Role of Homoeopathy in Chronic Renal Failure with miasmatic concepts

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Introduction-

Chronic renal failure is rapidly growing hazardous disorder of present era. The patient is habitually unaware of it and the diagnosis is frequently accidental. Often, the diagnosis is too late and the condition becomes irreversible or incurable, termed as ESRD (end stage renal disease). The other therapeutic regimens use only to clear up the toxins circulating with the blood stream by means of dialysis and supplementing the deficient ingredients along with symptomatic treatment if available. In contrary, Homoeopathic treatment, as usual, treats the individual as a whole and therefore has great percentage of cure. To achieve this high class goal, one must be acquainted with the knowledge of renal failure and homoeopathic science.

Review of literature-

Historical review-

The hydrological cycle regarding the earth as described by Aristotle was transferred to the human's body in order to explain human physiology. With food the human received the nutritional substances necessary for life. These substances were digested and classified into the useful ones that remained in the body and the useless ones that were eliminated. The final carrier of the food, the undigested elements of food as well as the remnants of digestion, was the blood. The blood should ultimately undergo catharsis. A healthy body realized this catharsis through the intestinal tube (that is, in the form of feces), through the lungs (a reference to a statement attributed to Aristogenes was given by Aristotle), through the kidneys by urine production, and through the skin by perspiration.

Hippocrates, the father of medicine, in his book, On Sufferings, referred to the cause of edemas and described them with exceptional detail: "An edema is mostly caused when catharsis does not occur, as in the case of a long-standing disease" ... "When an edema is attributed to the absence of catharsis, then the abdomen is filled with water and the legs up to the shins are swollen while the shoulders, the chest and the thighs languish".

According to Hippocrates, the humors and the fleshes were interchangeable both in health and in disease. The flesh could melt and become water and fill up the body's cavities. Hippocrates identified four forms of renal diseases. In his work On the Inner Sufferings, he described them as follows, "Renal diseases are caused when the kidneys, having received the phlegm or choler or pus that is to be excreted, cannot eliminate them, resulting in its accumulation inside the kidneys and thus the appearance of the disease occurs." To put it another way, this mechanism, which was actually suggested by Hippocrates, was identified with the reduction of the cathartic ability of the kidneys.

In his book On Inner Sufferings, Hippocrates referred to the treatment of all four categories of renal diseases. For all of them, apart from prescribing diuretics and cathartic drugs, the treatment included hot compresses, thermal baths, and steam baths.

Within this period, Ruphus from Efessus, whom the Byzantine doctor Oribasius called "a Great physician," appeared and prospered. Ruphus made an important reference in the section "on the sclerosis of kidneys" where he seemed to give a description of chronic renal failure:

"Whenever scleroses develop in the kidneys they are painless and, as someone would expect, the loins are hanging and the hips are restricted in their movements and the legs are weak; they discharge a small quantity of urine resembling greatly the conditions affecting patients with edemas. And these patients of course, in the course of time, are filled up with water as the other viscera become sclerosed, too". He added an interesting method for provoking perspiration in his work, On the Renal and Cystic Diseases, and in the paragraph on polyuria (urine diarrhea): "... because it is good for them to be able to perspire if diuresis stops. The best of all is a steam bath in a small vat with the head coming out from the top, so that, while the rest of the body is being heated, one can breathe cool air."

Medical review-

Introduction

The cells are surrounded by a watery environment that is probably similar in composition to the primordial sea in which life originated. The constancy of this 'internal environment' of extracellular fluid is a requirement of life, and the process of maintaining this constancy is called homeostasis. The kidneys, together with the lungs, are the most important organs ensuring a constant chemical composition of our extracellular fluid. The kidneys' importance can be gauged from the fact that they receive one-fifth of the cardiac output of blood, i.e. 1 litre per minute. The major role of the kidneys is to 'purify' blood by extracting waste products of metabolism; they must also help to control the osmolality, volume, acid–base status and ionic composition of the extracellular environment by modifying the composition of that part of the extracellular fluid (the blood plasma) that passes through them. The waste products extracted by the kidneys must be ejected from the body and, of course, this is done in the urine, a watery solution. The kidneys play important roles in controlling the production of red blood corpuscles and regulating blood-pressure.

The main function of the kidneys is to regulate the volume and composition of the extracellular fluid. This they do by filtering large volumes of plasma, retaining only plasma proteins, and then selectively reabsorbing from or secreting into the filtrate. The urine therefore contains 'unwanted' solutes in water. The processes of filtration, absorption and secretion are regulated homeostatically so as to minimize changes in extracellular fluid composition; in achieving this, urine of appropriate volume and composition is produced.

The kidneys also-

• excrete metabolic waste products including creatinine, urea, uric acid and some end products of haemoglobin breakdown

• excrete foreign substances and their derivatives, including drugs, and food additives – such substances are therefore excreted less efficiently when kidney function is impaired

• synthesize prostaglandins and kinins that act within the kidney

• function as endocrine organs, producing the hormones renin, erythropoietin and calcitriol, the active form of vitamin D.

Structure of the kidneys

The kidneys are paired, bean-shaped organs that lie behind the peritoneal lining of the abdominal cavity. Each kidney is surrounded by a thin capsule, which is usually removed when the kidney is used for culinary purposes. The capsule resists stretch and limits swelling. This has important consequences for the renal circulation. The renal artery and the renal vein, renal lymphatics and ureter enter and leave the kidney through its concave surface, at the **hilum**. When the kidney is cut in half longitudinally, an outer layer, the **cortex**, can be seen surrounding the **medulla**, which is made up of a series of conically shaped **pyramids**. The apical end of each pyramid, the **papilla**, opens into a space, the renal **pelvis**, which is continuous with the **ureter**. The ureter drains into the bladder.

Structure of the nephron

The basic unit of the kidney is the nephron, which is a blind-ended tubule running from **Bowman's capsule** into the ureter at the renal pelvis. There are about one million of them in each human kidney. Each nephron begins at the **glomerulus**, which comprises a tuft of glomerular capillaries contained within Bowman's capsule, which is the blind end of the nephron. The capillaries are derived from an afferent arteriole and drain into an efferent arteriole. The many branches of the capillaries form a cluster that invaginates into Bowman's capsule, like a fist pushed into a partially inflated balloon. All glomeruli are found in the cortex. The glomerulus produces a more or less protein-free filtrate of plasma. Fluid from Bowman's capsule flows into a coiled segment, the **proximal convoluted tubule**, and then into the **loop of Henle**, which courses down into the medulla forming a hairpin shape.

Two different populations of nephrons exist:

• **cortical nephrons** that have glomeruli in the outer two-thirds of the cortex and short loops of Henle that just dip into the outer medulla

• **juxtamedullary nephrons** that have glomeruli in the inner cortex and long loops of Henle that plunge deep into the medulla, as far as the tips of the papillae. The terms descending and ascending are used to describe the two limbs of the loop of Henle. The nephron first descends into the medulla and then ascends back into the cortex. The ascending limb of the loop of Henle leads into a second coiled section, the **distal convoluted tubule**. The distal convoluted tubule begins at a specialized structure known as the juxtaglomerular apparatus. Here the tubule passes between the afferent and efferent arterioles that supply the tubule's own glomerulus. This short section of tubule is known as the **macula densa** and senses the flow and composition of tubular fluid. It abuts onto a specialized region of the afferent arteriole whose granular cells secrete renin. The distal tubules of several different nephrons join to form a **collecting duct** that passes through the medulla to the papilla. Throughout its length, the nephron is composed of a single layer of epithelial cells resting on a basement membrane.

