Acute Renal Failure

:Lecturer

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TUCOM

:Objectives

- Definition of ARF.
- Pathogenesis of ARF.
- Clinical manifestations and diagnosis of ARF.
- Laboratory findings in patient with ARF.
- Treatment of ARF and its complications.
- Prognosis of ARF.

:Definition

Acute renal failure (ARF) is a clinical syndrome in which a sudden deterioration in renal function results in the inability of the kidneys to maintain fluid and electrolyte homeostasis. ARF occurs in 2–3% of children admitted to pediatric care centers and in as many as 8% of infants in the neonatal .intensive care unit

PATHOGENESIS

ARF has been conventionally classified into 3 categories: prerenal, intrinsic renal, and postrenal
Common Causes of Acute Renal Failure

PRERENAL

Prerenal ARF, also called prerenal azotemia, is characterized by diminished effective circulating arterial volume, which leads to inadequate renal perfusion and a decreased glomerular filtration rate (GFR). Evidence of kidney damage is absent. If the underlying cause of the renal hypoperfusion is reversed promptly, renal function returns to normal. If hypoperfusion is sustained, .intrinsic renal parenchymal damage may develop

Dehydration
Hemorrhage
Sepsis
Hypoalbuminemia
Cardiac failure

INTRINSIC RENAL

Intrinsic renal ARF includes a variety of disorders characterized by renal :parenchymal damage, including sustained hypoperfusion/ ischemia

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
Tumor lysis syndrome

POSTRENAL

Postrenal ARF includes a variety of disorders characterized by obstruction of the urinary tract. Relief of the obstruction usually results in recovery of renal function except in patients with associated renal dysplasia or prolonged .urinary tract obstruction

Posterior urethral valves
Ureteropelvic junction obstruction
Ureterovesicular junction obstruction
Ureterocele
Tumor
Urolithiasis
Hemorrhagic cystitis
Neurogenic bladder

CLINICAL MANIFESTATIONS AND DIAGNOSIS

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

The physical examination must be thorough, with careful attention to volume status. Tachycardia, dry mucous membranes, and poor peripheral perfusion suggest inadequate circulating volume and the possibility of prerenal .ARF

Peripheral edema, rales, and a cardiac gallop suggest volume overload and the possibility of intrinsic ARF from .glomerulonephritis or ATN

The presence of a rash and arthritis may suggest systemic lupus erythematosus (SLE) or Henoch-Schönlein purpura .nephritis

Palpable flank masses may suggest renal vein thrombosis, .tumors, cystic disease, or urinary tract obstruction

LABORATORY FINDINGS

:Laboratory abnormalities may include
anemia (the anemia is usually dilutional or hemolytic, as in SLE, renal-
.vein thrombosis, HUS)
.leukopenia (SLE)-
.thrombocytopenia (SLE, renal vein thrombosis, HUS)-
hyponatremia (dilutional); metabolic acidosis; elevated concentrations-
of blood urea nitrogen, creatinine, uric acid, potassium, and phosphate
.(diminished renal function); and hypocalcemia (hyperphosphatemia)
-The serum C3 level may be depressed
The presence of hematuria, proteinuria, and red blood cell or granular-
.urinary casts suggests intrinsic ARF, in particular glomerular disease
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 ARF from-
intrinsic ARF . 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 (FENa) <1%
(<2.5% in neonates) most likely have prerenal ARF. Those with a specific
gravity of <1.010, low urine osmolality (UOsm < 350 mOsm/kg), high urine
sodium (UNa > 40 mEq/L), and FENa greater than 2% (>10% in neonates)
.most likely have intrinsic ARF
Chest radiography may reveal cardiomegaly and pulmonary congestion-
.(fluid overload)
Renal ultrasonography may reveal hydronephrosis and/or hydroureter,-
.which are suggestive of urinary tract obstruction
Renal biopsy may ultimately be required to determine the precise cause-
of ARF in patients who do not have clearly defined prerenal or postrenal
.ARF

TREATMENT

MEDICAL MANAGEMENT

Bladder catheter should be placed to ensure adequate drainage of the urinary tract if there is suspicion of obstruction , also used to accurately .monitor urine output during ARF
Determination of the volume status is of critical importance . If there is no evidence of volume overload or cardiac failure, 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 points toward the presence of intrinsic or postrenal ARF

Diuretic therapy should be considered only after the adequacy of the circulating blood volume has been established. Mannitol (0.5 g/kg) and furosemide (2–4 mg/kg) may be administered as a single IV dose.

