

REVIVING RENAL FUNCTION THROUGH *PUNARNAVADI KASHAYA* *BASTI* - A NOTEWORTHY REDUCTION IN BUN (BLOOD UREA NITROGEN) & SERUM CREATININE

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ABSTRACT

CKD has become a common disease with a high Prevalence rate nowadays in Diabetic & Hypertensive people, with dialysis and renal transplant as its mainstream of treatment. But due to low financial backgrounds among large percentage of individuals in India, everyone cannot afford to live a good quality of life. Hence, every individual is not a candidate for renal transplant or dialysis. So, to overcome this issue, an alternate has to be taken keeping in mind a healthy life which is the need of hour. Ayurveda provides a comprehensive alternative,² emphasizing dietary and lifestyle interventions alongside the therapeutic potential of ayurvedic formulations and bio-balancing therapies. This study reflects the case of a 74-year-old male with CKD, Hypertension & DM₂, who had symptoms such as facial puffiness, pedal edema, constipation and generalized weakness since 4 months. Following Ayurvedic treatment, the patient reported significant symptomatic relief. Laboratory investigations revealed a notable reduction in serum urea and creatinine levels, further affirming the effectiveness of the intervention. This case underscores the potential of Ayurvedic management as a holistic, safe, and efficacious approach for CKD, offering promising avenues for integrated healthcare.

KEYWORDS: BUN, Serum Creatinine, *Punarnavadi Kashaya Basti*, Edema.

INTRODUCTION

Creatinine levels measure a waste product from muscle breakdown, indicating kidney function, with typical adult ranges around **0.7-1.3 mg/dL for men and 0.5-1.1 mg/dL for women**, though factors like muscle mass, age, and hydration affect this, often using the estimated **Glomerular Filtration Rate (eGFR)** for better kidney assessment.

High levels suggest kidney issues (like chronic disease or dehydration), while low levels can indicate low muscle mass or malnutrition¹. BUN (Blood Urea Nitrogen) is a key marker for kidney function, especially in Chronic Kidney Disease (CKD). The normal range for BUN is typically **6-24 mg/dL**. In CKD, elevated BUN levels can indicate; Reduced kidney function, Increased risk of complications.

Chronic kidney disease has no direct link to any of the Ayurvedic classics' clinical entities. All disorders cannot be labelled with a name, as stated in *Charaka Samhita Sutrasthana Trisothiyadhyaya*. Chronic kidney disease 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. In Ayurveda, it is viewed as an *Upadrava* of urinary disorders like *Mutraghata* or systemic conditions like *Prameha*. Vitiated *Dosha* affect the *Basti*, disrupting urine filtration and causing harmful metabolic waste retention, leading to systemic damage.

Here an attempt has been made to critically analyse the effect of *Punarnavadi Kashaya Basti*³ in the management of CKD.

CASE REPORT

Chief complaints

Puffiness of face on & off as well as bilateral pedal edema since 4 months; aggravated since 1 month.

Associated complaints

Pain & burning sensation during micturition since 1 month.

Generalised weakness & fatigue since 1 month.

Lower back pain.

Loss of appetite.

Constipation.

History of present illness

A male patient of 74 years with k/c/o Hypertension, DM₂ since 10 years and CKD since 4 months. All relevant blood examination done showed raised creatinine, BUN & Urea, for which was advised for Dialysis. As he was not willing for dialysis, he was prescribed for symptomatic medication. 2 months back suddenly he developed fever with chills along with painful and burning sensation while micturition. It was a squeezing type of pain which develop prior, during and even after voiding the urine associated with burning sensation. He developed puffiness of face (no periorbital swelling) and swelling over bilateral feet persisting throughout the day and aggravated since 1month. For which they consulted their physician and given symptomatic treatment. By which fever subsided; But puffiness of face, pedal edema, burning sensation and painful micturition persisted.

Simultaneously patient developed complaints of tiredness throughout the day, needs support to get up from bed, walking, climbing steps, feeling sleepiness after taking a little quantity of food, no interest to do any activities, reduced appetite. Pt also complaining of back pain (lumbar region) non radiating which he feels like originating from deep inside and numbness of left leg which will be during early morning, sitting for long hours.

For above mentioned complaints, patient was approached to Department of Panchakarma, GAMC, Bengaluru for further management.

Past History

H/O Left Cataract Surgery 4 years ago.

Family history

Not suggestive

Table 1: Showing subject's personal history.

