ACUTE RENAL FAILURE IN NEONATES

Acute renal failure (ARF) is a frequent clinical condition in sick neonates. There is a wide variation in the incidence of ARF across studies. It affects approximately 1% to 24% of newborns in the NICU.[1,2] In a recent report from a tertiary centre of Thailand, the prevalence of ARF among newborns was found to be 6.3%, with more than 65% developing within 7 days of birth.[3] ARF is an acute reduction in glomerular filtration rate (GFR) with both failure to remove solutes and water leading to concurrent net solute and water retention – oligo-anuric renal failure.[2]

Classification

Based on the urine output, it can be of two types:
1. Oligoanuric 2. Non-oliguric

Practical tip
Normal urine output can be found in up to one third neonates with ARF. Conversely, anuria can also occur in syndrome of inappropriate ADH secretion in the absence of ARF.

Based on the site of origin of insult it can be of types: [4]

1. Prerenal (75- 80%)
2. Intrinsic renal (10-15%)
3. Postrenal (5%)

Persistence of insult can convert pre renal or post renal failure to intrinsic renal failure. However, there is an increasing awareness that even moderate decrease in renal function is important in the critically ill and contributes significantly to morbidity as well as mortality.

Diagnosis

Plasma Creatinine

Neonatal ARF is defined as

1. Plasma creatinine more than 1.5 mg/dL for at least 24 to 48 hrs if mother's renal function is normal[2]
2. Serum creatinine raised more than 0.3 mg/dL over 48 hours
3. Serum creatinine fails to fall below maternal plasma creatinine within 5-7 days

Some studies say, if the neonate's creatinine increases two times between any two measurements, this is defined as ARF. The above definitions have reasonable accuracy in term neonates. In preterm neonates, there is a transient increase in serum creatinine, peaking on day 4, followed by a progressive decline to normal neonatal levels by a postnatal age of 3 to 4 weeks. This occurs due to re-absorption of creatinine across the permeable tubules.

Urine output

Oliguria: It has been defined as urine output less than 1 mL/kg/hr after first day of life for both term and preterm neonates. However, some term neonates may void for the first time at around 24 hrs of life. It has been seen that 17% of newborns void in the delivery room, approximately 90% by 24 hours, and 99% void by 48 hours.[1]

Practical tip
ARF can also present with normal renal output in one third of the cases, especially in asphyxiated neonates. Further, in VLBW infants without ARF, there could be oliguric phase that resolve spontaneously in the first few days of life.[1]

Concept of acute kidney injury (AKI)

An attempt has been made to define renal failure bringing uniformity across age, gender and body mass index and reduce the need for a baseline value of serum creatinine. The product of such an attempt is the concept of acute kidney injury (AKI).[5]

Definition of AKI

An abrupt (within 48 hours) reduction in kidney function currently defined as an absolute increase in serum creatinine of more than or equal to 0.3 mg/dL (≥ 26.4 µmol/L), a percentage increase in serum creatinine of more than or equal to 50% (1.5-fold from baseline), or a reduction in urine output (documented oliguria of less than 0.5 mL/kg per hour > 6 hours). Thus, the concept of AKI creates a new paradigm which encompasses not only established renal failure but also functional impairment relative to the physiological demand.[5]

Pre-renal versus intrinsic renal failure

- The usefulness of differentiating prerenal from intrinsic renal failure was believed to lie in the fact that in the former the damage to the kidneys is yet to begin, whereas in the latter it already has. However, with the increasing recognition of AKI as a continuum of volume responsiveness through unresponsiveness, this distinction has blurred out. There is a definite role of appropriate fluid therapy in reversing the renal damage in the former.[5]

- When a baby has not passed urine in the past 12 hrs, the first thing is to look for distended bladder by palpation of the abdomen or ultrasound (if available at bed side). *It is better to avoid catheterization of the bladder to prevent infection*, but it may be necessary in sick babies. If required, it has to be done with a 5 Fr lubricated feeding tube under strict asepsis. *Compression of the bladder (supra pubic pressure) should be avoided especially in preterm infants for the fear of VUR and rarely bladder rupture.* [1]

- After confirming the absence of urine in the bladder, a fluid challenge can be given. The common causes of pre renal azotemia are hypovolemia, systemic hypotension and hypoxia (in more than 80% of cases).[2] In the absence of obvious sign of fluid, a normal saline bolus of 10 mL/kg can be given over 20 min (or 20 mL/kg over 2 hrs). In spite of the fluid challenge, if urine output fails to ensue, frusemide can be given in a single dose of 1 mg/kg (in a non dehydrated patient).