If urine output is not improved, then a continuous diuretic infusion may be considered.

If there is no response to a diuretic challenge, diuretics should be discontinued and fluid restriction becomes 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.

Markedly hypervolemic patients may require further fluid restriction, omitting the replacement of insensible fluid losses, urine output, and extrarenal losses to diminish the expanded intravascular volume.

Hyperkalemia

In ARF, rapid development of hyperkalemia (serum potassium level >6 mEq/L) may lead to cardiac arrhythmia, cardiac arrest, and death. The earliest electrocardiographic change seen in patients with developing hyperkalemia is the appearance of peaked T waves. This may be followed by widening of the QRS intervals, ST segment depression, ventricular .arrhythmias, and cardiac arrest

Exogenous sources of potassium (dietary, intravenous fluids, total-.parenteral nutrition) should be eliminated

Sodium polystyrene sulfonate resin (Kayexalate), 1 g/kg, should be-given orally or by retention enema. A single dose of 1 g/kg can be expected to lower the serum potassium level by about 1 mEq/L. Resin therapy may be repeated every 2 hr, the frequency being limited primarily .by the risk of sodium overload

More severe elevations in serum potassium (>7 mEq/L), especially if accompanied by electrocardiographic changes, require emergency measures in addition to Kayexalate. The following agents should be administered:

- Calcium gluconate 10% solution, 1.0 mL/kg IV, over 3–5 min
- Sodium bicarbonate, 1–2 mEq/kg IV, over 5–10 min
- Regular insulin, 0.1 U/kg, with glucose 50% solution, 1 mL/kg, over 1 hr

Calcium gluconate counteracts the potassium-induced increase in myocardial irritability but does not lower the .serum potassium level

Administration of sodium bicarbonate and insulin and glucose lowers the serum potassium level by shifting potassium from the extracellular to the intracellular .compartment

A similar effect has been reported with the acute administration of β- adrenergic agonists in adults, but there .are no controlled data in pediatric patients

Persistent hyperkalemia should be managed by .dialysis

:Metabolic acidosis

Mild metabolic acidosis is common in ARF because of retention of hydrogen ions, phosphate, and sulfate, but it rarely requires treatment. If acidosis is severe (arterial pH <7.15; serum bicarbonate <8 mEq/L) or contributes to hyperkalemia, treatment is required. The acidosis should be corrected partially by the intravenous route, generally giving enough bicarbonate to raise the arterial pH to 7.20 . The remainder of the correction may be accomplished by oral administration of sodium bicarbonate after normalization of the serum calcium and phosphorus levels. Rapid correction of acidosis reduces the ionized calcium concentration and .may precipitate tetany

:Hypocalcemia

.Is primarily treated by lowering the serum phosphorus level
Calcium should not be given intravenously, except in cases-
.of tetany, to avoid deposition of calcium salts into tissues
.Low phosphorus diet-
Phosphate binders should be orally administered to bind -
any ingested phosphate and increase gastrointestinal
phosphate excretion. Common agents include sevelamer,
.calcium carbonate and calcium acetate

:Hyponatremia

Is most commonly a dilutional disturbance that must be corrected by fluid restriction rather than sodium chloride administration. Administration of hypertonic (3%) saline should be limited to those patients with symptomatic hyponatremia (seizures, lethargy) or those with a serum sodium level <120 .mEq/L

:Gastrointestinal bleeding

Because of uremic platelet dysfunction, increased stress, and heparin exposure if on hemodialysis or .continuous renal replacement therapy
Oral or intravenous H2 blockers such as ranitidine- are commonly administered to prevent this .complication

