Research Article

iMedPub Journals
www.imedpub.com

Journal of Renal Medicine

2025
Vol.8 No.2:21

Clinical Profile of Onconephrology in a Tertiary Care Center in South India

Sowmiya Mohan*

Department of Nephrology, Government Stanley Medical College Hospital, Chennai, India

***Corresponding author:** Sowmiya Mohan, Department of Nephrology, Government Stanley Medical College Hospital, Chennai, India; E-mail: sowmiyamohan22@gmail.com

Received date: September 25, 2024, Manuscript No. IPJRM-24-19710; **Editor assigned date:** September 27, 2024, PreQC No. IPJRM-24-19710 (PQ); **Reviewed date:** October 11, 2024, QC No. IPJRM-24-19710; **Revised date:** April 14, 2025, Manuscript No. IPJRM-24-19710 (R); **Published date**: April 21, 2025, DOI: 10.36648/ipjrm.8.2.21

Citation: Mohan S (2025) Clinical Profile of Onconephrology in a Tertiary Care Center in South India. J Ren Med Vol:8 No:2

Abstract

Patients diagnosed with cancer often face significant challenges beyond the disease itself, with acute or chronic kidney damage frequently emerging as a complication due to the cancer or its treatment protocols. This intersection of cancer and kidney damage is increasingly being explored within the field of onconephrology.

Keywords: Acute Kidney Injury (AKI); KDIGO (Kidney Disease Improving Global Outcomes); Onconephrology; Pathological mechanisms

Introduction

Specifically, Acute Kidney Injury (AKI) occurs with both solid tumors and hematologic malignancies, and the causes of AKI can be categorized as follows: Prerenal (e.g., volume depletion, hypercalcemia), intrarenal (e.g., cancer invasion, treatment toxicities), and postrenal (e.g., retroperitoneal or bladder cancer) [1,2]. The incidence of AKI in cancer patients is significant, as evidenced by previous large-scale studies [3]. For instance, a study involving over 37,000 patients with cancer in Denmark reported that 25.8% of cancer patients developed Acute Kidney Injury (AKI), with the risk peaking at 17.5% during the first year after diagnosis and rising to 27.0% over a five-year period [3].

This complex relationship between cancer and kidney damage underscores the need for integrated approaches to effectively manage both cancer and renal health.

This research paper aims to investigate the clinical characteristics of AKI among oncology patients. Moreover, this work seeks to explore the specific types of cancer associated with AKI, the underlying pathological mechanisms linked to each cancer type, the prevalence of dialysis dependency in these patients, risk factors contributing to the requirement for Renal Replacement Therapy (RRT), and the electrolyte disturbances associated with these conditions.

Materials and Methods

This 2-year prospective observational study was conducted at Kilpauk Medical College from 2022-2023, during which time we enrolled 120 patients with cancer who met the KDIGO (Kidney Disease Improving Global Outcomes) criteria for AKI, defined as any of the following: Increase in SCr by more than or equal to 0.3 mg/dl within 48 hours or increase in SCr to more than or equal to 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days. The baseline value was defined as the most recent creatinine measurement within one year prior to the cancer diagnosis. The inclusion criteria included the following: Patients who gave informed consent for participation, were aged 18 years and older, and met the KDIGO criteria for AKI with histologically proven carcinoma. We excluded patients who were aged less than 18 years, were diagnosed with carcinoma without histopathological evidence, and those who were not meeting the KDIGO criteria for AKI or declined to provide informed consent.

After selecting the eligible patients, we collected data from them, including a comprehensive patient history encompassing age, sex, comorbidities, type of carcinoma, urine output, previous renal function tests (blood urea, serum creatinine, and electrolytes), abdominal radiological imaging, details of chemotherapy and radiotherapy treatments, and we carried out investigations such as blood urea, serum creatinine and electrolytes, a complete blood count, and liver function tests. Details of any Renal Replacement Therapy (RRT) received were documented. We followed the patients requiring dialysis for a period of 6 months in our dialysis unit from the date of dialysis initiation to examine their dialysis dependency. Statistical analyses were performed using SPSS 2024 software, employing the *Chi-square* test to determine the statistical association between various risk factors for RRT requirements and our sample population with or without RRT requirements.

Results

In our study cohort of 120 patients, the median age was 62 years old. The male-to-female ratio was 3:1, with males accounting for 64% (n=77) and females for 36% (n=43) of the participants.

