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A Comprehensive Review on Revivification for ESRD Patients - Haemodialysis

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ABSTRACT

Renal system plays a vital role in removing metabolic waste product toxins like urea, uric acid, creatinine from blood and excrete it through urine. Abnormalities in Renal anatomical or in physiological condition can lead to the development of Acute kidney injury, it is reversible but in certain case due to progression of disease condition leads to CKD. Chronic kidney disease (CKD) which is characterized by structural and functional changes of the kidney with glomerular filtration rate of less than 60ml/min/1.73 m. sq. Here, Haemodialysis, extracorporeal procedure to eliminate waste from the body. During this procedure patient may experience various complications such vascular access stenosis, thrombosis of haemodialysis access, thrombus in the dialysis catheter, dialysis access infections, cardiovascular abnormalities, bone disorders, anemia, nutrition deficiency, fluid imbalance, hypotension, The changes in the lifestyle modifications and diet restriction should be maintained by haemodialysis patient to prevent further progression of the disease, complication and to improve the quality of life.

Key Words: Kidney, Chronic Kidney Disease, Haemodialysis, End Stage Renal Disease.

1. INTRODUCTION

The Human renal system plays a vital role in Homeostatic regulation of blood and remove metabolic waste product toxins like urea, uric acid, creatinine from blood and excrete through urine.[1] Kidneys are bean shaped organ retroperitoneally placed in the posterior abdominal wall on either side of the spine at the level of the upper lumbar vertebrae.[2] The renal parenchyma has cortex and medulla which contains nephrons, each consisting of a glomerulus and a tubule which is located in that region.[3]

In Glomerular network, blood enters Bowman capsule at high pressure via afferent arteriole. Blood is forced through glomerular capillaries for filtration. After filtration, blood leaves the glomerulus by an efferent arteriole at high pressure.[4] Glomerulus capsule is followed by proximal tubule (PT). The PT contains both a convoluted and a straight section after that filtrate descent to loop of Henle. Fluid then passes through the distal convoluted tubule (DCT), and then to connecting tubule [2] The peritubular capillary network surrounds the renal tubule as it tracks through the kidney, sustaining the tubule with oxygen and nutrients.[4]

Abnormalities in Renal anatomical or in physiological condition can lead to the

development of Acute kidney injury, it is reversible but in certain case due to progression of disease condition leads to CKD. [4] Chronic kidney disease (CKD) is a progressive condition characterized by structural and functional changes of the kidney due to various causes for more than three months characterized by decreased kidney function, and estimated glomerular filtration rate of less than 60ml/min/1.73 m. sq. [5]

According to KDIGO 2012 clinical practice guideline, CKD is classified into five stages considering the GFR level.

- Stage 1: Kidney damage with normal GFR (greater than 90ml/min)
- Stage 2: Mild reduction in GFR (60-89mL/min)
- Stage 3a: Moderate reduction in GFR (45-59mL/min)
- Stage 3b: Moderate reduction in GFR (30-44mL/min)
- Stage 4: Severe reduction in GFR (15-29mL/min)
- Stage 5: Renal failure (ESRD) (GFR less than 15mL/min).[6]

Renal replacement therapy referred as the treatment for the patient with ESRD. It include hemodialysis, peritoneal dialysis, hemofiltration, hemoperfusion and kidney transplantation.[7]

2. DIALYSIS:

In medicine, dialysis is a form of renal replacement therapy where the kidney's role of filtration of blood is supplemented by artificial equipment which removes excess water, solutes and toxins. The term dialysis is derived from the Greek word, "dia" meaning "through" and "lysis" meaning "loosening or splitting". It ensures the maintenance of homeostasis.[8]

2.1. TYPES:

There are 3 types of dialysis:

2.1.1. Haemodialysis:

Haemodialysis is the most widely recognized type of Dialysis. It uses a haemodialyzer to eliminate waste and additional liquid from the blood. The blood is taken out from the body and separated through the artificial kidney. The filtered blood is then sent back to the body with the assistance of a dialysis machine.

2.1.2. Peritoneal Dialysis:

Peritoneal Dialysis involves a medical procedure to implant a peritoneal dialysis catheter into the abdomen. During treatment, a dialysate streams into the peritoneum and assimilates waste. Once the dialysate removes waste out of the bloodstream, it is drained from the abdomn.