:Hypertension

May result from hyperreninemia associated with the primary disease process and/or expansion of the extracellular fluid volume and is most .common in ARF patients with acute glomerulonephritis or HUS
.Salt and water restriction is critical-
.Diuretic administration may be useful -
Isradipine (0.05–0.15 mg/kg per dose, maximum dose 5 mg qid) may be- .administered for relatively rapid reduction in blood pressure
Longer acting calcium channel blockers (amlodipine, 0.1–0.6 mg/kg/24- hr divided bid) or β blockers (propranolol, 0.5–8 mg/kg/24 hr divided bid .or tid may be helpful in maintaining control of blood pressure
Children with severe symptomatic hypertension (hypertensive urgency/- emergency) should be treated with continuous infusions of sodium .nitroprusside (0.5–10 μg/kg/min), labetalol (0.25–3.0 mg/kg/hr)

:Neurologic symptoms

In ARF it may include headache, seizures, lethargy, and confusion. Potential etiologic factors include hyponatremia, hypocalcemia, hypertension, cerebral hemorrhage, cerebral .vasculitis, and the uremic state
Diazepam is the most effective agent in controlling seizures,- and therapy should be directed toward the precipitating .cause

:Anemia

Anemia of ARF is generally mild (hemoglobin 9–10 g/dL) and .primarily results from volume expansion (hemodilution)
Children with HUS, SLE, active bleeding, or prolonged ARF- may require transfusion of packed red blood cells if their .hemoglobin level falls below 7 g/dL
In hypervolemic patients, blood transfusion carries the risk- of further volume expansion, which may precipitate hypertension, heart failure, and pulmonary edema. Slow (4–6 hr) transfusion with packed red blood cells (10 mL/kg) .diminishes the risk of hypervolemia

:Nutrition

Is of critical importance in children who develop .ARF
.Potassium, and phosphorus should be restricted-
Protein intake should be restricted moderately-
while maximizing caloric intake to minimize the .accumulation of nitrogenous wastes
In critically ill patients with ARF, parenteral-
hyperalimentation with essential amino acids .should be considered

DIALYSIS

Indications for dialysis in ARF include the following
Volume overload with evidence of hypertension and/or pulmonary -
edema refractory to diuretic therapy
Persistent hyperkalemia -
Severe metabolic acidosis unresponsive to medical management -
.Neurologic symptoms (altered mental status, seizures)-
Blood urea nitrogen greater than 100–150 mg/dL -
Calcium/phosphorus imbalance, with hypocalcemic tetany-
Inability to provide adequate nutritional intake because of the need for-
. severe fluid restriction

In patients with ARF, dialysis support may be necessary for days or for up to 12 wk. Many patients with ARF require .dialysis support for 1–3 wk

Intermittent hemodialysis is useful in patients with relatively stable hemodynamic status. Intermittent hemodialysis may be performed 3 to 7 times per week based on the patient's .fluid and electrolyte balance

Peritoneal dialysis is most commonly employed in neonates and infants with ARF, although this modality may be used in .children and adolescents of all ages

Continuous renal replacement therapy (CRRT) is useful in patients with unstable hemodynamic status, concomitant .sepsis, or multiorgan failure in the intensive care setting

PROGNOSIS

The mortality rate in children with ARF is variable- and depends entirely on the nature of the underlying disease process rather than on the renal .failure itself

Children with ARF caused by a renal-limited - condition such as postinfectious glomerulonephritis have a very low mortality rate (<1%); those with ARF related to multiorgan failure .have a very high mortality rate (>90%)

The prognosis for recovery of renal function- depends on the disorder that precipitated ARF. Recovery of renal function is likely after ARF resulting from prerenal causes, HUS, ATN, acute interstitial nephritis, or tumor lysis syndrome. Recovery of renal function is unusual when ARF results from most types of rapidly progressive glomerulonephritis, bilateral renal vein thrombosis, .or bilateral cortical necrosis