Chapter 16

ACUTE RENAL INSUFFICIENCY

DEFINITION

Rapid onset of biochemical disorders resulting from inability of renal function to cope with regulatory functions upon which homeostasis, nitrogen excretion, fluid and acid base balance depend. Consequentely oliguria (less than 250 ml per square meter per day) or anuria, azotemia and metabolic acidosis are dominant features together with other signs of precipitating cause such as shock, pallor and bleeding diathesis. Occasionally urine output is not reduced but concentration capacity is greatly diminished called as high output failure.

A. Prerenal:

This is due to hypovolaemia and poor renal plasma flow. Urine is of high osmolality and high specific gravity with oliguria potentially reversible by correcting inadequate haemoperfusion. Underlying parenchymal abnormality may coexist.

B. Intrinsic Renal:

Established renal parenchymal damage is reflected in oliguria with low osmolality of urine and uraemia, accumulation of metabolites, metabolic acidosis, hyperkalaemia and disturbed homeostasis.

C. Post Renal:

Occurs most commonly as complication of obstructive uropathy often due to spina bifida but also occurs in posterior urethral valve or bilateral pelvi ureteric strictures. It has been noted also in drug crystalluria.

ACUTE RENAL FAILURE IN NEWBORN

Renal insufficiency in neonate is masked by placental action. Gross renal abnormality may be present varying from agenesis of kidney through severe dysplasia to gross hydronephrosis due to posterior urethral obstruction.

Renal venous thrombosis may occur prenatally.

With division of umbilical cord placental renal function ceases and uraemia begins.

Agenesis of kidneys may be suspected during delivery when oligohydramnios and amnion nodosa is noted and Potter face may be present.

Intravenous urogram followed by diagnostic nephrosonography confirms absence of kidneys and not merely lack of function.

Routine examination of newborn includes palpation of bladder which if enlarged may indicate posterior urethral obstruction.

Common acquired lesions which produce ARF in newborn are renal venous thrombosis and gram negative septicaemia.

Prerenal azotaemia is common in neonates who become dehydrated particularly when this is due to lack of adequate fluid intake. Hypertonic dehydration with azotemia is common complication in newborn and may lead to renal venous thrombosis.

Recognition of ARF in napkin period is difficult. Alert nurse will spot unusually dry successive napkins. Alternatively tachypnea of metabolic acidosis draws attention.

Treatment:

If condition is mild then oral hydration will overcome prerenal azotemia with oliguria secondary to dehydration.

Lesser degrees of ARF should be given low sodium, low potassium and low protein milk feeds with contents of which no higher than breast milk.

If in doubt as to pathogenesis mannitol 10-20% solution should be given 0.5-1 gm per kg body weight. Increase in urinary volume indicates continuing renal function and need for increased fluid intake.

If oliguria is marked and serious disease is suspected then 1-3 ml per kg per hour of 0.25% saline with 5% dextrose is started.

If fluid overload is present 5-10 mg per kg of frusemide repeated 4 hourly and intake output recorded. Nursing neonate nude on weighing platform may be useful.

Hyperkalemia (7 mmol per L) may be corrected by administering cation exchange resin in calcium phase rectally or orally in dose of 1 gm per kg. Hyperkalaemia may be temporarily lowered by administering insulin and glucose intravenous. If other indications are present best method for correcting hyperkalaemia is by peritoneal dialysis.

Severe acidosis (pH less than 7.1) requires intravenous sodium bi carbonate which will incidentally increase hypernatraemia but is corrected by peritoneal dialysis.

Plasma phosphate level rising above 9.3 mg per 100 ml is indication for peritoneal dialysis since plasma calcium level should be kept above 8 mg% to prevent convulsions.

If fits are noted 5-10 ml of 10% calcium gluconate solution intravenous will control this.

Coliform septicaemia should be treated by appropriate antibiotic after culture of blood and urine.

Bilateral renal agenesis is lethal but work in Finland on newborn make it seem possible that effective dialysis followed by renal transplant could be life saving.

ARF IN INFANTS AND CHILDREN

In older child recognition of oliguria is easier. Urinary volume may drop less than 300 ml per meter square per day. Signs of underlying disease such as proteinuria, casturia, haematuria or dysuria may be reported.