AKI was identified in different time frames relative to the cancer diagnosis. Specifically, 52% (n=62) of cases of AKI were identified concurrently with the initial cancer diagnosis; 24% (n=29) of cases occurred within 6 months of the cancer diagnosis; and another 24% (n=29) occurred more than 6 months after the cancer diagnosis. Furthermore, upon presentation to the nephrology unit, the AKI severity was staged according to KDIGO staging: Stage 1 being an increase in serum creatinine 1.5-1.9 times from baseline, stage 2 being 2.0-2.9 times baseline, stage 3 being 3.0 times baseline OR an increase in serum creatinine to more than or equal to 4.0 mg/dl OR the initiation of renal replacement therapy. In our study population, stage 1 AKI was seen in 22% (n=26) of cases, stage 2 in 39% of cases (n=47), and stage 3 in 39% of cases (n=47).

In terms of the cancer diagnoses, the most prevalent cancers associated with renal failure in our study were cervical carcinoma (33%; n=33) and prostate carcinoma (13%; n=13). The other malignancies associated with AKI in this sample were penile carcinoma, endometrial carcinoma, ovarian carcinoma, and renal cell carcinoma (Table 1).

Table 1: Percentage of AKI across various carcinomas.

Type of malignancy	Number of cases (n)	Percentage of cases
Cervical carcinoma	40	33
Prostate carcinoma	16	13
Gastrointestinal malignancies	18	15
Oropharyngeal malignancies	12	10
Hematological malignancies	8	7
Solid organ malignancies	14	12
Other	12	10

The pathophysiological cause of AKI was determined for each patient based on clinical judgment. Renal biopsies were performed on 10 patients in our study, revealing light chain deposition disease in 4 cases, lymphomatous infiltration of the kidney in 2 cases, membranous nephropathy in 2 cases, and acute interstitial nephritis in 2 cases. The primary limitation to performing biopsies was thrombocytopenia, which was either disease-related or treatment-induced in these patients. The distribution of causes of AKI were as follows in Table 2 or mesangioproliferative pattern of glomerular injury.

Table 2: Pathophysiological cause of AKI.

Cause of AKI	Number of cases (n)	Percentage of cases
Obstructive nephropathy	52	43
Prerenal AKI	25	21
Drug-induced AKI	12	10
Sepsis-associated AKI	12	10
Tumour lysis syndrome	8	7
Tumour infiltration of the kidney	6	5
Other	5	4

Moreover, the most commonly implicated drugs causing drug-induced AKI were cisplatin, methotrexate, and immune checkpoint inhibitors.

Among the studied population, hyperkalaemia was the most prevalent electrolyte abnormality, as this was observed in 21% of cases (n=25), followed by hyponatremia, which was observed in 15% of cases (n=18). Across the sample, the mean serum creatinine level at the time of nephrology referral was 2.2 mg/dL, the mean hemoglobin level was 7.1 mg/dL, and the mean serum albumin was 3.2 mg/dL.

In terms of dialysis, approximately 35% (n=42) of patients required dialysis due to AKI, with 71% (n=30) of these patients remaining dialysis-dependent at the 6-month follow-up. The mean serum creatinine in the group requiring dialysis was 5.2 mg/dL. Additionally, two-thirds of the patients requiring RRT had obstructive nephropathy. Notably, most patients with drug-

induced AKI did not require dialysis. The risk factors associated with the need for dialysis, apart from traditional indications of RRT, in our study population include AKI caused by obstructive nephropathy in Table 3 as follows.

Table 3: Risk factors for RRT requirement.

Risk factors for RRT requirement	Cases of AKI requiring RRT (n)	Cases of AKI not requiring RRT (n)	P-value
Presence of sepsis	6	6	0.32
Obstructive nephropathy as the cause of AKI	32	20	0.00001
Tumour lysis syndrome	1	7	0.3708
AKI at the time of cancer diagnosis	35	27	0.2381
Drug-induced AKI	2	10	0.12476

Two patients experienced pseudo-AKI (normal kidney function with impaired tubular secretion of creatinine) attributed to palbociclib, with elevated serum creatinine levels detected at their initial consultation. However, upon re-evaluation using their serum cystatin levels, their kidney function was found to be normal. Another patient developed Acute Interstitial Nephritis (AIN) as a result of immune checkpoint inhibitor therapy and showed a favorable response to steroid treatment. On follow-up, out of 32 patients requiring RRT in the obstructive nephropathy group, 28 patients (87%) remained dialysis-dependent after 6 months of initiation.