2.1.3. Continuous Renal Replacement Therapy:

Continuous renal replacement therapy is used generally in the intensive care unit for those individuals who have acute kidney failure. It is also called hemofiltration [9]

2.2. HISTORY:

Thomas Graham in 1854 first described the concept of dialysis. It was first performed in laboratory animals in 1912. The first successful haemodialysis in humans was reported in 1943 by Dr. Willem Kolff who is considered the father of dialysis. Kolff's machine remained the standard for the next decade and is considered the first modern drum dialyzer. George Thorn received a set of blueprints of Kolff's kidney machine at the Peter Bent Brigham Hospital in Boston which led to the manufacture of the next generation of Kolff's dialyzer, a stainless-steel Kolff-Brigham kidney, that paved the way for the first kidney transplant in 1954.[10]

Early attempts at haemodialysis were complicated by the lack of reliable techniques for repeated circulatory access. Haemodialysis was at first performed using metal or glass cannulas that were inserted into an artery and a vein. A major advance occurred in 1960, when a young professor of medicine at the University of Washington, Dr. Belding Scribner and his associates introduced plastic indwelling vascular cannulas, the first long-term device for chronic dialysis that works by connecting the patient to the dialyzer using plastic tubes, one inserted into a vein and one into an artery.[10]The circulatory access would be kept open after the treatment by connecting the two tubes outside the body using a small U-shaped device, which would shunt the blood from the tube in the artery back to the tube in the vein.

In 1966, Cimino and his associates introduced surgical creation of an arteriovenous fistula that can withstand repeated needle puncture. Introduction of prosthetic materials for shunts and temporary use plastic dialysis catheters followed. The combination of improved access techniques, new plastic materials for dialyzer membranes, and technologic advances in production of dialyzers and dialysis machines has led to a remarkable growth in the number of patients on hemodialysis over the past 20 years. [10]

2.3. PRINCIPLE:

Dialysis involves altering the solute composition of the patient's blood by exposing the blood to a modified salt solution (dialysate) separated by a semipermeable membrane. Solutes and water pass across the semipermeable membrane through the processes of diffusion and ultrafiltration. The movement of solutes is directly proportional to the magnitude of existing concentration gradients. The concentration gradient can be manipulated by the measured addition of substances to the dialysate.

Passive diffusion occurs when a high to low concentration gradient is present between the patient's blood and dialysate used. Waste products in the blood will diffuse into the dialysate solution while essential minerals will diffuse into the blood; diffusion stops once equilibrium is achieved. The size of the molecule determines whether diffusion occurs: only low molecular weight solutes and water are able to pass through the semi-permeable membrane and therefore red blood cells are not lost.

Ultrafiltration is the second mechanism by which solutes are transported across semipermeable membranes which ensures excess fluid is cleared from the body through the use of a positive (blood) or negative (dialysate) pressure gradient, moving fluid from a high to low pressure region by either hydrostatic or osmotic forces.

Convection allows effective clearing of larger molecules from the blood by creating a higher hydrostatic pressure in the blood (using a blood pump), leading to the passive movement of solutes dissolved in fluid.

2.4. COMPONENTS:

2.4.1. WATER:

During a dialysis treatment, concentrated dialysate is mixed with water to produce the final dialysate. Water for dialysis is generated through a series of steps, including water softening, charcoal filtration, reverse osmosis, and deionization. In the final step, dialysate is generated by a proportioning system (central supply or

dialysis machine) that adds known amounts of dialysate concentrate to dialysis water. The dialysate has direct access to the patient's bloodstream across the semipermeable membrane, and with each dialysis session, the patient is exposed to approximately 120 L of dialysate. Thus, maintaining a quality water supply is a major focus in dialysis units.