Approach to a neonate with renal failure:

History:

a) Prenatal history:
   - History of maternal drug intake like enalapril or indomethacin which decrease glomerular filtration should be sought
   - Maternal uncontrolled diabetes is associated with genitourinary malformations.
   - Oligohydramnios may result from fetal oliguria due to bilateral congenital renal disease, bilateral/lower urinary tract obstruction or maternal drugs. Likewise, polyhydramnios may result from a defect in urinary concentration whereas hydrops may be the first sign of congenital nephrotic syndrome

b) Family history: May be present in cases of polycystic kidney disease, renal tubular disorders and congenital nephrotic syndrome.

c) Natal history:
   - Perinatal asphyxia, respiratory distress, sepsis and shock may predispose the kidneys to anoxic injury culminating in acute tubular necrosis.
   - Oliguria in asphyxia may result from prerenal failure mediated by endothelin, intrinsic renal failure (ATN) or SIADH.
   - Seizures may occur secondary to hypoxia, intracranial hemorrhage, hypoglycemia, hypocalcemia, hypertension and uremia.

d) Micturition history: As much as 7% newborns do not void in the first 24 hours. The most common cause of delayed micturition is inadequate perfusion of the kidneys. However, intrinsic renal disorders and urinary tract obstruction need to be ruled out.

Physical examination:

Examination must include assessment of hydration (edema/dehydration), vital signs including blood pressure and a search for dysmorphic features (abnormal ears, pre-auricular pits, ambiguous genitalia, hypospadias, abdominal wall defects, aniridia, Potter facies), which are associated with renal malformations. Spontaneous pneumothorax may be associated with renal abnormalities. Abdominal masses are present in 0.8% newborns, most of which are genitourinary in origin. A suprapubic mass could indicate a palpable bladder. In males, the urine stream should be carefully observed as thin stream, dribbling or post voidal residual bladder suggest posterior urethral valve.

Routine renal ultrasound for babies with single umbilical artery: what is evidence?

Studies indicate that 10% of babies with single umbilical artery (SUA) have an associated major congenital renal malformation. However a recent meta-analysis ascertains that 14 cases of SUA will have to be screened to pick up one major renal malformation, which could also be picked up with a good pediatric follow up. So the value of routinely screening all babies with SUA for renal malformations is not established.[6]

Laboratory investigations:

Babies with ARF must be investigated not only to look for the cause and but also to look at the complications. Apart from serum creatinine and blood urea, serum electrolytes, arterial blood gas analysis, urine sodium, urine creatinine must be done.

Role of indices

Differentiation of prerenal and intrinsic renal failure can be done basing on urinary indices (Table 1: Parameters to differentiate pre renal from intrinsic renal failure1. The important prerequisite is that the urine sample for measuring indices must be obtained *prior to fluid and diuretic challenge*. Among the indices available, fractional excretion of Na (FENa) is the most preferred. FENa more than 2.5% to 3.0% is associated with intrinsic ARF.

Preterm babies lose more sodium in the urine due to the tubular immaturity, hence a FENa of more than 6% can be used to define intrinsic ARF in babies born between 29-32 weeks of gestation.[7] Likewise renal failure index (RFI) more than 4 in term and more than 8 in preterm babies <32 weeks is suggestive of intrinsic ARF.

Table 1: Parameters to differentiate pre renal from intrinsic renal failure[1]

Parameters	Prerenal	Intrinsic renal
Urinary Na	≤20 mEq/L	>50 mEq/L
Renal failure index*	Low <1	High >4
Fractional excretion of Na$	≤1	>3

* Renal failure index (RFI): $\frac{\text{Urinary Na X plasma creatinine}}{\text{Urine creatinine}} \times 100$

$ Fractional excretion of sodium (FENa): $\frac{\text{Urinary Na X plasma creatinine}}{\text{Plasma sodium X urine creatinine}} \times 100$

Urine microscopic analysis: The presence of granular and hyaline casts, RBC, protein and tubular cells suggests an intrinsic cause. In asphyxia, there is an increase in epithelial cells and transient microscopic hematuria with leucocytes. The excretion of low molecular weight proteins like $beta_2$-glycoprotein is a sensitive indicator of tubular damage as in asphyxia.