There are characteristic differences in the structure of the cells along the length, which reflect their different functions. The cells form a selectively permeable barrier to diffusion into or out of the tubule; they are joined together to form the barrier by specialized tight junctions that limit diffusion between the cells.

Structure of the glomerulus

In the glomerulus, the filtrate of plasma has to pass through three layers:

• The fenestrated (perforated; from the Latin *fenestra* – a window) endothelium of the capillary which is the filtering membrane.

• The basement membrane of the Bowman's capsule which is mainly composed of connective tissue, but also contains mesangial cells those are both phagocytic and contractile. By contracting they are thought to be able to actively reduce glomerular filtration by reducing the area available for filtration.
• The epithelial cells of the capsule. These are known as podocytes because they have numerous foot-like projections (pedicels) that clasp the tubes of capillary endothelium. Substances that pass through the filtration slits (or pores) between the pedicels therefore pass close to the cell surface of the podocytes.

Structure of the tubule

The epithelial cells of the proximal tubules contain many mitochondria and have many microvilli at their luminal surface, called a **brush border**, which increase the surface area. Adjacent cells are joined together at their luminal (apical) ends by tight junctions. At their basal ends, there are gaps between them, known as lateral intercellular spaces. The descending limb of the loop of Henle and the first part of the ascending limb are thin walled: the epithelial cells contain relatively 720 few mitochondria and are flattened with few microvilli. The ascending limb becomes thick walled as it enters the cortex; here are many mitochondria and microvilli, but fewer than in the proximal tubule. Along the length of the distal tubule and collecting ducts, the numbers of mitochondria and microvilli decrease. In the late part of the distal tubule and collecting duct there are two specialized types of cells (**principal** and **intercalated**) that are involved in Na+–K+ balance and H+ balance.

Renal blood supply

As it enters the kidney, at its hilum, the renal artery branches to form interlobar arteries which radiate out towards the cortex.

At the boundary between the cortex and medulla, arcuate arteries branch off at right angles and from these arise the interlobular and afferent arterioles that supply the glomeruli. The efferent arterioles that drain the glomeruli branch to form a secondary capillary, or a portal system. Those from the cortical glomeruli give rise to a peritubular capillary network that supplies the renal tubules. Those from the juxtamedullary glomeruli give rise either to similar peritubular capillaries, or to capillaries which plunge deep into the medulla and form hairpin loops parallel with the loops of Henle. These vascular loops are called the **vasa recta**.

All the capillaries drain into a cortical venous system and then into the renal vein. The kidney is richly innervated. Postganglionic **sympathetic** noradrenergic nerve fibres supply the renal artery and its branches. The afferent and efferent arterioles of the glomeruli and the juxtaglomerular renin-secreting cells are particularly densely innervated. Sympathetic noradrenergic fibres also supply the proximal tubules, the thick ascending limb of the loop of Henle and the distal tubule.

Functions of the kidney

• The function of the kidneys is to regulate volume and composition of the extracellular fluid. This they do by the processes of filtration, reabsorption and secretion.
• The gross structure of the kidney is a cortex surrounding a medulla and an innermost cavity, the pelvis.
• The functional unit of the kidney is the microscopic nephron (1 million in each kidney).
• Fluid filters into the nephrons at a rate of about 180 litres/day; the vast majority is reabsorbed.

• Filtration is influenced by renal blood flow, which is subject to a high degree of autoregulation, and to control by enal nerves and the renin–angiotensin system.
• There is active reabsorption of substances from the nephron while water flows passively.
• Regulation of absorption is by endocrine factors including prostaglandins, the renin–angiotensin–aldosterone system, atrial natriuretic peptide and the antidiuretic hormone.
• The shape of the loop of Henle enables a process called countercurrent multiplication to produce a hyperosmotic extracellular fluid in the medulla. This is reinforced by movement of urea.
• The kidneys excrete the fixed acids formed and absorbed by the body.
• They control the acid–base balance of the body by reabsorbing bicarbonate, secreting hydrogen ions and forming ammonia at variable rates.
• In disturbances of acid–base balance, the kidneys and lungs act together to restore normality.

Renal Failure

Definition-

Renal Failure (CRF) is slow, insidious, and almost irreversible impairment of renal excretory and regulatory function. Renal failure is described in terms of its time-course and cause.

Acute renal failure manifests itself in the course of days, and sometimes is recognized within hours, as when a patient fails to pass any urine (anuria) postoperatively owing to complete loss of renalfunction as a result of processes operating during the anaesthetic and surgery. It may show rapid recovery when a treatable cause is addressed.

Chronic renal failure in contrast often unfolds over a period of months or years.

Etiology

- Diabetic glomerulosclerosis
    - CRF develops in about 30% of type I and type II diabetics
    - peak incidence at about 15 years after the development of diabetes mellitus
    - Untreated, the GFR in diabetic glomerulosclerosis progresses downward at a rate of about 10 to 12 mL/minute/year.
    - Predictors of the development of diabetic glomerulosclerosis are
        - hypertension
        - poor glycemic control
        - microalbuminuria
        - proliferative retinal vascular disease
- Hypertensive nephrosclerosis
    - Nephrosclerosis is as much as 25 times more likely to cause ESRD in the African-American than the white population.
- Glomerulonephritis
    - **Focal glomerulosclerosis** and membranoproliferative glomerulonephritis are the most likely chronic glomerulonephritides to progress quickly in adults.
- SLE, Wegener's granulomatosis
- Tubulointerstitial disease
- Reflux nephropathy (chronic pyelonephritis)
- Analgesic nephropathy

- Obstructive nephropathy (stones, BPH)
- Polycystic kidney disease
    - Very large cysts
    - onset of the disease at an early age
    - hypertension are associated with progression

Types of renal failure

These can be grouped as prerenal, renal and postrenal, referring to the flow of fluid from the circulation, through the kidneys and from the kidneys into the lower renal tract.

1- Prerenal renal failure

This is due to a failure of renal perfusion. The normal resting renal blood flow of about a fifth of the cardiac output provides an important buffer to protect the vital cerebral and coronary circulations in times of circulatory stress. Thus, when a patient suffers a serious haemorrhage, e.g. 20% of the blood volumes, non-vital circulations are reduced by the vasoconstrictor action of sympathetic nerves. Such vasoconstriction takes place in the skin initially and as thesituation deteriorates, i.e. the blood volume continues to decrease; the vasoconstriction spreads to the viscera, including the kidneys.

The combination of a fall in general arterial pressure and compensatory vasoconstriction of the renal resistance vessels (glomerular afferent arterioles) leads to a fall in glomerular capillary hydrostatic pressure (*P*GC), so that eventually it no longer exceeds the combined opposing pressures of the plasma oncotic pressure (*P*GC) and the hydrostatic pressure in the Bowman's capsule (*P*BC). Filtration and formation of urine then cease.