Name - XYZ	Micturition – Dysuria Burning micturition
Age – 74 years	Bowel habit – Irregular, Constipated
Sex – Male	Appetite – Reduced
Marital status – Married	Weight – 63 kg
Occupation – Farmer	Height - 158 cm
Sleep – Disturbed	Addiction - To oily and spicy foods. Chronic alcohol intake since 20 years(90ml/day)

Table 2: Showing Ashtasthana pareeksha.

<i>Nadi</i>	<i>Prakruta, 76bpm</i>
<i>Mutra</i>	6-7 times/day 1-2 times/night <i>Mutrakrichra & Ushnavata</i>
<i>Mala</i>	<i>Baddha Mala</i>
<i>Jihwa</i>	<i>Alpa-lipta</i>
<i>Shabda</i>	<i>Prakruta</i>
<i>Sparsha</i>	<i>Prakruta</i>
<i>Drik</i>	<i>Prakruta</i>
<i>Akriti</i>	<i>Madhyama</i>

Table 3: Showing Dashavidha pareeksha.

<i>Prakriti: Vata kapha</i>	<i>Satmya: Katu pradhana sarva rasa satmya</i>
<i>Vikriti: Tridosha</i>	<i>Ahara shakti: Avara</i>
<i>Sara: Madhyama</i>	<i>Vyayama shakti: Avara</i>
<i>Samhanana: Madhyama</i>	<i>Vaya: Vrdha-avastha (74 years)</i>
<i>Satva: Pravara</i>	<i>Pramana: Madhyama</i>

Physical Examination

- Temperature – Afebrile (98.6°F)
- Pulse Rate-92 bpm
- Blood Pressure-140/90 mm of Hg
- Respiratory Cycle-19 cpm
- Skin: cyanosis (-), jaundice (-)
- Head
- Eye: Conjunctiva anemic (+/+), sclera jaundice (-/-)
- ENT: secretions (-)
- Neck: lymph node not palpable, trachea central
- Thorax
- Heart: heart sound S1S2 regular, murmur (-)
- Pulmo: move symmetric, vesicular breathing sound (+/+), ronchi (-/-), wheezing (-/-)
- Extremities: swollen & pitting in pair of feet

Assessment of oedema for grading:

S.N.	Name of the test	Right	Left
1	Circumference at the level of medial malleolus.	23 cm	25.5 cm
2	Deepness of pitting and duration.	4mm >50 seconds	5mm >50 seconds
3	Fullness of extremities	Present	Present

Edema measurement chart

Grade	Depth	Rebound Time
1	<2 mm indentation	Disappears immediately
2	2-4 mm indentation	Last between 10-15 seconds
3	4-6 mm indentation	Can last more than 1 minute
4	6-8 mm indentation	Can last more than 2-5 minute

Pedal oedema of **GRADE 3** was observed.

Local examination

Inspection	Overlying skin -smooth, shiny, Taut and hairless, no normal skin wrinkles. Absence of normal dorsal Finger joint creases - Loss of normally appearing edges of medial malleoli. Rubor - absent Tumor - absent
Palpation	Pitting + on dorsum of foot, 5cm above medial malleoli and shin of tibia. Feeling of “valleys b/n hills” Pit refilling time (PRT) – More Than 50 seconds. Dolor - absent Calor - absent
Peripheral pulse - Dorsalis pedis Tibialis posterior	Feeble bilaterally Feeble bilaterally
Stemmer’s sign	Negative - both foot.

Systemic examination

- Central nervous system: Higher mental functions intact, no abnormality detected
- Cardiovascular system: S1 S2 heard, no abnormality detected
- Respiratory system: NVBS heard, no abnormality detected
- Gastrointestinal system: P/A- soft, non tender

Table 5: Showing Samprapti Ghataka.

Dosha	Tridosha (Kapha, Vata, Pitta)	Udbhavasthana	Amashaya
Dushya	Rasa, Rakta, Mutra	Sancharasthana	Sarva shareera
Agni	Jatharagni	Vyaktasthana	Mutravaha Srotos
Agnidushti	Jatharagni Mandya	Adhistana	Vrikka
Srotas	Rasavaha, Raktavaha, Udakavaha Mutravaha	Rogamarga	Bahya & Abhyantara
Srotodushti	Sangha, Vimargagamana	Sadhyasadhyata	Yapya

Table 6: Showing Treatment protocol adopted.

