



AYURVEDIC MANAGEMENT OF MUTRAVAH STROTAS VIKAR W.S.R. TO CKD- A SINGLE CASE STUDY

**Dr. Namrata
Kailas Tathe**

M.D. Final Year, Kayachikitsa, R.A. Podar Medical College Worli, Mumbai.

Dr. Arati Datye

Assistant Professor Kayachikitsa, R.A. Podar Medical College, Worli, Mumbai.

ABSTRACT Chronic Kidney disease is a syndrome that results from progressive and irreversible destruction of nephrons. Due to inflammatory processes, auto immune conditions, or vascular causes like atherosclerotic conditions. CKD is most common complication found in patients with Diabetes and hypertension .prevalence rate ranging from 31- 49 %. [1] End stage renal disease significantly increase morbidity and mortality which require multidisciplinary management. CKD due to both of these conditions is attributed to micro and macroangiopathy. Hence as per Avurveda this condition can be well correlated with dhamani pratichay associated with ras and raktagata sneha vruddi. Also vitiated kapha and pitta lead to samrambha janya shopha at sira and Dhamani, further agreviating micro and macro angiopathy conditions. The samprapti in initial stages can be considered as Margawarodhjanya samprapti leading to incomplete poorana of vrikka awayaw and eventually it leads to dhatukshaya over several years. Considering all above pathophysiological phenomenon treatment is targeted to reduce inflammation and reduce obstruction of strotas, and satisfactory result was seen after adding Ayurvedic management along with his conventional treatment, **Case Presentation:** A 72 year old male patient with CKD visited OPD with serum creatinine level 2.7 with eGFR 29. After 2 months of Ayurvedic treatment, Sr. creat. Reduced up to 1.8 and eGFR increased upto 40.

KEYWORDS : CKD, Sr. Creatinine, vrikka roga, dhamani pratichay, shaman and shodhan chikitsa in CKD

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, irrespective of the cause. [1] CKD is a state of progressive loss of kidney function, ultimately resulting in the need for renal replacement therapy, such as dialysis or transplantation. As the kidneys play a crucial role in vital physiological processes, damage to these organs can disrupt the balance of water and electrolytes, regulation of blood pressure, elimination of toxins, and metabolism of vitamin D. Treatment options such as dietary modifications and medications can help slow disease progression. Kidney Disease Improving Global Outcomes (KDIGO) guidelines recommend that glomerular filtration rate (GFR) measurement or estimation is a critical diagnostic tool for CKD [2]

The prevalence of CKD in the general population is estimated to be around 10% to 14%. Specifically, albuminuria and an eGFR less than 60 mL/min/1.73 m² have prevalence of about 7% and 4%, respectively.[3] Worldwide, CKD accounted for 2,968,600 (1%) of disability-adjusted life-years and 2,546,700 (1%-3%) life-years lost in 2012.[4]

CASE STUDY

A 72 years old male patient came to OPD with symptoms of

- 1) Bipedal pitting edema
- 2) Pain in bilateral lower limbs
- 3) Dysuria
- 4) Exertional dyspnea

Since last 3 months.

Patient was admitted to IPD of kayachikitsa department (M.A.Podar Hospital)

History Of Present Illness:

Patient was asymptomatic before 2 yrs. gradually he developed bipedal pitting edema, pain in bilateral lower limbs, dysuria. He had taken opinion of Nephrologist and diagnosed as CKD.

Past History:

K/C/O – DM (since 7-8 years)
K/C/O - HTN (since 3-4 years)
N/K/C/O - BA, PTB, Hypothyroidism, Dyslipidemia
No any major surgical illness
No history of Blood transfusion, PR bleeding.

Personal History

Marital status – married
No any specific Addiction History
No significant family history

Hetusevan:

Aaharaj:

Excessive protein intake
High salt diet (fermented food, processed food)

Vihar:

Diwaswap
Sedentary lifestyle

O/E (On Examination)

BP	120/70 mm of Hg
Pulse Rate	88/min
SPO2	99% on R.A.
Temperature	97.3
RBS	116

S/E

RS – AEBE clear
CVS – S1S2 normal
CNS – conscious and oriented
Urine – turbid,
24hr Input 1400 ml
Output 1700 ml
Stool – sticky, frequency - 2-3 times a day
Pallor -absent
Icterus -absent
P/A – liver, spleen, kidneys – no palpable

