WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
Coden USA: WJPRAP Impact Factor 8.63
Volume 15, Issue 17, 590-604. Review Article ISSN 2277–7105

CHRONIC KIDNEY DISEASE: AN AYURVEDIC PERSPECTIVE THROUGH THE LENS OF KAYACHIKITSA

[1]*Dr. Arpita Gupta, [2]Dr. Subhash Babu N.

[1]Phd Scholar, Department of Kayachikitsa, Glocal College of Ayurvedic Medical Sciences and Research Centre, Sharanpur, UP.

[2]Ditinguished Professor, Department of Kayachikitsa, Glocal College of Ayurvedic Medical Sciences and Research Centre, Sharanpur, UP.

Article Received on 04 August 2026,
Article Revised on 24 August 2026,
Article Published on 01 Sept. 2026,

https://doi.org/10.5281/zenodo.22159618

***Corresponding Author**

Dr. Arpita Gupta

Phd Scholar, Department of Kayachikitsa, Glocal College of Ayurvedic Medical Sciences and Research Centre, Sharanpur, UP.

How to cite this Article: [1]Dr. Arpita Gupta, [2]Dr. Subhash Babu N. (2026). Chronic Kidney Disease: An Ayurvedic Perspective Through The Lens Of Kayachikitsa. World Journal of Pharmaceutical Research, 15(17), 590-604.

ABSTRACT

Chronic kidney disease (CKD) represents a progressive loss of renal function over time and poses significant challenges in global healthcare. In *Ayurveda*, CKD is understood as an *Upadrava* (complication) of urinary system disorders or systemic conditions such as *Prameha* (diabetes). This review explores the *Ayurvedic* perspective on CKD, focusing on its pathogenesis, prognosis, and therapeutic approaches. The disease is characterized by the vitiation of *Dosha* and their impact on *Basti* (renal system), leading to impaired filtration and systemic toxicity. Early stages are considered *Krucchrasadhya* (difficult to treat), whereas advanced stages are *Yapya* (manageable with palliation) or incurable. Management strategies in *Ayurveda* emphasize purification (*Shodhana*), pacification (*Shamana*), and rejuvenation (*Rasayana*) therapies. Detoxification procedures like *Virechana* and *Basti* therapy are employed to eliminate vitiated *Dosha* and restore systemic balance. *Rasayana* drugs such as *Punarnava*, *Gokshura*, and *Shilajit* enhance renal function, act as antioxidants, and provide nephroprotective effects. Clinical studies indicate improvements in digestion, vitality, and quality of life with these interventions. The integration of *Ayurvedic* principles, including dietary and lifestyle modifications, offers a holistic approach to CKD, addressing its systemic effects and slowing disease progression. This study underscores the potential of *Ayurveda* as a complementary and cost-effective therapeutic framework for CKD, particularly in resource-

limited settings, by reducing reliance on conventional renal replacement therapies and improving patient outcomes.

KEYWORDS: CKD, *Ayurveda, Rasayana, Shodhana*, Nephroprotective.

INTRODUCTION

Chronic kidney disease (CKD) is characterized by the presence of kidney damage or an estimated glomerular filtration rate (eGFR) of less than 60 mL/min/1.73 m², persisting for 3 months or more. CKD involves a progressive loss of kidney function, often leading to the need for renal replacement therapy, such as dialysis or transplantation.[1] Globally, CKD is a major and escalating public health issue About 673.7 million people had chronic kidney disease (CKD) in 2021, along with 19.9 million new incident cases, 1.5 million fatalities, and more than 44 million disability-adjusted life years (DALYs) lost. Over the past three decades, there have been notable increases in incidence, mortality, and DALYs, despite a minor drop in age-standardized prevalence. Specifically, within India, CKD prevalence is notably high. A systematic review and meta-analysis of community-based studies from 2011 to 2023 found a pooled CKD prevalence of 13.24% among Indians aged 15 and above. This trend has surged—from 11.12% during 2011–2017 to 16.38% between 2018–2023—highlighting a worrying rise. Higher prevalence was observed in rural areas (15.34%) than in urban regions (10.65%), with a slight male predominance (14.80% vs. 13.51%).[2]

The management of CKD in modern medicine is largely limited to controlling risk factors, slowing disease progression, and addressing complications. Pharmacological interventions such as antihypertensives, antidiabetic drugs, and renin–angiotensin system blockers provide symptomatic relief but are insufficient to prevent progressive nephron loss. Once advanced stages are reached, renal replacement therapies such as dialysis and renal transplantation become the mainstay.