Panchakarma	Shamana Oushadhis
<i>Sarvanga Abhyanga with Dhanwantaram Taila followed by Triphala Kashaya Avagaha (For 3 Days)</i>	<i>Punarnava Choorna + Gokshura Choorna (3g BD) Chandraprabha Vati 1-0-1 A/F Triphala choorna 10g-HS</i>
<i>Punarnavadi Kashaya Basti followed by Anuvasana with Moorchita Tila Taila-80ml (Kala Basti)</i>	

Punarnavadi Kashaya Basti

Honey	50 ml
Saindhava	10 gm
Moorchita Tila Taila	70 ml
Gokshura+Punarnava+Haridra kalka	30 gm
Punarnavadi Kashaya Basti	340 ml
Total	500 ml

OBSERVATION AND RESULTS**Table 7: Showing Observation and Results.**

Treatment	Observation
<i>Sarvanga Abhyanga with Dhanwantaram Taila followed by Triphala Kashaya Avagaha</i>	Pedal edema reduced moderately Feeling of lightness Lower back pain reduced by 30-40%
<i>Punarnavadi Kashaya Basti followed by Anuvasana with Moorchita Tila Taila (Kala Basti)</i>	Pedal edema reduced (grade1), skin over the feet was shrunkend Facial puffiness reduced by 80% Lower back pain reduced by 80% Micturition was normal

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Lab Visit ID: Mf31	Collection Date: 04/Oct/2025 03:46PM
Barcode ID/Order ID: D...a...a.../1.../1.../1...	Report Date: 04/Oct/2025 10:56PM
Sample Type: Serum	Report Status: Final Report

BIOCHEMISTRY

Test Name	Result	Unit	Bio. Ref. Interval	Method
Kidney Function Test.				
Blood Urea Nitrogen	64	mg/dL	8.4-25.7	Urease
Urea	136.32	mg/dL	17.9 - 54.9	Calculated
Creatinine	5.67	mg/dL	0.7-1.3	Kinetic Alkaline Phosphate
Uric Acid	8.0	mg/dL	3.7-7.7	Uricase
Sodium	138	mmol/L	136-145	INDIRECT ISE
Potassium	5.20	mmol/L	3.5-5.1	INDIRECT ISE
Chloride	106.0	mmol/L	98-107	INDIRECT ISE
BUN/Creatinine Ratio	11.2	Ratio	12.1 - 20.1	Calculated

Critical Result-Creatinine : Please consult your doctor immediately.

Advice -Kindly correlate clinically.Repeat testing may be done on a fresh sample, if clinically indicated.

Comment:

BUN is directly related to protein intake and nitrogen metabolism and inversely related to the rate of excretion of urea. Blood urea nitrogen (BUN) levels reflect the balance between the production and excretion of urea. Increased levels are seen in renal failure, heart failure, hepatic failure, nephrotic syndrome, cachexia (low protein and high carbohydrate diets). Urea is a non-proteinous nitrogen compound formed in the liver from ammonia as an end product of protein metabolism. Urea diffuses freely into extracellular and intracellular fluid and is ultimately excreted by the kidneys. Increased levels are found in acute renal failure, chronic glomerulonephritis, congestive heart failure, decreased renal perfusion, diabetes, excessive protein intake, protein loading (BUN:Cr ratio > 20:1), metabolic acidosis, ketoacidosis, ketoacidosis, muscle wasting from starvation, nephritis, pyelonephritis, urinary tract obstruction, nephrotic drugs. Decreased levels are seen in inadequate dietary protein, low protein/high carbohydrate diet, malabsorption syndromes, pregnancy, severe liver disease, certain drugs. Creatinine is a catabolic product of creatine phosphate, which is excreted by filtration through the glomerulus and by tubular secretion. Creatinine clearance is an acceptable clinical measure of glomerular filtration rate (GFR). Increased levels are seen in acute/chronic renal failure, urinary tract obstruction, hypothyroidism, nephrotic drugs, shock, dehydration, congestive heart failure, diabetic ketoacidosis, metabolic acidosis.

BUN/Creatinine ratio (normally 12.1-20.1) is decreased in acute tubular necrosis, advanced liver disease, low protein intake, and in patients with metabolic acidosis. It is increased in heart failure, renal failure, and in patients with proteinuria. Uric acid levels show diurnal variation. The level is usually higher in the morning and lower in the evening. Increased levels are seen in starvation, strenuous exercise, malnutrition, or lead poisoning, gout, renal disorders, increased breakdown of body cells (as in cancer), and in patients with gout, renal failure, heart failure, or liver disease. Decreased levels are seen in chronic anemia, or heart failure, pre-eclampsia, liver disease (cirrhosis), obesity, psoriasis, hypothyroidism, low blood levels of parathyroid hormone (PTH), certain drugs, foods that are very high in purines - such as organ meats, red meats, some seafood and beer. Decreased levels are seen in liver disease, Wilson's disease, Syndrome of inappropriate antidiuretic hormone (SIADH), certain drugs.