ECG (31/01/2026) – No any significant changes

Chest X-ray PA view (31/01/2026) – No any significant abnormality

USG Done on 3/02/2026

Grade 1 fatty liver

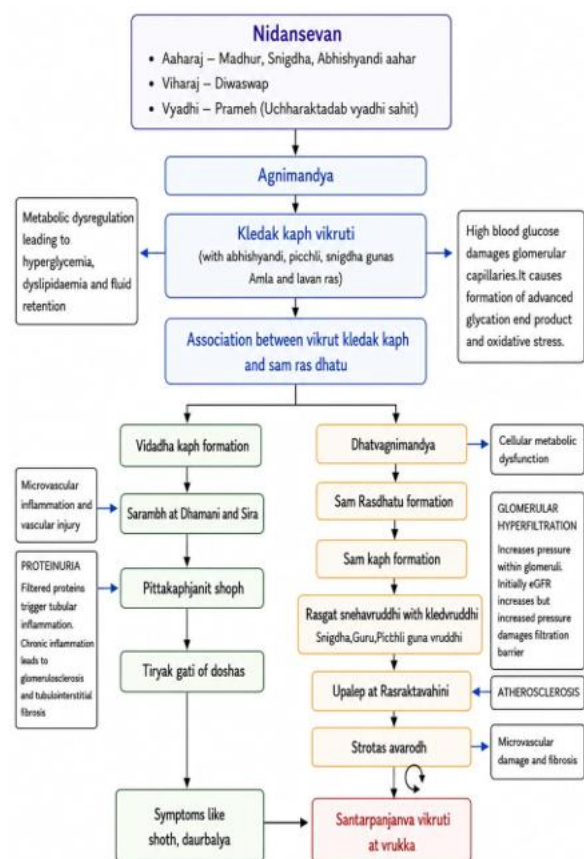
Left renal simple cyst (6.9 * 6.8 mm)

Bilateral mild renal cortical irregularity

Investigations done:

Blood Examinations:

Sr. No.	Parameter	02/02/2026	16/02/2026
1.	Hb	10.4	10.9
2.	Sr. Creat	2.7	1.8
3.	Sr. Urea	60	38
4.	eGFR	28.3	40.4
5.	FBS	142	134
6.	Total Chol,	83	106
7.	Sr. LDL	35	43
8.	Sr.VLDL	29	41
9.	Sr.Triglycerides	145	205
10.	Weight	81 kgs	77 kgs

Ayurvedic samprapti with modern correlation: [5]

Patient was treated with Shodhan and Shaman Chikitsa

Shaman Chikitsa with:

Sr.No.	Drug Name	Dose	Time	Duration
01.	Gokshuradi Guggul	250mg BD	After meal	2 months
02.	Punarnavashtak Kwath	20ml BD	1 hr Before meal (At morning and evening)	2 months

Shodhan Chikitsa with:

Shanik Snehan with Nadiswedan:

Kalbastikram: For 14 days (1 Anuvasan: 2 Niruh: 1 Anuvasan: 2 Niruh)

Niruh basti with Sarivadi, trunpanchmul, erandmul kwath (350 ml)

Anuvasan with Sahachar tail (60 ml)

DISCUSSION:**Role of Basti chikitsa;**

When basti is administered, drug comes in contact with the rectal and colonic mucosa, which has:

1. Rich vascular network
2. Thin epithelial lining
3. Good permeability for lipid and water soluble substances

Basti provides partial avoidance of hepatic first-pass metabolism, improving bioavailability of certain drugs.

Once the basti dravya is absorbed through rectal mucosa, it enters systemic circulation.

Parasympathetic supply to the proximal colon i.e. the intestinal branches originate from the posterior division of vagus nerve, which are secretomotor to glands and motor to muscular coats of gut. Thus, proximal colon has significant role in colonic motility and absorption.

Due to uniform positive pressure, homogeneous emulsion of basti dravya reaches quickly to proximal colon where it probably stimulates ICCSM, which in turn initiates colonic propagating activity and chain

of reactions like churning of contents in proximal colon and production of SCFA, absorption of electrolytes, water and other active principles through carrier mediated transport mechanism.

Certain components of basti formulations such as saindhava lavan, honey and other drugs of kwath dravyas create an osmotic effect.

