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LIFESTYLE MODIFICATION FOR PREVENTING THE PROGRESSION OF CKD

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ABSTRACT

Chronic kidney disease (CKD) is a progressive and debilitating condition affecting more than 10% of the global population. This article highlights the lifestyle modifications, optimal control of hypertension (HTN) and diabetes mellitus (DM), and pharmacological therapies play critical roles in slowing CKD progression. Regular walking, weight reduction, adherence to a low-protein diet (LPD), the alternate Mediterranean (aMed) diet, and the Alternative Healthy Eating Index (AHEI)-2010 are associated with delayed disease progression. In contrast, smoking and binge alcohol consumption significantly increase the risk of CKD worsening. In diabetic CKD persistent hyperglycemia, altered lipid metabolism, chronic low-grade inflammation, over activation of the renin-angiotensin-aldosterone system (RAAS), and overhydration contribute to accelerated renal decline. The Kidney Disease: Improving Global Outcomes (KDIGO) guidelines recommend maintaining blood pressure below 140/90mmHg in patients without albuminuria and below 130/80mmHg in those with albuminuria. Current medical therapies target fibrosis, inflammation and epigenetic alterations, with RAAS inhibitors, sodium-glucose contransporter-2 (SGLT2) inhibitors, pentoxifylline, finerenone and emerging endothelin receptor antagonists demonstrating promising reno protective effects.

KEYWORDS: CKD, Dietary modification, physical activity and weight loss, habit and safety modification.

INTRODUCTION

One of the leading causes of death globally is chronic kidney disease (CKD), a deadly progressive illness.[1,2] Chronic kidney disease (CKD) is a major cause of morbidity and mortality, with a prevalence of 9.1% to 13.4% worldwide.[3,4] CKD is the twelfth most common cause of death globally, with an estimated 1.2 million fatalities from CKD and 1.4 million deaths from CKD-related cardiovascular disease.[5]

In the past five years, the incidence of kidney illnesses has increased in our nation. In India's general population, the prevalence is 0.78%, and the most common cause of end-stage renal disease is diabetic kidney disease (41%). Hypertension (22%), glomerulus nephritis (16%), ischemic nephropathy (5.4%), drugs, particularly NSAID-induced (4.5%), obstructive uropathy (2.7%), miscellaneous (2.7%), and unknown etiology (1.4%) comprised the spectrum of additional etiological variables. Diabetes and high blood pressure are the most frequent causes of renal parenchymal disease. In addition, medications, viruses, toxic bacteria, kidney stones, genetics, polycystic kidney disease, and autoimmune all have a significant role.[6]

Two significant risk factors for chronic kidney disease (CKD) are diabetes mellitus (DM) and hypertension (HTN). Nephrotoxins, kidney stones, systemic infections, and environmental factors are additional risk factors.[7] Immunologic and genetic disorders, tissue hypoxia and reduced perfusion, medications, elevated glucose and other substances are the primary factors involved in the pathophysiology of kidney injury.[8] Patients with chronic kidney disease (CKD) may exhibit a range of symptoms, such as itching, anxiety, depression, pain, and generalized weakness.[9] When one or more of the following criteria are met for longer than three months, CKD is diagnosed:

1) Renal structural or functional abnormalities,
2) GFR (glomerular filtration rate) of less than 60 mL/min/1.73 m^2 and
3) Albuminuria of at least thirty milligrams daily.[10]

According to the GFR (TABLE:1), there are five stages of CKD.[10]

Table 1: CKD Stage.

Stages	Description	eGFR
1	Possible kidney damage (eg; protein in the urine) With normal kidney function	90 or above
2	Kidney damage with mild loss of kidney function	60 to 89
3a	Mild to moderate loss of kidney Function	45 to 59
3b	Moderate to severe loss of Kidney function	30 to 44
4	Severe loss of kidney function	15 to 29
5	Kidney failure	Less than 15

Chronic kidney disease; GFR, [glomerular filtration rate] Health issues that can be avoided and treated early in the course of the disease can complicate chronic kidney disease (CKD). CKD management strategies include treating specific renal diseases, controlling progression, preventing and treating complications, educating and preparing patients for renal replacement therapy (RRT), and screening high-risk groups (DM, HTN, kidney transplant, and family history of kidney disease) by testing for albuminuria, GFR, urine sediment, and serum creatinine (Cr).[11] In this review of the literature, we talked about how medications, controlled hypertension and diabetes, lifestyle changes, and nutritional interventions can slow the advancement of chronic kidney disease.

RISK FACTORS FOR CHRONIC KIDNEY DISEASE[CKD]

A) HYPERTENSION AND DIETARY SALT INTAKE

Uncontrolled high blood pressure may be a risk factor for the functional outcomes of renal surgery as well as for the progression of chronic kidney disease (CKD) to end-stage renal disease, cardiovascular disease (CVD), and death.[12,13] The hallmark of hypertension in patients with chronic kidney disease (CKD) is salt-sensitive hypertension, which is primarily caused by sodium reabsorption and volume excess. High salt consumption can exacerbate CKD.[14,15,16] This is due to the fact that it results in glomerular hyperfiltration and volume expansion, which eventually cause glomerular hypertension and focal glomerulosclerosis.

Patients lose nephrons more quickly as a result. Salt also has an impact on TGF beta production, an inflammatory cytokine that encourages myocardial and renal fibrosis. Extracellular volume rises as GFR falls in CKD patients. Put differently, extracellular volume and GFR are inversely correlated. Using three different salt dosages low salt (<125 mMol/day), medium salt (125–250 mMol/day), and high salt (>250 mMol/day) a post hoc analysis of the REIN 1 and 2 trials[17] examined the impact of salt on proteinuria CKD patients.

The progression of nephropathy, a critical outcome, was found to be significantly impacted by salt intake. Although dietary salt consumption is a modifiable risk factor, it can accelerate the development of nephropathy. A double-blind, placebo-controlled, randomized cross-over trial served as the foundation for this theory.[18] In patients with stages 3–4 CKD. examined how dietary sodium intake affected blood pressure, proteinuria, arterial rigidity, and extracellular volume.[18]

According to their research, lowering salt consumption considerably decreased extracellular volume, albuminuria, proteinuria, and 24-hour ambulatory blood pressure.[18] Compared to patients without CKD, these effects were even more noticeable. These findings highlight the possible advantages of non-pharmacological treatments, like limiting sodium intake, on trustworthy indicators of kidney disease, like proteinuria. Similar findings were also reported in proteinuria patients without diabetes[19], where the impact of sodium restriction (<90 mMol/day) on proteinuria was similar to that of Losartan 100 mg in the same patients.

