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# Update Management of Renal Failure Patients

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Chronic renal failure is defined as a disease with pathological damage to the kidney and persistantly diminished renal function. Typical patient with progressive chronic renal failure may be considered to pass through four stages.

**I. Mild Renal Insufficiency or Diminished Renal Reserve.**

A. 50% of nephron destroyed.
B. BUN rises but remains within normal limits.
C. Excetory and regulatory functions of the kidney are well preserved.

**II. Moderate Renal Insuficiency.**

A. Clearance decreased below 25cc/minutes, BUN 30-30 mg/dl., Creatinine 3.5 – 4.5 mg/dl.
B. Impaired concentrating ability with concomitant nocturia.
C. Mild anemia with hemoglobin approximately 10 gm% and hematocrit 30%.
D. Hyperphosphatemia – mild hypocalcemia.
E. Mild hypermagnesemia.
F. Functional reserve is decreased markedly. Even slight further insult to kidney like catabolic illness, volume depletion, congesetive heart failure may lead to severe azotemia.

**II. Severe Renal Insutficiency.**

A. Clearance decreased to 10 cc/Min, BUN 90-100 mg%, Creatinine 10 mg%.
B. Total loss of concentrating ability and development of inability to dilute the urine.
C. Severe anemia with hemoglobin 7.5 – 8.5 gms% Hematocrit 22-26 %.
D. Marked hyperphospatemia, More than 6.0 mgs% with hypocalcemia.
E. Metabolic acidosis with mild hyperkalemia.
F. Precarious balance between volume overload and hypertension vs volume depletion and worsening azotemia.

**IV. Uremic Syndrome or End Stage Renal Disease.**

A. Creatinine clearance 5cc/minute or less.
B. Deranged electrolytes with severe metabolic acidosis.
C. Hyperkalemia ensues with acidosis as urinary volume decreases.
D. Sequence of metallic taste, anorexia, weight loss, nausea and vomiting, pericarditis, peripheral neuropathy, various neuro-muscular symptoms, inability to concentrate, lethargy and coma.
E. Renal function is so little that without more aggressive therapy like dialysis or renal transplant patient would not live for more than few months.

Although variety of conditions can lead to chronic renal failure but the biochemical, endocrine and metabolic abnormalities occuring in renal failure are similar and these are due to accumulation of waste products which are normaly excreted by the kidney. Conservative management is directed towards decreasing consequences of accumulated waste products and possibly prevent or delay further loss of renal function. Certain goals for conservative therapy of renal failure are given in table I.

**TABLE – 1**

**GOALS FOR CONSERVATIVE THERAPY**

1. To establish the diagnosis
2. To correct reversible factors.
3. To correct mineral electrolyte and acid base abnormalities.
4. To maintain adquate protein and caloric balance.
5. To prevent further loss of renal function.

**Diagnosis:**

First step in the evaluation and management is to establish the diagnosis and to find type of pathological insult which has occured. It is also important to find whether the disease process is acute or chronic, how rapidly damage has occured or is occuring. Attempts should be made to find any reversible cause of renal failure. These cureable and treatable causes must be excluded early. These usually are disorders causing inadequate renal per-

fusion, like dehydration, congestive heart failure, obstruction to urine flow, accelerated hypertension, urinary tract infections and use of nephrotoxins. In addition there are diseases in which progressive loss of renal function can be prevented by appropriate therapy, like hypertensive nephrophathy, diabetic nephropathy, SLE and certain forms of arteritis. If treatable disease is suspected, diagnostic evaluation may include kidney biopsy. At the initial study patient's renal function is quantitated. Blood urea, creatinine, creatinine clearance, nerve conduction studies and bone survery is done. With the knowledge about the natural history of different renal diseases and by plotting reciprocal or logarithm of serial serum creatinine values against time, one can project the rate of progression of disease in a particular patient and this is very important for formulation of life plan for the patient.(1)

The rate of change of reciproval of serum creatinine is an estimate of the rate of loss of renal function. The reciprocal of serum creatinine declines linearly with the time. Besides this it has been found that logarithm of serum creatinine increases linearly with time in patients with progressive renal failure. One of these methods can be used to follow the effect of therapy and to estimate the prognosis.

