hypocitraturia, 6.5% hypematriuria, 43.5% hypokaliurea and 2.1% hyperoxaluria. Urea excretion average was 23.11±14.99 g per 24 hours. The study provided the basis for childhood reference pattern in urinary excretion of compounds related to various pathological conditions, in particular stone formation in this region (JPMA 48:241,1998).

Introduction

It is estimated that renal stones are atleast 10 times more common now than they were at the beginning of the century[1]. Nephrolithiasis is a multi factorial condition that occurs when the chemical environment favors crystalization, the urine must be supersaturated with the salts of the ciystals (e.g. Calcium, Oxalate and phosphate) and in the absence/low concentration of urinaiy inhibitors. These inhibitors include small ions such as magnesium and citrate as well as poiyanions of high molecular weight such as glycosaminoglycans.

Many epidemiologic variables can modify the urinary riskfactors, these include age, sex, heredity, occupation, social class, affluence, geographic location, climate and diet Of these, diet including fluid intake is the only one that can be easily changed and that has a markedeffect on all urinary risk factors[2]. Urolithiasis in children has received little attention in comparison with urolithiasis in the adult population. The development of the urinary tract stones in young patients has raised questions about causing factors and has generated controversy about evaluation and management. Concerns about urolithiasis inpaediatric patients are prompted by recent studies that have found hypercalciuna in 2.9 to 3.8% of otherwise healthy children and in 6.2% children brought to an acute-care clinic for minor complaints[3]. Relationship between idiopathic hypercalciuria and hematuria has been shown, suggesting that such children are predisposed to fonnation of stones[4]. As umlithiasis disease is increasing day by day all over the world among adults and children, it is interesting to investigate the various promoting agents such as uric acid crystals or inhibitors such as magnesium or citrate[5]. Three hundred children of this area were selected to find out the paediatric reference patterninurinary excretion of compounds in order to facilitate the monitoring of prophylactic treatment.

Materials and Methods

Collection of urine samles: 24 hours urine samples were collected from childrenbelongingto different areas of Quetta.
All the samples were stored in the refrigerator at below -20°C until analyzed. Three hundred children aged between 3-16 years (228 male and 72 female) were included in this study. The body weight was in the range of 11-50 kg and the urine excretion average was 987.5±452.5 ml/24 hours. Severn! metabolites of pathological importance such as urea, uric acid, phosphate, calcium, magnesium, oxalate, citrate, sodium and potassium were quantitatively determined using standard Boehringer kits. Citrate was determined spectrophotometrically by its selective complexation with copper. Copper reacts with citrate and forms ablue complex of bis-citrate-Cu (II). This complex has maximum absorption at 760 mn. The method is established in authors laboratory and has been applied to the determination of citrate in urine. The limit of detection of this method is 20 mg/dl. The results were correlated with the enzymatic method for citrate[6], which gave a correlation coefficient of 0.97.
The excretion per day of >300 mg calcium for men and >250mg for women is defined as hypercalciuria, while the excretion per day of >800mg uric acid for men or >750 mgfor women is defined as hypeniricosuria and the excretion of less than 300 mg per day is defined as hypophosphaturia . The excretion of less than 120 nig magnesium per day is defmed as hypomagnesuria. The excretion of 200 mg citrate per day is defined as hypocitraturia, more than200 mmolperday sodium (hypernatriuna), more than 50 mg per day oxalate (hyperoxaluna). The excretion of more than 20 mmol per day of potassium (hyperka!iuria) while excretion of less than 20 mmol per day of potassium (hypokaliuria).

Results

The percentage results among these three hundred children were 11.5% incidence of hypercalciuria, 8.0% hyperuricosuria, 2.0% hyperphosphatuna, 2.5% hypomagnesuria, 3.5% hypocitratuna, 6.5% hypernatriuria and 43.5% hypokaliuria. The urea excretion average was 23.11±14.99 gm per day.