There are several reasons why consuming a lot of salt can be detrimental to the kidneys. By activating the RAAS, a mechanism that reduces the effects of RAAS blockers on proteinuria, a high salt intake raises arterial pressure and proteinuria. Additionally, consuming a lot of salt raises reactive oxygen species, glomerular capillary pressure, local tissue inflammation, and endothelial dysfunction. Each of these processes worsens kidney function and increases proteinuria. Given this viewpoint and the fact that microalbuminuria is an early biomarker of renal damage, it is crucial to remember that in normotensive diabetic patients, the use of medications that act on the RAS system is appropriate during the microalbuminuria phase in order to prevent further clinical sequelae that lead to chronic kidney disease.[20]

It is also noteworthy to hypothesize that the Mediterranean diet has positive effects on blood pressure, diabetes, obesity and cardiovascular disease potentially leading to positive effects on the progression of chronic kidney disease (CKD), even though there are no specifically designed studies focused on this particular topic.[21] Additionally, the Mediterranean diet has benefits unique to chronic kidney disease (CKD), such as reduced inflammation, improved microbiota, and a lower dietary acid load.

Lowering blood levels of phosphorus and potassium can be achieved by choosing a diet richer in fruits, vegetables and whole grains and moderate in animal products. Limiting consumption of highly processed foods is associated with lower sodium, potassium, and phosphorus levels. Patients with chronic kidney disease (CKD) are advised to follow the Mediterranean diet pattern, with modifications based on their individual stage.[22]

It is still unclear exactly how hypertension accelerates the development of chronic kidney disease (CKD). Nonetheless, it is generally acknowledged that controlling hypertension is essential to preventing or delaying the development of CKD. Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) are two medications that can be used to treat hypertension in addition to restricting salt intake and maintaining a balanced Mediterranean diet.[23] These medications are well known for their ability to successfully lowering the blood pressure and reducing proteinuria.

There is little evidence that patients with advanced chronic kidney disease (CKD) benefit from the use of RAS inhibitors and current guidelines do not offer specific recommendations regarding the continuation or discontinuation of ACE inhibitors or angiotensin-receptor blockers for advanced CKD. RAS inhibitor discontinuation was not linked to a significant between-group difference in the long-term rate of eGFR decline in a multicenter trial where patients with advanced and progressive CKD were randomly assigned to either stop or continue therapy with RAS inhibitors.[24]

Therefore, the results of this trial support the theory that RAS inhibitors may not be as beneficial for patients with advanced and progressive CKD, even though their use has been shown to slow the decline in eGFR in patients with mild or moderate CKD. A class of medications known as SGLT2 inhibitors lowers blood sugar levels by stopping the kidneys from reabsorbing glucose. In patients with both CKD and type 2 diabetes, SGLT2 inhibitors slow the progression of CKD and lower the risk of heart failure and death. Furthermore, these inhibitors show protective qualities in CKD patients who are not diabetics.[25]

Reducing and maintaining a low salt intake is therefore crucial to maximizing the positive impact of ACE inhibition on CKD progression, since salt intake is a strong and independent predictor of cardiovascular and renal events in CKD patients. Sodium restriction should be prioritized in the management of patients with CKD to lower the risk of progression, even though additional research with longer intervention times and larger sample sizes is required to confirm these benefits. Theoretically, CKD patients' autonomy, clinical results and hypertension control could all be enhanced by a self-management strategy for salt intake.

In fact, the severity of the problem with excessive salt consumption in CKD patients is comparable to that of blood glucose regulation in diabetics. In order to achieve this goal, a multicenter study was conducted in which patients with hypertensive chronic kidney disease (CKD) self-monitored their salt intake, but the results were not entirely conclusive.[26] From a clinical standpoint, the primary issue with hypertension in CKD is that it is frequently challenging to treat, in addition to the mechanisms involved. Approximately 25% of patients with chronic kidney disease (CKD) still have uncontrolled hypertension despite being treated with three different anti-hypertensive medications, including a diuretic.

This group of patients with CKD and resistant hypertension is notable for having a larger expansion of extracellular volume and a higher risk of cardiovascular and renal events.[27] Nocturnal hypertension is the most significant component of hypertension in CKD. It is defined as either non dipping status or blood pressure (BP) above the target of 120/70 mmHg at night. With a combined prevalence of up to 60%, these fundamental alterations in the circadian blood pressure profile are more strongly linked to poor cardiorenal outcomes.[28,29] Patients with resistant hypertension have blood pressure levels that are 9% higher at night than those with other types of hypertensions, despite the fact that daytime blood pressure varies by only 4%.[30]

In patients with CKD, recent analyses found a relationship between night time blood pressure and 24hour urine sodium excretion.[31] Sleep apnea, which is primarily brought on by volume overload, is one of the major risk factors for nocturnal hypertension in CKD patients.[32,33] However, the current review does not address this important and frequently disregarded risk factor.

B) DIABETES MELLITUS

In both developed and developing nations, diabetes mellitus (DM) is the primary cause of CKD and ESRD.[34] According to the Turkish Society of Nephrology's registry, 37.3% of Turkish hemodialysis patients have diabetes.[35] Diabetic nephropathy affects half of newly diagnosed ESRD patients in the US, according to USRDS data.[36]

Reactive oxygen species, advanced glycosylation end products and hyperfiltration injury are some of the mechanisms that cause kidney disease in diabetics. The pathologic alterations linked to diabetic nephropathy are caused at the molecular level by a variety of cytokines, growth factors, and hormones, including angiotensin II and transforming growth factor-beta.[36]

Proteinuria is present in 8% of newly diagnosed type 2 diabetes patients. The 20-year risk of diabetic nephropathy is 41% in people who do not have proteinuria at first. The 10-year risk of progressive CKD is 11% after proteinuria first appears. As a result, 10% of people with type 2 diabetes will gradually lose their renal function and about half of them will develop nephropathy.[36]

C) NSAIDS IN CKD

Prostaglandins and the kidneys

NSAIDs offer their anti-inflammatory, analgesic, and antipyretic effects by blocking cyclooxygenase COX

enzymes. Arachidonic acid is converted by COX enzymes. released from the cell membrane to different eicosanoids, specifically prostaglandins and thromboxane. These fatty acids Derivatives have autocrine and paracrine effects locally. Mainly as modulators of the consequences of systemic hormones.[37]

