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Case Report

Chemotherapeutic Challenge: A Case of Acute Kidney Injury during Breast Cancer Management

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ABSTRACT

Acute Kidney Injury (AKI) represents a significant and potentially life-threatening complication, especially in patients undergoing treatment with chemotherapeutic agents. Numerous chemotherapy medications have been associated with nephrotoxicity through various mechanisms, including direct tubular injury, fluid depletion, and disturbances in electrolyte balance. This case analysis underscores the crucial importance of continuous renal function monitoring in cancer patients undergoing systemic chemotherapy. We present the case of a 45-year-old female diagnosed with early-stage breast cancer, who also had notable comorbidities such as type 2 diabetes mellitus, hypertension, and a prior cerebrovascular event. Following her initial cycle of combination chemotherapy using cyclophosphamide and doxorubicin, the patient experienced an episode of AKI. Clinical assessment revealed indications of dehydration and potential nephrotoxic effects. Laboratory tests revealed significantly elevated markers of renal function, while imaging studies showed mild to moderate hepatomegaly, along with alterations in the renal parenchyma. The timely recognition of her condition, alongside appropriate supportive measures, facilitated a complete recovery and successfully averted the risk of irreversible kidney damage.

Keywords: Acute kidney injury, Chemotherapeutic agents, Breast cancer, Comorbidities, Nephrotoxicity, Renal toxicity.

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INTRODUCTION

Renal dysfunction is a common issue among individuals diagnosed with cancer, particularly affecting older adults and those with pre-existing health conditions, including diabetes, hypertension, and cardiovascular diseases.[1,2] Breast cancer, which accounts for nearly one-third of newly diagnosed malignancies in women, often necessitates systemic chemotherapy. This treatment can lead to renal complications due to altered pharmacokinetics and impaired drug clearance.[3,4]

Acute Kidney Injury (AKI) associated with chemotherapy can arise from various factors, including tubular injury, dehydration-related pre-renal damage, immune-mediated glomerular dysfunction, and other indirect mechanisms.[5,3,2] Notably, nephrotoxic agents such as cisplatin, methotrexate, ifosfamide, and particularly cyclophosphamide, are recognized for their potential to induce acute tubular necrosis.[5,3] Monoclonal antibodies, such as trastuzumab, have also been linked to glomerulopathies and interstitial nephritis. The likelihood of experiencing acute kidney injury is heightened in patients with underlying conditions, such as diabetes mellitus and hypertension, and cardiovascular diseases.[1,2]

In the context of breast cancer treatment, combination therapy frequently involves the administration of paclitaxel, doxorubicin, and cyclophosphamide. Each of these agents carries a risk of nephrotoxicity, a risk that may be amplified when these drugs are used together.[3,6] Cyclophosphamide is particularly associated with dose-dependent renal toxicity, while doxorubicin has the potential to inflict vascular and oxidative damage to renal tissues. Although paclitaxel is seldom nephrotoxic, it may contribute to ischemic injury due to endothelial dysfunction. Moreover, trastuzumab has been demonstrated to cause glomerular impairment when used alongside other cytotoxic agents.[6]

The risk of AKI escalates significantly in patients with comorbid conditions such as diabetes mellitus and hypertension. Therefore, early detection and management of renal dysfunction, involving the monitoring of biomarkers like NGAL and cystatin C, are paramount in preventing long-term renal damage.[7,8]

This case report examines a breast cancer patient who experienced acute kidney injury shortly after receiving a combination

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chemotherapy regimen that included cyclophosphamide, doxorubicin, paclitaxel, and trastuzumab. The findings of this case highlight the critical need for awareness regarding the renal effects associated with specific agents, as well as the importance of initiating timely supportive care to facilitate renal recovery and the continuation of cancer treatment.[2,8]

CASE DETAILS

A 45-year-old female presented to the hospital with significant gastrointestinal distress, characterized by fifteen episodes of diarrhea and five to six vomiting incidents each day, and markedly reduced urine output, all commencing earlier that same day. Her pertinent medical history included type 2 diabetes mellitus managed with a daily regimen of glimepiride and metformin, hypertension controlled by Olmesartan medoxomil, cilnidipine, and chlorthalidone, and a history of cerebrovascular accident for which she was prescribed aspirin, clopidogrel, and rosuvastatin (20/75/75 mg) once daily at bedtime.

The patient had recently initiated chemotherapy for breast cancer, receiving a combination of agents five days before her admission, which included paclitaxel (280 mg IV), trastuzumab (540 mg loading dose followed by a 400 mg maintenance dose), doxorubicin (100 mg IV), cyclophosphamide (1000 mg IV), and pegfilgrastim (6 mg SC).