2- **'Renal' renal failure**

Here the cause of the renal failure lies within the kidneys themselves. Firstly, following on from the prerenal circulatory cause just mentioned, an even more severe failure of the renal circulation may, in addition to abolishing the filtration pressure gradient, lead to a blood flow so low that it is inadequate for the metabolic needs of the renal cells. This typically leads to serious damage or death (necrosis) of the highly active renal tubular cells (acute tubular necrosis) and hence acute (potentially reversible) renal failure.

A great variety of diseases can lead to gradual destruction of the kidneys. These include infectious and other inflammatory causes, the deposition of toxic material and in some cases over stretching when there is raised pressure due to obstruction of the urinary tract (this overlaps with the postrenal renal failure considered below). The end result of all these varied diseases is that the normal finely structured architecture of the kidney, on which normal function relies, is replaced by tiny scarred organs, or by abnormal material, or by thin-walled expanded sacs. Since structure and function are complexly and intimately related in the kidneys, it is not surprising that these abnormal organs steadily decline in their capacity to maintain homeostasis of the body fluids and eventually become worse than useless. Removal is often carried out when the kidneys are actually harming the body, e.g. by causing hypertension.

3- Postrenal renal failure

In this case the cause of the problem lies distal to the kidneys.

Pathophysiology of chronic renal failure

In CRF, the renal system experiences-

- Inflammation
- Ischemia

- Necrosis
- Sclerosis
- Fibrosis
- Scarring.
- Regardless of the primary cause of nephron loss, some usually survive or are less severely damaged.
- These nephrons then adapt and enlarge, and clearance per nephron markedly increases.
- If the initiating process is diffuse, sudden, and severe, such as in some patients with rapidly progressive glomerulonephritis (crescentic glomerulonephritis), acute or subacute renal failure may ensue with the rapid development of ESRD.
- In most patients, however, disease progression is more gradual and nephron adaptation is possible.
- Focal glomerulosclerosis develops in these glomeruli, and they eventually become non-functional.
- At the same time that focal glomerulosclerosis develops, proteinuria markedly increases and systemic hypertension worsens.
- This process of nephron adaptation has been termed the "**final common path.**"
- Adapted nephrons enhance the ability of the kidney to postpone uremia, but ultimately the adaptation process leads to the demise of these nephrons.
- Adapted nephrons have not only an enhanced GFR but also enhanced tubular functions in terms of, for example, potassium and proton secretion.

As failure is occurring, a number of substances that are normally excreted accumulate in the body, including nitrogenous waste, electrolytes, and uremic toxins. Eventually all organ systems are affected.

Signs and symptoms-

Patients are often not seen until **late in the course** of the disease, when much of their kidney function has already been lost Kidney adapts so well to progressive loss of nephrons and can maintain constancy of the internal environment until about 75% of renal function has been lost. Patients with uremic manifestations can have a myriad of different complaints referable to almost any organ system.

- All CRF patients with the exception of those with medullary cystic kidney disease have **fixed proteinuria (>200 mg/24 hours).**
- The syndrome may also come to attention because of an **elevated BUN or serum creatinine** concentration in laboratory testing done for a variety of reasons.
- Progressive **metabolic acidosis**
    - The major cause of the failure to excrete enough acid is diminished renal ammonia production and excretion.
    - Although the metabolic acidosis of CRF is commonly referred to as an anion gap acidosis, this gap does not develop until the serum creatinine concentration approaches 5 to 6 mg/dL.
    - Before this stage, serum chloride initially rises as the serum bicarbonate level falls.

    - High serum parathormone levels and extracellular fluid volume lead to proximal tubular acidosis but do not seem to fully account for the early hyperchloremic metabolic acidosis of CRF.
    - Patients who have hyperkalemic distal (type 4) renal tubular acidosis (e.g., in hyporeninemic hypoaldosteronism, common in diabetics) because of tubulointerstitial disease have a much more severe non-anion gap metabolic acidosis relative to the stage of progression of CRF.
- **Hypertension**
    - **Hypertension** develops in 95% of patients with CRF before ESRD does
    - is due to retention of NaCl, inappropriately high renin levels for the status of expended extracellular fluid volume, sympathetic stimulation via afferent renal reflexes, and impaired renal endothelial function with deficient nitric oxide and enhanced endothelin production.
    - If untreated, this type of hypertension is much more likely to enter the malignant phase than is essential hypertension.
- **Acute cardiovascular events,** especially stroke and myocardial infarction, account for about half of the deaths occurring in dialysis patients and also deaths after the first year post-transplantation.
- **Heart failure** is common and is due to sodium and water retention, acid-base changes, hypocalcemia and hyperparathyroidism, hypertension, anemia, coronary artery disease, and diastolic dysfunction secondary to increased myocardial fibrosis with oxalate and urate deposition and myocardial calcification. Uremia itself may also impair myocyte function.
- In the gastrointestinal tract, **anorexia and morning vomiting** are common.
    - In severe uremia, gastrointestinal bleeding may occur secondary to **platelet dysfunction** and diffuse mucosal erosions throughout the gut.
    - **Bloody diarrhea** can occur secondary to uremic colitis.
- **Uremic serositis** is a syndrome of pericarditis, pleural effusion, and sometimes ascites in any combination.
    - **Pericarditis** is fibrinous, hemorrhagic, and usually associated with a mild fever and may cause pericardial tamponade.
- **Pruritus** is a common and troublesome complication of uremia that is only partially explained by hyperparathyroidism and a high Ca × P product with increased microscopic calcification of subcutaneous tissues.
- **Renal osteodystrophy** is characterized by secondary hyperparathyroidism, which is due to hyperphosphatemia, hypocalcemia, marked parathyroid hypertrophy, and bony resistance to the action of parathormone; by inadequate formation of 1,25-dihydroxyvitamin D in the kidney resulting in osteomalacia in adults and rickets in children; and for as yet obscure reasons, by areas of osteosclerosis.
- High parathormone levels and high cytosol calcium concentrations probably contribute to **uremic encephalopathy,** myocyte dysfunction, and an impaired bone marrow response to erythropoietin.
- Severe syndromes termed **calciphylaxis** include metastatic calcification in soft tissues and small blood vessels and ischemic necrosis of skin and muscle. In such

circumstances, partial parathyroidectomy--removal of 3½ glands--may be required, but secondary hyperparathyroidism is best prevented.

- Other joint diseases include **secondary gout and pseudogout,** which may be associated with chondrocalcinosis.
- Patients in late CRF often **appear hypothyroid** and thyroid function tests may be abnormal, despite normal free levothyroxine; free triiodothyronine levels are low and binding of levothyroxine to thyroxine-binding globulin is diminished.
- Most women are **amenorrheic**--although occasionally menorrhagia can occur--and infertile, at least in the later stages of CRF. Impotence and oligospermia are common in men.
- Diabetic patients commonly require **less exogenous insulin** as CRF progresses because of diminished degradation by renal insulinase.
- As uremia progresses, subtle **mental and cognitive dysfunction** develops and, if untreated, progresses to coma.
- Neuromuscular abnormalities with **asterixis and muscle twitching** are common, as are muscle cramps.
- The **restless legs syndrome** is a manifestation of sensory peripheral neuropathy.
- Motor neuropathy is a late phenomenon in uremia.
- Progressively more severe **normochromic, normocytic anemia** develops as the GFR and renal erythropoietin secretion decrease.
    - In most patients, the hematocrit reaches about 20 to 25% by the time that ESRD develops.
- **Uremic coagulopathy i**s secondary to a defect in platelet function, as well as abnormal Factor VIII function.
- It is characterized by a prolonged bleeding time but usually normal prothrombin and partial thromboplastin times, platelet count, and clotting time.
- The **platelet dysfunction** responds to dialysis and to infusion of desmopressin. Epistaxis, menorrhagia, bruising, and purpura, as well as gut bleeding, may all occur.

