Chapter 17

CHRONIC RENAL INSUFFICIENCY

Chronic renal failure is clinical state in which deteriorating renal function occurs due to progressive reduction in number of functioning nephrons. Consequently essential renal functions such as excretion and concentration of nitrogenous products is impaired causing uraemia. Sodium, ammonia, hydrogen excretion and production of erythropoietin may be disturbed and metabolism of vitamin D3 impaired. Total body magnesium rises and growth retardation occurs with renal osteodystrophy.

CRF has 3 stages:

Stage 1:

Renal function more than 20% of normal. (GFR more than 20 ml per minute per 1.73 meter square). Symptoms mainly polyuria or polydipsia with or without enuresis. May be reversible or capable of arrest. Treatment of hypertension, anaemia, urinary tract infection, obstructive uropathy, renal osteodystrophy and metabolic acidosis may or may not be required.

Stage 2:

Renal function 5-20% of normal. (GFR 5-20 ml per minute per 1.73 meter square). Anorexia, nausea, vomiting now complicate polydipsia and polyuria with uncompensated metabolic acidosis increasing and anaemia. Protein intake must now be restricted.

Stage 3:

Renal function less than 5% of normal. (GFR less than 5 ml per minute per 1.73 meter square). Profound anaemia, oliguria, edema, hyperkalaemia and overhydration may occur. Life ceases to be supportable without artificial relief of uraemia. Decision for dialysis and renal transplantation is required.

CAUSES OF CRF

1. Glomerular nephropathy.
2. Hereditary nephropathy.
3. Renal hypoplasia.

4. Urinary tract malformations.
5. Vascular nephropathy.

DIAGNOSIS

When CRF is end product of known progressive renal disease such as idiopathic nephrotic syndrome, chronic pyelonephritis, reflux nephropathy, renal hypoplasia, obstructive uropathy, nephronothisis or cystinosis diagnosis is simple. Some children present with symptoms such as polyuria, polydipsia and return of enuresis, profound unexplained anaemia, dwarfism, growth retardation with or without renal rickets (renal osteodystrophy). Vague symptoms such as anorexia, dyspepsia and delayed puberty are common.

Some cases are detected on routine urine screening at school or clinic.

Urine examination shows low specific gravity (osmolality) of fixed type (isosthenuria). Volume is increased. There may not be proteinuria and hyaline or granular casturia. Other abnormalities depend upon primary cause of illness and complications such as systemic hypertension.

Plasma biochemistry reveals nitrogen retention with high levels of blood urea (more than 100 mg%), creatinine (more than 2 mg per dl), metabolic acidosis and variety of electrolyte disturbances such as hyponatraemia (serum sodium less than 125 m eq/L) and hyperkalaemia (serum potassium more than 7 m eq/L).

Uraemia term is applied to ill patient with above derangements of function usually accompanied by muddy pallor, sunken eyes and wasting as death draws nearer. Rarely uraemic pericarditis may occur and terminally haemorrhagic tendency may require attention.

MANAGEMENT

During period of assessment and diagnosis and until death, dialysis or renal transplant supportive measures and symptomatic treatment are required.

A. Diet:

No dietary restriction in stage 1 unless systemic hypertension mandates low sodium intake (25-50 mmols or 575-1150 mg daily).

In stage 2 and 3 dietary restrictions are applied to reduce excessive nitrogen intake, ensure adequate energy, minerals, vitamin intake, maintain homeostasis and acid base balance.

1. Protein Intake:

This is determined by two factors: age of patient and GFR.

In stage 2 CRF intake for young infant would be 1.25-1.5 gm per kg per day and 10 year old child 0.75 gm per kg per day. This is intended to provide nitrogen essential for growth and anabolism and reduce gluconeogenesis. Part of daily

nitrogen intake may take part of essential amino acid mixtures. Sulphated amino acids are principal source of H ions, reduction in specific protein intake is helpful.

In stage 3 CRF, diet of essential amino acids only with energy provided by carbohydrates and fat may be used but may not be palatable to child. When child reaches this terminal stage humanity should be allowed to triumph over science and child's whims be indulged during his remaining days desirably spent at home.

2. Energy Intake:

Preferably more than 100 kcal per kg per day should be given. This is difficult on low protein diet. Glucose, sugar, jam, honey, bread should be used freely.

3. Water and Electrolytes:

So long as urinary volume is adequate intake is dictated by thirst of patient who indeed is drinking to live by maintaining high output of isothenic urine. When urine volume decreases (around GFR 10%) intake of water must be reduced to equal output plus 300-500 ml daily.