Despite being lifesaving, dialysis has several negative effects, such as frequent infections, hemodynamic instability, malnourishment, a lower quality of life, and a significant financial burden. Better survival and rehabilitation are provided by renal transplantation; nevertheless, its effectiveness is limited by the scarcity of donors, high expenses, the risks associated with surgery, and the requirement for immunosuppressive medication for the remainder of one's life, which increases the risk of infections and cancer. As a result, the present strategy is more

supportive than curative, and despite these medical advances, the prevalence of CKD is still rising worldwide.

These limitations emphasize the importance of exploring complementary systems of medicine. Ayurveda, with its holistic understanding of disease, focuses on correcting the underlying *Dosha–Dushya–Srotas* imbalance. Concepts such as *Mutravaha Srotas dushti*, *Avarana of Vata*, and *Dhatukshaya* provide an alternative framework to understand the chronic and progressive nature of CKD. Therapeutic modalities including *Nidana Parivarjana*, *Shodhana* (detoxification), *Shamana* (conservative management), *Rasayana* therapy, and nephroprotective herbs like *Punarnava*, *Gokshura*, *Varuna*, and *Guduchi* hold potential in slowing progression, managing complications, and improving quality of life. Thus, an integrative approach combining evidence-based modern nephrology with *Ayurvedic* principles may provide a more sustainable and patient-centred management strategy for CKD.

AIM

To explore the *Ayurvedic* understanding and management of Chronic kidney disease (CKD) as described in classical texts, with a focus on its conceptual basis, pathogenesis, and therapeutic principles.

OBJECTIVE

- To review and analyze chronic kidney disease (CKD) through *Ayurvedic* principles, with emphasis on classical concepts, pathogenesis (*Samprapti*), and therapeutic approaches.
- To summarize *Ayurvedic* therapeutic modalities, including *Shodhana*, *Shamana*, *Rasayana*, and nephroprotective herbs, relevant to CKD management.
- To highlight the potential role of integrative approaches combining *Ayurveda* and modern nephrology in improving patient outcomes and quality of life.

MATERIAL AND METHODS

This review utilized classical *Ayurvedic* texts to analyse kidney-related concepts and management principles. Contemporary research articles from databases like PubMed and *AYUSH* portals were reviewed for evidence on *Ayurvedic* therapies in CKD. Key findings were synthesized to correlate classical wisdom with modern applications.

Understanding CKD through *Ayurveda:*

There is no direct connection between Chronic Kidney Disease (CKD)and any of the clinical entities found in the old *Ayurvedic* texts. According to *Sutrasthana Trisothiyadhyaya* and the *Charaka Samhita*, a foundational work for *Ayurvedic* medicine, not all illnesses can be given names. If the physician does not know the name of the ailment, they should never feel ashamed. After the location of the ailment and its causes are identified, the treatment should focus on treating the disease because the same *Dosha* frequently circulates to other parts of the body, creating other disorders. The ailments can be examined in terms of their locales, particular causes, and triggered *Doshas*.[3]

In *Ayurveda*, Chronic Kidney Disease (CKD) is understood within the framework of *Mutravaha Srotas* (urinary channels). The root involvement lies in *dosha* vitiation where obstruction (*Avarana*) and depletion (*Dhatukshaya*) occur simultaneously, the *Avarana* of *Vata* by *Kapha* and *Meda* impedes normal urine formation and excretion, gradually resulting in *Shotha* (edema), *Ojakshaya* (loss of vitality), and systemic deterioration, which parallel the progressive renal dysfunction observed in modern CKD.