## CORRECTION OF MINERAL ELECTROLYTE & ACID BASE BALANCE

### Calcium Phosphorus Metabolism

As the kidney funciton decreases regardless of the etiology, abnormality of calcium phosphorus and of hormones which regulate the concentration of these minerals in blood, begin to appear. As the GFR declines phosporus is retained in the body. Serum phosphorus level increases. With rise in serum phosphorus level serum calcium level decreases. This stimulates the paratharmone secretion. Increased level of PTH causes mobalization of calcium from bone resulting in renal osteodystrophy which is almost a universal feature of CRF. There are other factors also responsible for this but secondary hyperparathyroidisim is one of the major factors involved. Many other disabling symptums of CRF have been atributed to increased PTH. In fact PTH is considered as a uremic toxin Soft tissue calcification in muscles, arteries and vital organs like heart, lungs, & brain can occur. For these reasons control of serum phosphorus level is very important to prevent secondary hyperpasthyroidism.

Phosphate binders in the form of aluminium hydroxide and other aluminium compounds are given to keep phosphate in the range of 3.5 to 4 mg/dl. Antacids containing

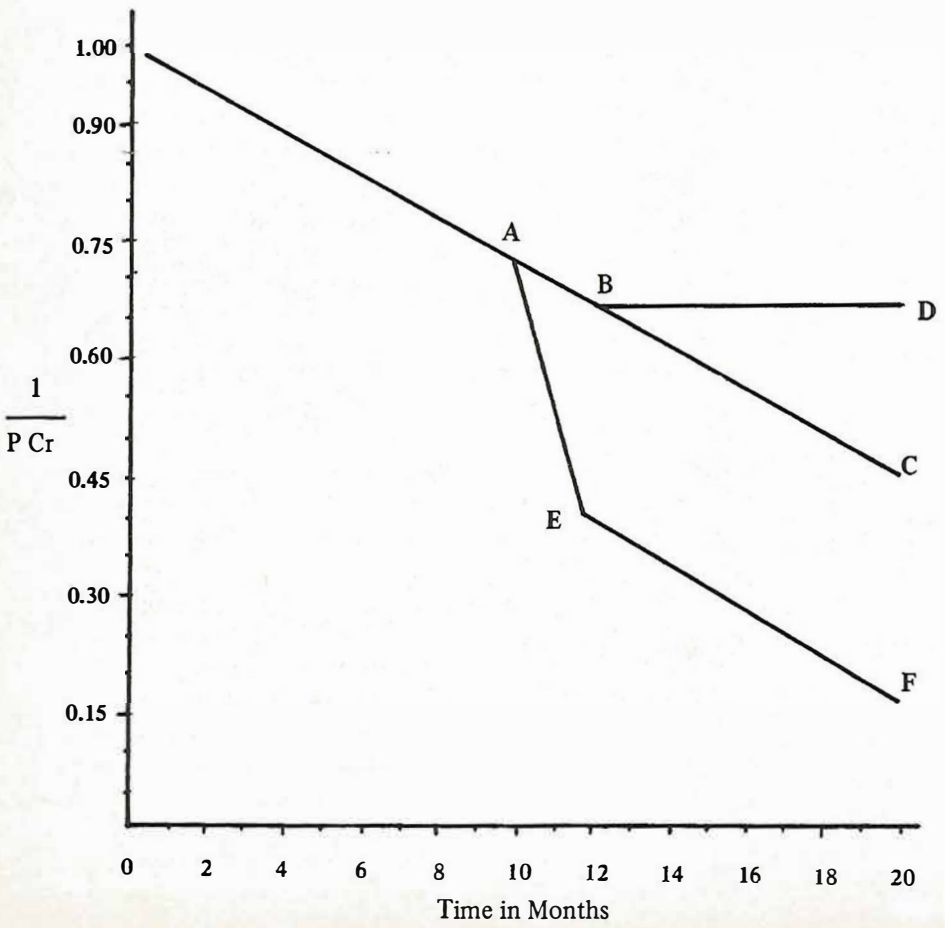


Fig. 1.
Rate of loss of renal function as measured by plotting reciprocal of serum creatinine against time.
ABC: Shows slowly progressive renal disease.

ABD: Improvement in renal function with therapy.

AE: Acute deterioration in renal function.

EF: stabalization of renal function again with treatment.

magnesium compounds should not be used as these patients already have tendency to retain magnesium and antacids with magnesium will aggravate hypermagnesemia.

Oral calcium carbonate at doses of 5-20 grams per day can effectively correct hypercalcemia and hyperphosphatemia of most patients without the risks of hyperaluminemia that is induced by Aluminum containing phosphate binders. Clinical side effects are gastrointestinal intolerance and soft tissue calcification (4).