COX-1 and COX-2, two COX isoforms, have distinct rate but overlapping roles. The expression of COX-1 is constitutively positive in numerous tissues and preserves baseline physiological functions, such as preserving kidney perfusion and function, control over platelet aggregation, and defense of the stomach mucosa.[38] COX-2 expression is altered by growth factors, cytokines and additional outside cues and is elevated in reaction to inflammation.[38] Despite the fact that Variations in the regulation of their genes result in more constitutive COX-1 expression and COX2 inducible expression in COX-2 is constitutively expressed in numerous tissues including the kidneys.[37–39]

Increased prostaglandin is mostly caused by COX-2 production in situations where augmentation is necessary of renal blood flow (RBF), including when there is a decrease in decreased GFR and effective circulating volume (ECV).[37] The locations of prostaglandin and COX-1 and COX-2 expression Figure 1 depicts action in human kidneys.[40] COX-2 is elevated in animal kidneys in reaction to volume contraction and angiotensin II levels in the blood, which rise prostaglandin production, which results in regional adaptation.[41–42] While there are variations in COX-2 localization between the strategic locations of this enzyme in humans and animals. And its elevation during physiological stress in both. According to models, COX-2 is essential for adaptive renoprotective actions. Therefore, NSAID inhibition of the nephrotoxicity of this is probably mostly caused by COX-2 class of drugs.[37–43]

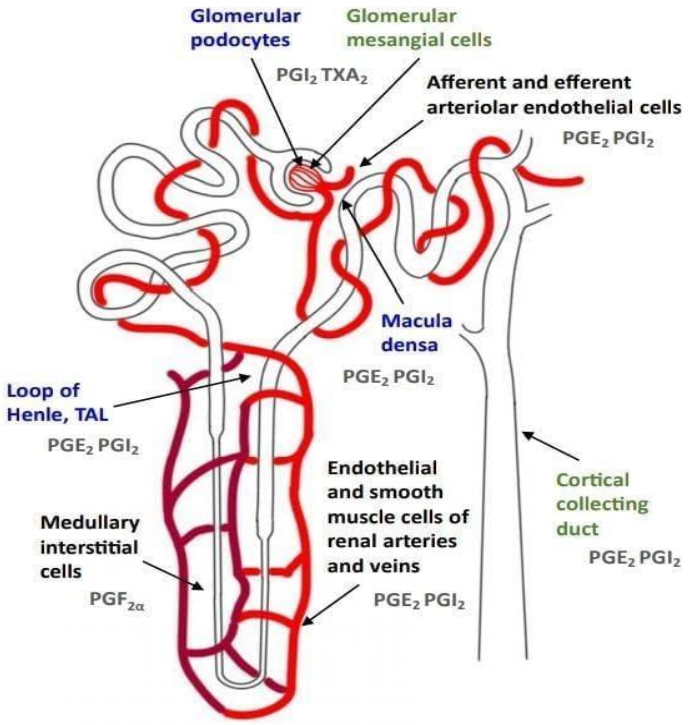


Figure 1: Cyclooxygenase (COX) iso-expression location develops in the kidney and the main prostaglandins (PGs) generated. Green text indicates areas where COX-1 expressed; COX-2 is blue; overlapping areas are black. expression of COX-1 and COX-2. Abbreviations: TAL, thick ascending limb; thromboxane A2 (TXA2).[40]

NSAIDs are viewed in the medical community as detrimental to CKD patients. Clinical recommendations currently advise against using NSAIDs for extended periods of time. Utilization in CKD with GFR > 30 mL/min/1.73 m^2 and com-Complete avoidance when GFR is less than 30 mL/min/1.73 m2.[44–45] A Patient concerns about NSAID-related nephrotoxicity with CKD emerged from the use of combination analgesics (a NSAID combination with phenacetin, paracetamol, or sali-cylamide and either codeine or caffeine) and is based physiologically and Logically, the absence of renal reserve in the "CKD" kidney.[46] Studies on epidemiology have indicated a rise in nephrotoxicity risk in patients using noncombination NSAIDs with CKD. Nevertheless, epidemiologic research looking at the NSAID-related risk for CKD progression are responsible for contrasting distinct populations that are characterized by their NSAID usage. Understanding of NSAID nephro-toxicity causes drug use to change, which results in selection and confounding prejudices.[47]

D) GLOMERULONEPHRITIS CONTRIBUTING TO CKD

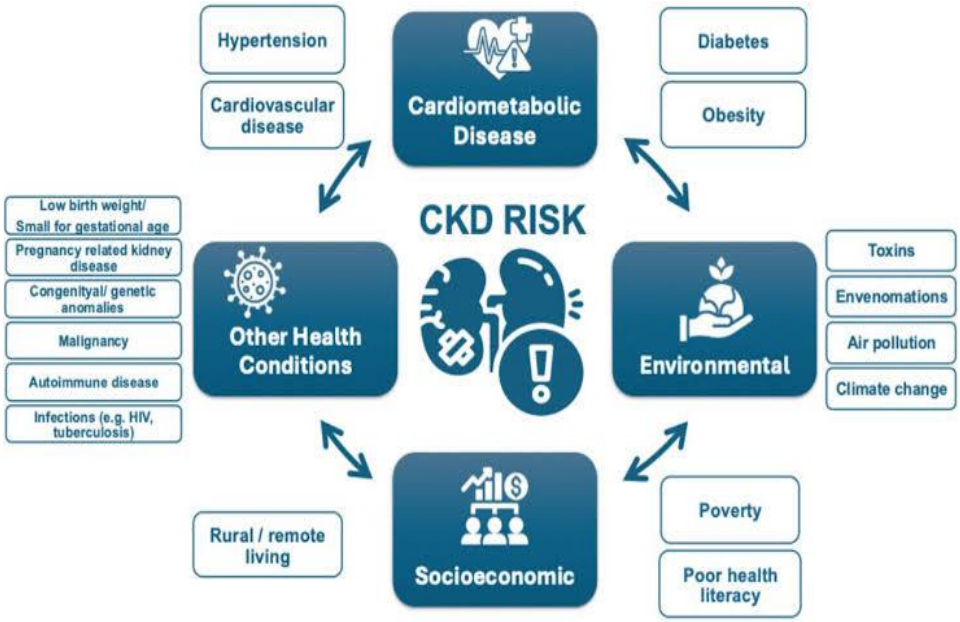
Additionally, glomerular disorders are a significant contributor to expanding CKD epidemic.[48] But the frequency and the prevalence of glomerulopathies varies greatly throughout the entire world. Like CKD, there aren't many national registries for mass documentation of glomerular diseases. The majority of the Single-center or multi-center studies are conducted on glomerular diseases. Therefore, precise information regarding the frequency and prevalence of Glomerular diseases are either nonexistent or in their early stages in the majority of nations. Additionally, the frequency of this has changed. Causes of CKD during the previous two to three decades. Long-term glomerulonephritis (GN) was a major contributing factor for dialysis patients in the early 1990s.[49–50]

