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CASE STUDY

Pivotal and Phenomenon role of Shodhan Chikitsa along with Shaman Ausadh in Chronic Kidney Disease - A Case Study

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ABSTRACT
Chronic Kidney Disease (CKD) is a progressive condition characterized by irreversible loss of renal function, commonly associated with comorbidities such as hypertension and diabetes mellitus. This case study evaluates the role of Shodhana Chikitsa (purificatory therapy) along with Shamana Aushadhi (conservative Ayurvedic management) in the management of CKD. A 65-year-old female patient presented with complaints of periorbital puffiness, bilateral pedal edema, oliguria, anorexia, weakness, hypertension, diabetes mellitus, and significant proteinuria. Laboratory findings revealed low hemoglobin (7.20 gm/dl) and elevated serum creatinine (6.17 mg/dl), indicating advanced renal impairment. The patient was treated with a combination of internal medications including Gokshuradi Guggulu, Rasayan Vati, Tapyadi Loha, and Punarnava Kwatha. Panchakarma procedures included Punarnava Kwatha Niruha Basti for 5 days followed by Gokshur Siddha Ghrita Matra Basti for 21 days. The treatment was continued for approximately one year along with appropriate pathya-apathya (diet and lifestyle modifications). Post-treatment assessment showed marked clinical improvement with reduction in edema, anorexia, and weakness. Hemoglobin level improved to 12.6 gm/dl and serum creatinine reduced to 3.20 mg/dl. The patient also demonstrated improvement in CKD staging from Stage 5 to Stage 4. This case highlights the potential efficacy of an integrative Ayurvedic approach combining Shodhana and Shamana therapies in slowing disease progression, improving quality of life, and enhancing clinical outcomes in CKD patients.
***Keywords**: CKD patients, GFR, Panchakarma*

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INTRODUCTION

The definition and classification of chronic kidney disease (CKD) have evolved over time, but current international guidelines define this condition as decreased kidney function shown by glomerular filtration rate (GFR) of less than 60 mL/min per 1·73 m2, or markers of kidney damage, or both, of at least 3 months duration, regardless of the underlying cause. Diabetes and hypertension are the main causes of CKD in all high-income and middle-income countries, and also in many low-income countries. Incidence, prevalence, and progression of CKD also vary within countries by ethnicity and social determinants of health, possibly through epigenetic influence. Many people are asymptomatic or have non- specific symptoms such as lethargy, itch, or loss of appetite. Diagnosis is commonly made after chance findings from screening tests (urinary dipstick or blood tests), or when symptoms become severe. The best available indicator of overall kidney function is GFR, which is measured either via exogenous markers (e.g., DTPA, iohexol), or estimated using equations. Presence of proteinuria is associated with increased risk of progression of CKD and death. Kidney biopsy samples can show definitive evidence of CKD, through common changes such as glomerular sclerosis, tubular atrophy, and interstitial fibrosis.

Complications include anemia due to reduced production of erythropoietin by the kidney; reduced red blood cell survival and iron deficiency; and mineral bone disease caused by disturbed vitamin D, calcium, and phosphate metabolism. People with CKD are five to ten times more likely to die prematurely than they are to progress to end stage kidney disease [1]. This increased risk of death rises exponentially as kidney function worsens and is largely attributable to death from cardiovascular disease, although cancer incidence and mortality are also increased. Health-related quality of life is substantially lower for people with CKD than for the general population, and falls as GFR declines. Interventions targeting specific symptoms, or aimed at supporting educational or lifestyle considerations, make a positive difference to people living with CKD. Inequity in access to services for this disease disproportionally affects disadvantaged populations, and health service provision to incentivise early intervention over provision of care only for advanced CKD is still evolving in many countries.
According to ayurveda utpatti of kidney(*vrikka)* from medovaha srotas *."Rakta va Meda sara bhago". Bhaishajya Ratavali* has mentioned *Hetu*, *Purvaroop*, *Roop*, *Upadrav* and *Chikitsa* under separate topic called *Vrukka Rog*.

