IJAYUSH
International Journal of AYUSH
AYURVEDA • YOGA • UNANI • SIDDHA • HOMEOPATHY
https://internationaljournal.org.in/journal/index.php/ijayush/
ISSN: 2349-7025
OPEN ACCESS
PEER REVIEWED
MONTHLY

Original Research Article	Volume 15 Issue 07	July 2026

ROLE OF KALA BASTI AND SHAMANA CHIKITSA IN THE AYURVEDIC MANAGEMENT OF CHEMOTHERAPY-INDUCED ACUTE KIDNEY INJURY: A SAMHITA SIDDHANTA-BASED PERSPECTIVE

***Dr. Deepak Kumar[1], Dr. Murali. K.[2], Dr. Praveen Nandal[3], Dr. Neha Saini[4]**

[1]PhD Scholar / Assistant Professor, Department of Samhita Siddhant, The Glocal College of Ayurvedic Medical Science and Research Centre, Mirzapur Pole, Saharanpur U.P / Shree Baba Mastnath Ayurvedic College & Hospital, Rohtak. Email id: deepakrockng@gmail.com, 0009-0003-5675-4741

[2]Distinguished Professor, Department of Kayachikitsa, The Glocal University, Research Wing, Mirzapur Pole, Saharanpur U.P. Email id: muralivaidyaka@yahoo.com

[3]PhD Scholar / Assistant Professor, Department of Kayachikitsa, The Glocal College of Ayurvedic Medical Science and Research Centre, Mirzapur Pole, Saharanpur U.P / Shri Baba Mastnath Ayurvedic College & Hospital, Rohtak. Email id: praveenandal320@gmail.com, 0000-0003-3305-3128

[4]Assistant Professor, Department of Kayachikitsa, Shree Baba Mastnath Ayurvedic College & Hospital, Rohtak. Email id: neha.saini408@gmail.com, 0009-0007-6498-3949

***Corresponding Author – Dr. Deepak Kumar**

PhD Scholar / Assistant Professor, Department of Samhita Siddhant, The Glocal College of Ayurvedic Medical Science and Research Centre, Mirzapur Pole, Saharanpur U.P / Shree Baba Mastnath Ayurvedic College & Hospital, Rohtak

Email id – deepakrockng@gmail.com, 0009-0003-5675-4741

Contact No.: 7610901760

Postal Address – Vill. Garhi Bohar Po. Asthal Bohar, Rohtak, Haryana 124001

ABSTRACT

Chemotherapy-induced Acute Kidney Injury (AKI) is a serious and potentially life-threatening complication associated with nephrotoxic anticancer agents. Renal toxicity develops due to direct tubular epithelial damage, oxidative stress, inflammatory mediator release, endothelial dysfunction, and impaired renal microcirculation. AKI significantly compromises continuation of chemotherapy, increases morbidity, and deteriorates patient quality of life. Modern management primarily includes supportive care, hydration therapy, electrolyte correction, and renal replacement therapy in severe conditions. However, satisfactory renal recovery remains a major challenge.

Ayurveda explains such pathological conditions under the concepts of Mutravaha Srotodushti, Basti Vikara, Vata-pradhana Tridoshaja Vyadhi, Dhatukshaya, Ama, and Srotorodha. Chemotherapy acts as an Agantuja Nidana leading to Agnimandya, Ama formation, Ojakshaya,

tissue depletion, and Vata aggravation ultimately affecting Mutravaha Srotas and renal physiology.

The present case report evaluates the efficacy of Kala Basti and Shamana Chikitsa in chemotherapy-induced AKI from a Samhita Siddhanta perspective. A 55-year-old male patient presented with generalized weakness, fatigue, anorexia, reduced urinary output, pedal edema, nausea, and disturbed sleep following chemotherapy. Initial laboratory investigations revealed severe renal dysfunction with serum creatinine 5.90 mg/dL and blood urea 115.8 mg/dL. The patient was treated with Kala Basti using Trinpanchamoola Kwatha and Chandana Bala Taila along with internal medications including Keedajadi Capsule, Neeri KFT Syrup, Amyron Tablet, Punarnava Mandura, and Eranda Bhrashta Haritaki.

After treatment, significant clinical and biochemical improvement was observed. Serum creatinine reduced from 5.90 mg/dL to 1.70 mg/dL, blood urea reduced from 115.8 mg/dL to 58.4 mg/dL, and overall quality of life improved substantially.

The present case demonstrates that Ayurvedic management based on Samhita Siddhanta principles may provide effective supportive nephroprotective care in chemotherapy-induced renal injury through Vatanulomana, Srotoshodhana, Rasayana, and Mutrala effects.

Keywords: Acute Kidney Injury, Chemotherapy-induced nephrotoxicity, Kala Basti, Panchakarma, Mutravaha Srotas, Ayurveda, Rasayana, Basti Chikitsa

INTRODUCTION

Acute Kidney Injury (AKI) is characterized by a sudden decline in renal function resulting in impaired excretion of metabolic waste products, electrolyte imbalance, acid-base disturbances, and fluid retention.[1] Chemotherapy-induced nephrotoxicity is increasingly recognized as a major dose-limiting complication in oncology patients receiving cytotoxic drugs. Various chemotherapeutic agents such as Cisplatin, Methotrexate, Ifosfamide, Cyclophosphamide, Carboplatin, Gemcitabine, and targeted biological therapies are known to produce nephrotoxicity through multiple mechanisms including oxidative stress, direct tubular epithelial toxicity, renal vasoconstriction, inflammatory cytokine release, endothelial dysfunction, and apoptosis or necrosis of renal tissue.[2] The kidneys are particularly vulnerable to these toxic effects because they receive approximately 25% of the cardiac output and are continuously exposed to circulating toxins and metabolites.

Modern management of chemotherapy-induced Acute Kidney Injury mainly focuses on supportive care measures including intravenous hydration, electrolyte correction, avoidance or modification of nephrotoxic drugs, dose adjustment of chemotherapeutic agents, and renal replacement therapy in severe conditions.[3] Despite considerable advancements in nephrology and oncology, long-term renal recovery still remains a major challenge in many patients, significantly affecting prognosis and quality of life.