Additionally, Chugh from India discovered chronic according to their study, GN was the primary cause of ESRD, followed by Chronic tubulointerstitial nephritis(14%) and diabetic nephropathy (14%) and nephrosclerosis (13%).[51] Another Indian study discovered chronic GN to be the primary reason for ESRD in adult patients (49.4%), followed by diabetic kidney disease (28.4%).[52] Additionally, Naicker is from South Africa. stated that the primary cause of chronicrenal failure is chronic GN.(CRF) and ESRD (25%), with hypertension (20%) coming in second.[53] On the other hand, information from the US Renal Data System (USRDS)demonstrates that the primary cause of ESRD is diabetes (42.9%), followed by glomerulonephritis (9.9%) and hypertension (26.4%) in the US populace.[54] In a study conducted in the middle of 2000, the glomerular disease made up just 9.9% of Pakistan's CRF. In this subsequent investigation the most CRF cases, the cause was unknown.

Very few of the diagnostic criteria have been mentioned in the aforementioned studies. of glomerular diseases, and the numbers could be regarded as at best as merely approximations of the actual image. According to Rizvi et al. Misclassification could be the cause of the lower incidence of GN. of long-term GN cases as unidentified hypertensive nephrosclerosis causes. Only biopsy-verified cases of GN or where chronic GN was strongly suggested by the history.[48]

Each and every m any glomerulopathies can cause chronic kidney disease (CKD). However, the rate of the percentage of patients who develop CKD varies, as does its progression. a great deal. It is essential to recognize glomerular early-stage illnesses. GN cases should also be referred. To specialized care in order to receive the best care possible.[55,56]

OVER VIEW OF CKD RISK



ROLE OF PROTEINS IN CKD

Consuming a lot of protein raises glomerular pressure, which is over filtration. Glomerular damage may result from this. Makes CKD worse. Consequently, a diet low in protein of 0.6 for the treatment of CKD, 0.8 g/kg/day is advised. We're examined the impact of low protein consumption on development CKD as well. This review explains why consuming a lot of protein may damage the kidneys, prolong the life of the kidneys, and why and how dietary protein restriction should be taken into account it is effective in managing chronic kidney disease.[57]

Increased renal blood flow results from a higher protein in take. And increasing intra glomerular pressure, which results in increased rate of glomerular filtration (GFR). "Glomerular hyperfiltration, which is brought on by a diet high in protein along with the ensuing rise in urine

albumin excretion, which could have unfavorable effects on the kidney and other organs.[58] Damage to the glomerulus caused by an increase in renal blood flow and intra glomerular pressure can result in sclerosis and ongoing glomerular damage.

A low in protein diet (LPD) lowers nitrogen waste products and reduces the workload on the kidneys by lowering the glomerular pressure, which could protect the kidneys, especially in people with decreased nephron capital and renal function. It has advantageous metabolic effects that can sustain renal health and uremic symptom management. However, Dietary Changes in modification of diet in renal disease (MDRD) study, the largest renal disease-controlled trial of dietary protein management in patients with chronic kidney disease (CKD) have not been able to demonstrate that LPD is unquestionably successful in reducing CKD progress.[59]

It was introduced to help shape the idea. that diet has a small but important impact on the management of CKD. Nevertheless, the MDRD study's secondary analysis demonstrated that for every 0.2 g/kg/day decrease, the drop-in Over time, GFR improved somewhat, to 1.15 mL/minute/1.73 square meters/year, as well as the chance of dying from renal failure is cut in half. Additionally, LPD helps to improve the metabolic acidosis in CKD. Acid is created when proteins are metabolized and Amino acids that contain sulfur and pre dialysis serum patients with lower bicarbonate concentrations increased intake of protein.

Acid is typically retained with reduced renal function leading to long-term metabolic Acidosis hinders the metabolism of proteins and increases muscle wasting and catabolism, as well as worsening of the uremic manifestations.[60] Many foods derived from plants and animals are abundant sources of protein. Fish, poultry, meat, and eggs are examples of animal proteins and dairy goods. Vegetable protein-containing foods include: grains, legumes, nuts, seeds, beans, and pea beans. Food in cans, boxes, jars, or bags are processed and typically contain a lot of salt and sugar and ought to be avoided. High consumption of Vegetable protein lowers CKD risk, but high Consuming animal protein raises the chance of developing CKD.

As a result, we require a harmonious blend of both. A cooked portion of meat, fish, or chicken is roughly two to three ounces, or roughly the same size as a deck of cards. For dairy products, a half-cup of yogurt or milk, or a cheese slice. About half a cup of cooked beans and nuts are served. Serve a quarter of a cup. Rice is one serving, and bread is a slice half a cup.[61]

ROLE OF LIPIDS IN CKD

The term "hyperlipidemia" refers to an excessively high level of triglycerides and/or cholesterol in the blood or they are transported by lipoproteins.[62] A 10% decrease in 0.6 mMol/L of cholesterol is equivalent to a 54% decrease in coronary heart disease (CHD) at age 40 age, but at 80 years old, it falls to 19%.[63] An expanding collection of research indicates that high cholesterol may be a factor in renal decline in function. Considering that cardiac events are the most cause of death in patients with renal disease, it is essential to maintain lipid levels under control. The proof that it could speed up the kidney failure's progression makes the case for therapy, including medication and diet.

Explaining mechanisms that connect an aberrant lipid profile to the renal failure progression involves mesangial cell stimulation of proliferation, expression of cytokines, oxidative insulin resistance and stress.[64] Regardless of conventional risk factors, the elevated risk of heart disease is linked to mild renal impairment and is most prevalent in individuals with ESRD who need dialysis. Individuals with Lipid patterns are distinctive in advanced CKD or ESRD of low HDL and hypertriglyceridemia levels of cholesterol, but LDL (low density lipoprotein) cholesterol levels are typical. The goal for healthy total cholesterol is LDL < 70 mg/dL and <200 mg/dL in CKD with diabetes and less than 100 mg/dL in CKD without diabetes are condemned. The ideal triglyceride level is less than 150 mg/dL.[65]

Foods that have a high concentration of monounsaturated fatty acids and omega-3 fatty acids and polyunsaturated fatty acids, like two fish portions or salmon twice a week, canola, peanuts, safflower, olives, Sunflower oil and soybeans are suggested. Foods comprising trans fats, margarines, and hydrogenated oils found in a lot of commercial snack foods ought to be limited.[66]

IMPORTANCE OF LIFESTYLE MODIFICATIONS

Changes in lifestyle are essential for controlling and preventing CKD because of their substantial impact on both the beginning and course of the illness. In contrast to non- Risk factors that can be changed, like age, genetics, and lifestyle elements like food, exercise, alcohol, and smoking Stress and consumption can be changed, providing a workable strategy to lower the risk of CKD and slowing its advancement.

