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Ayurvedic Interventions in the Management of Chronic Kidney Disease with Hypertension: A Single Case Study

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ABSTRACT:

Chronic Kidney Disease (CKD) is a progressive disorder characterized by structural and functional kidney abnormalities that persist for at least three months, regardless of whether there is a decrease in glomerular filtration rate (GFR). It is often accompanied by altered blood or urine composition. It commonly arises from diabetes and hypertension and usually remains asymptomatic until advanced stages, leading to serious complications. Early detection and timely management are crucial for slowing disease progression. This case study reports a 31-year-old hypertensive patient diagnosed with CKD, presenting with elevated serum creatinine (2.06 mg/dl), urea (56.20 mg/dl), uric acid (8.00 mg/dl), and BUN (26.18 mg/dl). The patient was treated with an Ayurvedic regimen comprising *Punarnavashtak Kwath, Gokshuradi Guggulu, Syrup Neeri KFT,* and *Sarpagandha Ghan Vati* for two months. Post-treatment, significant improvements were observed: creatinine reduced to 0.5 mg/dl, urea to 20.50 mg/dl, uric acid to 3.80 mg/dl, and BUN to 9.55 mg/dl. Clinical symptoms such as dizziness and headache, etc., subsided, and blood pressure stabilized at 120/80 mmHg. These findings suggest that Ayurvedic therapy can effectively improve renal function, regulate blood pressure, and enhance overall quality of life in CKD patients with hypertension.

KEYWORDS: Chronic kidney disease, CKD, *Gokshuradi Guggulu, Mutravaha Srotas dushti, Punarnavashtak kwath, Sarpagandha ghan vati.*

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INTRODUCTION:

Chronic kidney disease (CKD) describes abnormal kidney function and/or structure. It is common, frequently unrecognized, and often exists together with other conditions (such as cardiovascular disease and diabetes). CKD is usually asymptomatic. If not treated at an early stage or ignored at the primary level, it can convert into some life-threatening conditions. Many developing countries have a high prevalence of CKD. CKD classification into 5 stages uses the combination of an index of kidney function, the glomerular filtration rate (GFR), and markers of kidney damage to define the stages. Stages 3–5 were defined by a GFR less than 60 ml/min/1.73 m^2 with or without markers of kidney damage on at least 2 separate occasions separated by a period of at least 90 days. To delineate an increased risk of adverse outcomes, the 2008 NICE guideline on chronic kidney disease suggested 2 key changes to this classification: the subdivision of stage 3 into 3A (GFR 45–59 ml/min/1.73 m^2) and 3B (30–44 ml/min/1.73 m^2) [1]. Factors such as age, sex, hypertension, diabetes, atherosclerosis, overuse of painkillers, Obesity, hyperuricemia, area of residence, and economic status have a strong positive independent correlation with progressive CKD.[2]

Hypertension (HTN) is both a cause and a consequence of CKD. Mechanisms such as sodium dysregulation[3], reduced renal mass[4], sympathetic overactivity, and renin–angiotensin–aldosterone system[5] imbalance contribute to elevated blood pressure in CKD. Endothelial dysfunction[6] and arterial stiffness[7] further exacerbate this cycle, promoting renal injury. *Ayurveda* classics emphasize the pivotal role of *Agni* (digestive/metabolic fire) in maintaining homeostasis. The classics declare that *'Rogah Sarve Api Mandagnau'* — all diseases originate from an impaired *Agni* [8] This fundamental disturbance leads to the formation of *Ama* (toxins), which initiates a series of *Srotas Dushti* (vitiation of bodily channels), contributing to complex chronic conditions such as chronic kidney disease (CKD) and Hypertension (HTN). (Fig- 1)

CASE HISTORY:

A Patient of age 31 years with a factory job, who has had a history of HTN since 1 and 1/2yrs, is on irregular anti-hypertensive (Tab Amlong 5 mg)for the last 6 months, came to the outpatient department of *Kayachikitsa*.

He reported with headache and dizziness, uncontrolled hypertension with obstructed flow and decreased amount of urine during voiding, and a frothy appearance for one and a half years. Associated complaints of abdominal distension after meals, nausea, irregular or regular constipation, loss of appetite, heaviness, and body aches for 8-9 months.

Patient was diagnosed with grade 1 medico renal disease as per ultrasound reports on 16/11/23. (Fig 2) He had

known complaint of hypertension for 1 and a half years, took allopathic management for about 1 year, got mild relief, so stopped taking anti-hypertensives 6 months back and came to the OPD of *Kayachikitsa*, CBPACS.