Although chronic kidney disease (CKD) is not specifically addressed in *Ayurveda*, *Nidanapanchaka*, or the five-fold diagnostic approach, can apply Ayurvedic notions to it. An imbalance in the *Vata* and *Kapha Doshas*, as well as disruptions in other *Doshas*, are the main indicator of chronic kidney disease (CKD). All Dhatus and *Upadhatus* (tissues and sub-tissues) are involved after the initial vitiation of *Rasa* (plasma), *Rakta* (blood), *Mutra* (urine), and *Udaka* (water). *Mutravahasrotasa* (urinary channels) are impacted by the exacerbated Doshas and Dhatus as they circulate through the *Rasa* with *Vyana Vayu*, leading to *Kahavaigunya* (dysfunction). The main indicators of chronic kidney disease are thought to be the clinical manifestations of *Dosha* disruption.

Classical Ayurvedic compendia describe several conditions resembling CKD manifestations. *Charaka Samhita* explains disorders such as *Mutrakriccha* (dysuria), *Mutraghata* (urinary obstruction), and *Prameha* (metabolic disorders), which contribute to Mutravaha Srotas dushti. Similarly, *Sushruta Samhita* details *Ashmari* (urinary calculi), *Mutravruddhi*, and *Shotha*, emphasizing their chronic progression when untreated. Later commentaries like *Chakrapani's Ayurveda Dipika* (on *Charaka*) and *Dalhana's Nibandhasangraha* (on *Sushruta*) elaborate that long-standing vitiation of *Dosha* in *Mutravaha Srotas* leads to irreversible tissue damage.

Furthermore, *Astanga Hridaya* of *Vagbhata* explains that when *Mutravaha Srotas* are blocked or weakened, urine becomes altered in both quantity and quality, causing symptoms like reduced urine output, body swelling, and fatigue. These descriptions can be correlated with clinical CKD features such as oliguria, oedema, anaemia, and uremic symptoms, as highlighted in modern nephrology.

Chronic kidney disease (CKD) is not directly mentioned in Ayurveda, but Ayurvedic concepts can be applied to it through *Nidanapanchaka* (the five-fold diagnostic approach). The signs and symptoms of CKD primarily indicate an imbalance in *Vata* and *Kapha Doshas*, along with disturbances in multiple *Doshas*. Initially, there is vitiation in *Rasa* (plasma), *Rakta* (blood), *Mutra* (urine), and *Udaka* (water), followed by involvement of all *Dhatus* and *Upadhatus* (tissues and sub-tissues). The aggravated *Doshas* and Dhatus circulate through the *Rasa* with *Vyana Vayu*, affecting the *Mutravahasrotasa* (urinary channels), resulting in *Khavaigunya* (dysfunction). The clinical manifestations of *Dosha* disturbance are considered the primary signs of CKD.

Chronic kidney disease (CKD) is a highly complex condition classified as *Vyadhi Sankara.* Its signs and symptoms vary depending on the causative factors and the stage of the disease. The etiological factors contributing to CKD encompass the *Roopavastha*, *Upadrava*, and *Vyadhi Sankara* of the following conditions:

Prameha

In *Ayurveda,* the term "*Prameha*" refers to a group of disorders characterized by excessive urination (polyuria) and turbid urine. It encompasses conditions such as diabetes mellitus, obesity, and metabolic syndrome.[4] Additionally, certain conditions like nephrotic syndrome are occasionally regarded as variants within this group.[5] The pathogenesis of *Prameha* highlights the localization of vitiated *Doshas* and *Dushyas* in the *Basti*, indicating pathological changes (*Sroto-Vaigunya*) associated with this structure. During this process, *Dosha-Dushya Sammurcchana* (the interaction between vitiated *Doshas* and *Dushyas*) occurs,[6] marking the prodromal stage of the disease. A key early symptom related to renal dysfunction is glycosuria, observed as urine attracting *Pipilikas* (ants). If the condition advances, it results in the full manifestation of the disease, characterized by increased urine frequency accompanied by turbidity, indicative of polyuria with proteinuria.[7] This stage suggests nephropathy, a late complication of diabetes mellitus. Inadequate or inappropriate management at this stage can render the disease incurable.[8]

Mutraghata

Mutraghata refers to urinary retention accompanied by mild dysuria, caused by an obstruction in the urinary tract. This condition results in either a complete lack of urine excretion or minimal, difficult urination. It arises from the vitiation of *Basti* due to circulating aggravated *Doshas*.[9] Suppression of the urge to urinate in conditions like *Vatabasti*, *Mutratita*, *Mutrajathara*, and *Vatakundalika* leads to urine retention. Direct obstructions such as *Mutragranthi* and *Ashthila* also contribute to urinary retention.

