

## Intensive Care Nursery House Staff Manual

### Renal Disorders in the Newborn

**EPIDEMIOLOGY:** Renal disorders are a heterogeneous group of congenital and acquired conditions. Anomalies are detected in ~1% of fetuses by prenatal ultrasound, in <1% of newborns by physical examination and in 7-9% of individuals at autopsy. Factors that increase the risk of renal anomalies are maternal diabetes and maternal drug use, including alcohol. Early diagnosis of abnormalities of renal structure or function may help prevent complications including hypertension, obstructive or reflux uropathy, infections and renal failure.

**PRENATAL DIAGNOSIS OF RENAL DISEASE** is usually by fetal ultrasonograms that detect signs of obstructive uropathy. Fetal hydrops may occur with congenital nephrotic syndrome. Oligohydramnios occurs with severe urinary tract obstruction or renal agenesis, which is associated with pulmonary hypoplasia (Potter's syndrome).

**CLINICAL MANIFESTATIONS** of renal disease vary with the type and severity of abnormality. Certain findings are indicative or suggestive of renal disease:

- **Potter's syndrome** (renal agenesis and pulmonary hypoplasia) is a fatal condition that has typical physical abnormalities: flat nose, low set ears, receding chin, arthrogyposis and, often, a bell-shaped chest. With prolonged oligohydramnios due to other causes (*e.g.*, obstructive uropathy, prolonged rupture of fetal membranes), the infant may show similar physical features and the severity of pulmonary hypoplasia varies from absent to severe, depending on duration and severity of oligohydramnios.
- **Dysmorphic features** suggestive of renal disease include abnormal ears, single umbilical artery, hypospadias, anorectal abnormalities, polythelia (supernumerary nipples), vertebral anomalies and esophageal atresia (with or without tracheo-esophageal fistula).
- **Lateral abdominal mass:** polycystic or multicystic kidneys, hydronephrosis, tumor (Wilm's)
- **Ascites** (urinary) due to rupture of obstructed urinary tract
- **Suprapubic mass** may be an enlarged bladder secondary to urethral obstruction
- **Abdominal wall defects:** exstrophy of bladder, cloacal exstrophy, "prune belly" (absence of abdominal wall muscle due to fetal urinary ascites)
- **Failure to palpate kidney:** unilateral renal agenesis, renal malposition, horseshoe kidney
- **Hypertension** is frequently due to renal disease. The commonest cause of neonatal hypertension is renovascular disease secondary to clots or emboli from a "high" umbilical arterial catheter.
- **Anuria or oliguria:** However, only 90% of normal infants urinate in the first 24 hours after birth; **therefore, 10% of normal infants do not urinate on the first day.**

The presence of any of the above signs should alert one to the possibility of renal dysfunction and raise the possibility of further diagnostic work-up including, in addition to careful measurement of intake and urine output, serum creatinine, BUN and electrolytes, abdominal ultrasound.

**ACUTE RENAL FAILURE (ARF)**, defined as a serum creatinine >1.5 mg/dL, occurs in 6-23% of ICN patients and is described as either **oliguric ARF** (urine output <1 mL/kg/hr) or **non-oliguric ARF** (urine output is maintained despite decreased glomerular and tubular function). There are three categories of ARF:

- **Functional (Prerenal):** due to ↓ renal perfusion, not the kidney itself
- **Intrinsic (Renal):** usually renal tubular dysfunction caused by an acute insult
- **Obstructive (Postrenal):** due to anatomic urinary tract obstruction

### 1. Findings in ARF:

#### A. Clinical signs associated with ARF include:

- Oliguria or anuria
- Hematuria, proteinuria
- Fluid overload
- Hypertension
- Cardiac dysrhythmias (with ↑ K<sup>+</sup>)

#### B. Laboratory findings in ARF include:

- Creatinine > 1.5 mg/dL (An elevated creatinine on the first post-natal day is more likely due to elevated maternal creatinine or increased production of creatinine from tissue breakdown.)
- Abnormal electrolytes (especially, ↑ K<sup>+</sup>)
- ↑ BUN