2.4.2. ANTICOAGULATION:

Exposure of blood to dialysis membranes initiates the clotting cascade, and anticoagulation is used routinely to prevent occlusion of the dialyzer. Coagulation is monitored using the whole blood partial thromboplastin time. Most simply, a low-dose heparin infusion can be tried. Anticoagulant free dialysis is particularly used in patients with prolonged bleeding times; however, steps must be taken to decrease the incidence of clotting in the dialyzer. Frequent saline rinsing of the dialyzer (i.e., every 15 to 30 minutes) may be necessary to decrease clotting of dialyzer fibres. [11]

2.4.3. DIALYSATE SODIUM:

Sodium chloride is the most abundant salt in extracellular fluid that helps to determine plasma osmolality, intracellular tonicity and cell volume. Dialysate sodium should be set at 126.5 mEq/L, recognizing the importance of diffusive sodium removal to thirst and BP control. Potential unfavourable hemodynamic consequences of such a low dialysate sodium concentration can be offset by a high glucose concentration in the dialysate. A supra-physiologic dialysate glucose concentration (>1,800 mg/dL) was used to generate an

osmotic gradient for fluid removal. The combined osmolar effect of the dialysate glucose, and to a lesser extent dialysate sodium concentrations promoted hemodynamic stability during HD. [12]

2.4.4. DIALYSATE BICARBONATE:

Acid-base equilibrium is a complex and vital system whose regulation is impaired in chronic kidney disease. Metabolic acidosis, a common complication of CKD is typically due to the accumulation of sulphate,

phosphorus, and organic anions. Thus, to maintain the equilibrium pre-dialysis serum bicarbonate concentrations should be maintained at 22 mEq/L. [13]

2.4.5. DIALYSATE POTTASIUM:

The normal range for serum potassium levels is typically reported between 3.5 and 5.0 mEq/L in the general population, the optimal range of potassium concentration in patients on dialysis is higher. [14

2.4.6. DIALYSATE MAGNESIUM:

Magnesium ions is one of the main intracellular cations that plays a central part in the maintenance of normal neuroendocrine function, muscle contraction, and cell signal transmission [15] When the rate of glomerular filtration decreases, the reduction in magnesium excretion may bring about hypermagnesemia [16] thus the commonly used dialysate magnesium concentration of 0.5 (or rarely, 0.75 mmol/L) helps to maintain constant levels. [17]

2.4.7. DIALYSATE CALCIUM;

The dialysate calcium concentration for hemodialysis patients can be adjusted to manage more optimally the body’s Ca and phosphate balance, and thus improve bone metabolism as well as reduce accelerated arteriosclerosis and cardiovascular mortality. The appropriate dialysate Ca concentration allowing this balance should be prescribed to each individual patient depending on a multitude of variable factors relating to Ca load. Dialysate Ca concentration of 1.25 to 1.3 mmol/L will permit the use of vitamin D supplements and Ca-based phosphate binders in clinical

practice, with much less risk of Ca loading and resultant hypercalcemia and calcification. [18]

2.5. COMPLICATION:

2.5.1. VASCULAR ACCESS:

The lack of adequate vascular access (VA) was one of the major issues and factors contributing to hemodialysis (HD) failure.[19] The basis for performing hemodialysis is vascular access (VA), although this might cause complications and a decrease in the effectiveness of the procedure.[21]

2.5.1.1. TYPES OF VASCULAR ACCESS;

1.Arteriovenous Fistula(AVF).

2.Arteriovenous Graft(AVG).

3.Central Venous Catheter(CVC).

Patients with end-stage renal disease (ESRD) undergoing hemodialysis showed a correlation between the types of vascular access and survival (HD). Among the arteriovenous fistula (AVF), arteriovenous graft (AVG), and central venous catheter (CVC), AVF is the most preferred and recommended vascular access type because of its lower mortality and hospitalization rate. The use of CVC was linked to an increased risk of infection, which eventually led to a higher fatality rate. Therefore, we must stay focused to preventively prepare permanent vascular access in ESRD patients.[22]

An anastomosis between an artery and a vein is performed surgically to form an arteriovenous fistula. The vein passes through maturation once this anastomosis is made, and it is then used for dialysis therapy. In contrast, a polytetrafluoroethylene (PTFE) tube is anastomosed to the side of an artery and the end of a vein to form an arteriovenous graft. The PTFE material is used to insert needles for maintenance hemodialysis in individuals who have an arteriovenous graft. Finally, tunnelled hemodialysis catheters are effectively employed to administer dialysis therapy after being put into a major vein. Although various types of access for dialysis can offer the procedure successfully, they are also associated with a variety of problems that decreases the standard of the dialysis therapy. [23]

The optimal hemodialysis access would meet three requirements.

