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CHEMOTHERAPY-INDUCED THROMBOCYTOPENIA LEADING TO LIFE-THREATENING HYPOVOLEMIC SHOCK IN CERVICAL CANCER: A CASE REPORT

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ABSTRACT

Background: Hypovolemic shock due to acute hemorrhage is a life-threatening condition, particularly in cancer patients undergoing chemotherapy. Although recent studies have established protocols for managing hypovolemic shock in trauma and sepsis, the role of chemotherapy-induced thrombocytopenia in exacerbating post-procedural bleeding risks remains underexplored. This case emphasizes the clinical complexity of managing hemorrhagic shock in advanced-stage cervical cancer patients following percutaneous nephrostomy (PCN). **Case Presentation:** A 52-year-old female with stage IVB cervical cancer and liver metastasis presented with hypovolemic shock following a percutaneous nephrostomy (PCN) procedure. The hemorrhagic episode, initially triggered by the PCN, was significantly exacerbated by chemotherapy-induced thrombocytopenia, resulting in prolonged bleeding and hypovolemic shock. Initial management involved fluid resuscitation, blood transfusions, and correction of thrombocytopenia. Despite the challenges posed by her chronic kidney disease (CKD) stage IV. **Conclusion:** This case demonstrates that hypovolemic shock in this patient was caused by post-PCN hemorrhage, which was significantly worsened by chemotherapy-induced thrombocytopenia. The presence of chronic kidney disease further complicated the management. Early recognition and a multidisciplinary approach were key to achieving hemodynamic stabilization and recovery.

INTRODUCTION

Shock is a life-threatening condition characterized by a failure of circulation that leads to inadequate oxygen delivery through the bloodstream to tissues, ultimately resulting in organ failure if left untreated. The pathophysiology of shock is complex, manifesting from various etiologies and resulting in severe metabolic disturbances and permanent organ damage if not addressed promptly. Shock is primarily characterized by reduced oxygen delivery, increased consumption, and/or impaired utilization, leading to cellular and tissue hypoxia. It commonly presents as hypotension, defined by a systolic blood pressure lower than 90 mmHg or a mean arterial pressure (MAP) below 65 mmHg. There are four primary categories of shock: hypovolemic, distributive, cardiogenic, and obstructive, each with distinct hemodynamic profiles and underlying causes.[1,2]

Hypovolemic shock, the focus of this case, is marked by a significant reduction in intravascular volume, often due to blood loss, as seen in trauma, gastrointestinal (GI) bleeding, or surgical complications. The management of such cases requires prompt and aggressive interventions to

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restore organ perfusion and prevent irreversible damage. However, the treatment of hypovolemic shock becomes more complex when compounded by additional factors such as chemotherapy-induced thrombocytopenia, a condition where platelet production is impaired, leading to increased bleeding risks. This intersection of conditions presents a unique and severe clinical challenge, particularly in cancer patients undergoing intensive treatments like chemotherapy.[3,4]

This case report presents the management of life-threatening hypovolemic shock in a patient with advanced cervical cancer (stage IVB) and liver metastasis. The shock was precipitated by acute hemorrhage following a percutaneous nephrostomy (PCN), and critically aggravated by chemotherapy-induced thrombocytopenia. This hematological complication impaired hemostasis and contributed significantly to the severity of bleeding and subsequent shock. Chemotherapy regimens, particularly those involving drugs like Vincristine, Bleomycin, and Cisplatin, are known to cause severe hematological side effects, including anemia and thrombocytopenia, making post-procedural hemorrhage a critical concern.[5-7]

The importance of this case lies in its demonstration of the challenges faced in managing complex oncological emergencies, where a combination of factors such as cancer progression, chemotherapy side effects, and procedural complications must be simultaneously addressed. Hypovolemic shock, in this context, demands a multifaceted approach involving fluid resuscitation, transfusion of blood products, and careful monitoring of hemodynamic status to prevent further deterioration. Moreover, the interplay between chemotherapy-induced thrombocytopenia and acute hemorrhage post-PCN underscores the need for vigilant preoperative risk assessment and postoperative management in similar clinical scenarios.[8,9]

While the management of hypovolemic shock in various contexts is well-documented, there is a notable gap in the literature concerning the specific management of shock in cancer patients undergoing chemotherapy, particularly those with thrombocytopenia. Previous studies have primarily focused on sepsis-induced shock or trauma-related hemorrhage, with limited attention to the unique complications that arise in cancer patients with advanced disease and concurrent chemotherapy. This case report seeks to contribute to the literature by addressing this gap, offering insights into the clinical decision-making processes and therapeutic interventions necessary for high-risk patients.[10,11]

This case report aims to provide a comprehensive analysis of the management of hypovolemic shock in a cancer patient complicated by chemotherapy-induced thrombocytopenia, emphasizing the importance of early intervention and a multidisciplinary approach. By presenting this case, this report aims to highlight the need for further research into tailored management strategies for oncological patients, particularly those at high risk for hemorrhagic complications due to their treatment regimens. This contribution is vital for improving clinical outcomes and providing a framework for managing similar cases in the future.

CASE PRESENTATION

A 52-year-old female patient was referred to Eboni Ward at Andalas University Hospital on the third day of hospitalization with complaints of weakness for the past 12 hours and cold extremities over the last hour. Additionally, the patient reported reddish-brown urine output in her urine bag for 24 hours following a percutaneous nephrostomy (PCN) procedure. Five days prior to hospital admission, the patient experienced urinary retention due to the spread of cervical cancer to the bladder, which led to the PCN procedure on July 18, 2024, to evacuate urine.