A nutritious diet, especially one that emphasizes plant-based foods and lowers sodium intake foods, can reduce the strain on the kidneys and one of the main causes of kidney disease is blood pressure. Likewise, consistent exercise enhances cardiovascular health, controls body weight, and lowers the possibility of getting diseases like hypertension and diabetes, both of which are significant causes of chronic kidney disease.[67]

Both smoking and binge drinking are highly connected to kidney damage because they worsen oxidative stress and inflammation, hastening the course of a disease. Giving up smoking and lowering Drinking alcohol is essential for maintaining kidney function and improving general health.

Furthermore, overseeing Stress is necessary because long-term stress can increase blood pressure and raise the possibility of problems with the kidneys. Stress-reduction strategies like mindfulness and therapy, have been demonstrated to enhance mental and physical health outcomes in individuals with chronic kidney disease. Through adopting by making these lifestyle changes, people can greatly lower their chance of getting CKD or delay its advancement, enhancing their standard of living and lowering the possibility of problems. Additionally, these adjustments can reduce the financial strain on healthcare systems by lowering the requirement for additional intensive therapies such as kidney or dialysis transplantation.[68] Finally, lifestyle changes provide a comprehensive, practical, and accessible method for CKD management, enabling people to take charge of their health by making proactive behavioral adjustments.

LIFE STYLE MODIFCATION

A) WEIGHT LOSS

Central (waist circumference) and general obesity have a graded causal relationship with chronic kidney disease (CKD). incidence; a retrospective cohort study found a strong correlation between elevated body mass index (BMI) beginning at 25.0 kg/m2 and the likelihood of developing end-stage renal disease (ESRD).[69–70] A comprehensive analysis by Bolignano and Zoccali found that albuminuria (albumin-creatinine) was considerably reduced by weight loss. Ratio of ≥30 mg/g (3 mg/mmol)) and proteinuria, and it was primarily visible in weight loss after bariatric surgery in DM patients.[71] But inadvertent weight loss, especially in cases of malnutrition and muscle wasting, are linked to worse outcomes following kidney transplantation and a higher chance of passing away in ESRD patients.[72–73]

B) DIET

The Kidney Disease Outcomes Quality Initiative (KDOQI) of the National Kidney Foundation determined that following a low-protein diet (LPD) can improve the quality of life and slow the progression of end-stage renal disease (ESRD). CKD individuals.[74] According to a study by Hahn et al., there was a notable decrease in serum urea and serum uric acid in individuals following a very low-protein diet supplemented with keto-acid analogues (KA) (VLPD) (0.28-0.43 g/kg/body weight/day) after 15 months in contrast to the traditional LPD.[75] However, LPD has an unclear impact on serum levels when compared to a typical protein diet, creatinine.[76]

The alternate Mediterranean (Amed) diet is one of the other dietary interventions for CKD and the 2010 Alternative Healthy Eating Index (AHEI).[77] The Study of the Chronic Renal Insufficiency Cohort (CRIC) revealed that individuals who adhered to the Amed diet and AHEI-2010 the most had a decreased risk of CKD progression and reduces all-cause mortality, with Amed diets showing the best outcomes.[78] A salt-free diet (<2.3 g/day) reduces ESRD and the doubling of serum Cr from baseline. But the impact of a low- salt diet on proteinuria, all-cause mortality, and the rate of decrease in estimated glomerular filtration rate (eGFR) more research is necessary on mortality.[79]

C) PHYSICAL ACTIVITY

According to the Physical Activity Guidelines for Americans, adults should engage in at least 150 minutes of for significant benefits, engage in 75 minutes a week of vigorous exercise or moderate-intensity physical activity.

The risk of increased proteinuria and renal function made physical activity in CKD controversial. Impairment. Conversely, Villane go et al.'s meta-analysis discovered that low-intensity exercise enhances life quality without affecting renal function.[80] However, this research and another meta- Nakamura et al.'s analysis revealed no discernible variation in estimated GFR (eGFR). And proteinuria or the advancement of chronic kidney disease in the physically active exercise group and the control group without engaging in any physical activity.[80-81]

Patients with CKD who were not receiving dialysis made up both the exercise and control groups. Within Robinson-Cohen et al. discovered in another prospective cohort study of patients with stage 3 to4 CKD that there was an estimated 0.5% annual slower decline in eGFR for every 60-minute increase in weekly physical activity.

Additionally, the same study found that patients with no GFR declined more annually. ecreational activity in comparison to patients who engaged in physical activity as advised by guidelines; there was a 2.8% decrease in yearly eGFR between individuals who engage in no recreational physical activity and those who engage in ≥150minute/week of recreational physical activity after controlling for sociodemographic factors and common illnesses.[82] Patients with stage 3-5 CKD (mean age: 70 years) participated in an observational study by Chen et al. Walking was the most popular type of exercise, according to the majority of patients.

According to the study, walking was linked to decreased rates of RRT and overall mortality regardless of age, renal function, and the patients' comorbidities.[83]

These studies' conclusions imply that in older adults, physical activity may help reduce the risk of end-stage renal disease (ESRD) and slow the rate at which kidney function declines; Walking and overall mortality are associated in a dose-dependent manner (sub distribution hazard ratio).(SHR): 0.72, p=0.002 for patients who walked three to four times a week; 0.83, p=0.04 for patients who walked one to two times per week. Times /week; and 0.41, p<0.001 for individuals who walk more than seven times per week). Additionally, there is a dose-dependent decrease Walking during renal replacement therapy (stress hyperglycemia ratio, SHR: 0.81 for patients who walked 1-2 times per week and 0.56 for

patients who went for five to six walks a week). However, additional research is necessary to assess the minimal level of exercise necessary to slow the progression of CKD.[84]

D) STRESS MANAGEMENT

Stress on a psychological level is essential to the kidney disease progression and treatment. Prolonged Stress can trigger a series of detrimental physiological consequences, such as elevated blood pressure, inflammation, and metabolic abnormalities, all of which put stress on the kidneys and aid in the onset of chronic kidney disease. When people experience stress, their bodies react. Entails the release of hormones like cortisol, which can harm kidney function when it rises over time. Additionally, stress is connected to unhealthy habits like smoking, eating too much, and not exercising, which worsen kidney disease even more. Furthermore, stress can interfere with the current kidney's management conditions, making it more difficult for patients to follow plans for treatment and changes in lifestyle. Mental well-being Disorders like depression and anxiety are frequently observed in CKD patients and are linked to poorer health results.[85]

Research has indicated that stress management through methods like meditation, mindfulness, and Kidney health can be enhanced by cognitive behavioral therapy (CBT) by lowering blood pressure and lessening the effects of stress on the body. Taking care of mental health and putting Stress-reduction techniques are crucial elements of a comprehensive strategy for managing and preventing kidney illness.