Radiological Evaluation:

Ultrasonography and Doppler: Useful in ruling out congenital anomalies like polycystic kidneys, dysplasia of kidneys and obstructive causes like posterior urethral valves. Renal Doppler studies are useful in diagnosing vascular thrombosis.

Voiding cysto-urethrography can identify lesions of the lower urinary tract that cause obstruction, such as posterior urethral valves.

Etiology of renal failure

Having differentiated prerenal from intrinsic renal failure, look for the exact etiology of renal failure. There are several causes of ARF (Table 2).

Table 2: Etiology of neonatal renal failure:

I. Congenital malformations
- Renal agenesis
- Renal hypoplasia/dysplasia
- Cystic diseases of kidney e.g. autosomal recessive polycystic kidney

II. Acquired renal disorders
- Acute tubular necrosis
  - Perinatal asphyxia
  - Perinatal hypoxia due to respiratory distress syndrome, traumatic delivery
  - Sepsis
  - Hypovolemia due to dehydration, severe patent ductus arteriousus, intraventricular hemorrhage, post operatively, increased insensible water loss
- Vascular
  - Arterial thrombosis or embolism or stenosis
  - Venous thrombosis (Infants of diabetic mothers, dehydration, polycythemia)
- Drugs: Maternal use of ACE* inhibitors, indomethacin
  Neonate: indomethacin, aminoglycosides, radiographic contrast media

III. Urinary tract obstruction
Posterior urethral valves
Pelviureteric obstruction, ureterovesical obstruction

* ACE: angiotensin converting enzyme.

In one series of newborns with ARF, sepsis was the most common cause of AKI (30.9%) followed by hypovolemia (18.7%), kidney, ureter and bladder (KUB) anomalies (12.2%), congestive heart failure (12.2%) and birth asphyxia (11.5%).[3]

Some special considerations:
- A neonate with oliguric ARF, hematuria, hypertension with/without loss of femoral pulses suffers from bilateral renal artery thrombosis. Thrombolysis / thrombectomy is indicated in refractory hypertension in such a neonate
- Renal venous thromboses should be suspected in any newborn who presents with bilateral flank mass, proteinuria, hematuria with/without oliguria and thrombocytopenia in the presence of a setting like polycythemia, severe dehydration and maternal diabetes

Fluid management
- Fluids must be restricted to insensible water loss (IWL) along with urinary loss. The urinary loss must be replaced volume for volume. The insensible water loss in a term neonate is 25 mL/kg/day. In preterm neonates, this can vary between 40-100 mL/kg/day depending on gestation, postnatal age, use of radiant warmers, phototherapy etc.[8]

- Fluid requirement should be revised based on urine output, weight and assessment of extracellular volume status, preferably every 8 hourly.

- The insensible water losses should be replaced with 5-10% dextrose. The urine output should be replaced volume by volume with N/5 saline.[5]

- During the polyuric phase, hourly monitoring of urine output and serial monitoring of serum electrolytes with appropriate replacement of sodium, potassium and water are indicated to prevent dehydration, hyponatremia and hypokalemia.[1]

Electrolyte disturbances

Hyponatremia

Babies can have hyponatremia in oliguric renal failure.

Hyponatremia is due to dilution secondary to water retention hence has to be corrected with fluid restriction. In most of the cases, there is no sodium deficit.

- If serum sodium is between 120-135 mEq/L, restriction of fluids will suffice. Serum sodium must be monitored at least 12 hrly.

- If hyponatremia is associated with symptoms like seizures, or if serum sodium is less than 120 mEq/L, it requires prompt correction with 3% hypertonic saline over 2 hours, using the formula

Na required (mEq) = [Na desired – Na actual] x body weight (kg) x 0.8

- Hyponatremia unresponsive to above therapy is an indication for dialysis.

- Babies with non-oliguric ARF may have urinary sodium losses of up to 10 mEq/kg/day and these must be replaced.

- Care should be taken not to increase the serum sodium by more than 0.5 mEq/L/h.

Hyperkalemia

Hyperkalemia (K^+ > 6.5 mEq/L[1]): It is one of the most dangerous complications of ARF. It results from reduction in glomerular filtration rate, urinary potassium secretion, acidosis, immature tubular response to aldosterone and cellular breakdown.