Discussion

Among hospitalized patients with cancer, AKI is a prevalent complication that significantly impacts healthcare costs, the length of hospital stays, and patient outcomes [3]. Our study contributes to the current understanding of the relationship between cancer and AKI by examining various facets of AKI in the context of an oncological diagnosis, including epidemiology, aetiology, treatment implications, and associated complications.

For instance, critically ill cancer patients have been shown to have a higher incidence of AKI and subsequent dialysis requirements compared to patients with cancer who are not critically ill [4]. In a recent study of 200 patients with high-grade hematological malignancies and AKI, 68.5% had RIFLE-defined (risk, injury, failure, loss, end-stage kidney disease) AKI. Specifically, hemophagocytic lymphohistiocytosis, nephrotoxins, Acute Tubular Necrosis (ATN), hypoperfusion, and tumor lysis syndrome accounted for 91.4% of all patients with AKI [5].

In terms of the risk factors for AKI, advanced age (>65 years old) has consistently been identified as a significant risk factor for AKI in cancer patients, aligning with the results of our study, where the mean age was 62 years [6]. While renal cell carcinoma has historically been associated with AKI in the literature [6,7], obstructive nephropathy emerged as the predominant cause in our cohort, notably driven by cancers such as cervical and prostate carcinomas.

Chemotherapy-related nephrotoxicity remains a prominent contributor to AKI in patients with cancer across a spectrum of renal pathologies, including acute interstitial nephritis and acute tubular injury. The development of novel therapies such as immune checkpoint inhibitors has introduced additional complexities for renal damage, as such therapies cause distinct patterns of nephrotoxicity [8]. In our study, the drugs commonly associated with AKI were platinum compounds such as cisplatin, carboplatin, gemcitabine, ifosfamide, and bevacizumab methotrexate, in line with the work of Campbell et al. [2]. In addition to drugs, contrast agents used in diagnostic procedures have also contributed to AKI [9].

The kidney is responsible for the clearance of several cancer chemotherapy drugs, and the pharmacokinetics of these drugs are changed by AKI, potentially producing unsafe concentrations. Conversely, AKI that necessitates dialysis might result in sub-therapeutic doses of cancer medications and, perhaps, unsuccessful cancer therapy. As a result, even with significantly enhanced chemotherapeutic drugs, AKI may reduce their effectiveness. Consequently, drugs with lower renal toxicity should be taken into consideration when feasible, and kidney function should be regularly checked in these patients.

However, pseudo-AKI due to medication interference, as seen in cases involving palbociclib, highlights the complexities of diagnosing AKI in cancer patients receiving multiple therapies. Such instances also underscore the importance of employing complementary diagnostic tools, such as serum cystatin levels, to accurately assess kidney function in these scenarios.

Tumor lysis syndrome represents a unique and potentially life-threatening cause of AKI, particularly in patients with cancers with high cellular turnover rates. Our study identified TLS meeting the Cairo-Bishop criteria in eight cases, which highlights the importance of vigilance and prompt management to mitigate renal complications associated with rapid tumor cell lysis. Additionally, direct tumor infiltration of the kidneys, while less frequent, was observed in a subset of our patients, and such infiltration contributes to AKI through mechanical compression of the renal structures and the deposition of toxic free light

chains in cases of multiple myeloma. This issue highlights the diagnostic challenges and therapeutic implications of managing AKI in the presence of tumor infiltration.

Glomerular diseases, such as membranous nephropathy, represent another intriguing nexus between cancer and kidney pathology. In our study, membranous nephropathy was associated with carcinoma of the stomach in two cases, underscoring the diverse ways in which malignancies can affect renal function through immune-mediated mechanisms. According to an analysis of the Danish Kidney Biopsy Registry, which comprises all biopsies conducted in Denmark since 1985, individuals diagnosed with glomerulopathy face an increased risk of developing cancer compared to the general population. Specifically, at 1 year following the diagnosis of glomerulopathy, the risk of cancer is elevated by a factor of 2.4 compared to the general population, and between 1 and 4 years following the diagnosis, the risk increases further, with a factor of 3.5 compared to the general population.