E) YOGA

Yoga is a traditional Indian lifestyle practice that incorporates dietary modification, mental discipline, and specific techniques such as breathing exercises (pranayama) and physical postures (asanas). From a postural anatomy perspective, yoga poses primarily focus on muscle strengthening and stretching, thereby enhancing blood circulation. Research indicates that yoga plays an important role in the management of diabetes and hypertension. One study reported that Shavasana (corpse pose) reduces blood pressure and decreases the need for antihypertensive medication. Another study demonstrated that yoga is effective in lowering serum cholesterol, blood pressure, blood glucose levels, and body weight.

In a narrative review, Kashinath et al. described the benefits of yoga in patients with chronic kidney disease (CKD).[86] Since hypertension and diabetes are major risk factors for CKD, yoga appears to be a safe and beneficial complementary intervention. It may help improve renal function, reduce the need for hemodialysis, and enhance the quality of life in CKD patients. Participants were encouraged to adopt yoga-based lifestyle modifications and were trained in kidney-friendly yoga techniques and asanas as part of an integrated yoga program.

(1) Body poses/Asanas

(a) Standing asanas − Tadasana and Urdhva Hastasana (mountain posture with stretched arms), Tadasana and Urdhva Hastasana (with hands bound), Padahastasana (hand–foot posture), and Ardha Kati Chakrasana (half–waist rotation).
(b) Sitting asanas − Paschimottanasana (seated forward bend), Ardha-asana (sitting half with spinal twist), and Shashankasana (hare pose).
(c) Supine asanas − Setu Bandhasana (bridge pose), Naukasana (boat pose), Dhanurasana (bow posing), Makarasana (crocodile), and Shavasana(corpse).

(2) Practical breathing techniques

(a) Pranayama, breathing in and out, breathing with alternate nostril; Nadi Shuddhi, using his left nostril; Chandra Anuloma Viloma, breathing softly; and Bhramari pranayama, inhaling through the abdomen lying down.

Yoga significantly reduced oxidative stress in a study. Stress and a rise in (superoxide antioxidant activity catalase and dismutase) in CKD patients who were undergoing hemodialysis. Research indicates that elevated sympathetic tone can accelerate the development of CKD, encourage high blood pressure, as well as harm the intended organs.[87]

There is proof that yoga lowers sympathetic tone and enhances parasympathetic tone, which lowers heart rate, Diastolic blood pressure and systolic blood pressure (SBP)(DBP) and metabolic rate. This also highlights the crucial Yoga's function in managing renal dysfunction as determined by serum creatinine and blood urea levels. Individuals who underwent a yoga training regimen with Conventional therapy demonstrated notable decreases in Serum creatinine and blood urea levels over a six-month period time frame.[88]

F) SMOKING AND ALCOHOL CONSUMPTION

Both smoking and more consumption of alcohol are important lifestyle elements that have been connected to the CKD progression. Epidemiological research has been repeatedly demonstrated that smoking speeds up kidney decline in function, worsen hypertension, and raise the potential for kidney disease to worsen. The dangerous Tobacco's chemicals cause oxidative stress and inflammation, harming the sensitive filtering system of the kidneys Additionally, smokers are more likely to develop cardiovascular conditions that put additional stress on the kidneys.

Furthermore, smoking can reduce the efficacy of drugs used to treat chronic kidney disease (CKD)management more difficult. Likewise, excessive Negative kidney function is linked to alcohol consumption results. Dehydration can result from long-term alcohol consumption. Which eventually lowers kidney function. Additionally, alcohol contributes to liver disease and

high blood pressure, which can both make kidney issues worse. According to epidemiological data, heavy drinking consumption increases the risk of renal failure and renal disease in its final stages (ESRD).[89] However, it has been demonstrated that moderate alcohol consumption to have a kidney-protective or neutral effect health in certain research. Cutting back or giving up smoking and cutting back on alcohol consumption are crucial preventive actions for those who are suffering or at risk of suffering due to CKD.

G) AYURVEDA AND KIDNEY DISORDERS

Despite the fact that Ayurveda was written before 5000 BC, Vasti (urinary bladder) narration, Vrikka(kidney), Gavini (ureter), Mutra Parseka (urethra), etc. Were discovered in Ayurvedic literature. The development of ayurvedic urine mechanisms differ from modern ones. Comprehension of contemporary physiology. Urine is produced from Ahara rasa (processed food) following the separation of Kitta (waste) and Sara (essential for the body). The fluid Kitta (waste) is divided from Purisha solid (Stoll) in the Large Intestine of Pakshyasaya and transported via mutravaha dhamani (channels that transport urine) to the(bladder) Vasti. The development of kidney disorders is described under Mutra Vaha Sroto Dushti and in ayurveda and illnesses such as ashmari, mutraghata, and mutrakrcchra, Asthila, prameha, udavarta, soma roga, and ushna vata, can be taken into account. Ayurveda states that kidney disease is due to the obstruction of minute channels associated with urinary system, such as Mutravaha Srotas, are accountable for carrying urine. for the liquid that enters and exits the kidneys. If there are obstructions in the kidneys' incoming srotas fluids, shrinkage, and the emergence of kidney diseases. According to Ayurveda, kidneys are channels of fat tissue and feels that they are composed of meda (fat tissue) and rakta (blood). Dhatus and to treat kidney disorders, one doctor must prescribe meda kapha hara to balance these two. drugs.[90]

The causes of renal disorders as outlined in Mutra Vega dharana, are the suppression of urine urge, is an Ayurvedic practice. Ativyayama (excessive exercise), Anupa mamsa (over intake of fish and fish products), Teekna ausadha (strong medication).