In *Ushnavata*, urinary tract infections cause inflammation, resulting in both retention and dysuria. *Mutrakshaya*, characterized by oliguria due to poor hydration, can impair renal blood flow over time, causing damage to renal cells. *Vidvighata* involves faecal contamination of urine due to rectovesical fistulas. If this contamination ascends to the upper urinary tract, it may lead to infections and renal damage. *Bastikundala* resembles an atonic bladder, leading to urinary retention. In cases of complete obstruction of the urinary tract, symptoms like thirst, delirium, and breathlessness may emerge, resembling uremic syndrome.[10]

Chronic urine retention from these causes leads to urinary stasis, increasing the risk of infections and the formation of urinary calculi, which gradually harm the kidneys.

Probable *Samprapti* of Chronic kidney disease

Complicated urinary system disorders and other systemic diseases result in an imbalance of the three *Doshas* (*Tridosha*). *Acharya Sushruta* describes the process of urine formation as a pitcher immersed in water, where the *Doshas* enter the *Basti* (kidneys) in a similar manner, filling it from all sides.[11] These *doshas* impair the function of the kidneys. After digestion, the kidneys separate and differentiate urine as a metabolic by-product for elimination.[12] However, when the kidneys are diseased, they cannot properly differentiate and separate urine, leading to the retention of harmful metabolic waste products in the body, which then circulate and cause systemic harm.[13]

In modern medicine, the pathogenesis of chronic kidney disease (CKD) involves damage to renal cells due to various underlying causes, such as immune complex deposition, inflammation from specific types of glomerular nephritis, or toxin buildup in renal tubules and interstitium. The systemic harm is a complications arising from the loss of other renal functions, such as maintaining fluid and electrolyte balance, hormone regulation, and the progressive systemic inflammation that affects the vascular and nutritional systems.

Clinical features of chronic kidney disease

The 2012 Kidney Disease Improving Global Outcomes (KDIGO) CKD classification recommends specifying the cause of CKD and classifies the condition into 6 categories based on GFR (G1 to G5, with G3 split into 3a and 3b). In addition, it also includes staging based on 3 levels of albuminuria (A1, A2, and A3), with each stage of CKD subcategorized according to the urinary albumin-creatinine ratio (ACR; mg/g or mg/ mmol) in an early morning "spot" urine sample.[22]

The 6 CKD categories, known as stages 1 through 5, are described below (stage 3 is separated into 3a and 3b):

G1: GFR 90 mL/min/1.73 m2 and above with evidence of kidney disease, such as haematuria or proteinuria

G2: GFR 60 to 89 mL/min/1.73 m2

G3a: GFR 45 to 59 mL/min/1.73 m2

G3b: GFR 30 to 44 mL/min/1.73 m2

G4: GFR 15 to 29 mL/min/1.73 m2

G5: GFR less than 15 mL/min/1.73 m2 or treatment by dialysis

The 3 levels of albuminuria include an ACR

A1: ACR less than 30 mg/g (<3.4 mg/mmol)

A2: ACR 30 to 299 mg/g (3.4-34 mg/mmol)

A3: ACR greater than 300 mg/g (>34 mg/mmol)

Grades 1 and 2 of chronic kidney disease (CKD) typically do not present with noticeable symptoms related to the decline in glomerular filtration rate (GFR). However, symptoms may arise from the underlying kidney disease or other systemic conditions. As GFR declines to grade 3 or 4, the clinical and laboratory complications of CKD become more apparent. CKD impacts nearly all organ systems, but the most common complications include anaemia, leading to easy fatigue, decreased appetite, and progressive malnutrition. There are also disruptions in calcium, phosphorus, and the regulation of minerals like calcitriol and parathyroid hormone (PTH), as well as abnormalities in sodium, potassium, water balance, and acid-base homeostasis. Since CKD represents a complex syndrome with contributions from various diseases, the signs of renal failure often indicate a reduced life expectancy.

Progression of Chronic Kidney Disease (CKD)[12,13,14,15,16,17]

Characteristics: Decreased GFR, renal parenchymal disease, polycystic disease, glomerulonephritis, parenchymal and vascular diseases.