Normally sodium intake should be 1 mmol per kg per day but in hypertension or edema 0.5 mmol per kg per day is enough while sodium losers with interstitial disease require 5 mmol per kg per day. One must take account of sodium in medicaments such as sodium bi carbonate, intravenous infusions and sodium salts of penicillin.

Foods high in potassium should be eliminated from diet as GFR falls to 10 ml per minute per 1.73 square meter but below this specific daily intake must be maintained at about 1.5 m eq per kg per day.

B. Hypocalcaemia and Hyperphosphataemia:

Oral aluminum hydroxide 500 mg 6 hourly will reduce phosphate absorption.

Vitamin D 3 is used for renal rickets.

C. Renal Osteodystrophy:

Biochemically there is hyperphosphataemia, hypocalcimia and elevated serum alkaline phosphatase with increased level of serum parathyroid hormone.

Radiologically there are signs of secondary hyperparathyroidism with loss of lamina dura, bony erosions of metacarpals and generalized osteoporosis with or without radiological signs of rickets.

Initial treatment is with aludrox 500 mg daily which causes lowering of serum phosphorus with resultant elevation of serum calcium. Dietary calcium content should be 1 gm per day.

Vitamin D3 in dose of 400 iu daily is given orally.

Child may present with vague limb pains but more often with waddling gait due to associated myopathy of muscles of pelvic girdle.

D. Metabolic Acidosis:

This is most marked in predominantly medullary disease such as nephronopthisis.

Control of proteins especially sulphated amino acids is required. Sodium bi carbonate 0.5-2 mmol per kg per day orally in divided doses may be given. Risks are sodium overload and hypocalcimic tetany.

E. Gastrointestinal Symptoms:

Nausea and vomiting may be improved by administration of chlorpromazine 2-3 mg per kg per day. Intractable hiccough is similarly treated.

F. Dwarfism:

(Height less than 3rd centile)

Cause of growth retardation is multifactorial:

1. Poor nutrition.
2. Anaemia.
3. Metabolic acidosis.
4. Chronic sepsis.
5. Uraemia.
6. Renal osteodystrophy.

One etiological factor may be energy deficiency, therefore diet must provide more than 100 kcal per kg per day.

G. Blood Transfusion:

Packed cell transfusion raises PCV which may reduce renal plasma flow and may produce acute renal failure. This becomes emergency.

Erythropoietin has part to play in this condition since this is related to kidney function.

H. Hypertension:

Therapy consists of diuretics and hypotensive agents. Chlorthiazide and methyl dopa may be used.

I. General Supportive Measures:

Urinary tract infection is common. Intensive antimicrobial therapy is indicated.

In end stage CRF environment of child should be more pleasant.

J. Peritoneal Dialysis:

Soft canula can be left permanently in abdomen and continuous ambulatory peritoneal dialysis can be carried out in CRF.

It is of use in very young infant or preschool child. Or to tide patient over gap in available vessel access. Risk is peritonitis.

K. Haemodialysis:

Treatment is required for 12-18 hours per week at home or hospital. In addition to physical complications of poor growth and nutrition, delayed puberty and anaemia, psychosocial complications may arise in patient and parents. For every child so treated family's integrity is placed at risk and time for parental care of other siblings is lessened.

Child on regular dialysis therapy is supervised by team led by pediatric nephrologist but including surgeon, child psychiatrist, dietician, teacher, play therapist, social worker, technician, physiotherapist and pediatric nurse. Access to biochemical laboratory employing micromethods is also required.

Benefits of regular dialysis therapy are mixed with dangers of psychological problem, lack of growth and development, continual hospital admissions, pain, misery and inability to lead normal life or eat normal diet.

Diet during regular dialysis therapy should consist of:

1. Water 400-1500 per day.
2. Protein 1-2 gm per kg per day.
3. Sodium 25-150 mmol per day.
4. Calories 75-100 kcal per kg per day.

Patient must be weighed daily to monitor state of hydration as well as lean body mass.

Regular dialysis therapy is acceptable in young child only if subsequent renal transplant is possible, child mentally normal with stable home background and aged at least 5-10 years.

L. Renal Transplant:

Extraordinary success is obtained in transplanting healthy kidney from identical twin. Transplantation of healthy kidney from living or dead donor whether related or not is practical proposition provided tissue groups are compatible. Transplant holds out hope of living with normal diet and activity in child with terminal CRF.

Cadaveric donor is acceptable because requesting kidney from living parent amounts to moral blackmail since refusal is socially impossible.

Role of pediatrician is to assess whether or not child should be sent for regular dialysis therapy and transplant and to protect kidneys of parents and other potential donors from recklessness of overenthusiastic surgeons.

Average duration of transplant is 2-3 years.

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