#### C. Initial Evaluation of ARF:

- Careful perinatal & neonatal history
- Physical examination for signs suggestive of renal disease (see above)
- Abdominal ultrasound
- Laboratory tests:
  - Serum: electrolytes, BUN, creatinine, CBC, pH and blood gas tensions
  - Urine: pH, urinalysis, culture, gram stain Na<sup>+</sup>, K<sup>+</sup>, Cl<sup>-</sup>, osmolality

#### D. Diagnostic Indices in ARF (useful in determining type of ARF):

Test	Findings in	
	Pre-Renal ARF	Renal ARF
Urine osmolality (mmol/L)	>400	<400
Urine Na <sup>+</sup> (mEq/L)	31 ± 19	63 ± 35
Urine/Plasma Creatinine	29 ± 16	10 ± 4
Fractional excretion of sodium (FE <sub>Na</sub> , %)	<2.5	>2.5
	$\left( \frac{[\text{Urine Na}^+][\text{Serum Creatinine}]}{[\text{Serum Na}^+][\text{Urine Creatinine}]} \times 100 \right)$	

### 2. Causes of ARF:

**A. Functional Renal Failure**, the commonest type, is characterized by inadequate renal perfusion, and is often called prerenal azotemia. Causes are any conditions that lead to inadequate renal perfusion (*e.g.*, dehydration, hypovolemia, shock, myocardial failure, patent ductus arteriosus). **Treatment** is correction of the underlying cause and supportive care.

**B. Intrinsic Renal Failure:** The most common cause is acute tubular necrosis (ATN) resulting in renal tubular dysfunction. ATN may be precipitated by shock, prolonged prerenal state, or nephrotoxic drugs. Oliguria or anuria is prominent. With recovery, there may be polyuria with dehydration and electrolyte disorders.

**Treatment** includes:

- Strict I & O (urinary catheter)
- At onset, consider fluid challenge (10-20 mL/kg) to R/O functional ARF
- If no response to fluid challenge, restrict intake to insensible water loss and urine output.
- Consider one dose of furosemide
- Consider low dose dopamine (2-4 mcg/kg/min) to ↑ renal blood flow
- Restrict intake of  $K^+$  and  $PO_4$
- D/C any nephrotoxic drugs
- Obtain Nephrology Consult
- Dialysis as needed

**C. Obstructive Renal Failure** is due to bilateral urinary tract obstruction (in males usually posterior urethral valves). Obstruction in fetal life can result in cystic dysplasia of the kidney. Treatment consists of diagnosis and relief of the obstruction and careful supportive care. With relief of obstruction, there may be polyuria with electrolyte disorders. Infection is common and patients receive urinary antibiotic prophylaxis.

### **OTHER NEONATAL RENAL DISORDERS:**

**1. Renal tubular acidosis (RTA)**, caused by defects in reabsorption of  $HCO_3^-$  and secretion of  $H^+$  ions, generally presents metabolic acidosis and inappropriately high urine pH (>6.0). This occurs frequently in preterm infants and is transient. RTA can also be associated with a wide variety of other conditions.

**2. Syndrome of Inappropriate Secretion of Antidiuretic Hormone (SIADH)** is a very infrequent disorder of fluid and electrolyte balance due to excessive release of antidiuretic hormone leading to water retention and hyponatremia. Findings include oliguria, low serum osmolality, hyponatremia, concentrated urine and elevated urine sodium. Treatment is strict restriction of fluid intake.

**3. Renal Cystic Disease:** There are two types:

**A. Autosomal recessive polycystic kidney disease** presents with abdominal masses, hypertension, renal insufficiency, hepatic and biliary fibrosis and there may be pulmonary failure from lung hypoplasia.

**B. Autosomal dominant polycystic kidney disease** usually has its onset in the 3<sup>rd</sup> to 5<sup>th</sup> decade of life. In some newborns, it can present with bilateral flank masses, hypertension, renal insufficiency and cystic involvement of other organs.

**4. Congenital nephrotic syndrome** may be secondary to congenital infections, but most commonly is Finnish-type congenital nephrotic syndrome, an autosomal recessive disease. These infants present with proteinuria that may be severe enough to cause fetal hydrops.