Water is drawn from the intestinal mucosa and surrounding tissues into the colonic lumen. Distension of the colon due to increased fluid volume stimulates intestinal contractions, promotes expulsion of faeces and excess water content from body, thereby reducing fluid accumulation and edema in CKD.

Basti is prime therapy for vata and it helps restore normal functioning of vatadosha and supports proper urine flow, thereby maintain patency and function of urinary channels.

Basti may influence:

- Autonomic nervous system activity
- Colonic reflexes
- Fluid and electrolyte regulation [6]

In this patient, main important causative factors was santarpanjanya and abhishtyandi ahara, vihara, daily habit of diwaswap which is considered as one of the hetu of medovaha strotas dushti...Also, patient was having diabetes mellitus since 7-8 years and hypertension since 3-4 years, hence cumulatively induced strotovaigunya, vitiation of kapha and obstruction in the channels of vata and kidney damage with destruction of renal tissue was carried out.

Though, Tridoshas are important in pathogenesis of CKD, Kapha is highly responsible for obstruction of microvessels in kidney and vata dosha chiefly induces degeneration of structure of kidney which ultimately cumulatively lead to progressive CKD.

Therefore, drugs having properties like shophahar, strotoshodhak, aampachak, mutral were selected for the treatment.

Drugs For Niruh Basti:

Sr No.	Drug Name	Ras	Virya Vipak	Property
1.	Trunpanchmul	Kashay Tikta Madhur	Sheet	Shophahar Kledavahan[7]
2.	Sariva	Madhur Tikta	Sheet Madhur	Nephroprotective shophahar [8]
3.	Musta	Katu Tikta Kashay	Sheet Katu	Deepan pachan Antioxidant[7]
4.	Guduchi	Tilta Kashay	Ushna Madhur	Antiinflammatory Antioxidant[9]
5.	Punarnava	Madhur Tikta Kashay	Ushna Katu	Antiinflammatory Kledahara Nephroprotective [7]
6.	Gokshur	Madhur	Sheet Madhur	Diuretic[8] Nephroprotective
7.	Aargvadh	Madhur Tikta	Sheet Madhur	Aampachak
8.	Arjun	Kashay	Sheet Katu	Kledshoshak [7]
9.	Triphala	Lavanvarjit shadaras	Anushna Madhur	Rasayana Anulomaka Antioxidant [7]
10.	Erandmul	Madhur Tikta Katu	Ushna Madhur	Anti-inflammatory [7] Srotoshodhak

Niruha basti was given with the decoction of dravyas like Sariva, Musta, Guduchi, Punarnava, Gokshur, Aaragwadha, Arjun, Triphala and Trunpanchamula.

In which, Trunpanchamula acts by its mootral and sarambh nashak property, reduces inflammation at the level of capillaries, improving kledavahana.

Sariva also has anti-inflammatory anti-oxidant effect at the level of glomerulus.

Musta causes aampachan and reduces kledavruddhi.

Guduchi acts at the level of Rasdhatu, Raktadhatu and Medodhatu. It also has anti-inflammatory, nephroprotective action in CKD.

Punarnava helps in improving renal blood circulation, increases urine production, reducing edema associated with CKD, hence acts as shothahara.

Gokshuradi guggul:

Gokshuradi guggul acts as tridoshashamak, Deepan Pachana, Mutral, Shothahara, Kledaghna, Lekhaniya, Anulomaka, Strotoshodhak and raktaprasadak. [12]

In CKD, Gokshuradi guggul helps to reduce avarodh, it causes aampachan, thereby reducing the upalep at the level of Rasarakta vahini. By reducing inflammation and obstruction at the level of microvessels, it helps to increase urinary output, hence causing kledanirharan and acting a mutral drug

Punarnavashtak kwath:

It has Punarnava, Nimba, Abhaya, Amruta, Haridra, Patol, Tikta, and Shunthi.

It combinely acts as Shothahara, Strotoshodhak, Deepan Pachan, Kledaghna, and Anulomaka

Punarnava helps in improving renal circulation.

Nimba has specific active compounds such as Nimbidiol which helps to reduce renal fibrosis, improve kidney tissue damage and improving eGFR.

It also has antioxidant activity. It contains flavonoids, saponins that help neutralize free radicals and combat oxidative stress that drives CKD progression.