-It would be long-lasting,

- provide enough blood flow to complete the dialysis prescription,

-have a minimal incidence of related problems.

Currently no form of hemodialysis access perfectly achieves all three requirements. The native forearm arteriovenous fistula (AVF), which studies have shown to have the greatest 4-5-year patency rates and need the fewest interventions. [20]

2.5.2. VASCULAR ACCESS STENOSIS:

The most important complication that might develop with an arteriovenous access is the development of vascular access stenosis. Although vascular stenosis can occur in both grafts and fistulas, grafts are more prone to develop it. Neointimal hyperplasia is a significant factor in the development of stenosis in grafts and fistulas. The most common site for the development of stenosis is the venous anastomosis; however, thearterial anastomosis, peripheral, and central veins can also be affected. The patient may have several venous or coexisting venous and arterial lesions within the same access, which is important to observe because stenotic

lesions can occur as single or multiple lesions. [23]

80–85% of access thrombosis is caused by venous stenosis, while 1-2% is caused by arterial stenosis. The venous outflow tract's intimal and fibromuscular hyperplasia is considered to be the cause of access stenosis. Although the precise mechanisms of intimal hyperplasia are not yet known, it is believed that they include the interactions between coagulation, hemodynamic variables, and inflammation.[20] Blood flow in a dialysis access is directly decreased by this complication. It might therefore adversely affect the standard of dialysis treatment. In order to give a patient with the necessary treatments.[23]

2.5.3. THROMBOSIS OF HEMODIALYSIS ACCESS:

In both fistulas and grafts, stenosis is the major contributor to thrombosis, and the two conditions are closely related. It is known that underlying stenosis impacts more than 90% of thrombosis cases. Intervention lists frequently carry out thrombosed fistula and graft surgeries. To de-clot a thrombosed access, a variety of thrombectomy techniques have been used. Procedures for mechanical and pharmaco-mechanical thrombolysis can be carried out effectively. [23]

2.5.4.THROMBUS IN THE DIALYSIS CATHETER:

When extracorporeal catheter blood flow is insufficient to achieve adequate dialysis, late-developing catheter malfunction should be suspected. This failure can be caused by intrinsic and extrinsic thrombus formation. 300 ml/min is the basic minimum catheter blood flow rate, however 400 ml/min or more is desirable. Treatment for late catheter dysfunction should start right away and be gradual. A vigorous saline flush should be done first, and then tissue plasminogen activator should be injected (tPA).[20]

2.5.5. DIALYSIS ACCESS INFECTIONS:

A common issue that causes around 20% of access losses is dialysis access infection. Staphylococcus aureus is the most frequent infection, although empiric antibiotic therapy should also cover gram-negative pathogens including enterococci. The development of pseudoaneurysms or peri fistular hematomas (due to incorrect cannulation), use of the access by the patient for intravenous drug administration, and manipulation of the access during subsequent surgical operations are risk factors for AV access infection. Old, previously clotted AVFs and AVGs are a potential and usually ignored source of infection. Infection occurs in 5-20% of grafts in 61 AVGs, which is greater than in AVFs. Based on the results of the culture, local infections of a graft can be treated with 3 weeks of antibiotics and excision of the infected area. AVGs with severe infections should be effectively eliminated and given intravenous antibiotics as treatment. Regardless of the

severity of the infection, infected AVGs that are less than a month old should also be evacuated and given antibiotics. [20]

2.5.6. CARDIOVASCULAR ABNORMALITIES:

Patients with end-stage renal disease (ESRD) receiving haemodialysis frequently have cardiovascular disease (CVD), which is also the primary cause of mortality in these patients (HD). Many patients with maintenance HD have CVD, and death from CVD in this population is 20 times greater than in the general population. In addition to common risk factors such chronic volume overload, anaemia, inflammation, oxidative stress, chronic renal disease, mineral bone

disorder, and other components of the "uraemic milieu", this is probably caused by ventricular hypertrophy. Developing a better understanding of how these many variables affect CVD would be important for both prevention and therapy. [24]