According to Ayurveda, renal dysfunction can be understood under the concepts of Mutravaha Srotodushti, Basti Vikara, Mutrakrichra, Mutraghata, and Vata-pradhana Tridoshaja Vyadhi. Chemotherapy may be considered as an Agantuja Nidana which leads to Agnimandya, Ama formation, Dhatukshaya, Ojakshaya, Srotorodha, and aggravation of Vata Dosha.[4] The

pathological process ultimately affects Mutravaha Srotas resulting in impaired urinary function and accumulation of metabolic toxins within the body.

Acharya Charaka has stated "Basti Vataharanam Shreshtham," indicating that Basti is the most effective therapy for disorders predominantly involving Vata Dosha. Since Pakwashaya is considered the principal seat of Vata, Basti Chikitsa exerts systemic therapeutic action through regulation of Vata, elimination of morbid Doshas, restoration of Srotasa patency, and nourishment of depleted Dhatus.[5] Through systemic absorption and neurohumoral regulation, Basti influences multiple physiological systems including renal function. Kala Basti is a specialized schedule comprising Niruha and Anuvasana Basti administered in a sequential manner to achieve Vatanulomana, Srotoshodhana, Dosha Shamana, Dhatu Poshana, and Rasayana effects.[6]

Ayurvedic research on renal disorders highlights the role of Mutravaha Srotas, Dosha imbalance, Agni impairment, Ama formation, and Dhatukshaya in the pathogenesis of kidney dysfunction. Recent studies suggest that Ayurvedic interventions may support renal protection through antioxidant, anti-inflammatory, immunomodulatory, and Rasayana effects. Basti therapy has been described as an important modality for Vata disorders due to its systemic action on Dosha regulation and Srotas correction. However, limited clinical evidence is available regarding its role in chemotherapy-induced Acute Kidney Injury.

The novelty of the present article is the clinical exploration of Kala Basti along with Shamana Chikitsa in chemotherapy-induced AKI, integrating modern concepts of nephrotoxicity with Ayurvedic principles of Agantuja Nidana, Vata aggravation, Srotodushti, and Dhatu depletion. This study provides a unique Samhita-based perspective on supportive renal recovery and highlights the need for further research in Ayurvedic nephrology.

CASE REPORT

Patient Information

A 55-year-old male patient presented to the outpatient department of Shri Baba Mastnath General and Ayurvedic Hospital, Asthal Bohar, Rohtak, with complaints of generalized weakness, fatigue, reduced appetite, decreased urinary output, pedal edema, nausea, disturbed sleep, and body ache following chemotherapy for carcinoma urinary bladder. The symptoms gradually developed after chemotherapy and significantly affected the patient's daily activities and quality of life.

Chief Complaints

Table 1: Clinical Presentation of the Patient

Complaint	Duration
Generalized weakness	1 month
Reduced appetite	1 month
Reduced urinary output	20 days
Pedal edema	15 days
Fatigue	1 month
Disturbed sleep	20 days

Associated Complaints

- Nausea
- Lethargy
- Body ache
- Reduced physical activity
- Mild exertional breathlessness

History of Present Illness

The patient was apparently healthy before chemotherapy. Following multiple chemotherapy cycles, the patient gradually developed generalized weakness, anorexia, nausea, and decreased urinary output. Pedal edema and fatigue progressively increased over time. Laboratory investigations revealed marked elevation of serum creatinine and blood urea levels suggestive of severe renal dysfunction. The patient sought Ayurvedic treatment for supportive renal care and improvement in quality of life.

Past History

- No previous history of chronic kidney disease
- No history of diabetes mellitus
- No history of hypertension
- No major surgical history
- History of 4 cycles of chemotherapy (Carboplatin & Gemcitabine) for Ca Urinary Bladder

Personal History

Table 2: Details of Personal History

Parameter	Findings
Diet	Mixed
Appetite	Poor
Sleep	Disturbed
Bowel	Irregular
Micturition	Reduced quantity
Addiction	Absent

General Examination

Table 3: General Examination of the Patient at Baseline

Parameter	Findings
Pulse	84/min
Blood Pressure	126/84 mmHg
Temperature	Afebrile
Respiratory Rate	18/min
Pallor	Present
Pedal edema	Present

Parameter	Findings
Cyanosis	Absent
Clubbing	Absent
Icterus	Absent

Ashtavidha Pariksha

Table 4: Assessment of the Patient According to Ashtavidha Pariksha

Examination	Findings
Nadi	Vata-Pitta dominant
Mutra	Reduced quantity
Mala	Vibandha
Jihva	Slightly coated
Shabda	Normal
Sparsha	Slightly dry skin
Drik	Mild pallor
Aakruti	Madhyama

Dashavidha Pariksha

Table 5: Dashavidha Pariksha Evaluation of the Case

Parameter	Findings
Prakriti	Vata-Pitta
Vikriti	Vata predominant
Sara	Madhyama
Samhanana	Madhyama
Pramana	Madhyama
Satmya	Madhyama
Satva	Madhyama
Ahara Shakti	Avara
Vyayama Shakti	Avara
Vaya	Vriddha

Systemic Examination

Cardiovascular System: S1 and S2 heard normally. No added sounds.

Respiratory System: Bilateral air entry present equally.

Central Nervous System: Patient conscious and oriented.

Gastrointestinal System: Abdomen soft and non-tender.

Ayurvedic Diagnosis

- Mutravaha Srotodushti
- Basti Vikara

- Vata-pradhana Tridoshaja Vyadhi
- Dhatukshaya Janya Vyadhi

Investigations

Table 6: Initial Laboratory Investigations (25/01/2026)

Investigation	Result	Reference Range
Blood Urea	115.8 mg/dL	10–45 mg/dL
Serum Creatinine	5.90 mg/dL	0.70–1.30 mg/dL
Uric Acid	9.47 mg/dL	2.5–7.2 mg/dL
Hemoglobin	5.4 gm/dL	13–17 gm/dL
Serum Phosphorus	5.18 mg/dL	2.5–4.5 mg/dL
Potassium	4.52 mmol/L	3.5–5.5 mmol/L

These findings indicated **severe renal dysfunction** associated with **anemia** and **metabolic abnormalities**.