Symptoms: Generally asymptomatic with well-preserved GFR. Symptoms may arise from underlying diseases such as edema in nephritic syndrome or hypertension in polycystic kidney disease and glomerulonephritis.

Stages 3 & 4 CKD

Characteristics: Further decline in GFR, anaemia, calcium and phosphorus imbalances, abnormal mineral-regulating hormones (e.g., calcitriol, PTH), and disruptions in sodium, potassium, water, and acid-base homeostasis.

Symptoms: Easy fatigability, decreased appetite, malnutrition, and more evident complications in virtually all organ systems.

Stage 5 CKD

Characteristics: Severe decline in GFR leading to toxin accumulation, marked disturbance in daily activities, well-being, nutritional status, and water and electrolyte homeostasis.

Symptoms: Uremic syndrome, which is fatal without renal replacement therapy (dialysis or transplantation).

Etiology

Susceptibility Factors

- Advanced age
- Low income or education levels
- Racial or ethnic minority status
- Reduced kidney mass
- Low birth weight
- Family history of CKD are at higher risk

Initiation Factors

- Diabetes Mellitus
- Hypertension
- Glomerulonephritis

Risk Factors

- Age >65 years old
- Obesity
- Smoking
- Cardiovascular disease (CVD)
- Metabolic syndrome
- Hyperlipidemia
- Use of nephrotoxic drugs, long-term use of proton-pump inhibitors (PPIs), or analgesics.
- Family history of CKD or hereditary kidney disease.
- Gout
- Multisystem diseases with potential kidney involvement, e.g., Systemic Lupus Erythematosus (SLE).
- Structural renal tract disease, renal calculi, or prostatic hypertrophy.
- Opportunistic (incidental) detection of Hematuria or Proteinuria.

Clinical Features

Uremic Symptoms: Symptoms such as fatigue, weakness, shortness of breath, mental confusion, nausea, vomiting, bleeding, and anorexia are generally absent in stages 1 and 2, minimal during stages 3 and 4, and common in stage 5 CKD. Patients with stage 5 CKD may also experience itching, cold intolerance, weight gain, and peripheral neuropathies.

- Polyuria and Nocturia
- Proteinuria
- Haematuria
- Hypertension and fluid overload
- Anaemia
- Bone Disease (Renal Osteodystrophy)
- Secondary hyperparathyroidism
- Osteomalacia (reduced mineralization)
- Mixed renal osteodystrophy (both hyperparathyroidism and osteomalacia)
- Adynamic bone disease (reduced bone formation and resorption)

The prognosis of Chronic kidney disease (CKD) largely depends on the level of kidney function (estimated Glomerular Filtration Rate, eGFR), degree of albuminuria, rate of disease

progression, and associated comorbidities. Lower eGFR and higher urinary albumin excretion are independently associated with increased risks of all-cause mortality, cardiovascular events, and progression to kidney failure, with no apparent safe thresholds.

To standardize prognostication, KDIGO introduced the "heat map" approach combining GFR stages (G1–G5) with albuminuria categories (A1–A3), which stratifies patients into low, moderate, high, and very high risk groups for adverse outcomes. Patients with advanced stages and higher albuminuria require closer monitoring and earlier preparation for kidney replacement therapy. Prognostic models such as the Kidney Failure Risk Equation (KFRE), using variables like age, sex, eGFR, and albuminuria, accurately predict 2- and 5-year risks of kidney failure and have been validated across multiple international cohorts. These models help guide referral to nephrology, timing of vascular access creation, and patient education.

Other important modifiers of prognosis include the underlying etiology of CKD, blood pressure control, diabetes status, recurrent episodes of acute kidney injury, smoking, and presence of cardiovascular disease. Management strategies that reduce proteinuria and stabilize eGFR, such as renin–angiotensin system blockade and SGLT2 inhibitors, are associated with improved long-term outcomes.