Clinical findings:

General examination:

On general examination, the Patient was thin, with a weight of 42 kg, height 5'2", and BMI 16.4 kg/m^2.Blood pressure (B.P.) 156/104 mmHg, Pulse Rate 84/min, no edema, clubbing, cyanosis, or icterus was observed.

The Patient presented with previously conducted kidney function test reports, which revealed elevated renal parameters with serum creatinine of 2.06 mg/dl, serum urea of 56.20 mg/dl, and blood urea nitrogen(BUN) of 26.80 mg/dl.

USG Reports: shows the right kidney was smaller in size, 7.5*2.1 cm, with mild echogenicity and corticomedullary differentiation, also a hypoechoic cystic lesion with dense internal content measuring approximately. 24.9*19.9 mm noted at the upper pole of the left kidney on 16/11/2023. (Fig 2)

DIAGNOSTIC PROTOCOL:

Based on symptoms like *Atibaddha Mutra (*Obstructed flow*), Alpa Mutra (oligouria),* and *Bahalam Mutra (* frothy urine*)* with *Aruchi* (Anorexia)*, Hrilasa* (Nausea)*, Bhrama* (Dizziness)*, Gauravta (*Heaviness*)*, *Aangmarda* (Body aches*), Mandāgni* (Weak digestion), *Tandra (*Drowsiness*), Shiroruk* (Headache), indicates *Rasa [9] and Rakta [10] Dhatu Dusti* reflects through *Mutravaha Srotas Dusti*.

Based on modern signs and symptoms, along with investigations such as KFT (Sr. creatinine, Sr. urea, Sr. uric acid), Ultrasound reports, and Blood pressure, the patient was provisionally diagnosed with CKD and HTN. Based on the diagnosis, a treatment protocol was designed.

THERAPEUTIC INTERVENTIONS:

After a thorough evaluation of the patient and previous reports, the findings led to a provisional diagnosis of CKD with HTN. The patient's previous lab reports of KFT dated July 10, 2024 (Fig 3.1), show derangement, including serum creatinine (2.06 mg/dl), serum urea (56.20 mg/dl), and Blood urea nitrogen (26.18 mg/dl) with previous USG reports(Fig 2), confirming the diagnosis of CKD. e-GFR was not provided in the earlier reports; it was calculated using the NKF CKD-EPI calculator and found to be 48 ml/min/1.73 m^2, indicating Stage 3B CKD. Following confirmation, drug intervention was initiated, and the patient was treated according to the principle of *Chikitsa*, which was based on breaking the pathogenesis rooted in *Agni dushti* and *Ama* formation. Hence, the line of treatment adopted focused on *Agni Deepana* and *Ama Pachana* (to address the root), *Rasa-Rakta-Prasadana* (to improve dhatu quality), and *Mutravaha Srotoshodhana* (to correct the impaired renal function). The drug selected for the treatment was *Punarnavashtak Kwath*, *Gokshuradi*

Guggulu, Syrup Neeri KFT with *Sarpagandha ghan vati*.(Table 1, 2)

Instruction given to patient:

The patient was also advised to follow dietary and lifestyle modifications along with Ayurvedic medication for better management of the condition. He was advised to take a smaller amount of water, a salt-restricted diet. Also, to avoid protein-rich foods like pulses, meat, eggs, milk, and other dairy products.

Table 1: Drug administration with Dose, frequency, and *Anupana*

Drug intervention	Dose	Frequency	Anupana
Punarnavashtak Kwath	40ml	Twice a day, morning and evening, before food	-
Gokshuradi Guggulu	2 Tab	Thrice a day after food	lukewarm water
Syp. Neeri KFT	2 Tsf	Twice a day, after food	-
Sarpa Gandha ghan Vati	2 Tab	Twice a day, after food	lukewarm water

Table 2: Timeline of events:

Date	Details
16 NOV 2023	**USG REPORTS**: shows the right kidney was smaller in size, 7.5*2.1 cm with mild echogenic and corticomedullary differentiation, also A hypoechoic cystic lesion with dense internal content measuring approximately 24.9*19.9 mm noted at the upper pole of the left kidney.(Fig 2)
19 JULY 2024	Patient came to OPD Kayachikitsa for the first time with deranged limits in KFT and USG reports; he was diagnosed with CKD with Hypertension. Prescribed *Punarnavashtak kwath, Gokshuradi Guggulu, Syp. Neeri KFT, Sarpa Gandha ghan vati.* Advised to repeat KFT, Urine analysis Routine, and microscopy.
02 AUG 2024	1st follow-up visit, Mild relief in frequency and obstruction of micturition, burning sensation during micturition, nausea, dizziness, headache, loss of appetite, heaviness and body aches, but frothiness in urine and gaseous abdomen was not relieved. Investigation reports received on July 28, 2024, show Sr. Cr 1.8 mg/dl, Sr. uric acid 8.00mg/dl, Sr. urea 47.60 mg/dl, BUN 22.17 mg/dl, and Urine Pus cells-6-8 HPF, Urine Protein-Trace.
16 AUG 2024	2ND follow-up visit, improved frequency of micturition, relief in obstruction during micturition and burning micturition, nausea, headache, dizziness, body aches, and loss of appetite. Mild relief in frothiness in urine, gaseous abdomen. Advised to repeat KFT, Urine analysis Routine, and microscopy. Continue with the same medication.

13 SEP 2024	3rd follow-up visit, no fresh complaints, investigations reports received on SEP 11, 2024 show normal limits of KFT, Urine analysis Routine and microscopy i.e., Sr. Cr from 2.06 mg/dl to 0.5mg/dl, Sr. uric acid from 8.00mg/dl to 3.8 mg/dl, Sr. urea 47.60 mg/dl to 20.55 mg/dl, Urine pus cells 2-3HPF and Urine protein to Nil.

Table 3: Improvement in Objective Criteria

Investigatory method	**10 July 2024 Before treatment**	**28 July 2024 Follow up**	**11 September 2024 After treatment**
S. Creatinine	2.06 mg/dl	1.80 mg/dl	0.57 mg/dl
Sr. uric acid	8.00mg/dl	8.90 mg/dl	3.8 mg/dl
BUN	26.18 mg/dl	22.17 mg/dl	9.55 mg/dl
UREA	56.20 mg/dl	47.60 mg/dl	20.50 mg/dl
E-GFR	43ml/min/1.73m^2 (NKF CKD-EPI 2021 calculator)	51ml/min/1.73m^2 (NKF CKD-EPI 2021 calculator)	134 ml/min/1.73m^2 (NKF CKD-EPI 2021 calculator)
Urine Protein	NA	Trace	NIL
Urine Pus Cells	NA	6-8 HPF	2-3 HPF

Table 4.1 Improvement in Symptoms

Symptoms	Before Treatment	After Treatment
Aruchi (Anorexia)	+++	+
Hrilasa (Nausea)	++	-
*Gauravta (*Heaviness*)*	+	-
Aangmarda (Body aches*)*	+++	+
Mandāgni (Weak digestion)	++	-
*Tandra (*Drowsiness*)*	++	-
Shiroruk (Headache)	+++	+

Table 4.2 Improvement in symptoms

Symptoms	**Grading**	**Before treatment**	**After treatment**
Obstruction of urine	0- Normal urine flow 1- slight difficulty in urination 2- weak urine stream requiring effort 3- marked obstruction with straining, dribbling, or interrupted flow	2	0

Frequency of urine	0- normal urine output (1000-2000ml/day) 1- slight reduction in output (700-1000 ml) 2- noticeable reduction (400-700 ml)	1	0
Frothy Urine	0- absent 1- mild 2- moderate 3- severe	2	0