Treatment Protocol

Panchakarma Management

Kala Basti – It was administered according to classical principles.

Niruha Basti (N)

Trinpanchamoola Kwatha Basti content –

- Saindhava Lavana – 12 gms
- Honey – 30 gms
- Sneha (Chandana Bala Taila) – 40 ml
- Trinpanchamoola Kwatha – 450 ml
- Kalka Dravaya (Amalaki Powder) – 10 gms

Anuvasana Basti (A)

Chandana Bala Taila – 60 ml

Table 7: Kala Basti Schedule

Date	Basti Administered
27-01-26	A – Anuvasana Basti
28-01-26	N – Niruha Basti
29-01-26	A – Anuvasana Basti
30-01-26	N – Niruha Basti
31-01-26	A – Anuvasana Basti
01-02-26	N – Niruha Basti
02-02-26	A – Anuvasana Basti
03-02-26	N – Niruha Basti
04-02-26	A – Anuvasana Basti
05-02-26	N – Niruha Basti

Date	Basti Administered
06-02-26	A – Anuvasana Basti
07-02-26	N – Niruha Basti
08-02-26	A – Anuvasana Basti
09-02-26	N – Niruha Basti
10-02-26	A – Anuvasana Basti
11-02-26	A – Anuvasana Basti

Shamana Chikitsa

Table 8: Details of Shamana Chikitsa Prescribed to the Patient

Medicine	Dose	Duration
1. Cap. Keedajadi	1 gm twice a day	30 days
2. Syp. Neeri KFT	15 mL thrice a day	30 days
3. Tab. Amyron	500 mg twice a day	30 days

Follow-Up Medicines

Table 9: Details of Medicines Administered During the Follow-Up Period

Medicine	Dose	Duration
1. Erand Bhrashta Haritaki	1 gm at bed hours	5 days
2. Cap. Keedajadi	1 gm twice a day	10 days
3. Punarnava Mandura	250 mg twice a day	10 days

Observation and Results

Clinical Improvement

Table 10: Comparative Assessment of Laboratory Parameters Before Treatment, After Treatment, and During Follow-Up

Parameter	Before Treatment (25/01/2026)	After Treatment (26/02/2026)	Follow-Up (09/03/2026)
Blood Urea	115.8 mg/dL	96.5 mg/dL	58.4 mg/dL
Serum Creatinine	5.90 mg/dL	1.94 mg/dL	1.70 mg/dL
Uric Acid	9.47 mg/dL	6.94 mg/dL	5.39 mg/dL
Hemoglobin	5.4 gm/dL	8.1 gm/dL	10.2 gm/dL

Subjective Assessment of Clinical Parameters[7]

Table 11: Subjective Assessment of Clinical Parameters Before Treatment, After Treatment, and During Follow-Up

Clinical Parameter	Before Treatment (25/01/2026)	After Treatment (26/02/2026)	Follow-Up (09/03/2026)
Kshudha Hani (Loss of Appetite)	3	1	0
Daurbalya (Generalized Weakness)	3	1	1
Nidra Vaikrita (Disturbed Sleep)	2	0	0
Pada Shotha (Pedal Edema)	2	0	0

DISCUSSION

Chemotherapy-induced Acute Kidney Injury develops due to oxidative stress, endothelial dysfunction, inflammatory mediator release, and direct tubular epithelial injury, which collectively impair renal perfusion and urinary excretory function.[8] Persistent exposure to nephrotoxic chemotherapeutic agents leads to accumulation of metabolic waste products and progressive deterioration of renal physiology. According to Ayurveda, chemotherapy can be considered an Agantuja Nidana that initiates Agnimandya resulting in improper digestion and metabolism at both Jatharagni and Dhatwagni levels.[9] Impaired metabolic activity further leads to Ama formation, which circulates through the body and produces obstruction in various Srotasa. Simultaneously, continuous tissue injury and systemic toxicity cause Dhatukshaya and Ojakshaya, leading to aggravation of Vata Dosha. The aggravated Vata along with Ama produces Srotorodha predominantly in Mutravaha Srotas, ultimately impairing urinary function and renal physiology.[10] As the pathological process progresses, accumulation of metabolic toxins occurs leading to renal dysfunction manifested clinically as reduced urinary output, pedal edema, generalized weakness, elevated serum creatinine, and increased blood urea levels.[11]

The treatment protocol was planned with the objective of correcting Agnimandya, removing Ama, regulating aggravated Vata Dosha, clearing obstruction in Mutravaha Srotas, improving urinary function, and restoring Dhatu Bala. Kala Basti along with Shamana Chikitsa acted at multiple levels of the disease process and helped in reversing the pathological changes induced by chemotherapy.

Role of Kala Basti

Kala Basti was selected as the principal Panchakarma intervention because Basti is considered the best treatment for Vata predominant disorders. In the present case, chemotherapy-induced tissue depletion, metabolic toxicity, and obstruction in Mutravaha Srotas resulted in aggravation of Apana Vata leading to impaired urinary physiology. Kala Basti helped in regulating Apana Vata, removing Srotorodha, promoting proper elimination of metabolic waste products, and restoring normal urinary function.[12]

The sequential administration of Niruha and Anuvasana Basti provided both Shodhana and Brimhana effects. Niruha Basti helped eliminate accumulated Doshas and toxins, whereas Anuvasana Basti nourished depleted Dhatus and normalized Vata Dosha.[13] Through systemic absorption and neurohumoral regulation, Kala Basti improved renal circulation, reduced inflammation, corrected fluid imbalance, and enhanced overall physiological functioning.[14]

Mode of Action of Basti Drugs

Trinpanchamoola Kwatha, containing Kusha, Kasha, Shara, Darbha, and Ikshu, was used as the Niruha Basti Dravya because of its Mutrala, Shothahara, Vata-Kapha Shamaka, and Srotoshodhaka properties.[15] The formulation primarily acted on Mutravaha Srotas by relieving Srotorodha and facilitating proper urinary excretion. Its Mutrala action helped improve urinary output and eliminate accumulated metabolic toxins from the body, thereby reducing renal load in chemotherapy-induced Acute Kidney Injury. The Shothahara property contributed to reduction of inflammation and pedal edema associated with renal dysfunction.