MANAGEMENT

The modern management of CKD focuses on slowing disease progression, preventing complications, and improving quality of life. The key strategies include:

1. Lifestyle Modifications
2. Control of Underlying Risk Factors
3. Pharmacological Therapy
4. Monitoring and Early Detection of Complications
5. Renal Replacement Therapy (RRT)
6. Patient Education and Psychosocial Support

It emphasizes dietary strategies such as moderation of protein (≈ 0.8 g/kg/day), sodium restriction (< 2 g/day), alongside fluid balance, regular exercise, smoking cessation, and weight control. Pharmacologic approaches prioritize ACE inhibitors or ARBs for blood pressure and proteinuria management; SGLT-2 inhibitors and GLP-1 receptor agonists (e.g., semaglutide/Ozempic) for renal and cardiovascular protection; statins for dyslipidemia; diuretics to manage volume overload; ESAs (and HIF-PHIs where appropriate) along with

iron supplementation for anemia; phosphate binders, vitamin D analogs for mineral-bone disorders; and bicarbonate to correct metabolic acidosis. Continuous monitoring of eGFR, proteinuria, hemoglobin, electrolytes, and mineral status guides early detection and intervention. In advanced or symptomatic CKD, renal replacement therapy—hemodialysis, peritoneal dialysis, or transplantation—is initiated. Equally critical are structured patient education, dietary counseling, and psychosocial support to improve treatment adherence and outcomes.[18,19,20]

Ayurvedic Managemen[21,22,23,24,25]

Management is directed towards *Dosha-Dushti* correction, *Dhatu-poshana*, and *Srotoshodhana*, with the aim to restore kidney function, slow disease progression, and alleviate complications. The Ayurvedic management of CKD can be systematically approached through the following therapeutic strategies.

1. ***Nidana Parivarjana*** (Avoidance of causative factors)
2. ***Shodhana Chikitsa*** (Purification therapy)
3. ***Shamana Chikitsa*** (Pacification therapy)
4. ***Rasayana* Therapy** (Rejuvenation therapy)
5. ***Pathya-Apathya*** (Diet & Lifestyle)

Rakta and *Meda Dhatus* are the foundations of the *Vrukka* (kidneys), according to *Acharya Sushruta*. In order to improve *Dhatvagni* activity, therapeutic treatments should mainly focus on rectifying *Jatharagni*, as *Ahara* (dietary intake) is first converted into *Rasa Dhatu* and then feeds the other *Dhatus*, including *Rakta*. This ensures the production of *Rasadi Dhatus* that are properly nourished. In this regard, herbal remedies that specifically target *Rakta* and *Meda* Dhatus are thought to be beneficial for renal support. Mild purgation (*Mridu Virechana*) and *Basti Chikitsa* (medicated enemas) may be added as supportive measures after *Shamana Chikitsa* (palliative therapy) has helped to relieve weakness (*Daurbalya*) and tissue depletion (*Dhatu Kshaya*).

Shodhana chikitsa

Shodhana Chikitsa (purification therapy) is employed alongside *Shamana* measures to eliminate aggravated *Doshas* and restore balance in *Mutravaha Srotas*. *Mridu Virechana* (mild purgation) with gentle laxatives like *Trivrit*, *Haritaki*, or *Eranda Taila* helps in clearing *Pitta-Kapha Dushti* and reducing fluid overload, while *Basti Chikitsa* (medicated enemas) is

considered the most effective for *Vata Dushti*, using decoction-based *Niruha Basti* or oil-based *Anuvasana Basti* for detoxification, diuresis, and nourishment. In selected cases, *Raktamokshana* may be useful for uremic or *Pitta-dominant* symptoms, and light *Upavasa* (therapeutic fasting) can support *Agni* and reduce metabolic load. By correcting *Agni* and *Dosha Dushti*, Shodhana not only alleviates symptoms such as edema, oliguria, and fatigue but also prepares the body for effective *Rasayana Chikitsa*, thereby supporting renal function and long-term tissue rejuvenation.

Shamana Chikitsa

Shamana Chikitsa (palliative therapy) is the primary approach in CKD management when patients are weak, depleted, or unsuitable for *Shodhana*. It focuses on pacifying aggravated *Doshas*, supporting *Agni*, nourishing depleted *Dhatus*, and providing symptomatic relief.