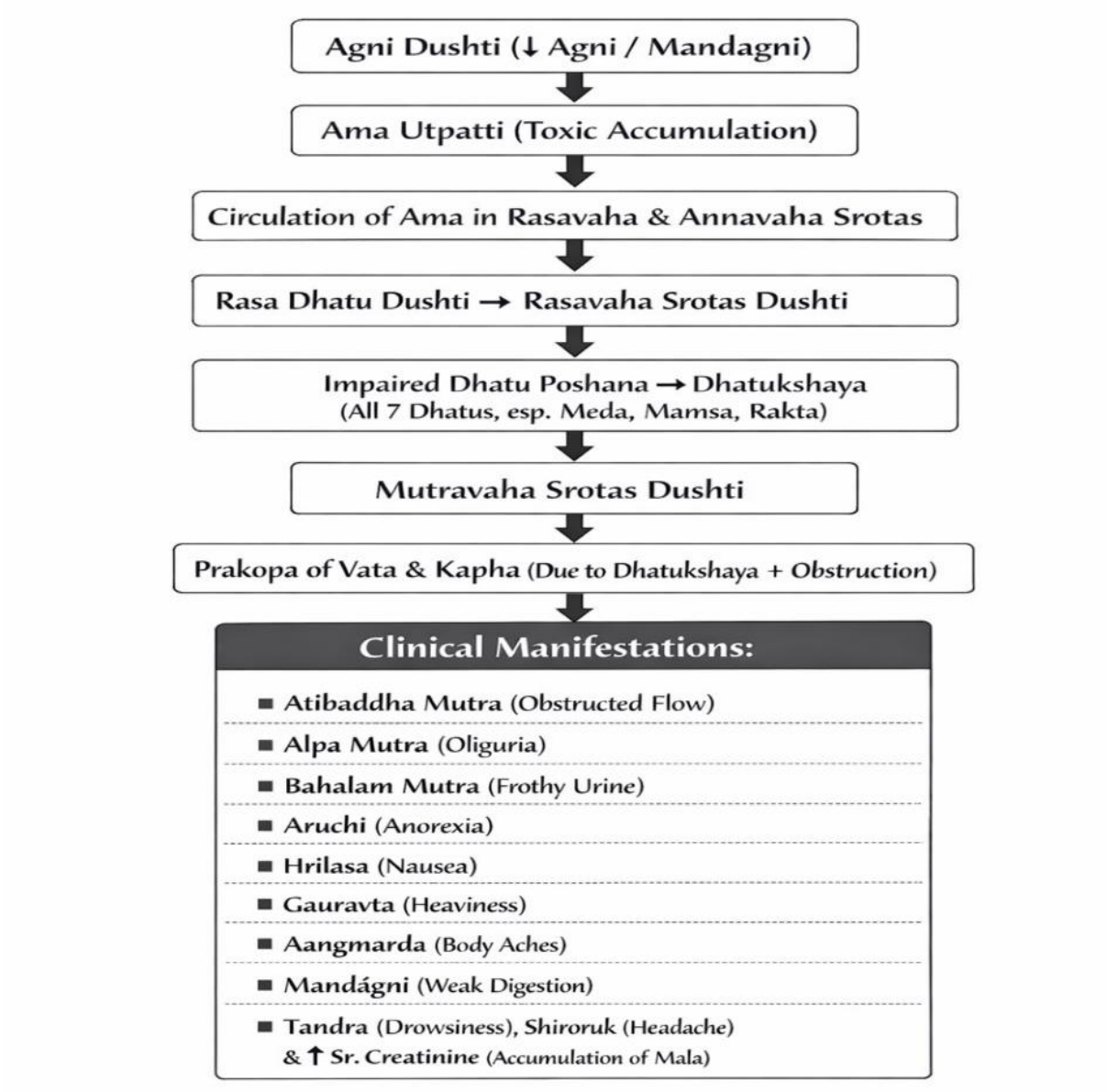
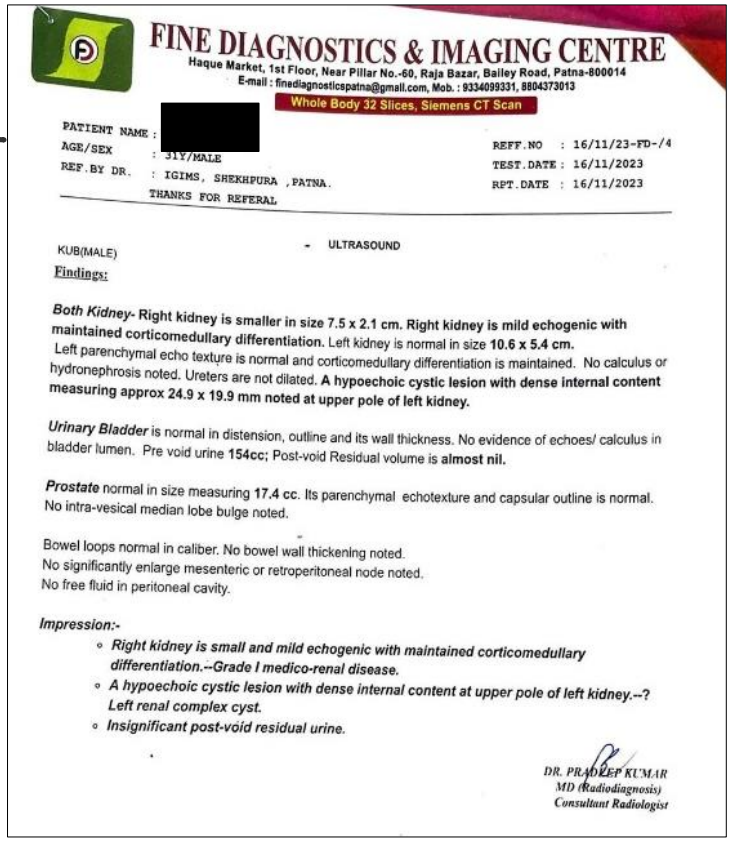