2.5.7. BONE DISORDERS:

Over the past three decades, there have been changes in the clinical and imaging characteristics of the bone disease associated with end-stage renal failure (ESRD) and therapy. Previously, symptoms of strong and persistent secondary hyperparathyroidism (bone resorption, osteosclerosis, and metastatic calcification) and vitamin D insufficiency (rickets/ osteomalacia) predominated; however, these characteristics are now seldom seen radiographically. This has happened thanks to advances in therapeutic treatment and a better knowledge of vitamin D metabolism. However, "adynamic" bone and metastatic calcification in soft tissues continue to be concerns. As a result of the therapy (dialysis and transplantation), new problems have emerged, including amyloid deposition, noninfective sponyloarthropathy, osteonecrosis, and osteopenia/ osteoporosis.[25]

2.5.8. ANEMIA:

Patients with chronic renal disease frequently have the complication of anaemia. Patient quality of life may be impacted if untreated. Anemia in this patient population has a variety of reasons. Patients may have iron shortage when their kidney function declines, which can be exacerbated by medicines and dietary restrictions. This reduces the amount of iron that the bone marrow receives (which is the body organ responsible for the production of different blood elements). Patients with chronic kidney disease may not be able to use their own body's iron stores efficiently, therefore many patients, especially those undergoing haemodialysis, may need supplemental iron therapy, which is often administered by infusion. Patients with chronic kidney disease may require extra therapy with erythropoietin, a substance that stimulates the bone

marrow to create its own blood, if kidney function continues to deteriorate. Any patient may ultimately need to be treated with injections of erythropoietin or other comparable medications. Several iron and erythropoietin products have recently received approval for the treatment of anaemia in people with chronic kidney disease.[26]

2.5.9. NUTRITION DEFICIENCY:

Malnutrition in these individuals is attributed to modifiable non-iatrogenic causes such as dietary insufficiency as per inadequate caloric and protein intakes due to poor appetite status, low food quality, high diet monotony index, and/or psychological and socioeconomic constraints .These elements ought to be included in a thorough nutritional assessment of the risk of malnutrition .It is essential for healthcare professionals to use the place of origin of malnutrition in dialysis patients to enable tailored patient care and identify country-specific malnutrition treatment solutions [27].

2.5.10. FLUID IMBALANCE:

Fluid overload is a frequent and significant problem in the treatment of hemodialysis patients with end-stage kidney failure. Fluid overload not only results in unpleasant symptoms for dialysis patients, but it also raises the risk of hospitalisation and fatality. It is critical that we find efficient and reliable ways to assess fluid status in patients with end-stage kidney failure who are receiving hemodialysis.[28]

2.5.11. HYPOTENSION:

People with ESKD that require maintenance hemodialysis, often three times per week, experience a special hemodynamic difficulty. Ultrafiltration objectives sometimes aim for the removal of the equivalent of a complete plasma volume in an effort to attain some level of euvolemia. In this situation, maintaining adequate end-organ perfusion requires the implementation of several complicated compensatory processes. Unfortunately, this compensation frequently falls short and leads to intradialytic hypotension as a result of a variety of patient- and dialysis-related factors. The range of negative effects linked to intradialytic hypotension, including increased cardiovascular and all-cause mortality, have come to be well recognized by both physicians and patients.[29]

IDH (intradialytic hypotension) happens in 10-12% of treatments, on average. Although there are multiple criteria for IDH, nadir systolic blood pressure has the highest correlation with the final result. Although patient features may play a role in the relationship between IDH and irreversible organ damage, it is also possible that reduced organ perfusion plays a role.[30]

2.5.12. NAUSEA AND VOMITING:

It is vital to look into and prevent nausea and vomiting since they might be problematic for hemodialysis patients. Dehydration, an electrolyte and water imbalance, is one of the most serious side effects of vomiting. The objectives of hemodialysis, namely to maintain a healthy and safe concentration of serum electrolytes, are disrupted by the electrolyte and water imbalance that results from vomiting. Despite the expensive cost of treatment, nausea and vomiting make dialysis unpleasant for patients and may cause them to quit the procedure early, which would reduce its effectiveness.[31]