Due to its Ushna Virya and Vata-Kapha Shamaka action, the formulation helped regulate aggravated Apana Vata and restore normal urinary physiology. The Laghu and Ruksha Guna

aided in clearing channel obstruction and improving microcirculation within the renal system. Trinpanchamoola Kwatha also acted on Rakta and Mutravaha Srotas by reducing internal toxicity, improving circulation, and supporting filtration and elimination functions of the kidneys. Additionally, its Vedanasthapana and anti-inflammatory actions helped reduce tissue irritation and oxidative stress produced by chemotherapeutic toxicity, thereby supporting gradual recovery of renal function.[16]

Chandana Bala Taila was used as Anuvasana Basti because of its Pittashamaka, Balya, Brimhana, Rasayana, Vatahara, and Dahaprashamana properties. The formulation provided systemic nourishment and rejuvenation to tissues depleted due to chemotherapy-induced toxicity. Bala acted as a potent Balya and Brimhana drug, helping improve muscle strength, vitality, and Dhatu integrity, while Chandana exerted Pittashamana and anti-inflammatory effects that reduced cellular irritation, burning sensation, and inflammatory changes associated with metabolic toxicity.[17]

The Sneha property of the Taila helped normalize aggravated Vata Dosha and improved microcirculation within the body. Through its Rasayana and Dhatu Poshaka actions, the formulation supported tissue regeneration, restoration of physiological balance, and improvement of Ojas.[18] Chandana Bala Taila also acted on Rakta and Mutravaha Srotas by reducing internal dryness, improving circulation, and supporting normal urinary physiology. Administration through Anuvasana Basti further enhanced nourishment of depleted tissues and contributed to overall improvement in strength, quality of life, and recovery from chemotherapy-induced debility.

Mode of Action of Internal Medicines

Cap. Keedajadi, containing single herb Keedajadi (Cordyceps sinensis), was administered for its Mutrala, Nephroprotective, Rasayana, Shothahara, and Vata-Pitta Shamaka properties. The formulation primarily acted by improving glomerular filtration and enhancing urinary excretion, thereby facilitating elimination of accumulated nitrogenous waste products and metabolic toxins from the body. Its Mutrala action helped improve urinary output and maintain fluid balance, while the Shothahara property reduced renal inflammation and pedal edema associated with chemotherapy-induced Acute Kidney Injury.[19]

The Rasayana effect of the formulation supported regeneration and protection of renal tissue from further chemotherapeutic damage. It also helped stabilize renal microcirculation and reduce oxidative stress-induced nephron injury. By balancing aggravated Vata and Pitta Dosha, Cap. Keedajadi restored normal renal physiology and improved the functional integrity of Mutravaha Srotas. The formulation further contributed to reduction in systemic weakness, improvement in strength, and enhancement of overall recovery and quality of life.

Syrup Neeri KFT contains important herbal ingredients such as Punarnava, Gokshura, Varuna, Pashanbheda, Palasha, Apamarga, and other Mutravirechaneeya and nephroprotective drugs. The formulation was administered for its diuretic, antioxidant, anti-inflammatory, and urinary system tonic properties in the management of chemotherapy-induced Acute Kidney Injury.[20]

The formulation primarily acted on Mutravaha Srotas by improving urinary output and facilitating elimination of accumulated metabolic waste products including urea and creatinine. Punarnava exerted potent Mutrala and Shothahara effects, thereby reducing pedal edema and

regulating fluid balance.[21] Gokshura and Varuna helped improve urinary flow, support renal function, and reduce obstruction within the urinary channels. Pashanbheda contributed to Srotoshodhana and helped maintain proper filtration and excretory function of the kidneys.[22]

The antioxidant and anti-inflammatory properties of the formulation helped reduce oxidative stress and inflammatory changes produced by chemotherapeutic toxicity, thereby protecting renal tubular cells from further injury. The combined action of the ingredients improved renal microcirculation, reduced Srotorodha, and supported recovery of damaged nephrons. Through its nephroprotective, Mutrala, and Rasayana effects, Syrup Neeri KFT significantly contributed to restoration of renal function, improvement in urinary physiology, and enhancement of overall patient recovery.

Tab. Amyron containing ingredients such as Ashwagandha, Shatavari, Amalaki, Guduchi, Gokshura, and other Rasayana and Balya drugs, was administered for its Balya, Rasayana, Dhatu Poshaka, Ojovardhaka, and Brimhana properties. The formulation acted as a restorative and rejuvenating therapy in the management of chemotherapy-induced debility and renal dysfunction.

Chemotherapy-induced Dhatukshaya and Ojakshaya resulted in fatigue, weakness, reduced vitality, and poor quality of life, which were effectively addressed through the Balya and Brimhana actions of Amyron. Ashwagandha and Shatavari helped improve strength, stamina, tissue nourishment, and immunity, while Amalaki and Guduchi exerted antioxidant and Rasayana effects that protected tissues from oxidative stress and supported cellular recovery. Gokshura contributed to maintenance of urinary system function and supported Mutravaha Srotas.[23]

The formulation improved metabolic activity, enhanced tissue repair, and promoted gradual restoration of depleted Dhatus. Through its Ojovardhaka and Rasayana effects, Amyron helped improve appetite, energy levels, physical strength, and overall well-being. Its nourishing and rejuvenative action significantly contributed to recovery from chemotherapy-induced weakness and improvement in quality of life.

