By Assist Prof. Dr.Amin Turki

Vesicoureteral Reflux (VUR)

Vesicoureteral reflux (VUR) is the retrograde flow of urine from the bladder up to the ureter or even up to the kidney. It is may be familial, as 30% to 40% of siblings of a child with VUR also have VUR with VUR also have VUR

Effect of VUR:

It cause hydrodynamic pressure on the kidney during voiding and predispose to UTI due to incomplete emptying of the bladder and ureter. This will facilitate the bacterial transport to the kidney causing pyelonephritis.

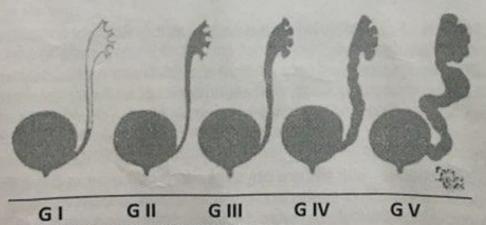
The inflammation reaction caused by pyelonephritis can result in renal injury or scarring termed Reflux nephropathy, which if it severe will cause end-stage renal disease.

Classification:

- 1. Primary VUR results from congenital incompetence of the ureterovesical (UV) iunction.
- 2. Secondary VUR e.g. secondary to distal bladder obstruction or other urinary tract anomalies like duplications of the ureter, neurogenic bladder, cystitis or acquired bladder obstruction such as foreign body or vesical stones.

Grading:

The severity of VUR is graded from grade I to V by contrast voiding cystourethrogram (VCUG) as following:



Grade I: R efflux into a Non-dilated ureter.

Grade II: Reflux into the upper collecting system (renal pelvis and calyces) without dilation.

Grade III: Reflux into dilated ureter and/or blunting of calyceal fornices.

Grade IV: Reflux into a grossly dilated ureter.

Grade IV: Reflux into a grossly dilated and sold and tortuosity and loss of the Grade V: massive VUR, with significant ureteral dilation and tortuosity and loss of the papillary impression.

The incidence of renal scarring in patients with low-grade VUR is low (15%) and increases with grade IV or V reflux (65%).

Grade I or II VUR is likely to resolve without surgical intervention, but VUR resolves in less than 50% grade IV or V.

Clinical manifestations:

VUR is often identified during radiologic evaluation after a UTI. The younger the patient with UTI, the more likely to have VIII. with UTI, the more likely to have VUR.

There are no clinical signs can differentiate children with UTI who have VUR from those without VUR

Treatment:

1. Long-term prophylactic antibiotic therapy (trimethoprim-sulfamethoxazole or nitrofurantoin) indication is controversial in mild to moderate VUR. But it may be useful in children with high-grade VUR and/or recurrent symptomatic UTI or in patient younger than one year of age.

2. Surgical correction: It might be indicated in sever VUS

Complications of VUR: hypertension and chronic kidney disease (CKD).

Nocturnal Enuresis (NE)

Is refer to occurrence of involuntary voiding at night after age of 5 yr, when voluntary control of micturition is expected.

Note: at age of 5 yr, 90-95% of children have complete control of micturition during the day time, and 80-85% have control at night.

Classification:

Primary NE (75-90% of affected children) in which the nocturnal urinary control never achieved.

Secondary NE (10-15% of cases) in which the child was dry at night for at least a few months and then enuresis developed.

75% of children with enuresis are wet only at night and 25% are wet at day and night which are more likely to have abnormality of the urinary tract and day time urological symptoms e.g. urgency, hesitancy, frequency or incontinence.

Epidemiology:

Approximately 60% of children with NE are boys (its more common in boys than girls). Family history is positive in 50% of cases and gene localized to chromosome 12 and 13. If one parent was enuretic, each sibling has a 44% risk of enuresis, and if both parents was enuretic risk will increase to 77%.

Causes:

- Delayed maturation of the cortical mechanisms that allow voluntary control of the micturition reflex
- Defective sleep arousal 2.
- Reduced ADH production at night, resulting in an increas UOP (nocturnal polyuria) 3. Genetic factors (gene localized to chromosome 12 and 13)
- 4.
- Constipation 5.
- Organic factors, such as urinary tract infection or obstructive uropathy 6.
- Sleep disorders e.g. secondary to enlarged adenoids 7.
- Psychologic factors more often in secondary enuresis 8.

Clinical features:

It can occur in any stage of sleep (but usually non-rapid eye movement sleep)

- All children are most difficult to arouse in the first third of the night and easiest to awaken in the last third, but enuretic children are more difficult to arouse than those with normal bladder control
- Enuretic children often are described as "soaking the bed" 3.

Family history is often positive for enuresis

Risk increased with developmental delay, attention-deficit/ hyperactivity disorder, 5. and autism disorder.

Diagnosis:

I. History:

1. Carful history of fluid intake at night and pattern of nocturnal enuresis.

2. History of other conditions which might associated with high UOP e.g. diabetes insipidus, D. mellitus and chronic renal disease

3. History of load snoring at night which suggest adenoidal enlargement.

II. Examination:

- 1. After voiding abdominal palpation and rectal examination to exclude chronic bladder distention.
- 2. Careful examination for neurological and spinal abnormalities.
- 3. Carful throat examination for possibility of adenoidal hypertrophy.

III. Investigations:

1. Urine culture to exclude bacteriuria especially in girls.

2. Urine sample after overnight fasting for specific gravity, osmolality and sugar to exclude poly urea as a cause of enuresis e.g. D. insipidus, and DM

3. If there are no day time associated symptoms (e.g. urgency, hesitancy, frequency or incontinence)the physical examination and urinalysis are normal and the urine culture is negative and no need for further evaluation.

4. Renal ultrasonography is indicated in older children or in children who don't response to therapy.

Differential diagnosis: Diabetes insipidus, diabetes mellitus and UTI.

Treatment:

I. General measures:

1. Reassure the child and his parents that the condition is self-limited.

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 Avoid punishment which can adversely affect on child's psychological development.
- 4. The child should void before go to the bed.
- Avoiding sugar and caffeine after 4 PM.
- 6. If the child have snoring and adenoidal enlargement should refer to otolaryngologist

II. Active therapy:

It should be avoided in children younger than 6yr of age because the enuresis is very common in young children, but it more successful in children near puberty. The active therapy include:

A. Motivation therapy:

1. Star chart for dry nights.

Waking child a few hours after he go to sleep for voiding.

B. Conditioning therapy:

- 1. Use of a loud auditory or vibratory alarm attached to moisture sensor in the underwear. This therapy success in 30-60% of children but with a significant relapse rate.
- 2. Bladder stretching exercise: use in children with day time enuresis in which the child ask to practice holding urination for longer and longer periods. This method should combined with reward system.
- C. Drug therapy: It regarded as second line therapy and it is not curative. It include:
 - 1. Desmopressin acetate (synthetic ADH analog): It reduces urine production over night. It available as nasal spray (which no longer use in children because it associated with hyponatremia and convulsion) and tablet form which save in children.
 - Dose of tablets: 0.2-0.6 mg at bed time for 3-6 mo, then tapering the dose and if enuresis recur, the dose should retain to initial high dose.
 - 2. Oxybutynin (anticholinergic therapy): 5 mg at bed time.
 - 3. Imipramine (tricyclic antidepressant): It has mild anticholinergic and alphaadrenergic effects, so it reduce UOP and affect the sleep pattern. Dose: 25 mg in children age 6-8yr, 50 mg in age 9-12yr, and 75 mg in older patients.

The success rate is 30-60%.

Side effects: Anxiety, insomnia, dehydration and cardiac arrhythmias.