2.5.13. HEADACHE:

One of the most common neurological complaints experienced during hemodialysis is headache. Dialysis-related headache, as described by the International Classification of Headaches, is that happens while receiving hemodialysis and lacks any distinguishing characteristics. Within 72 hours of the completion of the hemodialysis session, it resolves spontaneously. There aren't many research looking at the clinical characteristics of dialysis headache in the literature. Although the aetiology of headaches caused by hemodialysis is unknown, a number of triggers have been found, such as stress, coffee deprivation, and variations in blood pressure, serum salt, and magnesium levels during hemodialysis sessions.[32]

2.5.14. BACK PAIN:

A typical complaint among hemodialysis patients is low back discomfort. The primary risk factors for low back discomfort include advancing age, an elevated body mass index, and smoking. In hemodialysis patients, low back discomfort is also linked to a poor quality of life in terms of health. [33]

2.5.15. CHEST DISCOMFORT AND SHORTNESS OF BREATH:

Acute breathlessness can occur in haemodialysis patients for a number of reasons, including an allergic response to the dialyser or medicine administered during dialysis, acute coronary syndrome, catheter-related infection, pneumonia, and pericardial effusion. Therefore, clinical judgement is still crucial in assessing acute breathlessness in this population.[34] :

2.5.16. UREMIC PERICARDITIS:

The fibro-elastic sac that encircles the heart is called the pericardium. It is made up of a

"potential" space that divides the visceral and parietal layers. It is typical to have 15–50 mL of fluid for lubrication inside this potential region. This fibro-elastic sac's inflammation is known as acute pericarditis. There are several different factors that can cause pericarditis, including infections, autoimmune conditions, cancer, and uremia. Patients with end-stage renal failure and those who have severe azotemia (high blood urea nitrogen, or BUN), usually above 60 mg/dL, are more likely to develop uremic pericarditis. Chest discomfort, especially in the recumbent position a pericardial rub that is frequently audible, and in severe instances, cardiac tamponade may be present are all clinical symptoms of uremic pericarditis. The ECG, which often reveals generalised ST and T-wave abnormalities, is used to diagnose uremic pericarditis. Lowering BUN by dialysis is a common method of treating this disease. [35]:

2.5.17. DIALYSIS DISEQUILIBRIUM SYNDROME:

The neurological symptoms of Dialysis Disequilibrium Syndrome (DDS) are brought on by the rapid elimination of urea during hemodialysis. It is principally caused by an osmotic gradient that quickly hemodialysis causes to form between the brain and plasma. This causes cerebral edema, which causes neurological symptoms as headaches, nausea, vomiting, cramping in the muscles, trembling, and convulsions. Patients may pass away from progressive cerebral edema in extreme circumstances. Recent developments in cell biology suggest that the pathophysiological mechanism underlying this disease is urea disequilibrium, with a lesser contribution from organic osmolytes. [36].

2.5.18. CRAMPS:

Patients undergoing hemodialysis (HD) frequently experience cramps. The patient with cramps throughout the session was diagnosed with acute limb ischemia owing to thrombosis of a common femoral artery aneurysm. A high ultrafiltration rate and volume contraction have been linked in the aetiology, but the underlying mechanism is still not fully understood (CFAA). Although fake aneurysms (or pseudoaneurysms) that develop during femoral catheterization for diagnostic and therapeutic treatments are less prevalent, actual CFAAs are incredibly rare. that cramps cannot always be blamed on HD and are not always harmless. Rarely, it may be a symptom of or a cover for severe and fatal acute leg ischemia brought on by CFAA thrombosis. Notably, CFAA thrombosis is a very uncommon condition.[37]

2.5.19. OSTEODYSTROPHY:

Patients with chronic renal failure experience four types of common bone abnormalities, collectively known as Renal osteodystrophy (ROD). These alterations can take place early on in the course of renal disease and depend on a number of variables, including the calcium-phosphorus balance, the type of renal disease, and the frequency and dosage of potentially harmful medications. ROD often increases during hemodialysis, as renal insufficiency develops, and in the early post-transplant stage in the case of kidney transplantation. Although the later administration of immunosuppressive medication and a reduction in parathyroid hormone (PTH) allowed for a partial restoration of bone stability, ROD often lasts a lifetime and is related to a high incidence of bone fractures. Because there are now several novel treatment options available, it is crucial to be informed of and have a quick diagnosis

of renal osteodystrophy. Non-absorbable medicines that bind phosphate by ion exchange may replace previously utilised phosphate binders, which include either calcium or aluminium. Additionally, the injection of recently created calcimimetic drugs, which boost the sensitivity of calcium-sensing receptors in the parathyroid gland, can effectively suppress PTH synthesis. After transplantation or in individuals with high-turnover bone diseases, bisphosphonates may stop or reverse bone loss. [38].