Abhaya causes detoxification by acting with its anulomak and lekhanitya properties.

Guduchi is a potent immunomodulatory. Guduchi helps lower oxidative stress and mitigates inflammatory pathways in the renal tissues, which protects nephrons from further damage.

Curcumin has significant anti-inflammatory, anti-oxidant and various other features.

Shunthi stimulates Agni and acts as aam pachak. It is a rich source of antioxidants and is known to reduce inflammation.

This case demonstrates that Ayurvedic management improved renal function in a patient with chronic kidney disease (CKD). Following treatment, serum creatinine decreased from 2.7 mg/dL to 1.8 mg/dL, while the estimated glomerular filtration rate (eGFR) improved from 29 to 40 mL/min/1.73 m², accompanied by clinical improvement. After follow up treatment of 2 months, Sr. creatinine reduced upto 1.6 and not increased further. These findings suggest that a comprehensive ayurvedic approach may have a supportive role in improving renal function and quality of life in selected CKD patients

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CONCLUSIONS

LABORATORY REPORT - BIOCHEMISTRY REPORT

Name: Shivam P. K. Age: 35 M. OPD No: 3452. Date: 16/07/2024

Ref: 11100. Bed No: 07. Unit In-charge: Dr. R. D. J. R.

LABORATORY REPORT - BIOCHEMISTRY REPORT

Parameter	Results	Normal Range	Parameter	Results	Normal Range
BLOOD GLUCOSE			Blood Urea	60	15-40 mg/dL
Fasting (F)	142	70-110 mg/dL	S. Creatinine	2.7	0.6-1.2 mg/dL
Post Prandial (PP)	124	70-150 mg/dL	S. Uric acid	2.3	3.0-8.0 mg/dL
Random (R)		70-150 mg/dL	S. Calcium	8.8	8.8-10.4 mg/dL
S. Total Bilirubin	0.3	Up to 1.0 mg/dL	LIPID PROFILE		
S. Direct Bilirubin	0.1	Up to 0.4 mg/dL	S. Total Cholesterol	83	150-250 mg/dL
S. Indirect Bilirubin	0.3	Up to 0.6 mg/dL	S. HDL	19	30-60 mg/dL
SGOT (AST)	13	0-45 IU/L	S. LDL	35	40-100 mg/dL
SGPT (ALT)	16	0-40 IU/L	S. VLDL	28	10-30 mg/dL
S. Total Protein	6.2	6.0-8.3 G/dL	HDL : Chol. Ratio	1.40	Up to 1.5
S. Albumin	3.4	3.2-5.0 G/dL	S. Triglycerides	145	60-160 mg/dL
S. ALP Phosphatase	118	70-306 IU/L			

LAB TECHNICIAN: BIOCHEMISTS:

Before Treatment

LABORATORY REPORT - BIOCHEMISTRY REPORT

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LABORATORY REPORT - BIOCHEMISTRY REPORT

Parameter	Results	Normal Range	Parameter	Results	Normal Range
BLOOD GLUCOSE			Blood Urea	38	15-40 mg/dL
Fasting (F)	124	70-110 mg/dL	S. Creatinine	1.8	0.6-1.2 mg/dL
Post Prandial (PP)	114	70-150 mg/dL	S. Uric acid	2.3	3.0-8.0 mg/dL
Random (R)		70-150 mg/dL	S. Calcium	8.8	8.8-10.4 mg/dL
S. Total Bilirubin	0.3	Up to 1.0 mg/dL	LIPID PROFILE		
S. Direct Bilirubin	0.1	Up to 0.4 mg/dL	S. Total Cholesterol	105	150-250 mg/dL
S. Indirect Bilirubin	0.3	Up to 0.6 mg/dL	S. HDL	22	30-60 mg/dL
SGOT (AST)	10	0-45 IU/L	S. LDL	43	40-100 mg/dL
SGPT (ALT)	12	0-40 IU/L	S. VLDL	41	10-30 mg/dL
S. Total Protein	6.2	6.0-8.3 G/dL	HDL : Chol. Ratio	1.50	Up to 1.5
S. Albumin	3.4	3.2-5.0 G/dL	S. Triglycerides	205	60-160 mg/dL
S. ALP Phosphatase	118	70-306 IU/L			

LAB TECHNICIAN: BIOCHEMISTS:

After Treatment