Fig 1: Samprapati (Pathogenesis) of CKD with HTN:



FINE DIAGNOSTICS & IMAGING CENTRE

Haque Market, 1st Floor, Near Pillar No.-60, Raja Bazar, Bailey Road, Patna-800014

E-mail : finediagnosticspatna@gmail.com, Mob. : 9334099331, 8804373013

Whole Body 32 Slices, Siemens CT Scan

PATIENT NAME : [redacted]
AGE/SEX : 31Y/MALE
REF.BY DR. : IGIMS, SHEKHPURA ,PATNA.
THANKS FOR REFERAL

REFF.NO : 16/11/23-FD-/4
TEST.DATE : 16/11/2023
RPT.DATE : 16/11/2023

- ULTRASOUND

KUB(MALE)

Findings:

Both Kidney- **Right kidney is smaller in size 7.5 x 2.1 cm. Right kidney is mild echogenic with maintained corticomedullary differentiation.** Left kidney is normal in size **10.6 x 5.4 cm.** Left parenchymal echo texture is normal and corticomedullary differentiation is maintained. No calculus or hydronephrosis noted. Ureters are not dilated. **A hypoechoic cystic lesion with dense internal content measuring approx 24.9 x 19.9 mm noted at upper pole of left kidney.**

Urinary Bladder is normal in distension, outline and its wall thickness. No evidence of echoes/ calculus in bladder lumen. Pre void urine **154cc;** Post-void Residual volume is **almost nil.**

Prostate normal in size measuring **17.4 cc.** Its parenchymal echotexture and capsular outline is normal. No intra-vesical median lobe bulge noted.

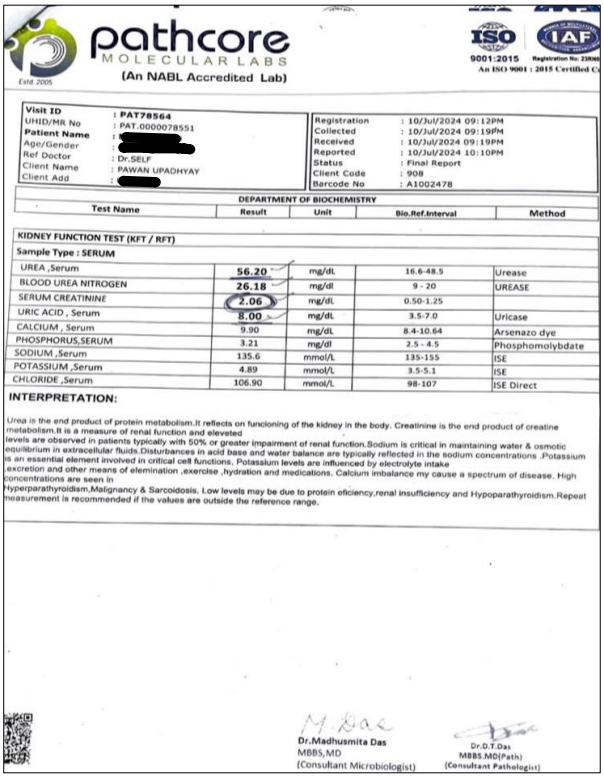
Bowel loops normal in caliber. No bowel wall thickening noted.
No significantly enlarge mesenteric or retroperitoneal node noted.
No free fluid in peritoneal cavity.

Impression:-

- ***Right kidney is small and mild echogenic with maintained corticomedullary differentiation.--Grade I medico-renal disease.***
- ***A hypoechoic cystic lesion with dense internal content at upper pole of left kidney.--? Left renal complex cyst.***
- ***Insignificant post-void residual urine.***

DR. PRADEEP KUMAR
MD (Radiodiagnosis)
Consultant Radiologist

Figure- 2: USG Report



pathcore MOLECULAR LABS (An NABL Accredited Lab)

Visit ID	: PAT78564	Registration	: 10/Jul/2024 09:12PM
UHID/MR No	: PAT.0000078551	Collected	: 10/Jul/2024 09:19PM
Patient Name	: [redacted]	Received	: 10/Jul/2024 09:19PM
Age/Gender	: [redacted]	Reported	: 10/Jul/2024 10:10PM
Ref Doctor	: Dr.SELF	Status	: Final Report
Client Name	: PAWAN UPADHYAY	Client Code	: 908
Client Add	: [redacted]	Barcode No	: A1002478