Upon admission, her vital signs were stable: blood pressure was 110/70 mmHg, pulse rate was 80 beats per minute, and respiratory rate was 20 cycles per minute, with no febrile response. The physical examination indicated generalized weakness and clinical signs of dehydration, but no pallor, jaundice, clubbing, or pedal edema were present.

Laboratory tests revealed significant renal impairment, indicated by a serum creatinine level of 4.0 mg/dL and a Blood Urea Nitrogen (BUN) of 150 mg/dL. Electrolyte analysis confirmed hyperkalemia (potassium 5.2 mEq/L), hypocalcemia (calcium 0.87 mEq/L), and metabolic acidosis as confirmed by arterial blood gas analysis (pH 7.26; bicarbonate 11.9 mEq/L). The laboratory findings at the time of admission are summarized in Table 1. The urinalysis was unremarkable, while ultrasonography identified mild hepatomegaly, grade I-II fatty liver, and grade II renal parenchymal changes.

A diagnosis of Acute Kidney Injury (AKI), likely attributed to recent chemotherapy, was established. Management commenced with intravenous fluid resuscitation utilizing normal saline and Ringer's lactate at a rate of 80 mL/hour. Empiric antibiotic therapy included sulbactam-cefoperazone (1.5 g IV twice daily) and metronidazole (500 mg IV three times daily). A bolus of 200 mL of sodium bicarbonate was administered to correct the acidosis, and insulin therapy was initiated to address hyperglycemia according to a sliding scale. Her existing medications, comprising antiplatelets and antihypertensives, were continued without alteration.

Supportive care and vigilant monitoring were sustained over the subsequent three days, during which progressive improvements in renal function, resolution of electrolyte abnormalities, and normalization of urine output were observed. The therapeutic interventions and clinical progress during hospitalization are outlined in Table 2. By the fourth day of hospitalization, the patient attained clinical stability, with full resolution of initial presenting symptoms.

DISCUSSION

Acute Kidney Injury (AKI) represents a notable, yet often reversible, complication of chemotherapy, particularly in patients with pre-existing conditions such as diabetes, hypertension, or cerebrovascular disease.[5] In our current case, the therapeutic approach included several agents notorious for their renal risks, such as cisplatin, ifosfamide, and methotrexate, alongside others that, while not typically highlighted for nephrotoxicity, can lead to renal complications.[3,6]

High-dose cyclophosphamide, administered at 1000 mg intravenously, has shown a clear link to AKI, primarily due to its direct impact on renal tubules. There's also a potential for secondary issues like hemorrhagic cystitis, which can further complicate the clinical picture. Doxorubicin, typically recognized

Table 1: Laboratory Findings on Admission.

Parameter	Result	Reference Range	Interpretation
Serum Creatinine	4.0 mg/dL	0.6-1.2 mg/dL	Elevated
Blood Urea Nitrogen (BUN)	150 mg/dL	7-20 mg/dL	Elevated
Potassium (K^+)	5.2 mEq/L	3.5-5.0 mEq/L	Mild Hyperkalemia
Calcium (Ca^{2+})	0.87 mEq/L	1.1-1.3 mEq/L	Hypocalcemia
Arterial pH	7.26	7.35-7.45	Metabolic Acidosis
Bicarbonate (HCO_3^-)	11.9 mEq/L	22-28 mEq/L	Low
Urinalysis	Unremarkable	-	Normal
Ultrasonography	Fatty liver, Grade II renal parenchymal changes.	-	Abnormal renal imaging

Table 2: Therapeutic Interventions and Clinical Course.

Day	Intervention	Details
Day 1	IV Fluids	Normal saline + Ringer's lactate at 80 mL/hour.
	Antibiotics	Sulbactam + Cefoperazone 1.5 g IV BID, Metronidazole 500 mg IV TID.
	Electrolyte Correction	Sodium bicarbonate 200 mL stat dose.
	Antihyperglycemics	Insulin therapy (sliding scale)
	Supportive Care	Continued aspirin, clopidogrel, rosuvastatin, antihypertensives.
Day 2-3	Continued monitoring and supportive therapy	Gradual improvement in renal function and urine output.
Day 4	Clinical stabilization	All symptoms resolved; lab values improved.

for its cardiotoxicity, can similarly affect renal function, induce oxidative stress, and damage the vascular endothelium. Furthermore, trastuzumab, a targeted monoclonal antibody, has been associated with glomerular damage and interstitial nephritis, particularly when used in conjunction with other cytotoxic agents. Although rarely documented as a nephrotoxic agent, paclitaxel may still play a role in renal impairment through effects like endothelial dysfunction and reduced renal perfusion.[3,2,6]