2.6. TREATMENT:

2.6.1. BONE DISORDER

Decline in renal function decrease the excretion of calcium and potassium. Eldecalcitol should be administered to CKD patients, with an initial dose of 0.75 μg/day, as the drug has a more potent serum Ca- and P-elevating effect more thanconventional active vitamin D preparations.Denosumab is effective at reducing the fracture [40] Now a days, antiresorptive agents are the most commonly prescribed drugs for the prevention and therapy of osteoporosis. [41]

2.6.2. UREMIC PERICARDITIS:

Intensive dialysis therapy is more effective in 50% of patients. NSAIDs or corticosteroids can be given to patients who do not respond to adequate and intensified dialysis. Currently, aspirin (750–1000 mg every 8 h for 1–2 weeks) or indomethacin (600 mg every 8 h for 1–2 weeks) has been used as first-line agents. Since, NSAIDs can cause bleeding low-dose corticosteroids (prednisone 0.2–0.5 mg/kg/d) can be considered in patients. [42]

2.6.3. DIALYSIS DISEQUILIBRIUM SYNDROME:

Management of DDS is supportive, Maintain the airway and consider hyperventilation. inIndividuals with cerebral edema mannitol or hypertonic saline should administered to reduce the osmotic gradient between the blood and brain.[43]

2.6.4. OSTEODYSTROPHY:

According KDIGO Guideline, current treatment of bone abnormalities in CKD-MBD include the maintenance of phosphate and calcium balance, administration of vitamin D, lowering of PTH levels and targeting bone remodelling with anti-resorptive or other osteoporosis therapies. [44]

2.6.5. ACCESS INFECTION:

The Centre for Disease Control and Prevention recommends the usage of polysporin triple antibiotic ointment (bacitracin/gramicidin/polymyxin B), povidone iodine ointment Mupirocin. [(39)]Antimicrobial Dressings**,** chlorhexidine-impregnated sponge dressings may be used as an alternative to ointments for prophylactic use in short-term, non-tunnelled catheters [39]

2.6.6. IRON DEFICIENCY:

In patients with CKD with anemia, high-dose IV iron is not associated with an increased incidence of adverse clinical outcomes

Optimization of iron status, ESA dose, and dialysis parameters improves anemia in many patients; however, a proportion of patients remain refractory to ESAs and are in need of alternative therapies.[46]

2.6.7. HYPOXIA-INDUCIBLE FACTOR INHIBITION:

HIF-PH Inhibitors inactivation of HIF-PHswhich ultimately stimulate production of endogenous EPO, improveand promote iron metabolism, fiveagents of HIF-PH include Roxadustat, daprodustat, vadadustat, molidustat, and enarodustat has been approved in China and Japan for the treatment of anemia caused due to CKD [46]

2.6.8. PAIN:

Non-opioid pharmacotherapies, including topical agents such as lidocaine, acetaminophen, anticonvulsants such as gabapentin, and certain antidepressants such as serotonin–norepinephrine reuptake inhibitors and tricyclic antidepressants, should also be considered and dose-optimized before considering opioid use. [49]

2.6.9. CARDIAC COMPLICATION:

Management of chronic heart failure over the past decades has improved survivalin patient with ESRD undergoing maintence hemodialysis.Treatment chart was shown in (table 2)[47]

Table 1. Management of heart failure in patients with CKD[47]

