RENAL REPLACEMENT TECHNIQUES IN RENAL FAILURE

Thesis

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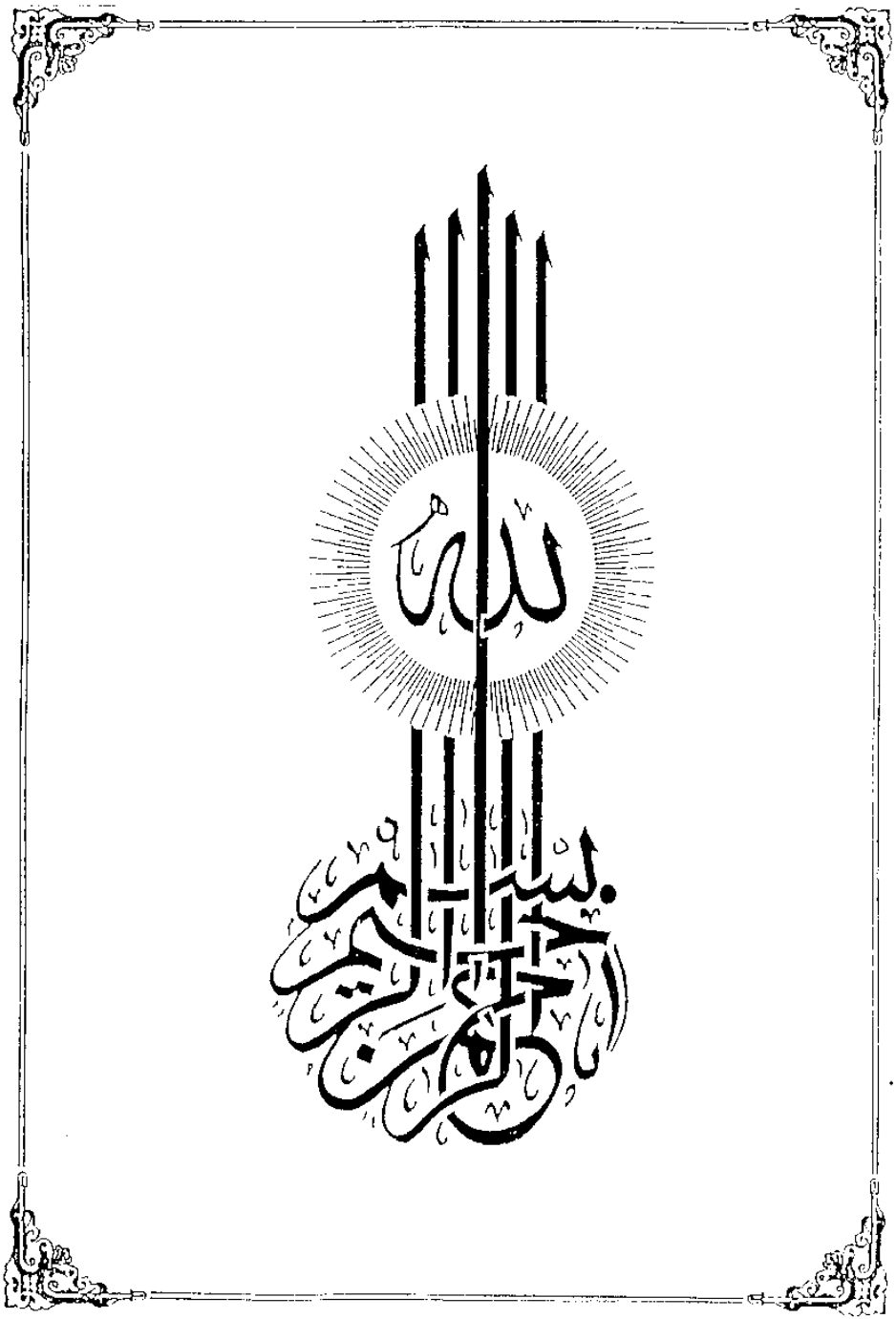

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INTRODUCTION

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Patients with end-stage renal failure are encountered commonly. In Egypt, the number of new patients accepted onto renal replacement therapy is 7.4 patients per million population each year . These patients have many unique problems and needs. To deal with these patients we must understand the pathophysiology of uraemia, the principles and mechanics of dialysis, the adjustments of medications required in dialysis patients, the placement and care of vascular access and peritoneal access and the selection and preparation of dialysis patients for kidney transplantation .

There are many methods for providing dialysis therapy. Extracorporeal haemodialysis can be offered in many forms with many different types of equipment . Dialysis therapy may utilize the capillaries of the peritoneum via intracorporeal peritoneal dialysis.

The alternative therapy for chronic renal failure patients are renal transplantation . During the past 15 years , much understanding has been gained of graft immunology, kidney preservation , the technical and logistical problems associated with renal transplantation , and general pre-operative selection and postoperative care of the transplant patient. Thus , it is the role of the physician to determine when renal replacement therapy is needed and whether maintenance haemodialysis, peritoneal dialysis, or renal transplantation would be the best approach. The relative merits and shortcomings of each type of treatment must be weighed against the medical and psychosocial condition of the patient.

RENAL FAILURE

RENAL FAILURE

- Causes
- Stages
- Clinical picture
- Pathology
- Pathogenesis
- Pathophysiology
- Diagnostic aids in renal failure

RENAL FAILURE

Renal failure is a generalized functional impairment in which the kidneys are unable to maintain the volume or composition of body fluids under conditions of normal dietary intake. Renal failure always implies bilateral disease, as one normal or nearly normal kidney has sufficient function to maintain homeostasis with normal metabolic load from exogenous intake and endogenous catabolism(Golden et al., 1977)[33].