CAUSES

- Haemolytic uraemic syndrome.
- Cortical necrosis.
- Glomerulonephritis.
- Nephrotic syndrome.
- Renal venous thrombosis.
- Acute tubular necrosis.
- Postoperative.
- Urolithiasis.
- Hyperuricaemic nephropathy.
- Obstructive uropathy.
- Nephroblastoma.
- Systemic lupus erythematosus.
- Anaphylactoid nephritis.
- Goodpasteur syndrome.
- Rapidly progressive glomerulonephritis.

Commonest cause is dehydration.

MANAGEMENT

A. General Measures:

1. Good nursing care.
2. Careful mouth toilet.
3. Barrier nursing.
4. Accurate assessment of weight, blood pressure, fluid balance and electrolytes.
5. Blood urea nitrogen and acid base state.

Diet should control azotemia yet be able to prevent catabolism.

Treatment of primary cause be it hypertonic dehydration, shock, poisoning, acute nephritis or haemolytic uraemic syndrome is essential.

Early peritoneal dialysis is advised.

Bladder catheterisation should be avoided.

B. Definition of Cause:

Primary cause may be obvious or obscure and prerenal, renal or postrenal. If primary cause is recognised appropriate specific therapy is applied together

with suitably modified nonspecific treatment. If actual cause is obscure then broad division into prerenal, renal and postrenal ARF may be made and appropriate nonspecific symptomatic therapy started.

Apart from usual diagnostic tests for renal disease, diagnostic radiology and ultrasound have much to offer and may indicate renal presence, size, location and morphology. Infusion urography may identify poorly functioning kidney and abnormalities in generalized and local function such as cortical necrosis or renal venous thrombosis. Assessment of size and morphology of functioning or nonfunctioning kidney and of renal pelvis may be obtained by pulsed ultrasound which will also show subcapsular perirenal haematoma or external blood from ruptured kidney.

C. Treatment of Specific Problems:

1. Hypovolaemia and hypotension are treated by correction of shock by restoration of circulation and hydration.

Insufficient renal perfusion due to haemodyanamic factors, reduced plasma volume, electrolyte disturbance and dehydration is usually readily reversible.

10-20 ml per kg of warm plasma should be given rapidly. If plasma is not available then same volume of physiological saline. When urination ensues glucose saline may be given 6-10 ml per kg per hour (can be dictated by plasma sodium level). If adequate urination does not occur than parenchymal damage must be suspected and fluid intake reduced to insensible loss (30 ml per kg per day) plus fluid loss assessed in urine, vomit, stools and sweat (20 ml per kg per hour).

In protein losing states such as nephrotic syndrome and burns, scalds, intravenous infusion of plasma or albumin in dose of 10-20 ml per kg may prove life saving.

2. Infection:

ARF may have been precipitated by septicaemia, acute pyelonephritis or other acute infections. Blood and other appropriate cultures should be sent to laboratory and vigorous antimicrobial chemotherapy begun on best guess principle intravenous.

3. Systemic Hypertension:

Diastolic blood pressure more than 105 mm Hg requires immediate therapy with hydrallazine 0.15 mg per kg per dose intravenous and may be repeated. Alternatively diazoxide 3-5 mg per kg intravenous or methyldopa 5-10 mg per kg intravenous may be used.

4. Anaemia:

This may be marked in haemolytic uraemic syndrome or following hemorrhage. Anaemia may be masked by dehydration and hypovolaemia. Correction is with small amount of packed cell transfusion slowly.

5. Left Ventricular Failure:

Digoxin should be used cautiously when there is hyperkalaemia. Correction of water and sodium overload by peritoneal dialysis is preferable.

6. Convulsions:

These may be due to uraemia, hyperphosphataemic hypocalcimia, water intoxication, hypernatraemia, hyponatraemia and systemic hypertension. Intravenous midazolam should be given slowly till convulsions cease. This should be followed by intramuscular phenobarbitone to obtain continuous anticonvulsant control until cause is defined and dealt with appropriately.

7. Overhydration:

This occurs when fluid intake is continued in face of oliguria. It may be associated with electrolyte imbalance. Cessation of fluid intake, attempts to produce diuresis by frusemide may work but peritoneal dialysis is usually indicated. Hyponatraemia due to excessive sodium loss following treatment of obstructive ARF should be avoided.