As the renal function declines the patient would need calcium suppliment and also active from of vitamin D. 1-25 dihydoxy vitamin $D_3$. In these patients diseased kidney can not activate 25 OH.$D_3$ to 1.25 $(OH)_2$ $D_3$. These compounds should be used carefully. If both serum phosphorus and serum calcium are elevated metastatic calcification can occur specially if calcium phosphorus product in the serum is above 60 (5,6,8)

Calcium suppliment or vitamin D should not be given if serum phosphorus level is high. This would aggravate soft tissue deposition of calcium & phosphorus. Serum phosphorus level should be brought down first. Hypercalcemic can lead to decrease in GFR serum calcium should be monitored during calcium and or vitamin D therapy.

**Regulation of Potassium**

In renal failure ability of kidney to excrete potassium may be deceased. These patients are acidotic. These factors could lead to hyperkalemia in CRF, which would require treatment. If hypekalemia is associated with cardiovascular abnormalities then acute therapy would include I.V. calcium gluconate or calcium chloride I.V. sodium bicarbonate, I.V. insulin and glucose. Each of these methods would not remove the potassium from the body but would reverse the effects of hyperkalemia on the heart temporarily. Further chromic therapy would be needed.

Dietary potassium restriction, typically 2 grm a/day is indicated if hyperkalemia appears or clinical settings suggest risk of potassium stress (diabetic nephropathy). Cation exchange resins as sodium polystyrene (Kayexelate) are useful adjuncts for mild hyperkalemia due to recurrent dietary indiscretion or intercurrent illness. Oral dose of 15-20 grams one to four times daily are given with 20 cc 70% sorbitol or another laxative to counteract constipation and potential fecal impaction. Kayexelate can also be given by retention enema.

Potassium exeretion is increased from the gastrointestinal tract as the kidney function decreases. Thus constipation could aggravate hyperkalemia and it should be avoided in patients with CRF. In treating the constipation magnesium salts should not be used. Magnesium exeretion is already reduced in these patients. Administration of magnesium salts could lead to magnesium intoxication.

**Metabolic acidosis**

As progressively fewer nephrons are available in CRF to excrete the normal daily acid load, patient usually develops metablic acidosis. Alkalinizing agents are generally indicated in uremic acidosis when serum bicarbonate level approaches or falls below 15 mEq/1. Sodium bicarbonate level should be maintained between 15-18 mEq/L. Gastrointestinal intolerance, sodium, volume overload are frequently dose limiting factors. Proportional reduction of dietrary sodium should be done in such cases.

**Sodium & Water**

During progressive renal failure, kidney adapts itself to exorete more sodium per functioning nephron. Sodium balance may be maintained in some patients. However the damaged kidney will not be able to change the sodium excretion repidly in response to changes in sodium intake. If sodium intake is restructed suddenly, the patient will continue to loose sodium for quite sometime before the kidney adapts to new salt intake. During this period patient could become salt and volume depleted leading to decreased renal perfusion and decrease in renal function.

Salt restriction is indicated in patients who have generalized edema or hypertension. Water intake should be restricted also in these patients who have edema with or without hypertension. If salt and fluid restriction do not reduce the edema then diuretics should be given.

## TREATMENT OF HYPERTENSION

Most of the patients with CRF develop hypertension. Different factors responsible for that are shown in Table-1

TABLE – 1

**FACTORS RESPONSIBLE FOR HYPERTENSION IN RENAL FAILURE**

1. Increased extracellular volume
2. Incsreased renin-angiotensin
3. Unknown
   - (a) Hormones
     - (i) Decreased renomedullary prostaglandins.
     - (ii) Increased aldosterone
     - (iii) Increased 18 hydroxy corticosterone and or/others.
   - (b) Vascular fixed structural changes
   - (c) Increased salt and water content in the vessal wall.
   - (d) Reset baroreceptors.

Sodium restriction in the diet will be needed. In some patients with salt loosing nephropathies salt restriction could lead to worsening of azotemia. Thiazide diuretics are ineffective when the G.F.R. is less than 30 cc per minute. Spironolactone and triamterene should not be used because of hazards of hyperkalemia. Antihypertensive drugs alpha methyl dopa, clonidine, prazocin, beta-blockers and vasodiators can be used. In severe hypertension minoxidil can be used. If the patient does not respond to these then bilateral nephrectomy may be needed (3). After bilateral nephrectomy patient could be maintained on dialysis or with renal transplant. With the availability of minoxidit, a very potent vasodilator, very few nephrectomies have been done to control the hypertension.