Key Principles

- ***Agni Deepana and Ama Pachana*:** Strengthening digestive and metabolic fire with mild *Deepana-Pachana* drugs like *Trikatu*, *Pippali*, and *Jeeraka* to improve assimilation.
- ***Mutrala* and *Shothahara* Herbs:** Use of *Punarnava* (Boerhavia diffusa), *Gokshura* (Tribulus terrestris), and *Varuna* (Crataeva nurvala) for diuretic and anti-inflammatory action.
- ***Rasayana* Therapy:** *Guduchi*, *Amalaki Rasayana*, and *Shilajit* to enhance immunity, reduce oxidative stress, and slow tissue degeneration.
- **Classical Formulations:** *Chandraprabha Vati*, *Punarnavadi Mandoor*, *Gokshuradi Guggulu*, and *Nephroprotective Rasayanas* for *Mutravaha Srotas* balance and renal support.
- **Symptom Management:** Correcting anemia with *Lauh preparations*; managing edema with *Shothahara* herbs; relieving fatigue with *Balya* and *Rasayana* drugs.
- ***Pathya-Apathya*:** Light, easily digestible diet (*Yusha*, *Mudga*, *Lauki*), restriction of heavy, oily, and salty foods; lifestyle modifications including yoga (*Bhujangasana*, *Pavanmuktasana*) and pranayama for circulation and stress reduction.

CONCLUSION

Chronic kidney disease (CKD) is the gradual decline of kidney function over months or years. In Ayurveda, it is viewed as an *Upadrava* (complication) of urinary disorders like *Mutraghata* or systemic conditions such as *Prameha* (diabetes). Vitiated *Dosha* affect the

Basti, disrupting urine filtration and causing harmful metabolic waste retention, leading to systemic damage. Early stages are *Krucchrasadhya* (difficult to treat), while advanced stages are *Yapya* (manageable) or incurable. Management includes purification (*Shodhana*), pacification (*Shamana*), and rejuvenation (*Rasayana*) therapies. *Rasayana* therapy, combined with psychological balance and a lifelong dietary regimen, can slow disease progression, improve kidney function, and enhance quality of life, even in advanced stages.

REFERENCES

1. Vaidya SR, Aeddula NR. Chronic Kidney Disease. [Updated 2024 Jul 31]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK535404/.
2. Talukdar R, Ajayan R, Gupta S, Biswas S, Parveen M, Sadhukhan D, Sinha AP, Parameswaran S. Chronic Kidney Disease Prevalence in India: A Systematic Review and Meta-Analysis from Community-Based Representative Evidence Between 2011 to 2023. Nephrology (Carlton), 2025 Jan; 30(1): e14420. doi: 10.1111/nep.14420. PMID: 39763170
3. Shukla V, Tripathi R, editors. Charaka Samhita Sutra Sthana, Chapter 18, verses 44–47. Delhi: Chaukhambha Sanskrit Pratishthan, 2014; 281.
4. Sharma H, Chandola HM. Prameha in ayurveda: Correlation with obesity, metabolic syndrome, and diabetes mellitus. Part 1-Etiology, classification, and pathogenesis J Altern Complement Med., 2011; 17: 491–6.
5. Patel MV, Patel KB, Gupta SN. Ayurvedic management of primary nephrotic syndrome J Res Educ Indian Med., 2017; 21: 16–22.
6. Agnivesha. Trikamji Acarya (ed.) Caraka samhita. Nidana sthana Ch. 4, Shloka 8. Varanasi: Chaukhamba Surbharati Prakashan, 2005; re-print, 213.
7. Vagbhatta. Paradakar Shastri (ed.), Astanga hrdaya. Nidana sthana Ch. 10, Shloka 7. 9th ed. Varanasi: Chaukhamba Surbharati Praka-shan, 2011; 502.
8. Agnivesha. Trikamji Acarya (ed.) Caraka samhita. Nidana sthana Ch.4, Shloka 8. Varanasi: Chaukhamba Surbharati Prakashan, 2005; re-print, 213.
9. Vagbhatta. Paradakar Shastri (ed.), Astanga hrdaya. Nidana sthana Ch. 9, Shloka 3. 9th ed. Varanasi: Chaukhamba Surbharati Praka-shan, 2011; 498.
10. Agnivesha. Trikamji Acarya (ed.) Caraka samhita. Siddhi sthana Ch. 9, Shloka 48. Varanasi: Chaukhamba Surbharati Prakashan, 2005; re-print, 719.