PATHOPHYSIOLOGY AND RISK FACTORS FOR CKD
The final common pathological manifestation of many chronic kidney diseases is renal fibrosis. Renal fibrosis represents the unsuccessful wound-healing of kidney tissue after chronic, sustained injury, and is characterized by glomerulosclerosis, tubular atrophy, and interstitial fibrosis.
Glomerulosclerosis is prompted by endothelial damage and dysfunction, proliferation of smooth-muscle cells and mesangial cells, and destruction of podocytes that normally line the glomerular basement membrane. Risk factors for progressive glomerulosclerosis include hypertension, dyslipidemia, and smoking. Glomerular microinflammation is initiated following activation of endothelial cells in response to hypertension, with inflammatory cells (including macrophages and foam cells) activating mesangial cells to proliferate. Transforming growth factor ß1 and other growth factors (including platelet-derived growth factor, fibroblast growth factor, tumor necrosis factor, and interferon gamma) stimulate mesangial cells to regress to mesangioblasts (immature mesangial cells). These mesangioblasts are capable of producing an excessive extracellular matrix, leading to mesangial expansion—an early sign of glomerulosclerosis (appendix). Stretching of podocytes leaves areas of the glomerular basement membrane exposed to Bowman's capsule with which it forms adhesions, thus contributing to glomerulosclerosis[4].
Tubular atrophy, interstitial fibrosis, and scarring are closely associated with GFR and proteinuria. Tubular epithelial cells are stimulated to synthesize inflammatory products including reactive oxygen species and chemokines by various abnormally-filtered urinary proteins, including complement, cytokines, and albumin. These agents attract inflammatory cells into the renal interstitium and initiate interactions with interstitial myofibroblasts. As fibrosis evolves, injured tubular epithelia lose their regenerative capacity and undergo apoptosis leading to tubular atrophy and creating non- functional glomeruli. Histologically, measures of tubular cell area are closely associated with GFR [2].
Kidneys are metabolically highly active with a high oxygen requirement. Early in CKD injury, interstitial capillaries become increasingly permeable (the kidney capillary leak syndrome) meaning that many plasma proteins that normally never reach the renal interstitium are able to do so and trigger an inflammatory response. A progressive decline in the surface area of interstitial capillaries leads to hypoxia within the kidney and affects the function of cells usually involved in the degradation of collagen which is synthesized (and degraded by matrix metalloproteinases, serine proteases, the adamalysin [ADAMTS] family, and lysosomal enzymes) in healthy kidneys. Collagens (particularly fibrillar collagen I and II), basement membrane proteins, proteoglycans, and glycoproteins become deposited in the chronically- damaged kidney; the area of fibrotic interstitium affected is closely associated with both renal function and long- term renal prognosis [4].

CASE REPORT
A 65 Years old female patient of Chronic Kidney Disease (CKD) came to *Brahma Panchakarma* and *Ayurvediya Chikitsalaya*,Vesma,Navsari. with the complaints of periorbital puffiness on face, pedal edema, hyper tension, diabetes mellitus, oliguria, massive proteinuria and high level of serum creatinine since 6 months.
Patient was healthy before two years, after that she started complaining of periorbital puffiness on face, pedal edema. after that she underwent to hospital and at that time she was diagnosed with Hypertension, diabetes mellitus. Investigation indicating massive oliguria, proteinuria and high level of serum creatinine. No any family history was present regarding CKD or any Renal disorders.

Physical Examination
Blood Pressure-160/90 mmhg.
Pulse rate-80 beats/min.
Respiratory rate-18/min.
Temperature-98.6^{o} C
Bowel-Irregular (Once in two days)
Appetite-Decreased (Less intake of food)
Micturition-2-3 times day
Sleep-Altered

Table 1. Prodromal symptoms

Nidranasha	Insomnia
Agnimandya	Anorexia
Netrapadanshashotha	Periorbital and Pedal oedema
Rakta sanchalanadhikya	Renal Hypertension
Vegavat nadi	Tachycardia
Tvag roukshya	Dry skin, Bruising

Table 2. Clinical symptoms

Chhardi	Nausea, Vomiting
Rakta panduta bhav	Anemia
Kati peeda	Back pain due to Mineral and bone disorders
Shoth	Oedema
Shir shoola	Headache
Matur nadi	Hematuria
Matur shavushna nadi yukta	Decreased, Painful and Burning micturition
Moha	Confusion

Ashtasthana Pariksha
Nadi(Pulse) – *Vata Pradhana kapha*
Mala(Stool) - *Asamyak*
Mutra(Urine) - *Alpa pravritti*
Jivha(Tongue) - *Lipta*
Shabda(Speech) – *Samyak Vaka prvariti*
Prakrita Sparsha(Tactilation) - *Ushna*
Druk(Eyes) - *Prakrut*
Prakruta Akriti(Anthropometry) – *Madhyama*

MATERIAL AND METHODS
Medications
1- *Gokshuradi Guggulu* 2-2-2
2- *Rasayan Vati* 2-0-2
3- *Tapyadi Loha* 1-0-1
4-*Punarnava Kwatha* 40ml-0-40ml
Panchakrma procedure
1-*Punarnava Kwath Niruha Basti* in dose of 1 Liter for 5 days
2-*Gokshur Siddha Ghrita Matra Basti* 40 ml for 21 days.

Treatment Underwent

Internal Medicines

Sr.No	Name of Medicine	Dose	Time	Anupana	No of Days
1	*Gokshuradi Guggulu*	2 Tab	3 Times After Food	*Ushnodak*	13 month
2	*Rasayan Vati*	2 Tab	2 Times Before Food	*Ushnodak*	13 month
3	*Tapyadi Loha*	1 Tab	2 Times After Food	Milk	13 month
4	*Punarnava Kwatha*	40 Ml	2 Times Before Food	Add 1 Tsp *Madhu*	13 month

Table 3. Internal medicines

Table 4. Showing details of *panchakarma* procedures given to patient

Sr.No	Procedure	Date	No of Days
1	Punarnava Kwath Niruha Basti in Dose of 1 Liter Through Anal Route	1-06-2023 - 5-06-2023	5
2	Gokshur Siddha Ghrita Matra Basti in Dose of 40 Ml Through Anal Route	6-06-2023 - 26-06-2023	21

Table 5. Showing Overall Result of Treatment

Symptoms	Before Treatment	After Treatment
periorbital puffiness on face	**4**	**1**
pedal edema	**4**	**1**
Anorexia	**4**	**2**
Weakness	**4**	**2**

*Note-Gradation Accroding to Dr.MS Baghel Developing guidelines for clinical research.