Practical tip

If hyperkalemia is associated with hypoglycemia, hyponatremia and hypotension, consider a diagnosis of adrenal insufficiency.

- The first step in the management of hyperkalemia is to stop all potassium in the fluids as well as drugs which can accentuate hyperkalemia (indomethacin, ACE inhibitors, potassium sparing diuretics)

- ECG will help in diagnosing cardiac effects of hyperkalemia. If ECG changes are evident, IV calcium gluconate ??? ml/kg 10% slowly with cardiac monitoring is given. This will decrease the myocardial excitability but will not lower the potassium levels.

- This should immediately be followed by methods to decrease the potassium levels (Table 3). Hyperkalemia which is unresponsive to medications is one of the most common indications for instituting dialysis.

Table 3: Management of Hyperkalemia[1]:

ECG strip of hyperkalemia

Level of K^+ at which it is instituted	Medication	Dose	Mechanism/ Degree of effect	Onset of action
ECG changes suggestive of hyperkalemia	Calcium gluconate (10%)	0.5 to 1 mL/kg over 5-10 min	Modifies myocardial excitability, no decrease in K levels	5-10 min
6.5-7.5 mEq/L	Glucose and insulin	0.5 g/kg/h of glucose and 0.1 U/kg/hr infusion of insulin	Intracellular uptake of potassium	30 min.
6.5-7.5 mEq/L	Salbutamol IV infusion	4 µg/kg over 20 min	Intracellular uptake of potassium	30-40 min
More than 6.0 mEq/L	Cation exchange resin (Na/Ca polystyrene sulfonate)	1g/kg intrarectally q 6 h	Exchange of K for Na or Ca. 1g/kg reduces K levels by 1 mEq/kg	1-2 hrs, may take upto 6 hours
More than 7.5 mEq/L	Exchange blood transfusion	2/3 Washed RBC reconstituted with 5% albumin	Uptake of K by RBC.	Minutes
K+ more than 7.5 mEq/L	Peritoneal dialysis	Use a dialysate with low K+ concentration	Dialysis	Minutes.

Practical tips[1]:

- Saturate the plastic tubing with insulin solution before infusing to the baby
- Oral administration of resins is associated with the risk of concretions, hypernatremia and fluid overload – avoid in VLBW infants and those with poor peristalsis
- Salbutamol aerosol may not be very effective in neonates

Hypocalcemia can develop in babies with ARF due to hyperphosphatemia and skeletal resistance to parathyroid hormone. Symptomatic hypocalcemia should be corrected by infusing 10% calcium gluconate at a dose of 0.5-1 mL/kg over 5-10 min under cardiac monitoring. Also, during the oliguric phase, no intake of phosphorous/ magnesium is to be provided.

Role of dopamine

At doses less than 5 mEq/L, dopamine acts via DA_1 and DA_2 receptors to increase renal blood flow. But preterm infants are hypersensitive to alpha receptors and hence even low doses of dopamine can cause vasoconstriction and raise renal vascular resistance.[9] This may explain the difficulty in dosing of dopamine for improving renal function. Dopamine when combined with frusemide causes natiruresis and diuresis in preterm infants with RDS and oliguria.[10]

Dopamine in ARF: what is evidence?

The Cochrane meta-analysis of three studies concluded that dopamine has no role in the management of acute renal failure due to indomethacin.[11]

In their meta-analysis, Friedrich et al., analyzed 61 randomized or quazi-randomized controlled trials of low dose dopamine and found no improvement of survival, no decrease in dialysis requirement, no improvement in renal function and improvement in urine output only on the first day of therapy in adults with ARF of any cause.[12]

Nutrition

- The goal is to provide 100 kcal/kg/day as babies with ARF are catabolic. Proteins or amino acids can be provided in a dose of 1-2 g/kg/day[13].
- If enteral feeding is possible, breast milk can be used, failing which, low phosphate formula can be given. Caloric density can be increased by adding medium chain triglycerides.
- In the baby is on parenteral nutrition, a central venous catheter may be needed to infuse hypertonic glucose in order to prevent hypoglycemia.