Punarnava Mandura was administered as a follow-up medicine because of its Mutrala, Shothahara, Panduhara, Rasayana, and Deepana-Pachana properties.[24] The formulation helped reduce pedal edema, improve urinary output, and restore renal function in the patient suffering from chemotherapy-induced Acute Kidney Injury. Punarnava acted as a potent diuretic and anti-inflammatory drug, thereby regulating fluid balance and reducing swelling associated with renal dysfunction. Mandura helped improve hemoglobin levels and tissue oxygenation, which supported recovery from chemotherapy-induced weakness and anemia.

The formulation also improved Agni and reduced Ama formation through its Deepana-Pachana action, thereby enhancing metabolism and tissue nourishment. From an Ayurvedic perspective, Punarnava Mandura acted on Rakta and Mutravaha Srotas by reducing Srotorodha, improving circulation, and facilitating elimination of metabolic toxins. Its Rasayana effect further helped nourish depleted Dhatus, improve strength, and support gradual restoration of renal function and overall quality of life.

Eranda Bhrashta Haritaki acted at the level of Rakta and Mutravaha Srotas by reducing Srotorodha and facilitating proper elimination of metabolic waste products. The Vatanulomana

and Anulomana properties helped regulate Apana Vata, thereby improving normal excretory functions and urinary flow. Haritaki contributed to Ama Pachana and Srotoshodhana, which helped clear obstruction within the channels and improve metabolic activity. Eranda Taila supported detoxification and maintained proper bowel evacuation, indirectly reducing toxin accumulation in Rakta Dhatu. The combined effect of the formulation helped restore physiological balance in Rakta and Mutravaha Srotas, improve circulation, reduce internal toxicity, and support renal function recovery.[25]

Overall Therapeutic Effect of Treatment

The combined administration of Kala Basti and Shamana Chikitsa produced significant clinical and biochemical improvement in the patient. The treatment helped achieve Vatanulomana and Srotoshodhana, thereby relieving obstruction in Mutravaha Srotas and restoring normal urinary physiology. Marked reduction in renal inflammation, pedal edema, and accumulation of metabolic toxins was observed along with improvement in urinary output and correction of fluid imbalance. The Rasayana and Dhatu Poshaka effects of the therapy helped improve strength, appetite, and overall quality of life. Significant improvement in renal function parameters, including serum creatinine and blood urea levels, indicated the nephroprotective and restorative potential of the Ayurvedic treatment protocol.

Thus, the treatment protocol effectively interrupted the pathogenesis of chemotherapy-induced Acute Kidney Injury and supported recovery through Ayurvedic principles of Dosha Shamana, Srotoshodhana, and Rasayana therapy.

CONCLUSION

Kala Basti along with Shamana Chikitsa demonstrated significant clinical and biochemical improvement in chemotherapy-induced Acute Kidney Injury. The treatment helped improve renal parameters, urinary output, appetite, edema, strength, and overall quality of life.

Ayurvedic management based on Samhita Siddhanta principles may serve as an effective supportive therapy in nephrotoxic complications associated with chemotherapy through Vatanulomana, Srotoshodhana, Mutrala, Rasayana, and Dhatu Poshaka effects.

Further clinical studies on larger sample sizes are recommended for scientific validation.

PATIENT CONSENT

Written informed consent was obtained from the patient for publication of clinical details and laboratory investigations.

ETHICAL CONSIDERATION

The treatment was conducted according to standard Ayurvedic clinical practice guidelines with patient consent.

CONFLICT OF INTEREST

None declared.

SOURCE OF FUNDING

No external funding was received for this study.

Declaration of Generative AI and AI-Assisted Technologies in the Writing Process

During manuscript preparation, the authors utilized AI-Tool for language editing, grammar correction, and improvement of manuscript presentation. The authors critically reviewed and revised all AI-generated content and accept full responsibility for the final version of the manuscript. AI tools were not used for data collection, data analysis, interpretation of results, or generation of scientific conclusions.

REFERENCES

1. Awdishu L, Wu S. Acute kidney injury. Renal/Pulmonary Critical Care. 2017; 2:7-26.
2. Hałka J, Spaleniak S, Kade G, Antosiewicz S, Sigorski D. The nephrotoxicity of drugs used in causal oncological therapies. Current Oncology. 2022 Dec 8;29(12):9681-94.
3. Konstantinidis I, Tsokkou S, Gavriilaki E, Delis G, Papamitsou T. Protective role of key micronutrients in chemotherapy-induced organ toxicity: a comprehensive review of mechanistic insights and clinical implications. Nutrients. 2025 Aug 31;17(17):2838.
4. Banthanal P, Sneha K, Kulkarni M. An integrative approach in understanding and managing cancer.
5. Teggi SM. A Study of Basti According to Therapeutic Action with Reference to Bruhatreyee (Doctoral dissertation, Rajiv Gandhi University of Health Sciences (India)).
6. Dwivedi PC. Basti – An Ayurvedic Concept. Chyren Publication; 2025 Nov 20.
7. Prashanth GS, Baghel MS, Ravishankar B, Gupta SN, Mehta MP. A clinical comparative study of the management of chronic renal failure with Punarnavadi compound. AYU (An International Quarterly Journal of Research in Ayurveda). 2010 Apr 1;31(2):185-92.
8. Lyrio RM, Rocha BR, Corrêa AL, Mascarenhas MG, Santos FL, Maia RD, Segundo LB, de Almeida PA, Moreira CM, Sassi RH. Chemotherapy-induced acute kidney injury: epidemiology, pathophysiology, and therapeutic approaches. Frontiers in Nephrology. 2024 Aug 9; 4:1436896.
9. Banthanal P, Sneha K, Kulkarni M. An integrative approach in understanding and managing cancer. World Journal of Pharmaceutical Research. 2026;15(1):720-728. ISSN: 2277-7105.
10. Sasane PU, Chandel A, Sahu SK, Nampalliwar A, Sinha CP, Namboothiri KP. Ayurvedic Management of Chronic Kidney Disease: Classical Texts, Herbal Interventions, and Clinical Evidence. KIDNEYS. 2026 Jan 30;15(1):54-64.
11. Nyarko JA, Larbie C, Ossei PP. Nephroprotective Activity of Dialium guineense Aqueous Extract Against Cisplatin-Induced Kidney Damage in Rats. Microbiol Infect Dis. 2024;8(1):1-7.
12. Basapur SN. A Critical Study on Mutravahasrotodusti vis-a-vis Urinary Tract Infections with Special Emphasis on Cystitis & Its Management Through Ayurvedic Anubhuta Yoga (Doctoral dissertation, Rajiv Gandhi University of Health Sciences (India)).
13. Dwivedi PC. Basti – An Ayurvedic Concept. Chyren Publication; 2025 Nov 20.
14. Sethi S, Borannavar S. Reviving Renal Function Through Punarnavadi Kashaya Basti – A Noteworthy Reduction in BUN (Blood Urea Nitrogen) & Serum Creatinine. World. 2026;5(1).
15. Dwivedi D, Mishra PK, Pandey AK. Management of chronic kidney disease through Ayurveda: A case study. World Journal of Pharmacy and Pharmaceutical Sciences. 2017;6(2):1318-1325. ISSN: 2278-4357.
16. Dwivedi D, Mishra PK, Pandey AK. Management of chronic kidney disease through Ayurveda medicine KF-7 & Urocid tablets: A case study. World Journal of Pharmacy and Pharmaceutical Sciences. 2017;6(2):1318-1325. doi:10.20959/wjpps20172-8537.