DISCUSSION

In this case of CKD patient had complains of peri orbital puffiness also called moon like face nd swelling over the face. than after few days' patient feels heaviness in both leg due to Bilateral pedal oedema with associated complains new Anorexia and Weakness in the body. After proper examination and diagnosis, the patient was successfully treated with classical ayurvedic principles i.e. shodhana and shaman chikitsa. This is the vyadhi of mutravaha srotas so for that firstly patient undergo for the Niruha Basti of Punarnava Kwath in dose of 1 liter for 5 days which is evacuate toxins from the body and stop further progression of disease and also specifically work on Pedal oedema and puffy face because punarnava has shothaghna effect which reduces shoth.

Serum creatinine value is 6.17 mg/dl. which is very high and also reports suggest that massive oliguria and proteinuria which is clearly indicate patient suffering from chronic kidney disease.

Etiopathogenesis-

1-Diseases causing Glomerular pathology

A-Primary glomerular pathology

B-Systemic glomerular pathology

2-Diseases causing tubulointerstitial pathology

A-Vascular causes

B-Infectious causes

C-Toxic causes

D-Obstructive causes

Regardless of the initiating cause, CKD evolves progressively through 4 stages:

1-Decreased renal reserve;

2-Renal insufficiency;

3-Renal failure;

4-End-stage kidney(CKD).

Gokshuradi guggulu specifically work on obstructive causes in tubulointerstitial pathology. Rasayan vati works on increase and maintain immunity and stop further progression of disease.

Tapyadi loha plays most important role in this disease because in this stage of disease their is a severe hemolysis occurs which leads drastically falls RBCs and Hb level decreased produces sever anaemia.Hb

level of pation at 1 june 2023 was 7.20 mg/dl. patient also taken Inj.Erythropoietin 4 times on 1-month (weekly once) for maintain RBCs and Hb level. After started Tapyadi Loha in dose of 1 tab two times per day with milk for next 3-month Hb level is increased and reach at 12.6 mg/dl. Which is Indicate significant result of Tapyadi loha in this case and patient is safe and prevent further massive hemolysis.
Punarnava kwath also work for its shothaghna, Mutral efficacy in this disease. Punernava kwath was given in dose of 40 ml twice with added 1 tsp of madhu. kwath reduces swelling in next 15 days of time period.
Punarnava Kwath Niruha Basti in Dose of 1 Liter Through anal route given at first 5 days this basti works in multi aspects firstly its work on shrotorodh and evacuation of toxins which are accumulate in body as well as specifically in mutravaha srotas and vrikka.
After niruha basti Gokshur siddha ghrit matra basti in dose of 40 ml after meal started for next 21 days. Matra basti work on stop the toxic(*visha*) accumulation. Ghrit is antidote of vish explained by our acharyas in classics, so matra basti of ghrit stop further toxins accumulation and give strength to overall immune system. Intake of some drugs and its adverse effect on kidney can consider as toxic accumulation such as Analgesics, which leads "Chronic Analgesic Nephritis".

Table 6. Investigations

DATE	LABORATORY INVESTIGATION Before Treatment	LABORATORY INVESTIGATION Before Treatment	eGFR	STAGES OF CKD
13/04/2023	Hb-7.20 gm/dl S. Creatinine-6.17 gm/dl	Hb-12.6 gm/dl S. Creatinine-5.71mg/dl	8/ml/min/1.73m	5TH
04/08/2023		Hb-12.5 gm/dl S. Creatinine-5.25mg/dl	9/ml/min/1.73m	5TH
08/12/2023		Hb-12.7 gm/dl S. Creatinine-4.75mg/dl	10/ml/min/1.73m	5TH
05/05/2024		Hb-12.6 gm/dl S. Creatinine-3.20mg/dl	16/ml/min/1.73	4TH

CONCLUSION

In this case report, we presented a patient with Chronic Kidney Disease (CKD), highlighting the clinical course, management strategies, and outcomes. Our findings underscore several critical aspects of CKD management: 1) Early Detection and Management, 2) Individualized Treatment, 3) Multidisciplinary Approach (*Shodhan* along with *Shaman*), 4) Patient Education and Adherence. On the basis of this case study it can be determined that *Niruha Basti* and *Matra basti* and *shaman* treatment along with *pathya-apathya palan* was effective in the management of Chronic Kidney Disease. Patient responded well with above medication and treatment. It is evidently endorsing that patient is shifted from Stage 5 chronic kidney disease to stage 4 chronic kidney disease.
In conclusion, this case report reinforces the importance of a comprehensive and individualized approach to CKD management. By applying the *Ayurvedic* management from this case, healthcare providers can better address the complexities of CKD and enhance patient care.

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