Tikna madya (overuse of concentrated alcohol), Druta Pratyayana (long sitting) and traveling in a vehicle with a high spread), Ksheena purusha (immunocompromised or malnourished) and abhighata (Mutra Baha srotos injury). The signs of Renal conditions include Shotha (periorbital, pedal, whole body swelling), Chardi (vomiting), Shirashoola (headache), Panduvarnatwak(uremic Tinge), Raktahrasa (anemia), and Jvara (fever) Swedabhava (Reduced Sweating), Agnimandhya (Loss of Appetite), Twacha Roukshya(Uremic Frost), and Peeda Kattayam (low back pain), Udara Peeda (Abdominal pain), Vrukkadesha Peeda (Renal angle pain), Nadi Vegata (high tension), Nadi Nyuna (weak pulse), Peedayukta, Bindurupa Mutra (Scanty urine), and Painful burning mimicturition is known as Ushna Mutra.[91] Renal disease incidence has been rising. in the last ten years in an Ayurvedic hospital. The most typical Patients have nephritis, renal calculus, and chronic kidney disease.

H) PANCHAKARMA AND CKD

In CKD, panchakarma therapy plays a specific role that Ayurvedic doctors place less emphasis on it. Vata is abnormal in Sakha (the entire skin) and Pakshyasaya (large intestine). Hypertension is still present, and resistance hypertension in the majority of CKD cases. This is clear from clinical practice experiences that Abhyanga (Body message), sarbang swedana (sudation therapy), and sarbanga swedana and patra-potali-swedana have alter the self-healing process and lower blood pressure, BUN, and creatinine in chronic kidney disease patients with illnesses. Goshura and Punnavadi Kasya Vasti Clinical evidence suggests that Punnava Vasti reduces micro albumin and enhance the removal of creatinine.[92]

Procedure for Panchakarma Administered to patients

1. Avagaha Sweda

Procedure: The patient sits in a tub of 42°C warm water enriched with therapeutic herbs for thirty to Sixty minutes.[93]

Physiology: Vasodilation is induced by warm water. Boosting blood flow and encouraging perspiration to eliminate toxins; herbal properties are absorbed via the skin.[94]

Mode of Action: The heat triggers the hormones released by the sympathetic nervous system that increase fat breakdown, metabolism, and toxin removal (ammonia, creatinine, urea). Described It mobilizes Acharya Charaka as Sagni Sweda and liquefies Doshas lodged in the body’s microchannels (Srotas).[95]

2. Gokshuradi Siddha Sneha Matra Basti

Procedure: Warmed Gokshuradi Siddha Sneha oil is administered via the rectal route in a dose of 90 ml and retained to improve absorption.[96]

Physiology: The oil permeates the rectal mucosa, providing lubrication to the intestines, which supports digestion and facilitates bowel evacuation.[97]

Mode of Action: Matra Basti helps regulate Vata dosha by promoting the elimination of flatus, feces, and urine. Once the oil reaches the large intestine (Pakwashaya), it disperses throughout the body, exerting a systemic Vata-pacifying effect. Gokshura, known for its diuretic properties, enhances bodily strength and maintains balance among all three Doshas (Tridosha), producing both local and systemic actions through the circulatory system.[98]

3. Guduchyadi Kashaya Niruha Basti

Procedure: A warm Guduchyadi herbal decoction, measuring 300 ml, is administered through the rectal route and retained for a predetermined period.[99]

Physiology: Niruha Basti acts at the primary seat of Vata dosha in the large intestine (Pakwashaya), thereby regulating Vata across the body and effectively managing various Vata-related disorders.[100]

Mode of Action: According to Acharya Sushruta, the potency (Virya) of Basti disseminates throughout the body via the Srotas (channels), producing systemic effects based on the Kedarikulya Nyaya principle. Guduchi helps maintain balance among all three Doshas, enhances immune function, and exhibits anti-inflammatory and anti-diabetic activities, thereby contributing to overall well-being.[101]

4. Shiro Abhyanga (Head Massage)
Procedure: Warm medicated oil is gently applied and massaged over the scalp and neck region for a duration of 20–30 minutes.[102]

Physiology: The therapy promotes increased blood circulation and improves lymphatic drainage in the head and neck areas, thereby supporting detoxification processes.[103]

Mode of Action: The treatment enhances lymphatic flow and influences tryptophan activity, leading to increased secretion of melatonin and serotonin. This results in relaxation, better sleep quality, mood stabilization, reduction of anxiety, and improved metabolic function.[104]

5. Shiro Pichu with Brahmi Oil
Procedure: A cotton pad soaked in warm Brahmi oil is placed over the forehead and maintained for a period of 30–60 minutes.[105]

Physiology: Brahmi oil permeates the skin layers, providing nourishment to the underlying tissues and producing a calming effect on the mind.

Mode of Action: The therapy helps relieve headaches, migraines, and mental exhaustion by promoting vasodilation, which enhances cerebral blood circulation and oxygen delivery. Additionally, the sedative properties of Brahmi reduce stress, elevate mood, and induce muscle relaxation, offering both therapeutic and psychological benefits.[106]

6. Lepam using Dashmool, Trikatu, and Shunthi
Procedure: An herbal paste prepared from Dashmool, Trikatu, and Shunthi is applied over the affected areas, retained for a predetermined duration, and then washed off.
Physiology: This external application enables direct absorption of the active herbal constituents through the skin, thereby bypassing hepatic metabolism.

Mode of Action: The formulation promotes localized perspiration, which facilitates the elimination of metabolic waste products such as urea, creatinine, and uric acid. This process helps reduce edema and alleviate pain. Dashmool exhibits anti-inflammatory, antioxidant, and analgesic properties; Trikatu helps regulate Vata and Kapha doshas while providing anti-inflammatory effects; and Shunthi acts as an effective anti-inflammatory and analgesic agent.[107]

Ayurveda based Reno medications
Ayurveda has long employed medicinal plants along with metals and minerals in the prevention and management of kidney-related disorders. Ayurvedic formulations are generally viewed as cost-effective, widely available, and comparatively safe, offering a rich source of biologically active substances useful in therapeutic applications. The Ayurvedic approach to treatment extends beyond oral medicines and also includes regulated diet, physical activity, and Panchakarma procedures as supportive therapeutic measures. Many herbal preparations used in Ayurveda contain phytoconstituents such as flavonoids, vitamins, resveratrol, anthocyanins, curcumin, and phenolic acids, which are known to exhibit antioxidant activity.