17. Pokhrel SM, Sharma A, Singh M. Uttarabasti in bladder neck contracture following transurethral resection of prostate: A case report. Journal of Ayurveda and Integrative Medicine. 2023 Jul 1;14(4):100772.
18. Jaitwar AP, Jatav R, Verma R. Holistic healing: the principles and practices of ayurvedic medicine. Academic Guru Publishing House; 2024 Sep 24.
19. Aanchal G, Kant SC, Monika S. Enhanced production of Cordyceps militaris fruiting bodies using brown rice substrates for therapeutic purposes. Research Journal of Biotechnology. 2025 Jan;20:1.
20. Gautam G, Parveen B, Khan MU, Sharma I, Sharma AK, Parveen R, Ahmad S. A systematic review on nephron protective AYUSH drugs as constituents of NEERI-KFT (A traditional Indian polyherbal formulation) for the management of chronic kidney disease. Saudi Journal of Biological Sciences. 2021 Nov 1;28(11):6441-53.
21. Bhowmik D, Kumar KS, Srivastava S, Paswan S, Sankar A, Dutta D. Traditional Indian herbs Punarnava and its medicinal importance. Journal of Pharmacognosy and Phytochemistry. 2012 May 1;1(1):52-7.
22. Shukla BP. A Comparative Clinical Study in the Management of Mutrashmari Through Pashanabhedadi Kashaya and Virataradi Kashaya WSR to Urolithiasis (Doctoral dissertation, Rajiv Gandhi University of Health Sciences (India)).
23. Mingrone G, Greco AV, Capristo E, Benedetti G, Giancaterini A, Gaetano AD, Gasbarrini G. L-carnitine improves glucose disposal in type 2 diabetic patients. Journal of the American College of Nutrition. 1999 Feb 1;18(1):77-82.
24. Yaji VV. A Clinical Comparative Study on Efficacy of Gudadya Gutika and Punarnava Mandura in Iron Deficiency Anaemia WSR Pandu (Doctoral dissertation, Rajiv Gandhi University of Health Sciences (India)).
25. Gandhi AJ. Industrial approach of process validation of Bharjana (frying) process WSR to Eranda Bhrishta Haritaki: An experimental observation. International Journal of Green Pharmacy (IJGP). 2016;10(04).

REPORT

Patient ID : OMC26/509
[redacted]
Age/Gender : 56 Yrs/Male
Ref. Dr. :

Sample ID : 2601509
Registration Date : 25/01/2026 12:23 PM
Report Date : 25/01/2026 01:09 PM

HAEMATOLOGY REPORT

Test Description	Result	Unit	Biological Reference Range
COMPLETE BLOOD COUNT			
Haemoglobin	5.4	gm/dL	13.0 - 17.0
Total WBC Count	7100	cell/cu.mm	4000 - 11000
Rbc	2.00	mill./cu.mm	4.35 - 5.50
RBC Indices			
Hematocrit HCT	15.1	%	36 - 48
Mean Corp Volume MCV	75.5	fL	83 - 101
Mean Corp Hb MCH	27.0	pg	27 - 32
Mean Corp Hb Conc MCHC	35.8	gm/dL	31.5 - 34.5
RDW-CV	18.0	%	11.0 - 16.0
Platelet Count	1.40	lac/cmm	1.5 - 4.5
DIFFERENTIAL LEUCOCYTE COUNT			
Neutrophils	80	%	40 - 70
Lymphocytes	13	%	20 - 40
Monocytes	05	%	02 - 08
Eosinophils	02	%	01 - 04
Basophils	00	%	00 - 01
Absolute Differential Count			
Absolute Neutrophils Count	5680	/cumm	2000 - 7000
Absolute Lymphocyte Count	923	/cumm	1000 - 3000
Absolute Eosinophil Count	142	/cumm	40 - 440
Absolute Monocyte Count	355	/cumm	200 - 1000

NOTE:
1. As per the recommendation of International council for Standardization in Hematology, the differential leukocyte counts are additionally being reported as absolute numbers of each cell in per unit volume of blood.
2. Test conducted on EDTA whole blood.