Concerning the types of AKI and their prevalences, prerenal AKI is common among cancer patients and can result from intravascular volume depletion due to vomiting or diarrhea, especially in individuals with gastrointestinal or oropharyngeal cancers. Medications causing mucositis can also lead to prerenal AKI. Furthermore, hypercalcemia, which occurs in 20% to 30% of malignancies, can cause prerenal AKI by inducing kidney vasoconstriction and promoting volume depletion through diuresis. In our study, 21% of patients (n=25) had AKI due to prerenal causes, most of which were secondary to gastrointestinal fluid losses and the consequent intravascular volume depletion.

In addition to prerenal AKI, obstructive post renal AKI is more frequent in individuals with cancer than in the general population. In particular, the most prevalent causes of post renal AKI are bladder outlet obstruction and ureteral obstruction. Furthermore, the most common solid organ cancers causing obstructive processes are bladder, prostate, uterine, and cervical cancers. External compression caused by retroperitoneal lymphadenopathy can also produce ureteric obstruction. In our study, cervical carcinoma was the most common cause of obstructive nephropathy, as well as the most common cause of AKI overall.

Our findings are in line with those of similar studies from different geographic regions, emphasizing the universal challenges and epidemiological patterns of AKI in oncological settings. For example, factors associated with AKI requiring dialysis in our study, such as age, elevated serum creatinine, sepsis, and electrolyte disturbances, align closely with findings from the study by Valenca et al.

There was around 87% (n=28) dialysis dependency in the obstructive nephropathy group (n=32) on six-month follow-up, which adds to the significant burden of the CKD population. The dialysis dependency was mainly attributed to the inability to relieve the obstruction surgically, as it was not amenable at that stage of the disease.

In terms of the recommendations from this work, although the causes of AKI in oncology patients are multifaceted, obstructive nephropathy, mainly associated with cervical cancer, stands out as a significant and preventable factor in our region. As a consequence, promoting HPV vaccination and enhancing screening programs to detect cervical cancers early could potentially lower the incidence of obstructive nephropathy and subsequent AKI, thus reducing healthcare costs and improving patient outcomes in the long term. Additionally, future research should focus on developing tailored strategies for early AKI detection, optimizing nephron protective interventions, and understanding the impact of evolving cancer therapies on kidney health.

Conclusion

In our study, it was found that AKI in patients with cancer arises from various factors, with obstructive nephropathy emerging as the predominant cause in our study population. Importantly, obstructive nephropathy also serves as a significant predictor for the need for dialysis and dialysis dependency at 6-month follow-up, and hyperkalaemia is the most common electrolyte abnormality found in patients with AKI and cancer.

Conflicts of Interest

In compliance with the ICMJE uniform disclosure form, all authors declare the following:

Payment/services info: All authors have declared that no financial support was received from any organization for the submitted work.

Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work.

Other relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Meraz-Munoz A, Langote A, Jhaveri KD, Lee T, Wald R, et al. (2021) Acute kidney injury in the patient with cancer. Diagnostics 11:611
2. Campbell GA, Hu D, Okusa MD (2014) Acute kidney injury in the cancer patient. Adv Chronic Kidney Dis 21:64-71
3. Christiansen CF, Johansen MB, Langeberg WJ, Fryzek JP, Sørensen HT (2011) Incidence of acute kidney injury in cancer patients: A Danish population-based cohort study. Eur J Intern Med 22:399-406.
4. Libório AB, Abreu KLS, Silva GB, Melo GCF, Cordeiro TM, et al. (2011) Predicting hospital mortality in critically ill cancer patients according to acute kidney injury severity. Oncology 80:160-166
5. Canet E, Zafrani L, Lambert J, Thieblemont C, Darmon M, et al. (2013) Acute kidney injury in patients with newly diagnosed high-grade hematological malignancies: Impact on remission and survival. PLoS One 8:e55870
6. Rosner MH, Perazella MA (2019) Acute kidney injury in the patient with cancer. Kidney Res Clin Pract 38:295-308.

7. Cho A, Lee JE, Kwon GY, Huh W, Kim YG, et al. (2011) Post-operative acute kidney injury in patients with renal cell carcinoma is a potent risk factor for new-onset chronic kidney disease after radical nephrectomy. Nephrol Dial Transplant 26:3496–3501

8. Tian R, Liang J, Li R, Ma J, Liu H, et al. (2022) Acute kidney injury induced by immune checkpoint inhibitors. Kidney Dis 8:190-201

9. Heiken JP (2008) Contrast safety in the cancer patient: Preventing contrast-induced nephropathy. Cancer Imaging 8:124