Agents	CKD Stages 1– 3	CKD Stages 4 and 5
ACE Inhibitors	Should be used in all patients with HFrEF, with monitoring of creatinine and potassium	May be used in HFrEF, with monitoring of creatinine and potassium. Dose modification may be necessary
β-Blockers	Should be used in all patients with HFrEF	May be used in HFrEF
Mineralocorticoid receptor antagonists	Should be used in HFrEF, with careful monitoring of potassium	May be used in HFrEF, with caution and monitoring of potassium
ARBs	Should be used in all patients with HFrEF with caution	May be used in HFrEF, with monitoring of creatinine and potassium
Ivabradine	May be used in patients with HFrEF with sinus rhythm and who are stable on ***β***-blockers	Unknown effects
Angiotensin receptor and neprilysin inhibitor	May be used in patients with HFrEF instead of ACEis/ARBs	Unknown effects
Sodium-glucose cotransporter 2 inhibitor	Can be used patients with HFrEF used in or without diabetes	Unknown effects
Hydralazine and isosorbide dinitrate	Should be considered in patients with HFrEF who are intolerant to ACEis/ARBs	May be considered in patients with HFrEF who are intolerant to ACEis/ARBs

ACEi, angiotensin-converting enzyme inhibitor; HFrEF, heart failure with reduced ejection fraction; ARB, angiotensin receptor blocker.[47]

2.6.10. BLOOD PRESSURE:

Beta-blockers, angiotensin receptor blockers (or, possibly, ACE inhibitors), and dihydropyridine calcium channel blockers are all reasonable choices. For HD patients with questionable medication compliance, beta-blockers such as atenolol given three times a week following dialysis may be useful.In the majority of patients, aim for a pre-HD BP between 130/60 and159/99mm Hg and post-HD BP between 120/70 and 139/99mm Hg so long as it is tolerated. If patient experiences intradialytic hypotension, the ultimate goal is to raise BP.Encourage adherence amongst patients and dialysis staff with recording of BP measurements is mandatory**[48]**

2.6.11. ACUTE CHEST PAIN AND BREATHLESSNESS:

Tissue plasminogen activator as an intrapleural fibrinolysis agent was also shown to be safe and useful in complicated pleural effusion including retained haemothorax Despite of concern about bleeding, allergic and other adverse events while using intrapleural fibrinolytic therapy various studies have determined it was safe with a low risk of adverse.[50]

2.6.12. NUTRITIONAL SUPPORT:

For patients undergoing hemodialysis who are clinically stable, a minimum of 1.0 g/kg/d protein should be recommended Two major types of IDPN solutions exist currently a commercial admixture-based solution and a compounded admixture-based solution. In clinical practice, the use of commercial mixture remains a popular option because of the costly and time-consuming procedure to compound a customized mixture [51]

2.6.13. VASCULAR CALCIFICATION:

The discontinuation of calcium and vitamin D supplementation as well as use of low-calcium dialysate may help in the management of calciphylaxis.Bisphosphonates increase osteoprotegerin production which in turn inhibits osteoclastic bone resorption and inhibit arterial calcification. Hence, they can be beneficial even without changing calcium or phosphate levels in the treatment of calciphylaxis.[52]

2.7. DIET MODIFICATION:

Along with the management of ESRD , in maintenance hemodialysis patients it is mandatory to control the diet in their daily routine[53,54]

Table 2 – Dietary Management

PARAMETERS	DAILY INTAKE	FOODSTO CONSUME IN CASE OF INCREASED LEVELS	FOODS TO CONSUME IN CASE OF DECREASED LEVELS
Protein	1.2-1.3 g/kg/day	Fruits except dried fruits, all vegetables except peas, beans	Chicken, egg whites, fish, paneer, beans
Calcium	800-1000 mg/day	Fresh fruits, vegetables, Avoid dairy products	Dairy products, spinach, sardines
Phosphorus	800-1,000 mg/day	Green beans, egg white, apple	Nuts, whole grains, Oats.
POTASSIUM	2.7-3.1 g/day	Beans, cabbage, cauliflower	Kiwi, mangoes, oranges, banana
SODIUM	2g/day	Fresh fruits, vegetables.	Pickles, salted nuts

3. CONCLUSION

In Individuals with CKD-ESRD, hemodialysis is been used as an extracorporeal method to eliminate waste from the body. Patient on maintenance hemodialysis experience various complication that may decrease the effectiveness of hemodialysis. Continous monitoring, life style and dietary restriction improve the patient quality of life.

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