DR. KARAN BHUTANI
MBBS., MD (Med.)
Holy Heart Superspeciality & Trauma Centre Rohtak
Reg. No. HR110827

Figure 1: Laboratory Investigation Report

Patient ID : OMC26/509
[redacted]
Age/Gender : 55 Yrs/Male
Ref. Dr. :

Sample ID : 2601509
Registration Date : 25/01/2026 12:23 PM
Report Date : 25/01/2026 01:09 PM

BIOCHEMISTRY REPORT

Test Description	Result	Unit	Biological Reference Range
KIDNEY FUNCTION TEST(KFT)			
Urea	**115.8**	mg/dl	10.0 - 45.0
Serum Creatinine	**5.90**	mg/dl	0.70 - 1.30
Uric Acid	**9.47**	mg/dl	2.5 - 7.2
Sodium	142.6	mmol/L	135 - 146
Potassium	4.52	mmol/L	3.5 - 5.5
Calcium	8.93	mg/dl	8.5 - 10.7
Blood Urea Nitrogen-BUN	**46.3**	mg/dl	7 - 20
S.Chloride	105.5	mmol/L	98 - 107
S.Phosphorous	**5.18**	mg/dl	2.5 - 4.5

Interpretation :
Kidney function tests help to screen the individual for renal disease and to determine the extent or progression of renal disease. These tests also aid in determining drug dosage for drugs excreted through the kidneys. The clinical syndrome resulting from decreased renal function and azotemia is called uremia Renal azotemia: glomerular nephritis and chronic pyelonephritis. Prerenal azotemia: severe dehydration, hemorrhagic shock, and excessive protein intake. Post renal azotemia: urethral stones or tumors and prostatic obstructions Measurement of urea in dialysis fluids is widely used in assessing the adequacy of renal replacement therapy.

In these prerenal situations, the plasma creatinine concentration may be normal.In obstructive post renal conditions, both plasma creatinine and urea concentrations will be increased, although there is often a greater increase in plasma urea than creatinine because of the increased back diffusion.These considerations give rise to the principal clinical utility of plasma urea, which lies in its measurement in conjunction with that of plasma creatinine and subsequent calculation of the urea nitrogen/creatinine ratio. This ratio has been used as a crude discriminator between prerenal and postrenal azotemia. Significantly lower ratios usually denote (1) acute tubular necrosis, (2) low protein intake, (3) starvation, or (4) severe liver disease

DR. KARAN BHUTANI
MBBS., MD (Med.)
Holy Heart Superspeciality
& Trauma Centre Rohtak
Reg. No. HN110607

Figure 2: Laboratory Investigation Report

Patient ID : OMC26/1367
[redacted]
Age/Gender : 54 Yrs/Male
Ref. Dr. :

Sample ID : 2602703
Registration Date : 26/02/2026 09:39 AM
Report Date : 26/02/2026 03:38 PM

HAEMATOLOGY REPORT

Test Description	Result	Unit	Biological Reference Range
COMPLETE BLOOD COUNT			
Haemoglobin	**8.1**	gm/dL	13.0 - 17.0
Total WBC Count	**42700**	cell/cu.mm	4000 - 11000
Rbc	**2.77**	milli./cu.mm	4.35 - 5.50
RBC Indices			
Hematocrit HCT	**22.3**	%	36 - 46
Mean Corp Volume MCV	**80.5**	fL	83 - 101
Mean Corp Hb MCH	29.2	pg	27 - 32
Mean Corp Hb Conc MCHC	**36.3**	gm/dL	31.5 - 34.5
RDW-CV	**19.5**	%	11.0 - 16.0
Platelet Count	**5.75**	lac/cmm	1.5 - 4.5
DIFFERENTIAL LEUCOCYTE COUNT			
Neutrophils	**94**	%	40 - 70
Lymphocytes	**04**	%	20 - 40
Monocytes	**01**	%	02 - 08
Eosinophils	01	%	01 - 04
Basophils	00	%	00 - 01
Absolute Differential Count			
Absolute Neutrophils Count	**40138**	/cumm	2000 - 7000
Absolute Lymphocyte Count	1708	/cumm	1000 - 3000
Absolute Eosinophil Count	427	/cumm	40 - 440
Absolute Monocyte Count	427	/cumm	200 - 1000

NOTE:
1. As per the recommendation of International council for Standardization in Hematology, the differential leukocyte counts are additionally being reported as absolute numbers of each cell in per unit volume of blood.
2. Test conducted on EDTA whole blood.

DR. KARAN BHUTANI
MBBS., MD (Med.)
Holy Heart Superspeciality
& Trauma Centre Rohtak
Reg. No. HN110607

Figure 3: Laboratory Investigation Report

Patient ID : OMC26/1367
[redacted]
Age/Gender : 54 Yrs/Male
Ref. Dr. :

Sample ID : 2602703
Registration Date : 26/02/2026 09:39 AM
Report Date : 26/02/2026 03:38 PM