In this patient's scenario, the emergence of AKI within just five days post-chemotherapy strongly indicates a drug-induced origin. The well-documented nephrotoxic properties of cyclophosphamide and doxorubicin, coupled with the high doses utilized, suggest they are likely the main culprits behind the renal injury. Meanwhile, trastuzumab might have exacerbated the situation, particularly concerning glomerular harm. While the nephrotoxic potential of paclitaxel is rarely acknowledged, its possible cumulative effect via endothelial damage shouldn't be overlooked. Compounding these risks, the patient's considerable volume depletion from gastrointestinal fluid losses likely heightened the nephrotoxic effects, leading to pre-renal azotemia and further tubular damage.[3,2]

Swift identification of renal impairment, along with timely interventions, including intravenous fluid resuscitation, correction of electrolyte abnormalities, and diligent renal function monitoring, led to the patient achieving complete clinical and biochemical recovery.[7,8]

This case highlights the crucial need for comprehensive renal risk assessments before treatment, along with proactive renal monitoring, especially for those with multiple underlying conditions. It underscores the significance of carefully weighing nephrotoxic risks when formulating chemotherapy regimens. A team-oriented approach involving oncologists, nephrologists, and supportive care staff is essential to optimize patient outcomes and minimize renal complications during cancer treatment.

CONCLUSION

Chemotherapy-induced Acute Kidney Injury (AKI) may not be a common occurrence with all chemotherapy agents, but it certainly raises significant concerns, particularly among patients who already have conditions like diabetes, hypertension, or cerebrovascular disease. Taking this case into account, the AKI likely originates from a mix of factors. Cyclophosphamide and doxorubicin are known for their direct nephrotoxic effects, while trastuzumab may contribute to glomerular injury, compounded by hypovolemia due to severe gastrointestinal losses.

This scenario highlights the critical need to perform a comprehensive renal risk assessment before initiating treatment. It's essential to tailor therapeutic approaches for patients undergoing combination chemotherapy. Early identification of any renal issues is vital. This can be achieved through diligent monitoring, keeping a close eye on renal function, serum electrolytes, and utilizing early biomarkers such as Neutrophil Gelatinase-Associated Lipocalin (NGAL), Kidney Injury Molecule-1 (KIM-1), and cystatin C. These measures can play a pivotal role in avoiding any irreversible damage to the kidneys.

Collaboration among oncologists, nephrologists, and supportive care teams can greatly improve patient outcomes through timely interventions and renal support. Additionally, it's crucial for cancer survivors who experience AKI during their treatment to receive ongoing nephrological care, this follow-up can significantly lower the risk of developing chronic kidney disease.

This case serves as an important reminder of the nephrotoxic risks linked to widely used chemotherapy drugs and underscores the necessity of providing comprehensive supportive care to help protect renal function throughout the cancer treatment journey.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

AKI: Acute Kidney Injury; **BP:** Blood Pressure; **BUN:** Blood Urea Nitrogen; **IV:** Intravenous; **SC:** Subcutaneous; **NGAL:** Neutrophil Gelatinase-Associated Lipocalin; **KIM-1:** Kidney Injury Molecule-1; **NS:** Normal Saline, **RL:** Ringer's Lactate; **ABG:** Arterial Blood Gas.

DECLARATION OF PATIENT CONSENT

The authors confirm that informed consent has been duly obtained from the patient through a signed consent form. The patient has agreed to the publication of their clinical details and understands that while their name and initials will not be disclosed, efforts will be made to ensure their anonymity.

SUMMARY

This case report highlights a rare but important complication of chemotherapy-Acute Kidney Injury (AKI) in a 45-year-old female with breast cancer and multiple comorbidities. AKI developed shortly after administration of a multi-agent regimen including cyclophosphamide, doxorubicin, paclitaxel, and trastuzumab. The likely contributing factors included direct nephrotoxicity from the chemotherapeutic agents and volume depletion secondary to gastrointestinal fluid loss. Prompt diagnosis, supportive care, and electrolyte management facilitated full recovery. This report emphasizes the necessity of vigilant renal monitoring in cancer patients receiving nephrotoxic drugs, especially those with underlying health conditions.

AUTHOR CONTRIBUTIONS

Dr. K. Ravi Shankar provided overall guidance during the development of the manuscript and offered substantial input in refining its structure and content. Dr. Nandini Palivela was responsible for drafting the manuscript, ensuring clarity and coherence throughout. Lakshya Gayathri Reddy Karri contributed by collecting and organizing the clinical data presented in the case report. Ultimately, all authors participated in a comprehensive review of the manuscript and approved its final version for publication.

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