Discussion

Eleven percent hypercalciuna among the 200 children studied is of significant value. Although a correlation was attempted between high dietary calcium intake and an increased risk of stone formation[8], there were difficulties in comparing data on dietary intake and urinaiy excretion of calcium. Most studies have indicated that a decrease in calcium intake by stone forming patients results in decreased calcium excretion[9]. However, these studies have also indicated that decreasing the calcium intake without also decreasing the oxalate intake leads to increased oxalate excretion, which carries an even greater risk for subsequent stone formation. This increase is the result of greater concentmtion of free oxalic acid in the intestine. Excess calcium and oxalate usually form complexes in the intestine that are thenexcreted in the faeces. Whenthe calcium intake is limited, the reduced amount of unabsorbed calcium left in intestine results in more free oxalates, which is absothed by passive diffusion in the colon. The risk in urinary oxalate excretion disappears when dietary oxalate restriction is also imposed[10], while 2. 1%hyperoxaluriais significant. Oxalate is a high risk factor for formation of stones. The dietary components of urinary oxalates is derived from the break-down of dietary oxalate. We have also found 8.0% hyperuricosuria which is also significant and several recent studies have implicated hyperuricosuria as an important risk factorforcalciumoxalate stone formation[11]. Other significant parameters include 3.5% hypocitraturia and 43.5% hypokaliuria. There is a relation between hypokaliuna and hypocitraturia. Hypematuria 6.5% is also a risk factor as there is a close associationbetween urinary excretionof sodium and calcium because of many common sites of

absorption along the renal tubules. People with calcium stones are more sensitive to the calciuric effects of sodium than the rest of the population[12]. This is also worth mentioning that the major source of available drinking water of this area is fountains, lakes and mostly underground water in the form of wells (both openand tube wells). As the available source of drinking water is highly salt rich, containing salts of calcium, phosphates, cathonates and bicarbonates, it is assumed that there is a definite role of such contaminated salt constituents in the elevated rate of these risk factors.

References

1. Laerum E. Urolithiasis in clinical practice: Occurrence, etiology, investigation and preventive treatment. Urology, 1996; 116:2879-902.
2. Kriger iN, Kronmal RA, Coxon Vet at. Dietary and behavioral risk factors for urolithiasis : Potential implications for prevention. Am. J. Kidney Dis., 1996;28:195-201.
3. Kruse K, Kracht U, Kruse U. Reference value for calcium excretion and screening for in children and adoLescents. Fur. J. Pediatr., 1984; 143:25-31.
4. Kaliq A, Travis LB. Brouhard BH. The association of idiopathic hypercakiuria and a symptomatic gross hematuria in children J,Pediatr., 1981 ;99:71 6-19.
5. Cupiste A, Morelli E, Lupetti S et al. Low urine citrate excretion as main risk factor for recurrent calcium oxalate nepbrolithiasis in males. Nephron, 1992;6 1:73-6,
6. Toflegaard NT. A method for enzymatic determination of citrate in serum and urine. Scand. J. Clin. Lab. Invest., 1976;36:5 13-19.
7. Coe FL. Treated and untreated recurrent calcium nephrolithiasis inpatients with idiopathic hypercalciuria, hyperuricosuria or no metabolic disorder. Ann, Intern. Med., 1977;87:404-10,
8. Peacok M And Hodgkinson A. Importance of dietary calcium in the definition of hypercalciuria. Br. Med. J. 1967;3:469-7 1.
9. Batsille P, gregorie J. Felstonn B et at. Calcium restriction in idiopathic hypercalciuria. Contrib. Nephrol., 1984;37:17-21.
10. Tisellius HG. Oxalate and renal stone formation. Scand. 3. Urol. Nephrol., 1980;53: 135-48.
11. Coe FL. Karalach AG. Hypercalciuria and hyperuricosuria in patients with calcium nephrolithiasis. N. Engi. 3. Med., 1974;29: 1344-60.
12. Muldowney FP, Freancy R. Importance ofdietary sodium in the hypercalciuria syndromes. Kidney Int., 1982;22:292-96.