Vesicoureteral Reflux

Vesicoureteral reflux (VUR) describes the retrograde flow of urine from the bladder to the ureter and kidney. The ureteral attachment to the bladder normally is oblique, between the bladder mucosa and detrusor muscle, creating a flap valve mechanism that prevents VUR . VUR occurs when the submucosal tunnel between the mucosa and detrusor muscle is short or absent.

Affecting 1–2% of children, VUR usually is congenital and often is familial. VUR is present in approximately 30% of females who had a urinary tract infection and in 5–15% of infants with antenatal hydronephrosis.

VUR predisposes to kidney infection (pyelonephritis) by facilitating the transport of bacteria from the bladder to the upper urinary tract .The inflammatory reaction caused by pyelonephritis can result in renal injury or scarring, also termed **reflux nephropathy.** In children with a febrile urinary tract infection (UTI), those with VUR are three times more likely to develop renal injury than those without VUR, and can result in hypertension, renal insufficiency and end stage renal disease

Classification

VUR severity is graded using the Classification of I-V and is based on the appearance of the urinary tract on a contrast voiding cystourethrogram (VCUG) . The higher the VUR grade the greater the likelihood of renal injury. VUR severity is an indirect indication of the degree of abnormality of the ureterovesical junction.

Clinical Manifestations

VUR usually is discovered during evaluation for a UTI. In other children, a VCUG is performed during evaluation of bladder–bowel dysfunction, renal insufficiency, hypertension, or other suspected pathologic process of the urinary tract. Primary VUR also may be discovered during evaluation for antenatal hydronephrosis.

whose VUR is diagnosed following a UTI. The UTI may be symptomatic.

Diagnosis

Diagnosis of VUR requires catheterization of the bladder, instillation of a solution containing iodinated contrast or a radiopharmaceutical, and radiologic imaging of the lower and upper urinary tract: a contrast VCUG or radionuclide cystogram, respectively

Treatment

The goals of treatment are to prevent pyelonephritis, VUR-related renal injury, and other complications of VUR. Medical therapy is based on the principle that VUR often resolves over time and that if UTIs can be prevented, the morbidity or complications of VUR may be avoided without surgery. Medical therapy includes

Observation

In children undergoing observation, therapeutic emphasis is directed at minimizing the risk of UTI by behavioral modification

- **Antimicrobial therapy**

The risk of recurrent UTI is highest in patients with grade III or IV reflux, those with BBD, and those whose first reflux-associated UTI was *febrile rather than just symptomatic without fever*. *Antibiotic prophylaxis* after a reflux-associated UTI decreases the risk of recurrent UTI but may increase the risk of developing resistant bacteria.

- **Surgery**

The purpose of surgical therapy is to minimize the risk of febrile UTI from ongoing VUR and nonsurgical therapy (observation or prophylaxis with followup testing).

Renal Failure

Acute Kidney Injury

Acute kidney injury (AKI) has been traditionally defined as an abrupt loss of kidney function leading to a rapid decline in the glomerular filtration rate (GFR),accumulation of waste products such as blood urea nitrogen (BUN) and creatinine, and dysregulation of extracellular volume and electrolyte homeostasis. The term AKI has largely replaced acute renal failure (ARF),because the latter designation overemphasizes the discrete event of a failed kidneys.

AKI is defined as:

Stage 1: S.Cr.1.5-1.9 times baseline, OR≥0.3 mg/dL increase
U.V.:<0.5 mL/kg/hr for 6-12 hr

Stage 2:S.Cr. 2.0-2.9 times baseline
U.V. : <0.5 mL/kg/hr for ≥ 12 hr

Stage 3:S.Cr 3.0 times baseline, ORSCr ≥ 4.0 mg/dL, OR
Initiation of renal replacement therapy, OR
eGFR < 35 mL/min per 1.73 m2 (< 18 yr)
U.V. : <0.3 mL/kg/hr for ≥ 24 hr, OR
Anuria for ≥ 12 hr

Increase in serum creatinine by ≥ 0.3 mg/dL from baseline within 48 hr; *or*
Increase in serum creatinine to ≥ 1.5 times baseline within the prior 7 days;
or
Urine volume ≤ 0.5 mL/kg/hr for 6 hr

Common Causes of Acute Kidney Injury

PRERENAL

Dehydration
Gastroenteritis
Hemorrhage
Burns
Sepsis
Capillary leak
Hypoalbuminemia
Cirrhosis
Abdominal compartment syndrome
Cardiac failure
Anaphylaxis