DEPARTMENT OF BIOCHEMISTRY

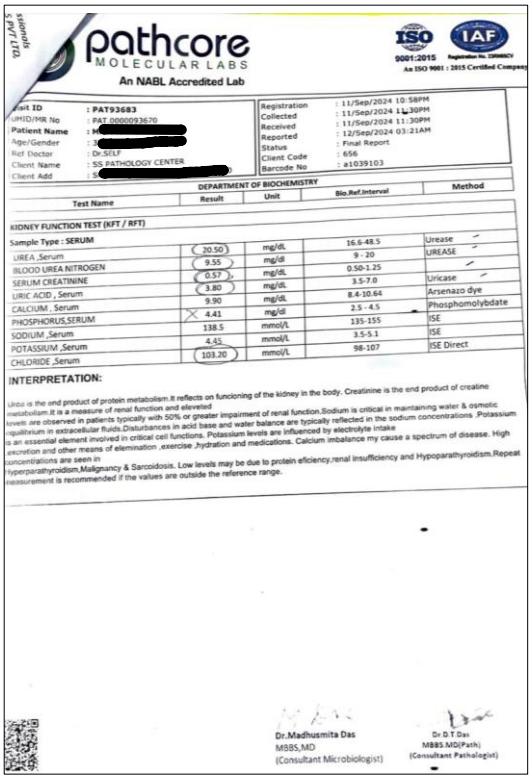
Test Name	Result	Unit	Bio.Ref.Interval	Method
KIDNEY FUNCTION TEST (KFT / RFT)				
Sample Type : SERUM				
UREA ,Serum	56.20	mg/dL	16.6-48.5	Urease
BLOOD UREA NITROGEN	26.18	mg/dl	9 - 20	UREASE
SERUM CREATININE	2.06	mg/dL	0.50-1.25	
URIC ACID , Serum	8.00	mg/dL	3.5-7.0	Uricase
CALCIUM , Serum	9.90	mg/dL	8.4-10.64	Arsenazo dye
PHOSPHORUS,SERUM	3.21	mg/dl	2.5 - 4.5	Phosphomolybdate
SODIUM ,Serum	135.6	mmol/L	135-155	ISE
POTASSIUM ,Serum	4.89	mmol/L	3.5-5.1	ISE
CHLORIDE ,Serum	106.90	mmol/L	98-107	ISE Direct

INTERPRETATION:

Urea is the end product of protein metabolism.It reflects on funcioning of the kidney in the body. Creatinine is the end product of creatine metabolism.It is a measure of renal function and elevated levels are observed in patients typically with 50% or greater impairment of renal function.Sodium is critical in maintaining water & osmotic equilibrium in extracellular fluids.Disturbances in acid base and water balance are typically reflected in the sodium concentrations .Potassium is an essential element involved in critical cell functions. Potassium levels are influenced by electrolyte intake ,excretion and other means of elemination ,exercise ,hydration and medications. Calcium imbalance my cause a spectrum of disease. High concentrations are seen in Hyperparathyroidism,Malignancy & Sarcoidosis. Low levels may be due to protein eficiency,renal insufficiency and Hypoparathyroidism.Repeat measurement is recommended if the values are outside the reference range.

Dr.Madhusmita Das MBBS,MD (Consultant Microbiologist) Dr.D.T.Das MBBS.MD(Path) (Consultant Pathologist)

Fig 3.1: Lab. Reports Before treatment



pathcore MOLECULAR LABS An NABL Accredited Lab

Visit ID	: PAT93683	Registration	: 11/Sep/2024 10:58PM
UHID/MR No	: PAT.0000093670	Collected	: 11/Sep/2024 11:30PM
Patient Name	: [redacted]	Received	: 11/Sep/2024 11:30PM
Age/Gender	: [redacted]	Reported	: 12/Sep/2024 03:21AM
Ref Doctor	: Dr.SELF	Status	: Final Report
Client Name	: SS PATHOLOGY CENTER	Client Code	: 656
Client Add	: [redacted]	Barcode No	: a1039103

DEPARTMENT OF BIOCHEMISTRY

Test Name	Result	Unit	Bio.Ref.Interval	Method
KIDNEY FUNCTION TEST (KFT / RFT)				
Sample Type : SERUM				
UREA ,Serum	20.50	mg/dL	16.6-48.5	Urease
BLOOD UREA NITROGEN	9.55	mg/dl	9 - 20	UREASE
SERUM CREATININE	0.57	mg/dL	0.50-1.25	
URIC ACID , Serum	3.80	mg/dL	3.5-7.0	Uricase
CALCIUM , Serum	9.90	mg/dL	8.4-10.64	Arsenazo dye
PHOSPHORUS,SERUM	4.41	mg/dl	2.5 - 4.5	Phosphomolybdate
SODIUM ,Serum	138.5	mmol/L	135-155	ISE
POTASSIUM ,Serum	4.45	mmol/L	3.5-5.1	ISE
CHLORIDE ,Serum	103.20	mmol/L	98-107	ISE Direct