Uremic patients should be regarded as i**mmunocompromised**, and infection is an important cause of death in CRF and dialysis patients.

Effects of Renal Failure

The effects of renal failure are due to impairment of the range of normal functions, which can be grouped under the headings:
(a) Fluid and electrolyte balance;
(b) Excretion; and
(c) Endocrine functions.
The distinction between (a) and (b) is that balance is maintained by great variation in the amounts of various substances lost in the urine, whereas excretion refers particularly to unwanted substances which, as far as possible, are totally eliminated from the body. Failure of fluid and electrolyte balance Balance is maintained in terms of sodium chloride, which determines extracellular fluid volume, osmolality; whichdetermines total body water, potassium, and hydrogen ions (acid–base balance).

Sodium chloride

Sodium chloride has been called the skeleton of the extracellular fluid. The reason is that its ions constitute the great bulk of the dissolved particles in extracellular fluid. Osmoregulation will determine that these ions are dissolved in an appropriate volume of water, thereby determining extracellular fluid volume. Extracellularfluid volume tends to rise in renal failure because mostpeople take more salt than they need in their diet and the kidney can no longer excrete the surplus. The extracellular volume may increase until the body is seriously waterlogged, with massive dependent oedema and the risk of circulatory overload (blood plasmavolume rises and falls with extracellular volume) and fatal pulmonary oedema. Less commonly, the body may lose extracellular fluid, e.g. With diarrhoea or vomiting and in this case the kidney may make matters worse by failing to conserve salt.

Potassium

Potassium is normally secreted in the urine in accordance with body needs, by a pump which exchanges absorbed sodium for secreted potassium or hydrogen ions. As the system ails, the body is at the mercy of the amount of ingested ionfor its content of that ion. Potentially, either deficiency or excess of potassium could result, but in practice an excess of potassium is much more common, especially in diets which restrict salt and protein in or der to minimize accumulation of salt and the toxic products of protein. Potassium can rise quickly, particularly if there is breakdown of body cells as inacute renal tubular necrosis due to ischaemia. Major cardiac problems are a serious risk and are often preceded by increasingly high T waves in the electrocardiogram.

Hydrogen ions

Hydrogen ion accumulation is one of the most serious problems of renal failure. The degree of accumulation approaches that in diabetic ketoacidosis.

Failure of endocrine functions

Major endocrine functions of the kidney include control of red cell formation via erythropoietin and control of arterial blood pressure via the renin–angiotensin system. Renal failure can lead to anaemia and hypertension.

Anaemia

Anaemia in renal failure, particularly severe renal failure, is related mainly to deficiency of *erythropoietin*. Erythropoietin is believed to be formed in the renal cortex, in metabolically very active cells able to sense the hypoxia due to anaemia or arterial desaturation.

Hypertension

Hypertension has long been recognized as a complication of renal disease, including renal failure. The mechanisms involved are complex. Major causes are likely to be secretion of inappropriately large amounts of renin and inability to excrete adequate amounts of salt and water. Particularly in early renal failure, parts of the kidney may suffer from inadequate circulation (ischaemia) and secrete *rennin* from the juxtaglomerular cells. The renin activates a circulating peptide toangiotensin I and this is converted in the circulation, particularly the pulmonary capillaries, to angiotensin II with its dual actions of vasoconstriction and stimulation of the salt- andwater-retaining hormone aldosterone from the zonaglomerulosa of the adrenal cortex. This would account for the hypertension in early renal failure. Later in renal failure, retention of salt

and water probably plays a role – the patient's blood pressure can bereduced during dialysis by the removal of salt and water from the circulation.

Investigations

Diagnostic Tests-

Urine:

Acidic pH, low osmolality

Fixed specific gravity.

Proteinuria

Casts, WBCs and RBCs may be present in sediment.

Serum:

Decreased pH, bicarbonate, magnesium

Increased potassium, sodium, hydrogen, phosphate, calcium ions.

Increased uric acid, blood urea nitrogen, osmolality.

Decreased iron and iron-binding capacity

Decreased creatinine clearance.

Complete blood count:

Decreased hemoglobin, hematocrit, RBC survival time

Reduced platelets and decreased adhesiveness.

X ray of kidneys, ureter, and bladder:

Signs of contracted kidneys and associated lesions.

Ultrasound:

Small contracted kidneys.

The diagnosis of renal failure may be suggested in a number of clinical situations, e.g. failure to pass urine postoperatively, or gradual development of weakness and drowsiness in some one with recurrent urinary infections. Biochemical studies however are needed for confirmation. Quantitative confirmation of failure and assessment of its severity are obtained by measuring the glomerularfiltration rate. Inulin clearance is regarded as the gold standard. The *creatinine clearance* is also useful and is much easier to measure. Glomerular filtration rate equals creatinine clearance, which equals [urinary creatinine concentration] · [urinary volume/minute]/ [plasma creatinine concentration]. The average adult value is around 120–150 ml/minute, so a value below 100 suggests possible early impairment, a value below 50 definite failures and a value around 5–10 ml/minute indicates severe failure, requiring dialysis.

Normal values vary with body size, sex and age, with much smaller values in infants and young children. As usual, *serial measurements* are particularly helpful in deciding whether the condition is getting worse or improving.

Once the diagnosis is established, and particularly in severe failure, details of the condition and guidance to treatment can be obtained from plasma measurements of various electrolytes, including sodium and potassium, together with acid–base assessment by measuring arterial blood pH and blood gases, and bicarbonate levels.

Haemoglobin levels will indicate whether anaemia ispresent, and, if so, its severity.

X-ray studies may be used to detect abnormality of the kidneys. If required, the function of each kidney can be assessedseparately by collecting its urine from a ureteric catheter and measuring creatinine clearance.

Finally, a simple but fundamental test, not often used in view of more precise measurements, is to assessthe *range of urinary concentration*. This can be done by

depriving a person of fluids for up to 24 hours to assess maximal concentration (normally sparse dark-yellow urine with a high specific gravity, around 1.030 or more, and an osmolality around 1000 mOsm/kg H2O) and then obtaining a urinary sample when the person has taken a surplus litre of fluid when already fullyhydrated, to assess minimal concentration (copious clear urine with a specific gravity around 1.001 and an osmolality around 100 mOsm/kg H2O). In everyday life we can observe these variations.

Potential Complications

All organ systems are affected by end-stage renal disease, and death is imminent without renal transplantation although life may be prolonged with dialysis and/ or hectic treatment.

Goals for Management of Chronic Renal Failure-

- Diagnose and treat the underlying cause if possible
- Avoid factors that exacerbate CRF
- Slow the natural progression of CRF
- Manage the uremic syndrome

Treatment options

Treatment can be in four forms:

• Conservative
• Haemodialysis
• Peritoneal dialysis
• Renal transplantation.

These treatments deal with the problem in very different ways.