Acidosis

Mild metabolic acidosis is common in babies with ARF. If pH is <7.2 and bicarbonate <18 mEq/L, sodium bicarbonate is given in a dose of 1-2 mEq/kg over 3-4 hrs. But monitoring for fluid overload, hypernatremia, intracranial hemorrhage and hypocalcaemia is needed. Babies with persistent acidosis require dialysis.

Hypertension

Fluid overload in neonatal ARF can result in hypertension, which can be controlled with fluid restriction and antihypertensive agents. The development of severe hypertension in the setting of neonatal ARF should raise the suspicion for renal artery or venous thrombosis.

Commonly used antihypertensives in newborns are oral amlodipine (0.1-0.3 mg/kg/dose q 12-24 hourly), enalapril (0.1-0.4 mg/kg/day q 6-12 hourly, with careful monitoring of potassium and renal functions) and intravenous diazoxide (2-5 mg/kg/dose over 5 min q 4-24 hourly)[1].

Renal replacement therapy

The indications of renal replacement therapy are:

- Hyperkalemia refractory to medication
- Hyponatremia with volume overload (pulmonary edema, severe hypertension)
- Metabolic acidosis (TCO_2 < 16-18 mEq/L)
- Hypocalcemia

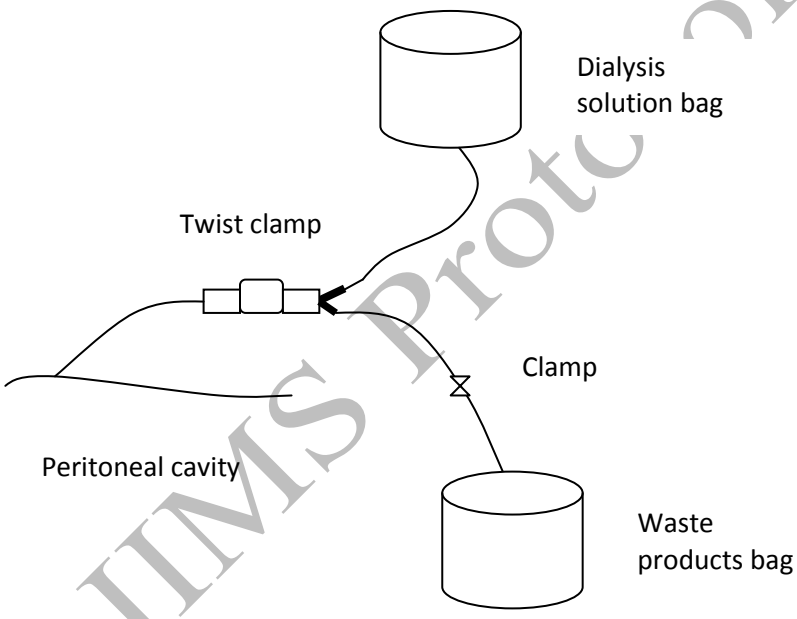
- Hyperphosphatemia refractory to therapy
- Inability to provide adequate nutrition due to fluid restriction.

The two purposes of renal replacement are ultrafiltration (removal of water) and dialysis (removal of solutes). Dialysis is a process of removal of plasma solutes by diffusion down their concentration gradient across a semi permeable membrane. Filtration involves removal of protein free plasma across a membrane by convection.

Peritoneal dialysis (PD) catheters: Peritoneal access in most institutes is achieved by a stiff catheter and trocar, but when used beyond 48-72 hours, infection rates are high.[14] Risk of infection and visceral injury is less with soft PD catheters made of silicone polymer of methyl-silicate, either in curled or straight configurations[15,16] (Fig. 1).

Most of the catheters have side holes that allow for easy ingress and egress of fluid. Permanent catheters have cuffs. Straight Tenckhoff and coiled Tenckhoff catheters are available. Coiled Tenckhoff catheters are useful for chronic dialysis. Detailed description of drug dose modification in ARF is available in literature[5].

Figure 1: Peritoneal dialysis circuit



Procedure:

- The first step involves creating a fluid filled reservoir by infusing 20-30 ml/kg dialysate into the peritoneum using a cannula.
- After this, the catheter is inserted into the peritoneal cavity and connected to a three way cannula. The common sites of insertion are in the midline below the umbilicus, right or left lower quadrant of the abdomen. Urinary bladder must be emptied before insertion of the catheter.
- The dialysate fluid is connected to a pediatric burette set and its terminal end is connected to one of the ports of three way cannula. The remaining port of the three way is connected to a intravenous (IV) set, the end of which is let into a sterile container (empty IV fluid bottle).