INTRINSIC RENAL

Glomerulonephritis
Postinfectious/poststreptococcal
Lupus erythematosus
Henoch-Schönlein purpura
Membranoproliferative
Anti–glomerular basement membrane
Hemolytic-uremic syndrome
Acute tubular necrosis
Cortical necrosis
Renal vein thrombosis

Rhabdomyolysis
Acute interstitial nephritis
Tumor infiltration
Toxin and drugs
Tumor lysis syndrome
Vasculitis

POSTRENAL

Posterior urethral valves
Ureteropelvic junction obstruction
Ureterovesicular junction obstruction
Ureterocele
Tumors
Urolithiasis
Urethral strictures
Hemorrhagic cystitis
Neurogenic bladder
Anticholinergic drugs

Clinical Manifestations and Diagnosis

carefully taken history is critical in defining the cause of AKI. An infant with a 3-day history of vomiting and diarrhea most likely has prerenal AKI caused by volume depletion, but hemolytic-uremic syndrome (HUS) must also be a consideration. A 6 yr old child with a recent pharyngitis who presents with periorbital edema, hypertension, and gross hematuria most likely has intrinsic AKI related to acute postinfectious glomerulonephritis. A critically ill child with a history of protracted hypotension or with exposure to nephrotoxic medications most likely has ATN. A neonate with a history of hydronephrosis seen on prenatal ultrasound studies and a palpable bladder most likely has congenital urinary tract obstruction, probably related to posterior urethral valves.

Laboratory Findings

Anemia

Leukopenia

Thrompocytopenia

Elevated blood level of BUN, uric acid, creatinine, potassium, phosphate

Metabolic acidosis

Hyponatremia, and hypocalcemia

The presence of hematuria, proteinuria, and red blood cell or granular urinary casts suggests intrinsic AKI, in particular glomerular disease and ATN. The presence of white blood cells and white blood cell casts with low-grade hematuria and proteinuria suggests tubulointerstitial disease

Urinary indices may be useful in differentiating prerenal AKI from intrinsic AKI (Table 550.4). Patients whose urine shows an elevated specific gravity (>1.020), elevated urine osmolality (UOsm > 500 mOsm/kg), low urine sodium (UNa < 20 mEq/L), and fractional excretion of sodium < 1% (<2.5% in neonates) most likely have prerenal AKI. Those with a specific gravity of < 1.010, low urine osmolality (UOsm < 350 mOsm/kg), high urine sodium (UNa >40mEq/L) and fractional excretion of sodium> 2%(> 10% in neonates) most likely have intrinsic AKI.

Chest radiography

Renal ultrasound

Renal biopsy

TREATMENT:

Determination of the volume status is of critical importance when initially evaluating a patient with AKI. If there is no evidence of volume overload or cardiac failure, the intravascular volume should be expanded by intravenous administration of isotonic saline, 20 mL/kg over 30 min. In the absence of blood loss or hypoproteinemia, colloid-containing solutions are not required for volume expansion. Severe hypovolemia may require additional fluid boluses. After volume resuscitation, hypovolemic patients generally void within 2 hr; failure to do so suggests intrinsic or postrenal AKI.

Diuretic therapy should be considered only after the adequacy of the circulating blood volume has been established. Furosemide (2-4 mg/kg) may be administered as a single intravenous dose

If urine output is not improved, then a continuous diuretic infusion may be considered. To increase renal cortical blood flow, many clinicians administer dopamine (2-3 µg/kg/min) in conjunction with diuretic therapy, although no controlled data support this practice. There is little evidence that diuretics or dopamine can prevent AKI or hasten recovery.

Mannitol may be effective in the prevention of pigment (myoglobin, hemoglobin)-induced renal failure. Atrial natriuretic peptide may be of value in preventing or treating AKI, although there is little pediatric evidence to support its use.

If there is no response to a diuretic challenge, diuretics should be discontinued and fluid restriction is essential. Patients with a relatively normal intravascular volume should initially be limited to 400 mL/m2 /24 hr (insensible losses) plus an amount of fluid equal to the urine output for that day. Extrarenal (blood, GI tract) fluid losses should be replaced, milliliter for milliliter, with appropriate fluids.

Nutrition is of critical importance in children who develop AKI. In most cases, sodium, potassium, and phosphorus should be restricted. Protein intake should be moderately restricted while maximizing the caloric intake to minimize the accumulation of nitrogenous wastes. In critically ill patients with AKI, parenteral hyperalimentation with essential amino acids should be considered hypo. Hyperkalemia, hyponatremia, hypocalcemia, and hypertension should be treated.