

stone formation: Natural inhibitors of urinary stone formation include citrate, magnesium, and pyrophosphate⁽³⁻⁷⁾.

In 20 to 25 percent of children with UL, urinary tract infection (UTI) is detected or there is a history of a UTI. Infection may be the primary cause of a stone or occur concomitantly with an underlying urinary metabolic abnormality or structural abnormality⁽⁸⁾.

Congenital and structural abnormalities that are accompanied by urinary stasis are associated with UL. Urinary stasis predisposes to crystal and stone formation^(2,9). Patients who have surgically augmented bladders are at risk for nephrolithiasis, most commonly bladder stones composed of struvite⁽¹⁰⁾.

The incidence of UL in children varies worldwide with the highest incidence occurring in endemic areas, such as in Turkey and Thailand. Stones were more commonly found in Caucasian children and rarely in African-American children. Incidence of UL is lower in children than in adults⁽⁴⁾. The Objectives of this study were to evaluate pediatric UL in a group of Iraqi children regarding some demographic characteristics, clinical presentation, laboratory findings, metabolic disorders and characteristics of stones and chemical composition.

Methods

This cross sectional study was based on 96 children with UL for the period from 1st of January 2009 to the end of December 2011. Those patients with UL were evaluated, treated, and followed up in the pediatric nephrology clinic in Al-Imamian Al-Kadhymian Medical City in Baghdad.

Presence of stone disease was confirmed in all cases; radiologically by renal sonography with or without plain abdominal radiograph or patient passed at least one urinary calculus.

All children with renal stones whether newly diagnosed or recurrence were included. Children with renal tubular acidosis or nephrocalcinosis were excluded from the study.

Full recording of the patient characteristics and stone data was done. All children were

examined physically and underwent the following investigations: Urinalysis, urine culture. Blood biochemistry test: urea, creatinine, sodium, potassium, calcium, phosphorus, uric acid, and alkaline phosphatase level. Twenty-four hour urine determination of urinary calcium, oxalate and uric acid was performed for all children.

Hypercalciuria (HCA) was defined as urine calcium excretion $>4 \text{ mg/kg/24 h}$, Hyperoxaluria (HOx) was defined as urine oxalate excretion $>55 \text{ mg/1.73 m}^2/24 \text{ h}$, Hyperuricosuria (HUr) was defined as uric acid excretion $> 815 \text{ mg/1.73 m}^2/24 \text{ h}$ ^(3,4,11-17).

Serum and urine amino acid excretion were tested for all children using paper chromatography and the nitroprusside test for diagnosis of cystinuria^(3,13,17).

Stones from 33 patients that were removed surgically or obtained by spontaneous passage were analyzed chemically.

Voiding cystourethrography (VCUG) and intravenous pyelography (IVP) were done to some patients as indicated. VCUG was done in patients with recurrent urinary tract infection (UTI), and suspected vesicoureteral reflux (VUR). IVP was performed in some cases of suspected renal anomalies.

All of the urinary stones were classified into four groups according to the predisposing risk factor for their occurrence^(4,13,15-18).

(1) Metabolic stones: stones predisposed by metabolic disorder.

(2) Anatomic stones: stones that formed with anomalies of urinary system.

(3) Infection stones: children with recurrent UTI with one or more of the following: **A.** staghorn calculi **B.** urine culture of *Proteus* **C.** Stone chemical analysis of calcium phosphate carbonate, or magnesium ammonium phosphate **D.** associated VUR

(4) Idiopathic stones: The stones that are formed without metabolic, anatomic or infectious etiology.

Patients received follow-up testing every 1-2 months; serial US was used to track UL status and was scheduled at every 4-6 months.