Conservative treatment

This refers to the adjustment of food and fluid intake to minimize the load on the kidneys. Because protein provides the bulk of dietary toxins, it is restricted to around a quarter of normal. Because the patient's energy requirements must be met to prevent breakdown of the tissues (releasing amino acids) the carbohydrate and fat content must be fairly high. Fluids should be adjusted to balance the patient's urinary output, and electrolytes adjusted according to the plasma levels. Usually this means low sodium content. Overall this diet is difficult to maintain, unpalatable and of limited effectiveness, but the general principles are applied, in a rather more relaxed manner, during long-termdialysis as a back-up to this therapy.

Haemodialysis

Introduction of this treatment has dramatically extended life in patients with severe renal failure. The principle is simple. The patient's blood is withdrawn from the circulation and passed through tubing surrounded by a dialysate fluid. The tubing is permeable to water and to the smaller particles in the blood, including ions, glucose, urea and creatinine, but the tubing does not allow plasma proteinsand cellular elements to be lost from the blood. The dialysate fluid is free of unwanted items such as urea and contains appropriate amounts of various ions. Thus, if there isneed to lose sodium, the dialysate will have a low sodium content. The dialysate should also be free of unwanted materials and care is needed to avoid infection. The patient's 'purified' blood is then returned to the circulation. Advancing technology has led to increasingly efficient systems which, rather like the kidney, contain multiple fine tubes in a very small space.However, the simple principle of equilibration with a dialysate is much different from the sophistication of normal renal

function with its filtration, reabsorption, excretion, medullary osmotic gradient, complex vasculature and hormonal control.

The concept of an *arteriovenous shunt* was developed. Initially a tube connected a forearm artery and vein. The tube rather than the artery and vein could then be punctured for dialysis. However, this tubing was uncomfortable and there was a considerable risk of bleeding. Finally, a *surgical arteriovenous fistula* was devised. An opening, usually in the radial artery, was connected to a nearby vein so that the forearm veins draining the fistula became dilated and carried an adequate flow for dialysis. Haemodialysis using such 'arterialized' veins can maintain health for long periods, provided there are no complications with thrombosis or infection.

Peritoneal dialysis

This is an alternative to haemodialysis – it uses the capillaries of the peritoneal cavity as the tubing, and fluid passed into the peritoneal cavity and withdrawn after an equilibration period as the dialysate. The dialysate is supplied in plastic bags and is passed into the peritoneal cavity under the influence of gravity by raising the bag above the level of the patient's abdomen. The peritoneal cavity is capable of holding several litres of fluid without any difficulty. In practice, fluid is kept in the peritoneal cavity almost continuously. About four times a day, the patient drains as much fluid as possible by connecting an empty bag to the peritoneal cavity and placing the bag on the floor. When drainage has ceased, a fresh 2-litre bag is hung up well above the patient's abdomen and the fluid run in. Thus solute exchange can proceed throughout the day and night by a procedure analogous in slow motion to gas exchange in the alveolar air, replenished by the tidal ventilation. This process has the advantage of relative simplicity compared with haemodialysis but it is laborious for the patient and still carries the risk of infection.Treatment with erythropoietin in renalfailure is also neccessory. It is, of course, not required with successful renal transplantation and this is nowthe definitive treatment which can liberate patients from the onerous demands of either form of dialysis treatment.

Renal transplantation

Renal transplantation is now well established. The requirements are:

1. Connection of the renal artery of the transplanted organ to any convenient artery in the recipient
2. A corresponding venous connection
3. Connection of the donor ureter to the patient'sbladder, and
4. Prevention of rejection of the kidney.

In practice, the donor kidney is usually placed in one of the iliac fossae, with attachments to the neighbouring major blood vessels. Prevention of rejection is achieved by as close a match as possible for cellular antigens (identical twins have provided a perfect match on rare occasions) and by drugs, including glucocorticoids, which suppress immune responses. The donor organ may come from a relative, friend, or from the body of someone who has died in circumstances where the kidney can be removed prior to post-mortem deterioration. The organ must then be preserved prior to transplantation, sometimes during a considerable journey, to a well-matched recipient. It is kept in isotonic solution at around 4–5°C. This temperature is high enough to avoid freezing, with the disastrous formation of destructive ice crystals, and low enough to reduce the metabolic rate of the renal cells to ensure survivalfor several hours. Once the organ has been 'plumbed in', it will begin to function and produce urine. While various blood tests

may give clues about transplant rejection, measurements of glomerular filtration rate by creatinine clearance provide the definitive indication of function. A substantial and gradually rising clearance indicates good function, whereas a falling clearance suggests that rejection has begun. Prior to transplantation, the kidney had provided half the renal function and the initial glomerular filtration rate of the transplanted kidney will be about half normal. However, as the sole kidney in the recipient, the organ will undergo gradual hypertrophy with an increase in glomerular filtration rate over the next 2–3 months. All functions of the kidney, including appropriate formation of erythropoietin, can be expected to be normal.

Dietary Therapy

a. Protein restriction - potential benefits include:
    i. Decreases glomerular hyperfiltration, which may slow progression of glomerulosclerosis.
    ii. Protein restricted diets are phosphorus restricted, which delays onset of renal secondary hyperparathyroidism and may slow progression of glomerulosclerosis.
    iii. Moderate protein restriction reduces proteinuria in glomerulopathies.
    iv. Protein restriction may reduce net acid load and renal ammoniagenesis, which may slow progression of CRF.
    v. May reduce serum lipids.
    vi. May reduce immune cell activity and intraglomerular coagulation within the kidney.
    vii. Improves the symptoms of uremia - this is probably the most significant advantage!
b. Recommendations
    i. Moderate protein restriction is recommended for all uremic patients, and may be beneficial in early CRF patients. Examples are Hill's K/D, Eukanuba Veterinary Diets Nutritional Kidney Formula Early Stage, or Purina Veterinary Diets N/F.
    ii. Be cautious to avoid malnutrition by monitoring body weight, serum albumin, anemia, haircoat and BUN:creatinine ratio.
    iii. For severe uremia or intractable hyperphosphatemia, there are diets with severe protein and phosphorus restriction (e.g. Hill's U/D or Eukanuba Nutritional Kidney Formula Advanced Stage).
    iv. If a patient is not meeting his energy needs with food intake, he will catabolize body proteins for energy. This contributes to acidosis, renal ammoniagenesis, and uremia. Adequate 'bad' calories are better than inadequate 'good' calories!
c. Nutritional support "Beyond the Can"
    i. Enteral nutrition is preferred for uremic patients if it is at all possible. This approach nourishes the gut as well as the body, reducing the risk of bacterial translocation and sepsis.
    ii. Feeding tubes (PEG, esophagostomy, nasoesophageal) are very helpful in the medical management of CRF. Bigger tubes can be

used for blenderized canned renal diets. Esophagostomy tubes are often preferred due to lack of specialized equipment, short anesthetic time required, and simplicity of placement and care.

iii. Patients with severe vomiting and/or hypoalbuminemia may benefit from PPN or TPN. Strict intravenous catheter care, nutritional knowledge, and critical care nursing support are essential for success with these therapies. A local human hospital pharmacy will sometimes prepare the prescribed solution for the veterinarian.

Here, I am giving some cases treated by me with Homoeopathy.