8. Hypernatraemia:

Sodium overload may exist with hyperosmolar hypernatraemic dehydration with normal plasma and extracellular levels when concomitant sodium overload is present and even with hyponatraemia when gross overhydration exists.

Edema, ascites and increased heart rate and respiratory rate may be present along with pulmonary congestion. Peritoneal dialysis is required.

9. Hyperkalaemia and Hypokalaemia:

ECG shows peaked T waves, widened QRS segment, ST elevation and arrhythmias which can lead to cardiac arrest.

Treatment is with cation exchange resin in calcium phase, glucose and insulin infusion, calcium gluconate and peritoneal dialysis. Hyperkalaemia complicates various types of ARF including traumatic, burns, general anesthesia and use of aldosteron antagonists.

Hypokalaemia (serum potassium less than 3.5 mmol per L) may result from overtreatment with cation exchange resin or peritoneal dialysis with low potassium in dialysate. It may be corrected by peritoneal dialysis by adding potassium chloride 3.5 mmol per L to dialysate fluid prophylactically or 7 mmol per L therapeutically until plasma potassium level rises to 5 mmol per L.

10. Hypocalcimia:

This is secondary to hyperphosphataemia and may produce convulsions. Plasma phosphate level more than 9.3 mg per 100 ml or calcium level less than 8 mg per dl may be indication for peritoneal dialysis.

11. Metabolic Acidosis:

pH less than 7.1 calls for immediate intravenous dose of 5-20 mmol of 8.4% sodium bi-carbonate followed by peritoneal dialysis. Sodium bi-carbonate increases hypernatraemia.

12. Intravascular Coagulation:

Renal venous thrombosis and haemolytic uraemic syndrome are characterized by thrombocytopenia and increased FDP. Heparin is indicated in RVT.

In rapidly proliferating glomerulonephritis combination of anticoagulant with immunosuppresant has been tried (heparin with steroids).

Repeated plasmapheresis is of benefit in Goodpasteur syndrome.

13. Acute on Chronic ARF:

Child with anaemia and less than 3rd centile for height raises question of undetected CRF underlying acute incident. Large palpable cystic kidneys, renal rickets, bruising and pruritus may be found.

Precipitating factor may be infection or hypertension. Infection may be renal or extrarenal producing exaggerated metabolism and perhaps pyrexia.

Immediate appropriate antibiotic therapy is indicated with high fluid intake.

Other precipitating factors are reduced fluid intake (due to stomatitis) or increased fluid loss (due to enteral infection). These conditions must be treated.

14. Maintaining Nutrition:

Requirement is 400 kcal and 300 ml fluid per meter square body surface area per day to prevent protein and fat catabolism.

Use of central venous line permits 25% or 50% glucose to be delivered to blood. 2.4 kcal per ml of fluid low in sodium and protein is given between dialysis until diuretic phase.

15. Peritoneal Dialysis:

Indications:

1. Uncontrolled hyperkalaemia (serum potassium more than 7.1 mmol per L).
2. Convulsions.
3. Left ventricle failure.
4. Progressive uraemia.
5. Uncontrolled hypertension.
6. Water and sodium overload.
7. Uncontrolled hyponatraemia (serum sodium less than 125 mmol per L) or hypernatraemia (serum sodium more than 160 mmol per L).

Peritoneal dialysis should begin early and not regarded as desperate last in moribund water logged patient.

DEATH, TRANSPLANT OR LONG TERM DIALYSIS

When ARF is irreversible and lethal there are two choices:

1. Permitting dignified death.
2. Attempted survival.

When attempted survival is justifiable again there are two choices:

1. Renal transplantation.
2. Long term dialysis.

Parents of child with irreversible ARF are greatly influenced by attitudes of medical, nursing and auxiliary personnel.

Few parents wish their offspring to suffer physical and psychological trauma of long term dialysis including chronic invalidism, dwarfing, psychiatric upsets, social and economic disasters and likely family problems.

Transplant is justified for children of 5 years or more if medical, social and parental aspects are right.

Multifactorial ethical, clinical, financial, social and psychological pressures result in differing answers.

Most parents are willing to accept dignified death for loved child rather than continued but low quality existence for few extra months or year followed by death. ❑