INTERPRETATION:

Urea is the end product of protein metabolism.It reflects on funcioning of the kidney in the body. Creatinine is the end product of creatine metabolism.It is a measure of renal function and elevated levels are observed in patients typically with 50% or greater impairment of renal function.Sodium is critical in maintaining water & osmotic equilibrium in extracellular fluids.Disturbances in acid base and water balance are typically reflected in the sodium concentrations .Potassium is an essential element involved in critical cell functions. Potassium levels are influenced by electrolyte intake ,excretion and other means of elemination ,exercise ,hydration and medications. Calcium imbalance my cause a spectrum of disease. High concentrations are seen in Hyperparathyroidism,Malignancy & Sarcoidosis. Low levels may be due to protein eficiency,renal insufficiency and Hypoparathyroidism.Repeat measurement is recommended if the values are outside the reference range.

Dr.Madhusmita Das MBBS,MD (Consultant Microbiologist) Dr.D.T.Das MBBS.MD(Path) (Consultant Pathologist)

Fig 3.2: Lab. Reports After treatment

DISCUSSION

The patient presented with chronic uncontrolled hypertension for over 18 months, accompanied by obstructed urine (*Atibaddha Mutra)* , decreased urine output (*Alpa-Alpa Mutrata*), frothy urine (*Bahalam Mutra* indicative of proteinuria), and deranged kidney function tests. According to *Ayurveda*, this reflects *Mutravaha*, *Rasavaha*, and *Raktavaha Srotas dushti*, where impaired *Agni* and *Ama* accumulation obstruct normal *Dhatu poshana*, leading to *Mala sanchaya* and altered excretory function. These manifestations correlate with the modern understanding of reduced glomerular filtration and progressive renal damage.

Mode of action of *Punarnavashtak Kwath*:

Punarnavashtak Kwath exhibits a multidimensional mode of action in the management of Chronic Kidney Disease (CKD). The *Tikta–Kashaya Rasa* and *Laghu–Ruksha* or *Snigdha–Laghu Guna* of herbs such as *Punarnava(Boerhavia diffusa)*, *Nimb(Azadirachta indica)*, *Patol(Trichosanthes dioica)*, *Kutaki(Picrorhiza kurroa)*, and *Guduchi(Tinospora cordifolia)* support *Ama Pachana*, *Srotoshodhana*, and the reduction of inflammatory changes within *Mutravaha Srotas*. *Ushna Virya* herbs like *Punarnava, Shunthi, Guduchi*, and *Deodar* stimulate metabolism, promote diuresis, and reduce fluid retention, while *Sheeta Virya* herbs such as *Nimb* and *Kutaki* provide hepatoprotective and antioxidant benefits. *Katu Vīpaka* drugs contribute *lekhana* and *shoshana* actions, assisting in *Kleda shoshana* and maintaining fluid balance, whereas *Madhura Vipaka* herbs, such as *Guduchi*, *Shunthi*, and *Haritaki*, provide nourishment and rejuvenation. Importantly, *Punarnava* has demonstrated nephroprotective effects [11] by lowering serum creatinine and urea levels, improving renal histopathology, and reducing fibrosis [12] in experimental CKD models. Additionally, *Punarnavashtak Kwath* enhances the functioning of *Rasavaha Srotas* by improving nutritional assimilation and the quality of *rasa dhatu*. At the same time, its action on *Raktavaha Srotas* helps purify blood, reduce oxidative stress, and maintain vascular health. Collectively, these properties establish *Punarnavashtak Kwath* as a formulation with significant therapeutic and nephroprotective potential in CKD.

Mode of action of *Gokshuradi Guggulu*:

The components include: *Gokshura (Tribulus terrestris), Haritaki (Terminalia chebula), Bibhitaki (Terminalia Bellerica), Amalaki (Emblica officinalis), Mustaka (Cyperus rotundus), Shuddha Guggulu (Commiphora Mukul), Sunthi (Zingiber officinale), Maricha (Piper nigrum), and Pippali (Piper longum)*.