S. no.	Case no.	DOA	Pt's name	Sex / age	Address	Diagnosis	1st Remedy	2nd Remedy	Result
1	9482	1/1/2001	Prit Pal Singh	M 35	New Jersy, USA	CRF, RPD	Ser ang 30	Apis Q	Cured
2	9487	1/1/2001	Jagdish Pd Agrawal	M 72	Ganj Market, Kashipur	CRF	Med 1M	Rhus t 200	Got Relieved ++++
3	9268	3/2/2001	P. K. Goel	M 47	Jigar Colony, Moradabad	DM, CRF, Gangrene	Opium 30	Acet ac 30	First Got Relieved then Stable
4	9291	12/2/2001	Shahzad Akhtar	M 45	Muslim College, Moradabad	CRF	Opium 30	Urea 30	First Got Relieved then Stable
5	9319	20-02-01	Jitendra Kumar	M 50	Vat. College, Pantnagar	CRF, HT, COAD	Phos 30	Kali c 30	First Relieved then died
6	9324	22-02-01	Shish Ram	M 58	Mandawar, Bijnor	CRF	Opium 30	Apis Q	Got Relieved +++
7	9325	22-02-01	Nathu Ram Sharma	M 59	Phool Bagh, Pantnagar	Nephrolithiasis, CRF	Ser ang 30	Aur met 3x	Cured
8	9340	24-02-01	Mahendra S Rajput	M 65	21, Naveennagar, Moradabad	CRF, DM, Tub	Helon q	Opium30	Not reported
9	9372	6/3/2001	Vijendra Kumar	M 19	Govindnagar, Moradabad	CRF	Nit ac 30	Apis Q	First Got Relieved then died
10	9379	8/3/2001	Saroj	F 18	Deputy Parao, Moradabad	CRF, Poliomyelitis	Opium 30	Apis Q	Got Relieved ++++
11	9416	19-03-01	Santosh Pandey	F 39	Pakka Kot, Kashipur	CRF	Helon q 30	Merc sol	Got Relieved +++
12	9420	19-03-01	O P Gupta	M 60	13, Punjabi Mohalla, Kichchha	CRF	Opium 30	Apis 200	Got Relieved +++
13	9426	20-03-01	Harish Chandra	M 65	Garhinegi, Kashipur	CRF, DM, HT	Opium 30	Nux v 200	Relieved +++
14	9455	24-03-01	Muktesh Mathur	M 66	Kotpurvi, Sambhal	CRF	Stram 30	Opium 200	Cured
15	9475	29-03-01	Ashok K Agrawal	M 57	Moradabad Road, Kashipur	CRF, DM	Ars a 200	Apis Q	Died
16	9478	30-03-01	Mahesh Ch Rastogi	M 68	53, warden Road, Bombay	CRF, CAD	Ser ang 30	Dig Q	Did not Get relieved
17	9486	31-03-01	Rafeeq	M 32	Tanda, Rampur	CRF, Tubercular Pyelonephritis	Colch 30	Merc sol 30	Got Relieved ++++
18	9500	4/4/2001	Manoj	M	Negpur, Bareilly	CRF, Post pyretic	Nit ac 30	Helon Q	Got Relieved

			Kumar	20						+++
19	9505	5/4/2001	Mulakraj	M 60	Garhinegi, Kashipur	Diabetic Nephropathy	Ser ang 30	Dig Q	Progressively Deteriorated and Died
20	9522	9/4/2001	Rajiv Grover	M 22	Balram Nagar, Gadarpur (USN)	CRF, Ureter Calc	Opium 30	Canth Q	Cured
21	9525	10/4/2001	Surendra Singh	M 50	Doraha, Bazpur (USN)	HT, CRF, Sensory auditory deficit	Opium 30	Lith 3x	Not reported
22	9537	13-04-01	V K Bhatnagar	M 50	Phoolbagh, Pantnagar	CRF	Phos Q	Lach 30	Got Relieved but left
23	9543	14-04-01	Sudhakar Prasad	M 23	Pantnagar	CRF	Arg n 3x	Urea 30	Cured
24	9560	18-04-01	Sonu Sharma	F 18	Meerganj, Bareilly	CRF, Ascitis, RPD	Colch 30	Apis Q	Did Not Get relieved
25	9597	25-04-01	Aviral Tyagi	M 6 m	Lal Sagar, Jodhpur	ARF	Cal carb 6	Podo 200	Cured
26	9609	30-04-01	R L Shah	M 62	Tallital, Nainital	CRF, HT, ITA, DM	Opium 30	Plumb 3x	Not Reported
27	9637	5/5/2001	Kalawati Padiyar	F 50	GGIC, Kashipur	RPGN, CRF	Opium 30	Canth Q	Not reported
28	9648	9/5/2001	Ashok Goel	M 48	Ganesh Chawk, Hapur	CRF, DM, Gangrene	Opium Sec c 30	200	Got Relieved ++++
29	9649	9/5/2001	Saroj Arora	F 49	Patel nagar, Kashipur	CRF, DM, HT, Lt UR Stone	Nux v 30	Pariera b Q	Cured
30	9700	19-05-01	Vinod Bhatia	M 53	Neelam Cinema, Faridabad	DM CRF Ascitis Membranous	Acet ac 30	Apis Q	Got Relieved ++++
31	9713	23-05-01	Ashok Bajaj	M 40	Congress Block, Gadarpur (USN)	Glomerulo-nephritis, CRF	Merc sol 30	Iris v 30	Got Relieved ++++
32	9833	23-06-01	Rajrani	F 60	Maheshpura, Kashipur	CRF, Tub. Lungs	Opium Urea 30	200	Got Relieved +++
33	9834	25-06-01	Amar Singh	M 25	Bhagat Singh Cawk, Rudrapur (USN)	CRF, RMD	Opium Med 1M	30	Cured
34	9863	3/7/2001	Sardar Singh	M 60	Bijli Farm, Bilaspur, Rampur	CRF, DM	Opium 30	Apis Q	Not Reported
35	9998	37142	Dr. Joginder Singh	M 43	P 292, Awas Vikas, Rudrapur (USN)	CRF, ESRD	Med 1M	Ser Ang 30	Got Relieved ++++
36	10323	24-10-01	Mohd. Yaqi	M 56	Liberty Tailors, Kashipur	DM, CRF,	Opium 30	Helon Q	Not Reported
37	10324	25-10-01	Kripal Singh	M 48	Haldi Farm, Pantnagar	CRF	Opium 30	Helon Q	Not Reported
38	10038	20-08-01	Kamla Devi	F 26	Kundeshwari, Kashipur	CRF, ESRD	Veart vir 30	Canth Q	Not Reported
39	10115	8/9/2001	Harprit Singh	M 15	Dhakia, (USN)	CRF	Terebinth 30	Acid Nit Q	Got Relieved +++
40	10091	36900	Gopal Singh	M 47	Jawahar nagar, Pantnagar	CRF, DM, ESRD	Opium 30	Helonias Q	Cured
41	10401	14-11-01	Harjeet Kaur	F 45	Gulzarpur, Khikhratal, Kashipur	CRF, Ca Cervix	Kreosote Q	Acid Nit 30	Got Relieved +++
42	10530	26-12-01	Srivastava	M 28	Engg. College Office, Pantnagar	CRF, ESRD, RPGN	Terebinth 30	Helon Q	Got Relieved +++
43	10434	23-11-01	Chandra	M	Subhash Chawk,	CRF, CRF, SA	Opium	Apis Q	Got Relieved