Although extensive experimental research on Panchakarma is limited, existing studies indicate its potential role in managing resistant renovascular hypertension and supporting improved creatinine elimination.[108,109] Panchakarma therapies are believed to enhance systemic cellular activity, promote relaxation of peripheral blood vessels, correct endothelial dysfunction, and reduce inflammatory mediators, thereby facilitating the body's natural healing processes.[110]

The combined use of Ayurvedic medications and Panchakarma therapies shows notable benefits in renal disorders, emphasizing the importance of promoting Ayurvedic interventions in the management of chronic kidney diseases. Evidence from traditional systems of medicine, including Chinese herbal therapy, suggests improved long-term outcomes in patients with chronic kidney disease. Ayurvedic treatments have similarly demonstrated usefulness in the management of CKD.[111,112]

Panchakarma interventions have also been found beneficial in cases of resistant renovascular hypertension. In Ayurvedic literature, drugs that support renal health and are used in disorders of the Mutravaha Srotas are categorized into groups such as Mutrasangrahaneeya, Mutravirajaneeya, Mutravirechaneeya, Ashmarighna, and Pramehagna dravyas.[113]

Ayurvedic Medicines

A) Chander Vati: Helps regulate Pitta dosha, alleviates symptoms of urinary tract infections, and promotes diuretic action.
b) GFR Powder: Assists in reducing renal inflammation and supports the body's detoxification processes.
C) Asthi Purak Vati: Supports tissue repair, lowers oxidative stress, and contributes to cardiovascular health.
D) Trivrit Avaleh: Facilitates cleansing of the system, helps control inflammation, and promotes kidney function.
E) Divya Shakti Powder: Improves digestive function, enhances metabolic activity, and aids in the elimination of toxins
F) CKD Syrup: Exhibits diuretic effects; ingredients such as Shatavari and Giloy help reduce inflammation and support tissue healing.
G) Sarvatobhadra Vati: Contributes to the management of chronic kidney disease through its anti-inflammatory and detoxifying properties.
H) Kidney Care Syrup: A polyherbal formulation designed to reduce inflammation, support renal health, and assist detoxification.
J) URI Plus: Contains antioxidant and diuretic components that improve urinary function and facilitate toxin clearance.

The integrative use of Ayurvedic formulations offers a holistic strategy for the management of chronic kidney disease by addressing both clinical symptoms and underlying doshic imbalances. This approach supports renal function and improves overall patient well-being. Although further scientific studies are required to validate these observations and optimize therapeutic protocols, this case report underscores the potential role of Ayurvedic interventions as a supportive option in CKD management, particularly in settings with limited healthcare resources.[114]

Impact of Lifestyle Modifications and Exercise on Hypertension in CKD patients

There is mounting evidence that lifestyle modifications, such as consistent exercise, greatly improve blood pressure control in people with chronic kidney disease (CKD). Studies have shown that patients with CKD achieve better blood pressure control when they follow dietary approaches such as the DASH diet.[115] Frequent exercise has also been linked to improvements in blood pressure control, endothelial function, and general cardiovascular health. However, research on exercise regimens designed especially for people with chronic kidney disease (CKD) is still scarce and in its early stages.[116,117]

Numerous studies assessing the effects of resistance and aerobic training have shown positive results in the control of hypertension in CKD patients.[118] It is thought that mechanisms like improved endothelial function, decreased vascular stiffness, and reduced inflammatory responses mediate these effects. However, differences in study design, exercise modalities, and participant characteristics highlight the need for additional research to determine the best exercise regimens for people with hypertension and chronic kidney disease.[119]

Impact of Lifestyle modification on type 2 diabetes in CKD

In addition to medication, lifestyle changes are essential for the prevention and treatment of type 2 diabetes mellitus (T2DM). Guidelines from the American Diabetes Association and other international organizations emphasize the role of dietary modification, regular physical activity, and behavioral interventions. By encouraging weight loss, enhancing insulin sensitivity, and improving glycemic control, these tactics aid in addressing metabolic abnormalities. Research suggests that long-term lifestyle modifications can improve clinical outcomes for patients with established type 2 diabetes and postpone or prevent the onset of diabetes in high-risk individuals.[120] A major part of managing type 2 diabetes mellitus (T2DM) is dietary modification. A balanced diet emphasizing whole grains, lean protein sources, healthy fats, and fiber-rich vegetables contributes to improved glycemic control and reduced insulin resistance. Calorie restriction and better food choices also help people lose weight, which is crucial for managing diabetes. Exercise is equally important because it improves cardiovascular health and muscle glucose uptake. Both aerobic and resistance exercises have been shown to reduce HbA1c levels and lower the risk of diabetes-related complications.[121] Behavioral and psychological support, such as motivational interviewing, structured goalsetting, and technology-assisted tools, is essential for adherence to lifestyle interventions, and addressing psychological and socioeconomic barriers through tailored strategies that consider individual preferences, cultural context, and resource availability is crucial to optimize clinical outcomes.[122]

CONCLUSION

A key component of preventing and delaying kidney damage in chronic kidney disease (CKD) is changing one's lifestyle. The main modifiable risk factors that contribute to the progression of chronic kidney disease (CKD) are directly addressed by interventions like stress management, regular physical activity, dietary control, weight management, and abstinence from alcohol and tobacco. While dietary control, especially lowering sodium and protein intake, reduces metabolic and hemodynamic stress on the kidneys, weight loss in overweight people lowers glomerular hyperfiltration and proteinuria. Frequent exercise enhances cardiovascular health, insulin sensitivity, and blood pressure regulation, all of which indirectly protect renal function. Stress reduction and getting enough sleep improve metabolic stability and treatment compliance, which further protects the kidneys. By lowering psychological stress, enhancing autonomic balance, and promoting blood pressure and glycemic control, yoga and mind-body techniques offer extra advantages. These methods are

beneficial supplements to traditional lifestyle interventions because they are safe, economical, and enhance general well-being. Avoiding alcohol and quitting smoking are two of the best ways to prevent kidney damage because they both cause oxidative stress, endothelial dysfunction, and worsening hypertension. Renal and cardiovascular outcomes are greatly improved by removing these risk factors. By emphasizing systemic balance, metabolic regulation, and detoxification, Ayurvedic-based renal medications and Panchakarma therapies provide a comprehensive and supportive approach. When used carefully under professional supervision, some Ayurvedic formulations may help lower inflammation and enhance quality of life. In stable or early stages of CKD, panchakarma procedures, especially gentle and customized therapies like Basti, may support metabolic balance and stress reduction. However, these therapies should be used as supplemental rather than primary treatment options because of the lack of extensive clinical evidence and possible risks in advanced disease. In terms of efficacy, lifestyle change continues to be the safest, universally applicable, and evidence-based method of preventing kidney damage in CKD. While Ayurveda-based interventions may offer supportive benefits when carefully integrated, yoga and stress management increase the efficacy of lifestyle changes. The best way to slow the progression of chronic kidney disease (CKD) and improve long-term results is through a comprehensive, patient-centered approach that combines lifestyle modification with specific complementary practices.

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