All things considered, it has diuretic, immunomodulatory, antioxidant, and anti-inflammatory qualities. *Gokshur* is an important ingredient in

Mutravirechaniya Mahakashaya [13], a category of medications known for their diuretic properties (*Mutrala*), which can aid in reducing fluid overload in the body, an important factor linked to high blood pressure. Additionally, *Gokshur* acts as a *Basti Shodhak* by purifying the *Srotas marga*, which helps reduce unnecessary and excessive secretions in the *Mutravaha Srotas marga*. This makes it effective for treating issues like *Mutrakriccha*, *Mutraghat*, and *Ashmari*. Clinical studies have also demonstrated its antihypertensive [14] properties.

Mode of action of *Sarpagandha Ghan Vati:*

In *Siddha Yoga Sangraha, Vaidya Yadavji Trikamji* describes it as an herbal preparation. *Sarpagandha* has been extensively studied in various research. *Vata* and *Kapha* doshas are corrected by *Katu vīpaka*, *Ushna Virya*, and *Nidrajanana Prabhava*. Its anti-hypertensive properties have already been demonstrated in earlier research. In addition to *Sarpagandha(Rauwolfia serpentina)*, which is the main component, this mixture also contains *Cannabis(Cannabis sativa)*, *Khurasani ajwain(Trachyspermum ammi)*, *Jatamansi(Nardostachys Jatamansi)*, and *Pippli Mula(Piper longum)* . Modern pharmacological studies attribute its efficacy mainly to alkaloids like reserpine, which deplete catecholamines from sympathetic nerve endings and reduce peripheral vascular resistance. The additional ingredients—*Jatamansi* with its nervine sedative effects, Cannabis with hypotensive and anxiolytic actions [15] , and *Khurasani Ajwain* with antispasmodic properties [16] —synergistically enhance its blood pressure–lowering capacity.

In the context of chronic kidney disease (CKD), uncontrolled hypertension is a major factor in accelerating glomerular injury, vascular remodeling, and nephron loss. [17] By lowering systemic and intraglomerular pressure through sympathetic inhibition and vasodilation, *Sarpagandha Ghana Vati* reduces glomerular hyperfiltration and helps preserve renal function.

Mode of action of Syp. Neeri KFT*:*

It is a combination of multiple herbs formulated based on a scientific principle. It has a safe general therapeutic effect on a variety of urinary problems, including cystitis, urinary calculi, UTIs, and disorders related to the prostate. Various phytoconstituents, including arbutin, tannins, quinolone derivatives, bioflavonoids, and glucosides, are abundant in these extracts. The following chemical substances are utilized as ingredients in this syrup: *Giloy*(*Tinospora cordifolia)*, *Makoya(Solanum nigrum), Palash Pushp(Butea monosperma), Sirisa(Albizzia lebbeck), Haridra(Curcuma Longa), Shigru(Moringa oleifera), Dhania(Coriandrum Sativum), Varun(Cratavea Nurvala), ShwetParpati, Lal Chandan(Pterocarpus Santalinus), Gokshur(Tribulus Terristris), Punarnava(Boerhavia diffusa)*, and so on. Neeri-KFT contains a combination of

herbs with notable antioxidant, nephroprotective, and immunomodulatory properties. These actions support patients with renal impairment or chronic kidney disease (CKD) by safeguarding kidney structure, enhancing renal physiology, and contributing to the restoration of altered renal architecture. [18]

CONCLUSION:

The present case study demonstrates that an Ayurvedic treatment regimen comprising *Punarnavashtak Kwath, Gokshuradi Guggulu, Syrup Neeri KFT*, and *Sarpagandha Ghan Vati* resulted in notable improvement in renal function parameters and blood pressure control in a patient diagnosed with CKD with hypertension. Significant reductions in serum creatinine, urea, uric acid, and blood urea nitrogen levels were observed within two months of therapy, along with relief from associated symptoms such as headache and dizziness. These findings suggest that Ayurvedic management may help in disease stabilization, prevention of further renal deterioration, and improvement in quality of life.

Limitation of the study:

However, as this is a single-case observation, the findings cannot be generalized to a larger population. Additionally, long-term follow-up could not be assessed as the patient discontinued treatment and did not report for subsequent visits after improvement. Therefore, further well-designed clinical studies with larger sample sizes and longer follow-up periods are required to validate the efficacy and safety of Ayurvedic interventions in Chronic kidney disease and Hypertension.

Acknowledgement:

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Consent of the patient:

The informed written consent of the patient has been taken before publication of reports without disclosing the identity of patient.

Conflict of interest: The author declares that there is no conflict of interest.

Guarantor*:* The corresponding author is the guarantor of this article and its contents.

Source of support: None

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