				Shekhar	17	Ramnagar (USN)	Glomerulonephritis, Koch's Lungs	30		++++
44	10439	24-11-01	Khem Singh	M 40`	Irrigation Dep, Banbasa, Champawat	CRF, ESRD	Apis Q	Opium 30	Got Relieved ++++
45	10453	27-11-01	Sunita Khurana	F 41	Nai Line, Sabji Mandi, Ramnagar (USN)	CRF, IDDM	Helon Q	Aur iod 30	Cured
46	10458	29-11-01	Anil Kumar	M 35	128, Old Prempuri, Station Road, Meerut	CRF	Opium 30	Apis Q	Got Relieved +++
47	10517	24-12-01	Lalit Kumar Sharma	M 21	Pantnagar	Recc. Nephrosis Syndrome,CRF	Apoc 30	Arg nit 6x	Cured
48	10543	31-12-01	Ramesh Ch. Sharma	M40	C/O Y P Sharma, Pantnagar	LVH, CRF	Dig Q	Ser Ang 30	Relieved +++
49	10529	25-12-01	Doongar Singh	M 80	Sharifnagar, Thakurdwara, Moradabad	CRF, Stevens Johnson Syndrome	Ars alb 30	Antim Sulph 6x	Got Relieved ++
50	10532	26-12-01	Satbir Singh	M3 4	624, Sect. 6, Gurgaon	CRF	Colch 30	Merc sol 30	Cured
51	10562`	37377	Urmila	F 52	Rama Mandir, Ramnagar (Nainital)	CRF, RA, HT	Colch 30	Helon Q	Got Releived +++
52	10572	37561	Sarfuddin Alam	M 46	Nawab Ganj, Bareilly	CRF, ESRD	Opium 30	Dig Q	First Releived then died
53	10579	14-01-02	Sudesh Kumar	M 27	Sahaswan, Budaun	CRF, ESRD, Post Renal Tx Case	Arg. Nit. 30	Calc phos 30x	Got Releived ++++
54	10595	18-01-02	Nidhi Gupta	F 22	Sambhal (Moradabad)	HT, CRF, ESRD	Ser ang 30	Helon Q	Got Relieved ++
55	10674	37592	Kamla Bagadwal	F 35	Haldwani	CRF ESRD	Opium 30	Helon Q	Got Relieved but left
55	10675	37592	Prushottam Sharma	M 45	Ghaziabad	CRF ESRD CGN	Apis Q	Opium 30	Got Relieved but left
56	10779	23-03-02	Rajendra Mehra	M 40	Haldwani	CRF, ESRD	Apis Q	Ser ang Q	Cured
57	10849	23-04-02	Jang Bahadur	M 45	Baheri (Bareilly)	CRF	Canth Q	Urea 30	Got Relieved +++
58	10952	10/6/2002	Islam	M 60	Bareilly	CRF	Ser ang 30	Apis Q	Got Relieved but left
59	10965	37596	Savitri	F 38	Bareilly	CRF, ESRD,	Opium 30	Canth Q	Not reported
60	11036	37536	Seema	F33	Pantnagar	CRF	Sec c 30	Opium 200	Got Relieved ++++
61	11071	25-07-02	Ram Kumar	M2 5	Bijnor	CRF	Nux v 30	Pariera b Q	Got Relieved ++++
62	11129	15-08-02	Dr.S.M.Puri	M6 2	Rudrapur	CGN,CRF,	Acet ac 30	Apis Q	Got Relieved but left
63	11144	21-08-02	.ShaifaliAdhikari	F60	Dineshpur	CGN,CRF	Merc sol 30	Iris v 30	Got Relieved ++++
64	11210	18-09-02	Kamal Singh	M3 0	Kashipur	ESRD,CRF	Urea 30	Opium 200	Got Relieved +++
65	11194	37446	Satyapal Sharma	M5 2	Dhampur	ESRD,CRF	Med 1M	Opium 30	Cured

Area wise sorting of the patients-

Kashipur-	13	Sambhal-	02	Ghaziabad-	01
Pantnagar-	10	Bijnor-	02	Gurgaon-	01
Bareilly-	06	Bombay-	01	Hapur-	01
Moradabad-	06	Badaun-	01	Jodhpur-	01
Rudrapur-	04	Champawat-	01	Kichha-	01
Ramnagar-	03	Dhampur-	01	Meerut-	01
Gadarpur-	02	Dineshpur-	01	Nainital-	01
Haldwani-	02	Bazpur-	01	USA-	01
Rampur-	02	Faridabad-	01		

By making Zones-

We see that there are only four cities which have minimum 06 kidney failure patients. We can name these four placs as zones. The surrounding places of these four zones can be added to the adjacent zone for studies.

1- Pantnagar Zone-	21	32.30%
2- Kashipur Zone-	17	26.15%
3- Moradabad Zone-	11	16.92%
4- Bareilly Zone-	07	10.76%
5- Solitary Cities-	09	13.84%

Summary-

	No. of Patients	Percentage
Total no. of Patients registered	65	(Male- 51, Female- 14)
Not reported	09	
Result Awaited	none	
Effective No. of patients	**56**	
Cured	14	25%
Got Relieved ++++	14	25%
Got Relieved +++	14	25%
Got Relieved but left	05	8.92%
First relieved and stable	02	3.57%
First relieved and died	03	5.35%
Did not Get relieved	02	3.57%
Progressively deteriorated and died	01	1.78%
Died	01	1.78%

Role of Homoeopathy in CRF

Response	No. of Patients	Percentage
Positive	52	92.85%
Neutral	2	3.57%
Negative	2	3.57%

Total no. of Remedies Employed in 65 Cases 31

S. No.	Remedy	No. of Cases as First Choice	No. of Cases as Second Choice	Total No. of Cases Where the Remedy is Used	Proposed Grade
1	Opium	19	10	29	1
2	Apis	3	13	16	1
3	Serum angullae	6	3	9	2
4	Helonias	3	8	11	2
5	Urea	2	2	4	2
6	Acetic acid	2	1	3	3
7	Cantheris	1	4	5	3
8	Colchicum	4		4	3
9	Digitalis	1	3	4	3
10	Medorrhinum	4		4	3
11	Merc sol	1	3	4	3
12	Nit acid	2	2	4	3
13	Nux Vom	2	1	3	3
14	Phosphorus	2		2	3
15	Arg nit	2	1	2	4
16	Aur met		1	1	4
17	Ars alb	2		2	4
18	Calc carb	1		1	4
19	Iris v		2	2	4
20	Kali carb		1	1	4
21	Lachesis		1	1	4
22	Lithium carb		1	1	4
23	Pariera breva		2	2	4
24	Plumbum		1	1	4
25	Podophyllum		1	1	4
26	Rhus tox		1	1	4
27	Secale corn	1		1	4
28	Strammonium	1		1	4
29	Aur iod		1	1	4
30	Kreosote	1		1	4
31	Terebinth	1		1	4

Here we see that Opium is the top rank remedy for CRF. The second one is Apis. Totality of the symptoms based on symptoms of CRF, underlying causes and constitutional symptoms of the patients simulates these 31 great remedies rendering their value to top in our repertory for CRF.