- The abdomen is filled with 20-30 mL/kg of dialysis fluid infused over 10 min. A dwell time of 20 to 30 min is used before draining the fluid over 10 min. The dwell time can be reduced in case of respiratory compromise.
- A total of 20-40 cycles can be used or it can be continued till the desired effect is obtained.
- Blood sugar, serum electrolytes and blood gas should be monitored every 6 hourly and serum creatinine every 24 hourly. At the end of the procedure the catheter can be removed and the tip and the fluid are sent for culture.

Dialysate Fluids:

The common dialysate fluid contains 1.7% dextrose with lactate. If higher gradient is required as in case of fluid overload 3% solution can be used. This can be prepared by adding 25 mL of 50% dextrose to one liter of 1.7% PD fluid.

- In case of liver failure as in asphyxia, lactate free bicarbonate containing fluid has to be used as these babies may be unable to metabolize lactate quickly. If baby becomes hypokalemic during the procedure, add one mL of KCl to one liter of dialysate fluid.

Composition of dialysis solutions[5]:
Osmotic agent: Dextrose 1.4- 3.9 g/dL or icodextrin 7.5 g/dL or amino acids 1.1 g/dL
Base: Lactate 35-40 mEq/L or bicarbonate 34 mEq/L
Sodium 132-134 mEq/L, Calcium 1.25-1.75 mM/L,
Magnesium 0.25-0.75 mM/L, Chloride 95-103.5 mEq/L

Complications of PD:

PD is invasive procedure and the following complications/contraindications need to be remembered.

- The chief complication of PD is peritonitis, the common organisms being coagulase negative Staphylococcus, *S. aureus* and gram negative bacteriae.[5]
- Catheter related bleeding, catheter malfunction, perforation of abdominal viscera, adhesion of catheter tip to momentum.
- Hyperglycemia can occur when higher concentrations of dextrose are used.
- PD cannot be done in babies with necrotizing enterocolitis, babies who underwent abdominal surgery and in those with severe respiratory compromise as it may worsen with abdominal distension. This may be circumvented with smaller volume cycles.
- Hypothermia must be prevented by using pre-warmed dialysis fluid.

  PD will be less effective in poor cardiac output or gut hypo perfusion.

Hemofiltration and hemodiafiltration are effective in neonates with ARF in whom PD is contraindicated. The complication rates are less. Hemofiltration is particularly useful in the

presence of fluid overload, but it needs a vascular access with large sized catheters and adequate mean arterial blood pressure. Hemodiafiltration is more useful in the presence of fluid overload and azotemia with electrolyte disturbances.[2]

Outcome

Non oliguric renal failure has a better prognosis when compared to oliguric renal failure. Mortality ranges from 25 to 78% in oligo anuric renal failure.[17] Long term abnormalities in GFR and tubular function are common in babies who survive ARF and is secondary to hyperfilteration in the surviving nephrons.