Captopril should be used with caution. It could lead to hyperkalemia and also by inhibition angiotensin II formation, in some patients, it may reduce the GFR.

## DIETARY THERAPY

**Protein requirement**

The patient with renal failure have tendency towards catabolism. These patients must get 0.3 to 0.5 grams proteins per kg/day plus one grm for every gram of urinary proteins. . The dietary proteins should be high quality containing high proportion of essential amino-acids. Institution of protein restirction in CRF is delayed until uremic symptoms become manifest. In practice total protein restriction to less than 40 grams high biological value protein per day is associated with negative nitrogen balance and can no loger be considered reasonable.

**Aminoacid suppliment and aminoacid analogs**

Role fo essential aminoacids and their ketoacid analogues in renal failure is currently being evaluated. These compounds are supposed to provide nutritional needs with lower nitrogen intake and thus lower urea production rates.

**Calories**

Adequate caloric intake is also very imprtant and this could be provided through carbohydrates and polyunsaturated fats. By giving adequate calories and high quality proteins catabolism would be reduced. Patients whould stay in positive nitrogen balance have less anemia, more immunoglobulins, less hyperkalemia and less acidosis.

## TREATMENT OF ANEMIA

Anemia is commonly present in patients with CRF several factors are responsible :—

i. Poor nutrition due to averexia vomiting
ii. Vitamin difficiencies
iii. Altered iron metabolism
iv. Increased blood losses due to bleeding
v. Decreased red cell production due to decreased erthropoiten production by damaged kidney.

Treatment would include adequate protein of high quality, vitamin suppliment and iron preparations. Absorption of medical ferrous salts is impaired by antacids and food. These iron suppliments should be taken between meals and temporally separate from antacids. Histidine supplimentation has been shown to have beneficial effect on anemia in certain studies. Androgens are also used. Androgens are supposed to increase the erthroid cell mass and stimulate erythropoiten production. To date there has been no adequate treatment for anemia of E.S.R.D. except for some impact of androgens. Repeated blood transfusions have been only way. Exposure to hepatitis or other infectious agents, transplant sensitization and volume overload are the major hazards. Fortunately biologically active erythropoiten has been isolated and should be available for therapy trials. This would be a major advance and particualr importance to patients with severe anemia and ESRD.(7)

## TREATMENT OF PRURITUS

The patient with CRF usually gets pruritus and for that antihistaminic drugs can be given. In certain cases activated charcoal in capsules taken orally has been shown effective. For intractable itching parathynoidectomy may be required.

## TREATMENT FOR NAUSEA AND VOMITING

Patient will have nausea of vomiting for whch antiemetics are given.

## TREATMENT FOR END STAGE RENAL DISEASE

In the End Stage of Renal failure the kidney function is so low that without more aggressive therapy, like dialysis or transplantation, patient would not survive for more than few months.

## INDICATIONS FOR DIALYSIS AND TRANSPLANTATION

Following are the indications for dialysis or transplantation.(2)

1. Creatinine clearance less than 5 ML/M., unless well compensated, higher in systemic disease like diabetes mellitus.
2. Clinical motor neuropathy.

3. Pericarditis.
4. Bleeding diathesis.
5. Convulsive seizures.
6. Repeated pathological fractures.
7. Intractable vomiting, continuous nausea.
8. Inability to perform usual work, worsening intractable pruritis.

Prior to 1960, End Stage Renal failure was a uniformly fatal disease, but now with three very different effective therapies, renal transplantation, peritoneal dialysis and haemodialysis, a uremic patient can be kept alive for years These therapies are far from being perfect. There are alternative approaches to the uremic patients now in trial and they may improve substantially the future treatment of End Stage Disease. These are hemofiltration, a modification of standard haemodialysis, with advantages of effeciently removing more solutes in the middle molecular range. Similarly hemoperfusion, alone or in combination with haemofiltration is another approach under trial.

In the end, this point is to be stressed that in the management of renal failure every effort should be made to detect the disease early and to prevent or at least slow down the progression of renal damage so that the patient does not reach the stage where he would need dialysis and transplant. Both these modes of treatment are very expensive and facilities for these are limited in the country. Early detection of the disease is possible through periodic complete examination of first void morning urine specimen.

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