Repertorial Rubrics in covering Cardinal Signs and Symptoms of Chronic Renal Failure (CRF)

1 MIND - DULLNESS
2 MIND - IRRITABILITY
3 MIND - RESTLESSNESS
4 GENERALS - UREMIA
5 GENERALS - MEDICINE - allopathic - abuse of

6 GENERALS - DROPSY - kidney disease, from
7 GENERALS - DROPSY - albuminuria, with
8 GENERALS - SLUGGISHNESS of the body
9 GENERALS - ANEMIA
10 GENERALS - ANEMIA - nutritional disturbance, from
11 GENERALS - ANEMIA - disease; from exhausting
12 GENERALS - CONVULSIONS - uremic
13 GENERALS - HYPERTENSION
14 SLEEP - SLEEPINESS
15 GENERALS - PULSE - frequent (= accelerated, elevated, exalted, fast, innumerable, rapid)
16 GENERALS - PULSE - irregular
17 GENERALS - DROPSY - external dropsy (= anasarca, edema)
18 GENERALS - DROPSY - internal
19 GENERALS - SWELLING - general, in
20 GENERALS - TUBERCULOSIS - lupus vulgar
21 GENERALS - WEAKNESS (= enervation)
22 STOMACH - VOMITING
23 STOMACH - HEARTBURN
24 STOMACH - HICCOUGH
25 STOMACH - THIRST
26 STOMACH - THIRSTLESS
27 ABDOMEN - DROPSY - ascites
28 RECTUM - DIARRHEA
29 RECTUM - CONSTIPATION
30 STOOL - BLACK
31 URINE - SUGAR
32 URINE - SEDIMENT - bloody
33 URINE - SEDIMENT - mucous
34 URINE - ALBUMINOUS
35 URINE - SEDIMENT - purulent
36 URINE - SEDIMENT - renal calculi
37 CHEST - DROPSY
38 KIDNEYS - INFLAMMATION - cold; from
39 KIDNEYS - CATARRH
40 KIDNEYS - INFLAMMATION
41 KIDNEYS - INFLAMMATION - acute parenchymatous
42 KIDNEYS - INFLAMMATION - bloody, ink-like, albuminous urine, with
43 KIDNEYS - INFLAMMATION - cardiac and hepatic affections, with
44 KIDNEYS - INFLAMMATION - suppurative
45 KIDNEYS - INFLAMMATION - toxemic
46 KIDNEYS - SUPPRESSION of urine
47 KIDNEYS - SUPPRESSION of urine - dropsy, and
48 KIDNEYS - SUPPRESSION of urine - convulsion with
49 KIDNEYS - SUPPRESSION of urine - stupor, with
50 KIDNEYS - SUPPRESSION of urine - violent
51 KIDNEYS - SUPPRESSION of urine - gonorrhea; from suppressed

Result of analysis of 51 Cardinal Rubrics of CRF with Synthesis Repertory

Opium.

Other Remedies in Decreasing Order of Their Value In CRF

apis, dig., canth., colch., sulfa., hell., helon., plb. and crot-h.

A. Rubrics Covered with Opium

1. MIND - DULLNESS
2. MIND - IRRITABILITY
3. MIND - RESTLESSNESS
4. GENERALS - UREMIA
5. GENERALS - MEDICINE - allopathic - abuse of
6. SLEEP - SLEEPINESS
7. GENERALS - PULSE - frequent (= accelerated, elevated, exalted, fast, innumerable, rapid)
8. GENERALS - PULSE - irregular GENERALS – SWELLING – genera, in
9. GENERALS - DROPSY - external dropsy (= anasarca, edema)
10. GENERALS - SLUGGISHNESS of the body
11. GENERALS - CONVULSIONS - uremic
12. GENERALS - WEAKNESS (= enervation)
13. STOMACH - VOMITING
14. STOMACH - HEARTBURN
15. STOMACH - HICCOUGH
16. STOMACH - THIRST
17. STOMACH - THIRSTLESS
18. GENERALS – DROPSY - Internal
19. RECTUM - DIARRHEA
20. RECTUM - CONSTIPATION
21. STOOL - BLACK
22. URINE - SUGAR
23. URINE - SEDIMENT - mucous
24. URINE - ALBUMINOUS
25. CHEST - DROPSY
26. KIDNEYS - SUPPRESSION of urine

B. Rubrics Covered with Apis

1. MIND - DULLNESS
2. MIND - IRRITABILITY
3. MIND - RESTLESSNESS
4. GENERALS - UREMIA
5. GENERALS - ANEMIA
6. GENERALS - CONVULSIONS - uremic
7. SLEEP - SLEEPINESS
8. GENERALS - PULSE - frequent (= accelerated, elevated, exalted, fast, innumerable, rapid)
9. GENERALS - PULSE - irregular
10. GENERALS - DROPSY - external dropsy (= anasarca, edema)
11. GENERALS - DROPSY - internal
12. GENERALS – DROPSY, kidney disease, from
13. GENERALS - SWELLING - general, in
14. GENERALS - TUBERCULOSIS - lupus vulgar

15. GENERALS - WEAKNESS (= enervation)
16. STOMACH - VOMITING
17. STOMACH - HEARTBURN
18. STOMACH - THIRST
19. STOMACH - THIRSTLESS
20. ABDOMEN - DROPSY - ascites
21. RECTUM - DIARRHEA
22. RECTUM - CONSTIPATION
23. STOOL - BLACK
24. URINE - SEDIMENT - bloody
25. URINE - ALBUMINOUS
26. CHEST - DROPSY
27. KIDNEYS - INFLAMMATION
28. KIDNEYS - INFLAMMATION - acute parenchymatous
29. KIDNEYS - SUPPRESSION of urine

C. Rubrics Covered with Ser- ang

1 KIDNEYS - INFLAMMATION - cold; from
2 GENERALS - PULSE - frequent (= accelerated, elevated, exalted, fast, innumerable, rapid)
3 GENERALS - PULSE - irregular

D. Rubrics Covered with Helonias

1. MIND - DULLNESS
2. MIND - IRRITABILITY
3. MIND - RESTLESSNESS
4. GENERALS - DROPSY - kidney disease, from
5. GENERALS - DROPSY - albuminuria, with
6. GENERALS - ANEMIA
7. GENERALS - ANEMIA - nutritional disturbance, from
8. GENERALS - ANEMIA - disease; from exhausting
9. SLEEP - SLEEPINESS
10. GENERALS - DROPSY - external dropsy (= anasarca, edema)
11. GENERALS - WEAKNESS (= enervation)
12. ABDOMEN - DROPSY - ascites
13. URINE - SUGAR
14. URINE - ALBUMINOUS
15. KIDNEYS - INFLAMMATION
16. KIDNEYS - INFLAMMATION - acute parenchymatous

A. **Rubrics Covered with Urea**

1 GENERALS - DROPSY - external dropsy (= anasarca, edema)
2 GENERALS - DROPSY - internal
3 GENERALS - SWELLING - general, in
4 GENERALS - TUBERCULOSIS - lupus vulgar
5 GENERALS - WEAKNESS (= enervation)

Conclusion

Apart from these, the constitutional symptoms of the particular individual patient play vital role in deciding the final choice of the remedy. Thus we see that Homoeopathy has a miraculous role in field of Nephrology, especially CRF, where other pathies have only option to transplant a new kidney to support a life.

Further researches are needed for new discoveries and to make Homoeopathy more valuable in this regard.