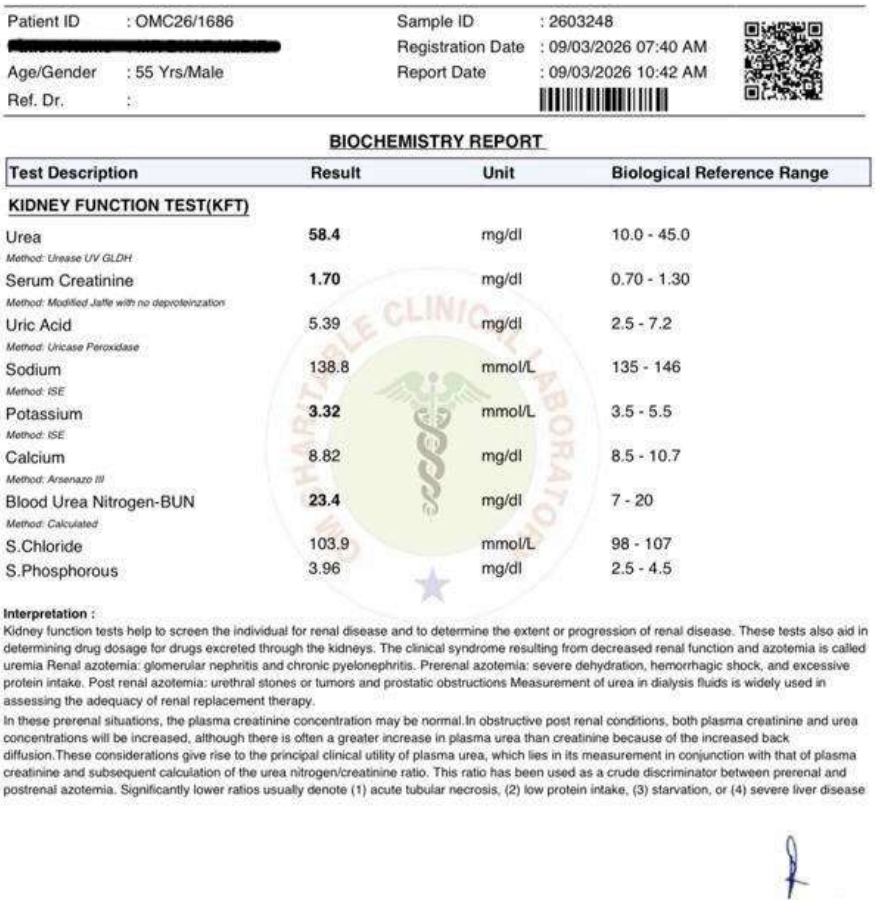
BIOCHEMISTRY REPORT

Test Description	Result	Unit	Biological Reference Range
KIDNEY FUNCTION TEST(KFT)			
Urea	**96.5**	mg/dl	10.0 - 45.0
Method: Urease UV GLDH			
Serum Creatinine	**1.94**	mg/dl	0.70 - 1.30
Method: Modified Jaffe with no deproteinzation			
Uric Acid	6.94	mg/dl	2.5 - 7.2
Method: Uricase Peroxidase			
Sodium	**130.4**	mmol/L	135 - 146
Method: ISE			
Potassium	**5.58**	mmol/L	3.5 - 5.5
Method: ISE			
Calcium	8.72	mg/dl	8.5 - 10.7
Method: Arsenazo III			
Blood Urea Nitrogen-BUN	**38.6**	mg/dl	7 - 20
Method: Calculated			
S.Chloride	103.8	mmol/L	98 - 107
S.Phosphorous	4.50	mg/dl	2.5 - 4.5

Interpretation :
Kidney function tests help to screen the individual for renal disease and to determine the extent or progression of renal disease. These tests also aid in determining drug dosage for drugs excreted through the kidneys. The clinical syndrome resulting from decreased renal function and azotemia is called uremia Renal azotemia: glomerular nephritis and chronic pyelonephritis. Prerenal azotemia: severe dehydration, hemorrhagic shock, and excessive protein intake. Post renal azotemia: urethral stones or tumors and prostatic obstructions Measurement of urea in dialysis fluids is widely used in assessing the adequacy of renal replacement therapy.

In these prerenal situations, the plasma creatinine concentration may be normal.In obstructive post renal conditions, both plasma creatinine and urea concentrations will be increased, although there is often a greater increase in plasma urea than creatinine because of the increased back diffusion.These considerations give rise to the principal clinical utility of plasma urea, which lies in its measurement in conjunction with that of plasma creatinine and subsequent calculation of the urea nitrogen/creatinine ratio. This ratio has been used as a crude discriminator between prerenal and postrenal azotemia. Significantly lower ratios usually denote (1) acute tubular necrosis, (2) low protein intake, (3) starvation, or (4) severe liver disease

Figure 4: Laboratory Investigation Report



Patient ID : OMC26/1686
[redacted]
Age/Gender : 55 Yrs/Male
Ref. Dr. :

Sample ID : 2603248
Registration Date : 09/03/2026 07:40 AM
Report Date : 09/03/2026 10:42 AM

BIOCHEMISTRY REPORT

Test Description	Result	Unit	Biological Reference Range
KIDNEY FUNCTION TEST(KFT)			
Urea	**58.4**	mg/dl	10.0 - 45.0
Method: Urease UV GLDH			
Serum Creatinine	**1.70**	mg/dl	0.70 - 1.30
Method: Modified Jaffe with no deproteinzation			
Uric Acid	5.39	mg/dl	2.5 - 7.2
Method: Uricase Peroxidase			
Sodium	138.8	mmol/L	135 - 146
Method: ISE			
Potassium	**3.32**	mmol/L	3.5 - 5.5
Method: ISE			
Calcium	8.82	mg/dl	8.5 - 10.7
Method: Arsenazo III			
Blood Urea Nitrogen-BUN	**23.4**	mg/dl	7 - 20
Method: Calculated			
S.Chloride	103.9	mmol/L	98 - 107
S.Phosphorous	3.96	mg/dl	2.5 - 4.5

Interpretation :
Kidney function tests help to screen the individual for renal disease and to determine the extent or progression of renal disease. These tests also aid in determining drug dosage for drugs excreted through the kidneys. The clinical syndrome resulting from decreased renal function and azotemia is called uremia Renal azotemia: glomerular nephritis and chronic pyelonephritis. Prerenal azotemia: severe dehydration, hemorrhagic shock, and excessive protein intake. Post renal azotemia: urethral stones or tumors and prostatic obstructions Measurement of urea in dialysis fluids is widely used in assessing the adequacy of renal replacement therapy.

In these prerenal situations, the plasma creatinine concentration may be normal.In obstructive post renal conditions, both plasma creatinine and urea concentrations will be increased, although there is often a greater increase in plasma urea than creatinine because of the increased back diffusion.These considerations give rise to the principal clinical utility of plasma urea, which lies in its measurement in conjunction with that of plasma creatinine and subsequent calculation of the urea nitrogen/creatinine ratio. This ratio has been used as a crude discriminator between prerenal and postrenal azotemia. Significantly lower ratios usually denote (1) acute tubular necrosis, (2) low protein intake, (3) starvation, or (4) severe liver disease

Figure 5: Laboratory Investigation Report