11. Sushruta. Trikamji Acarya (ed.) Sushruta samhita. Nidana sthana Ch. 3, Shloka 23, 24. 7th ed. Varanasi: Chaukhamba Orientalia, 2003; 279, 280.
12. Agnivesha. Trikamji Acarya (ed.) Caraka samhita. Dipika commentary by Cakrapannidutta. Cikitsa
13. sthana. Ch. 25, Shloka 17. Varanasi: Chaukhamba Surbharati Prakashan, 2005; re-print, p. 515.
14. Agnivesha. Trikamji Acarya (ed.) Caraka samhita. Dipika commentary by Cakrapanidutta. Sharira sthana. Ch. 6, Shloka17. Varanasi: Chaukhamba Surbharati Prakashan, 2005; re-print, 333.
15. .Inker LA, Astor BC, Fox CH, Isakova T, Lash JP, Peralta CA, Kurella Tamura M, Feldman HI. KDOQI US commentary on the 2012 KDIGO clinical practice guideline for the evaluation and management of CKD. Am J Kidney Dis., 2014 May; 63(5): 713-35.
16. Matsushita K, van der Velde M, Astor BC, et al. Association of estimated glomerular filtration rate and albuminuria with all-cause and cardiovascular mortality: a collaborative meta-analysis. *Lancet*., 2010; 375(9731): 2073-81.
17. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of CKD. *Kidney Int Suppl*., 2024.
18. Tangri N, Stevens LA, Griffith J, et al. A predictive model for progression of chronic kidney disease to kidney failure. *JAMA*., 2011; 305(15): 1553-9.
19. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of CKD. *Kidney Int Suppl*., 2013; 3(1): 1-150.
20. Heerspink HJL, Stefánsson BV, Correa-Rotter R, et al. Dapagliflozin in patients with chronic kidney disease. *N Engl J Med*., 2020; 383(15): 1436-46.
21. Agnivesha. Charaka Samhita, Sutrasthana, Chapter 10, Verses 11–20. In: Trikamji Acharya, editor. *Charaka Samhita with the Dipika commentary of Chakrapanidatta*. Varanasi: Chaukhamba Surbharati Prakashan, 2005; 66–7.
22. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. [Guideline update developed with patient partners, highlighting new therapies and care models]. Kidney Int. 2024 Jan 13 [supplement]; S117–S314. [cited 2025 Sep 09].
23. Chebib FT, et al. GLP-1 receptor agonists and weight-loss drugs (e.g., semaglutide/Ozempic) have demonstrated reductions in CKD progression, kidney failure, and cardiovascular outcomes in large-scale trials and meta-analyses. Lancet Diabetes

Endocrinol. 2024 16–22 % reductions in kidney-related events; overall reduction ~19 % [cited 2025 Sep 09].

24. KDIGO 2025 Clinical Practice Guideline for Anemia in Chronic Kidney Disease (ESA and HIF-PHI recommendations: lowest effective dose, cautious Hb targets, individualized route and frequency of administration), [cited 2025 Sep 09].
25. Sudheendran, A., Shajahan, M. A., & Premlal, S. (2021). A comparative diuretic evaluation of fruit and root of Gokshura (Tribulus terrestris Linn.) in albino rats. Ayu, 42(1): 52–56. https://doi.org/10. 4103/ayu.AYU_154_17
26. Prashanth, G. S., Baghel, M. S., Ravishankar, B., Gupta, S. N., & Mehta, M. P. (2010). A clinical comparative study of the management of chronic renal failure with Punarnavadi compound. Ayu, 31(2): 185–192. https://doi.org/10.4103/0974-8520.72388
27. Sushruta. Trikamji Acarya (ed.) Sushruta samhita. Cikitsasthana Ch.33, Shloka 3,32. 7th ed. Varanasi: Chaukhamba Orientalia, 2003; 515, 519.
28. Ibid. Cikitsasthana Ch.XXXV, Shloka 3,4,6, 527.
29. Agnivesha. Trikamji Acarya (ed.) Caraka samhita. Dipika commentary by Cakrapanidutta. Siddhi sthana. Ch.9, Shloka 8. Varanasi: Chaukhamba Surbharati Prakashan 2005; re-print, 717, 718.