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Customer Name	Age/Gender	Lab Visit ID	Barcode ID/Order ID	Sample Type	Collected Via	Referred By	Collection Date	Report Date	Report Status
JN	74/Male	Mf31	D...a...a.../1.../1.../1...	Serum	TATA 1MG BANGALORE	Dr.	04/Oct/2025 03:46PM	04/Oct/2025 10:56PM	Final Report

BIOCHEMISTRY

Test Name	Result	Unit	Bio. Ref. Interval	Method
Kidney Function Test.				
Blood Urea Nitrogen	36	mg/dL	8.4-25.7	Urease
Urea	77.04	mg/dL	17.9 - 54.9	Calculated
Creatinine	4.62	mg/dL	0.7-1.3	Kinetic Alkaline Phosphate
Uric Acid	5.3	mg/dL	3.7-7.7	Uricase
Sodium	141	mmol/L	136-145	INDIRECT ISE
Potassium	5.30	mmol/L	3.5-5.1	INDIRECT ISE
Chloride	115.0	mmol/L	98-107	INDIRECT ISE
BUN/Creatinine Ratio	7.7	Ratio	1.1 - 20.1	Calculated

Note: Mildly elevated potassium levels without symptoms may be referred with a fresh sample if needed. Causes can include kidney disease, dehydration, certain medications (e.g. ACE -1 inhibitor, potassium sparing diuretics), high potassium foods (e.g. bananas, spinach, avocados), tissue damage, or blood co - action errors. Persistent high levels should be discussed with your physician to explore potential underlying or medication effects.

Comment:

BUN is directly related to protein intake and nitrogen metabolism and inversely related to the rate of excretion of urea. Blood urea nitrogen (BUN) levels reflect the balance between the production and excretion of urea. Increased levels are seen in renal failure (acute or chronic), urinary tract obstruction, dehydration, shock, burns, GI bleeding, nephrotic drugs. Decreased levels are seen in starvation, strenuous exercise, malnutrition, or lead poisoning, gout, renal disorders, increased breakdown of body cells (as in cancer), and in patients with gout, renal failure, heart failure, or liver disease. Decreased levels are seen in chronic anemia, or heart failure, pre-eclampsia, liver disease (cirrhosis), obesity, psoriasis, hypothyroidism, low blood levels of parathyroid hormone (PTH), certain drugs, foods that are very high in purines - such as organ meats, red meats, some seafood and beer. Decreased levels are seen in liver disease, Wilson's disease, Syndrome of inappropriate antidiuretic hormone (SIADH), certain drugs.

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DISCUSSION

Ayurveda offers a comprehensive approach in the treatment of CKD. *Nidan Parivarjana* is the first and foremost advice given to the patients in Ayurveda. Management of Systemic Derangements (*Bheshaja Chikitsa*), aims at treating *Amotpatti* by *Jathragni* improvement there by removing *Srotorodha*. Management in form of Shaman Chikitsa and Shodhan Chikitsa can be done.

In Ayurveda CKD, can be considered as a *Mootravaha Srotas Vikara* which causes oedema. Both Kidney are root of *Medovaha Srotas*. The pathogenesis of kidney disease is not separately mentioned. It can be included in *Prameha*, *Mutra dosha*, *Mutra krichchra*, *injury of Vankshana*, *Ashmari* (renal stones) and oedema (*Shopha*) etc. Chronic kidney disease is a very complex *Vyadhi Sankara*. So we need drugs which helps in *Basti Shodhana* as well as *Lekhana*, *Mootrala & Rasayana* property.

Before carrying out *Samshodhana Chikitsa*, *Deepana Pachana* was done. *Basti* is the treatment mostly effective on *Pakvashaya* in which urine formation carried out. It is not only effective in treating *Vaataja Rogas* but is also effective in *Pittaja*, *Kaphaja*, *Sansargaja*, *Sannipataja* and *Raktaja Rogas*. Ayurveda emphasizes the importance of *Trimarma* (*Siras*, *Hrudaya* and *Basti*) which are to be protected, if not it may lead to death. *Basti Marma* although structurally similar to bladder but here the entire renal functioning is to be considered among which kidneys plays a major and vital role. So, considering *Basti Marma* is affected in CKD and keeping in mind importance of *Marma Paripalana*, *Basti Karma* was considered as treatment of choice.