Follow up

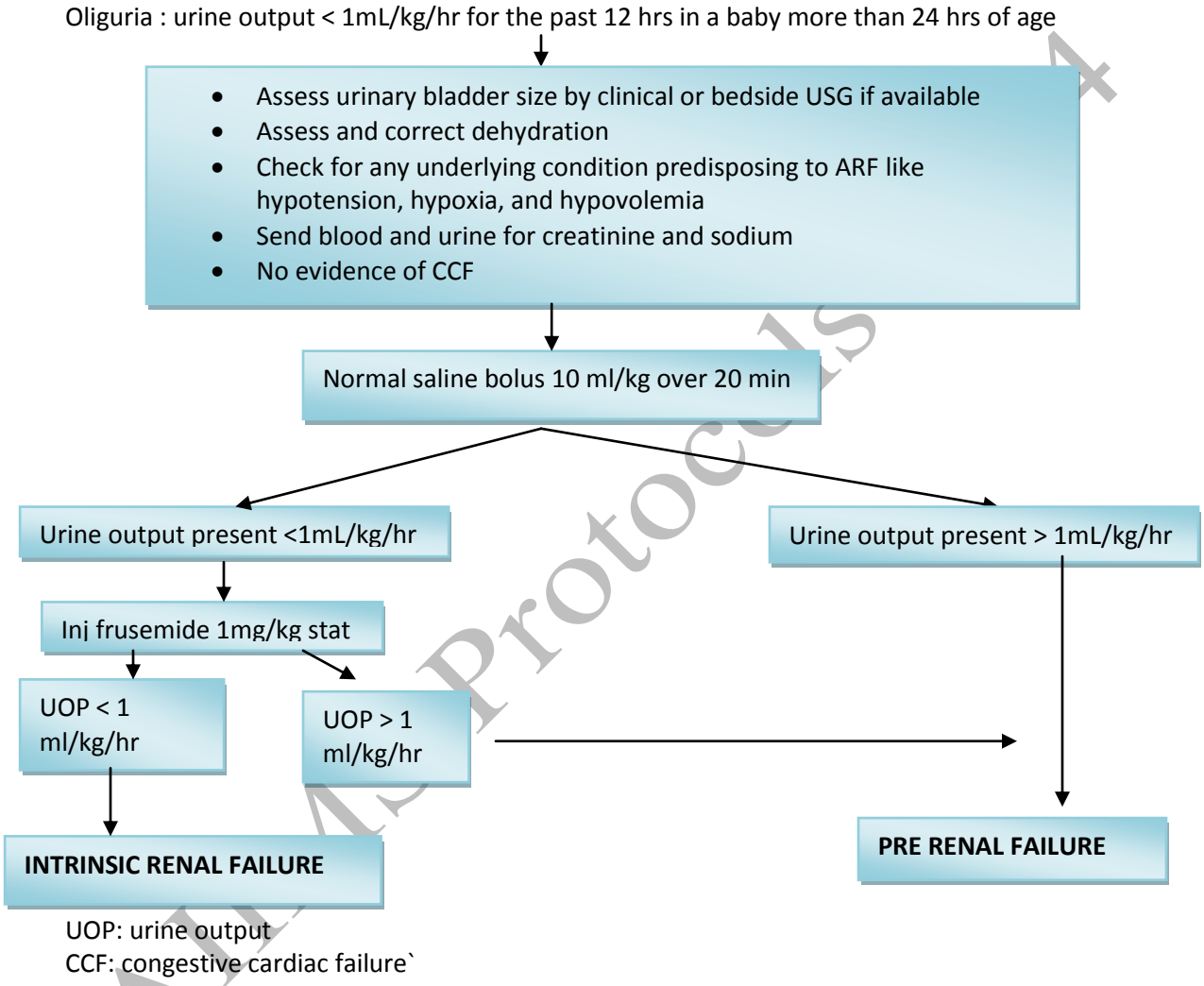
All babies who develop ARF need follow up. Adequacy of growth and nutrition, blood pressure, and renal function status has to be monitored. Newborns who have had ARF are predisposed to the development of chronic renal failure in the future. Long-term follow-up of extremely low birth weight infants who had neonatal ARF has shown that the risk factors for progression of renal disease at 1 year of age included a random urinary protein/creatinine ratio of greater than 0.6, serum creatinine greater than 0.6 mg/dL and a tendency to obesity with a body mass index greater than the 85th percentile.[18]

Research priorities:

- Role of novel therapies like ATP – Magnesium chloride/ thyroxine / peptide growth factors/ cytokines/ calcium channel blockers in intrinsic AKI

Research Question	Subjects	Study design	Proposed Outcomes
The role of *biomarkers* (urinary neutrophil gelatinase-associated lipocalin, cystatin C, urinary interleakin 18 and L-type fatty acid binding protein) in the diagnosis and prognosis of neonates with ARF/AKI (like cystatin C)	Newborns with acute kidney injury as defined in the protocol	Cohort study	Use as a diagnostic test, compared to existing gold standards (urine output and serum creatinine) Prognosis – to predict risk of progression, chronic renal failure or mortality
Role of early treatment strategies– Intravenous fluid bolus and diuretic challenge	Newborns with acute kidney injury , preferably early AKI	Randomised controlled trial	To assess the benefit (response measured as improvement in urine output or creatinine) and risks
Usefulness of novel therapies such as recombinant human growth factors, erythropoietin, atrial natriuretic peptide or N-acetyl cysteine in	Newborns with acute kidney injury	Randomised controlled trial	To assess the benefit (response measured as improvement in urine output or creatinine)

Flow chart



UOP: urine output
CCF: congestive cardiac failure`

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