Causes

I. Local causes :

Diseases in which the kidneys are predominantly involved and in which the presenting features are usually those of renal disease :-

1. Proliferative glomerulonephritis .
2. Membranous glomerulonephropathy .
3. Chronic pyelonephritis .
4. Renal calculi .
5. Polycystic disease .
6. Medullary cystic disease .
7. Renal hypoplasia .
8. Upper urinary tract obstruction.
   - Hydronephrosis .
   - Retroperitoneal fibrosis .
   - Neoplasm .

II. Lower urinary tract obstruction :

Presenting features often those of bladder dysfunction, but may present in renal failure:-

1. Prostatic enlargement .
2. Urethral stricture .
3. Urethral valves .
4. Bladder neck obstruction .
5. Neurogenic bladder .

III. Systemic diseases and intoxications :

a. In which renal failure is not infrequently a presenting feature : -

1. Malignant essential hypertension .
2. Polyarteritis nodosa .
3. Disseminated lupus erythematosus .
4. Primary and secondary amyloidosis .
5. Cystinosis and oxcalosis .
6. Consumption coagulopathies :-
   - Haemolytic-uraemic syndrome .
   - Thrombocytopenic purpura .
   - Postpartum renal failure .
7. Lead poisoning .

b. In which renal failure is usually a late feature or overshadowed by other manifestations :-

1. Benign essential hypertension .
2. Systemic emboli .
3. Gout .

4. Diabetes .

5. Heart failure .

6. Cirrhosis (Kerr , 1975)[40] .

Stages of renal failure

1. Early on , renal failure simply reflects decreased renal reserve. There may be no symptoms or prominent biochemical disturbances, hyperuricaemia, proteinuria, and hypertension may or may not be present. Diminished creatinine clearance may be the only observable change.
2. As illness progress and creatinine clearance falls below 50 ml./min., one can speak of second stage , renal insufficiency. There may be mild azotaemia , impaired ability to concentrate urine, nocturia, and mild anaemia .
3. The third stage is frank renal failure. This usually does not occur until creatinine clearance has fallen to 10 to 15 ml./min. or less. At this stage anaemia may become severe. Hypocalcaemia and hyperphosphataemia occur, polyuria, hypochloraemia, and hyponatraemia are common. Hyperkalaemia may also occur, although it is not as common as might be expected .
4. The fourth and final stage is uraemia. The patient has symptoms referable to major organ systems such as nausea vomiting, and diarrhea . Frank pericarditis, neuropathy , and metabolic encephalopathy may be evident. Oliguria, bone disease, and bleeding disturbances may coexist. Laboratory examination

shows pronounced azotaemia and life threatning hyperkalaemia (Knochel ,1978)[43].

Clinical picture

The patient of chronic renal failure noticed for some time that he passes large amounts of urine, that his urine is exceptionally pale and that he is obliged to get up several times during the night to micturate , he starts to feel weak and tired, loses appetite, suffers from nausea and sometimes vomits. Centrally determined hiccups can develop.

Symptoms and signs :

1. Dyspnea:

   The patient begins to suffer from dyspnea, often after the slightest exertion. This is caused by anaemia and acidosis. In this situation , resting respiration is also generally deeper than usual, but typical Kaussmaul's breathing is found only with severe acidosis .

2. Pruritis .
3. Vomiting .
4. Muscular twitching .
5. Consciousness :

   Step by step, consciousness begins to be obscured . He gradually loses contact with outside world, falls into a state of semiconsciousness and finally, after weeks of suffering,dies in coma.

On examination :

1. On physical examination the attention is immediately drawn to the patient's appearance and to the curious odour of his breath , foetor azotaemicus . The mucous membranes and epidermis are strikingly pale, owing to anaemia, and the skin is dirty yellow in colour . The skin is abnormally dry , marked with abrasions, petechiae and large extravasations of blood can be found , particularly in places exposed to pressure and around the sites of injections. The tongue, especially in the later phase, when Kaussmaul's breathing is present, is dry with a thick, white or brownish coating .

2. Lungs :

Signs of pulmonary oedema may sometimes appear and may be due to left ventricular failure or of uraemic origin. The patient's diminished resistance can naturally also lead to terminal bronchopneumonia .

3. Circulatory system :

Aside the consequences of arterial hypertension , abnormalities may be found in the pulse . In very severe anaemia, the pulse can be noticeably heaving and jerky ; other signs of hyperkinetic circulation (quinke's pulse, warm pulsating finger tips) can be present. A pericordial friction rub denoting uraemic pericarditis may be detected . Clinically significant exudate, with signs of cardiac tamponade, is a rare development .

4. Abdomen :

In the presence of potassium deficiency the abdomen can be distended and signs of paralytic ilieus can also appear .

5. Neurological findings :

The neurological findings include lively tendon reflexes. In patients whose life is prolonged by the chronic use of the dialytic therapy , a polyneuritis may develop which can proceed from the initial tingling and paraesthesia in the feet to pain , wasting and paresis which eventually may affect all the extremities and even be followed by death (Brod ,1973)[12] .

Pathology

The principal kidney diseases that lead to chronic renal failure produce kidneys that have many morphologic features in common. In fact, their similarities are often more stiking than their differences, and it is frequently impossible to determine morphologically the etiology of chronic renal disease .

Gross picture :

Most frequently , the kidney is small in size and appears pale. It has a granular cortical surface with small pitted scars and elevated grayish nodules.
On cut section , the cortex is thin and frequently poorly demarcated from the medulla.