Abhyanga followed by Kashaya Avagaha

Abhyanga: *Snehana* induces improvement in blood circulation & remove waste material. It decreases swelling in local tissue & acts as an excellent muscle relaxant. It accentuates the effect of *Niruha Vasti*, which was administered later.

Dhanwantaram Taila is indicated for *Mutraghata* as well as 80 types of *Vata Vyadhi*.

Avagaha sweda: Warm water induces vasodilation, increasing blood flow and promoting sweating to eliminate toxins; herbal properties are absorbed through the skin.

Mode of Action: The heat activates the sympathetic nervous system, releasing hormones that boost metabolism, fat breakdown, and toxin elimination (urea, creatinine, ammonia). Described by *Acharya Charaka* as *Sagni Sweda*, it mobilizes and liquefies *Doshas* lodged in the body's microchannels (*Srotas*).

Triphala is widely recognized in Ayurvedic and modern research for its potential nephroprotective benefits due to its antioxidant and anti-inflammatory properties, with studies suggesting it may improve kidney function and reduce damage in conditions like diabetic nephropathy.

Punarnavadi Kashaya Basti

Basti helps in removal of morbid *Doshas* which thereby helps in achieving equilibrium of *Doshas* in body. Progression of CKD leads to CRF where uremic toxins start developing and showing effect on body. The researches have shown that uremic toxins originate in gut. Microbes in colon produces compounds, normally excreted by the kidneys, which are potential uremic toxins. Solutes made by colon microbes may contribute to uremic illness. Such putrefaction products were originally detected in urine and subsequently shown to accumulate in plasma when the kidneys failed.

Probable mode of action of Basti can be understand

- (1) by absorption mechanism
- (2) by system biology concept
- (3) by neural stimulation mechanism
- (4) by excretory mechanism.

Pharmacodynamics outcome of Basti Karma may be due to functioning of the one or combined effect of all the four mechanisms.

Punarnavadi kashaya predominantly has *tikta, kashaya and madhura rasa, laghu guna and ushna veerya*. This drug act as *shothahara*. By the diuretic action, it flushes out the toxin and reduces excess fluid retention. **Beta sitosterol** is one of the active principles present in this which helps in easy metabolism of cholesterol and has anti-inflammatory effect. Thus helps in reducing pedal edema, burning micturition and improves urine output.

Moorchita Tila Taila alleviates *Vata* without aggravating *Kapha*. It acts as *Yogavahi*, provides therapeutic benefits in *Niruha Basti*.

Gokshura choorna

Gokshura is one among the drug of *Mutravirechaniya Gana*, and it act as *Anulomaka* of *Apama Vayu*. *Gokshura* is *Sheeta Veerya, Balya, Basti Vishodhaka, Madhur Vipaki, Deepana, Vrushya, Pushtikara* and *Ashmarihara, Pramehahara, Shvasahara, Kasahara, Arshahara*, also useful in *Hridroga* and *Vatahara* in nature. *Gokshura* is a widely used *mutrala dravya* in the clinical practice. It has *madhura rasa, guru-snigdha guna* and *sheeta veerya*. The studies suggest diuretic properties of *gokshura* are due to large quantities of nitrates and potassium salts.

Chandraprabha vati

Chandraprabha vati *Rogadhikara* in *Mutrughata, Katishoola, Vibandha, Mandagni, Aruchi* as mentioned in *Sharangadhara Samhita-Madhyama Khanda-Vata Kalpana* 7/40-49. It also act as *Vrshya & Rasayana*. The way it functions is like; Cleanses the Urinary Tract, Prevents and Manages Infections, Reduces Swelling and Retention, Balances Blood Sugar Levels, Supports Stone Prevention.

CONCLUSION

This case of elevated BUN & Serum creatinine, based on its presentation and *Dosha* involvement was diagnosed under *Mutravaha srotas Vikara* and it was treated according to the *Chikitsa Sutra* of *Mutragnata*. *Mutrala* and *Tridosha shamaka* mainly *Kapha* and *Vata* *shamaka dravyas* were used in the management of the disease. With the use Ayurveda medicines this condition was well managed and symptomatically improvemet was observed in this case, but such cases require frequent follow ups and regular medication until the KFT comes under normal range.

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