The patient had a history of cervical cancer diagnosed in 2023, for which a hysterectomy was performed, followed by chemotherapy and 20 sessions of radiotherapy. The patient was undergoing her second round of chemotherapy with a planned regimen of five cycles. She had no history of coagulation disorders or bleeding difficulties.

Data were obtained from both patient-reported symptoms and objective clinical assessments. Laboratory tests, imaging studies, and daily clinical follow-ups were utilized to evaluate the patient's progress. Vital signs and laboratory parameters were closely monitored during hospitalization to track the response to treatment, as well as potential complications.

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On physical examination, the patient was alert but in poor condition, with a blood pressure of 63/45 mmHg, heart rate of 119 beats per minute, respiratory rate of 26 breaths per minute, and temperature of 36.8°C. The patient had cold extremities, capillary refill time longer than two seconds, and urine output of 300 mL/24 hours, which appeared dark brown. A detailed physical examination of the lungs, heart, and abdomen showed no acute abnormalities.

Initial laboratory tests revealed anemia (hemoglobin of 11.6 g/dL), leukocyte (4,930/mm3), thrombocyte (104,000/mm3), and kidney dysfunction (serum creatinine 2.7 mg/dL). Blood gas analysis showed compensated metabolic acidosis (pH 7.39, HCO_3^- 8.5 mmol/L, base excess -15.2). Imaging studies, including a chest X-ray and electrocardiogram (ECG), showed no significant findings.

The primary diagnosis was hypovolemic shock secondary to acute hemorrhage due to hematuria following PCN. The secondary diagnoses included normocytic normochromic anemia, chemotherapy-induced thrombocytopenia, stage IVB cervical cancer with metastasis to the liver and bladder, hyponatremia, chronic kidney disease stage IV, and malnutrition.

The patient was immediately placed on oxygen therapy with a nasal cannula at 3 L/min and positioned in the Trendelenburg position. A fluid resuscitation challenge was initiated with 200 mL of Ringer's lactate, followed by 1,000 mL over 24 hours. The patient was also placed on a high-calorie high-protein diet (ML TKTP). Serial laboratory investigations and urinalysis were ordered to monitor renal function, electrolytes, and coagulation status. An ultrasound of the inferior vena cava (IVC) was performed to assess volume status. To manage anemia and thrombocytopenia, the patient received two units of packed red blood cells (PRC) and five units of platelet concentrate (TC) transfusions. Norepinephrine was administered at 3 mcg/kg/min to maintain a mean arterial pressure (MAP) above 65 mmHg.[12,13]

Daily clinical assessment and laboratory evaluations were performed to monitor the patient's condition as shown in Table 1. Fluid balance, urine output, and vital signs were measured periodically. Central venous ultrasonography was performed to evaluate central venous pressure. Blood tests, including hemoglobin, leukocyte count, platelet count, electrolytes, and renal function tests, were analyzed to track treatment response and guide further management decisions.

On the second day, the patient's symptoms began to improve, with less weakness and warmer extremities. Laboratory results showed improvement in anemia and thrombocytopenia, with hemoglobin increasing to 9.0 g/dL and thrombocyte decreasing to 36,000/mm^3. Renal function also improved, with creatinine levels declining from 2.7 mg/dL to 1.1 mg/dL by the fourth day of treatment. Norepinephrine dosage was gradually tapered based on the MAP readings.

By the fifth day, the patient showed significant clinical improvement, with normalization of vital signs and urine output. Laboratory results showed improved hemoglobin and platelet counts. The patient was discharged on the sixth day, after stabilization of her condition and completion of antivenom therapy. A follow-up visit one week after discharge showed no significant clinical manifestations of envenomation or local complications at the PCN site.

The results were measured by daily monitoring of clinical symptoms, vital signs, and laboratory markers, allowing for adjustments in the therapeutic regimen as necessary. This case demonstrates the effective management of hypovolemic shock in a high-risk oncological patient, providing insights into handling similar cases in the future.

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Table 1. Laboratory Results and Monitoring

Parameter (Units)	Preoperative	Post Operation			
		Day 1	Day 3	Day 5	Day 6
Hematology					
Hemoglobin (g/dL)	11.6	9.0	13.3	11.6	11.6
Hematocrit (%)	35	26.8	40	25.7	36.7
Leukocyte (mm^3)	4.930	4.800	8.580	7.700	5.000
Thrombocyte (mm^3)	104.000	36.000	34.000	42.000	30.000
MCV (FI)	90	-	-	87	89
MCH (Pg)	29	-	-	28	28
MCHC (%)	32	-	-	32	31
Blood Chemistry					
Sodium (mmol/L)	131	126	-	-	-
Potassium (mmol/L)	4.1	3.8	-	-	-
Chloride (mmol/L)	96	92	-	-	-
Urea (mg/dL)	87	133	-	106.6	-
Creatinine (mg/dL)	2.7	3.4	-	2.0	-
SGOT (U/dL)	19	-	-	-	-
SGPT (U/dL)	3.8	-	-	-	-
Albumin (g/dL)	3.9	-	-	-	-
Random blood glucose (mg/dL)	140	-	-	-	-

RESULTS AND DISCUSSION

This case highlights the complex and multifactorial nature of hypovolemic shock secondary to acute hemorrhage in a patient with advanced cervical cancer, post-percutaneous nephrostomy (PCN). The main contributing factors to this condition were hematuria post-PCN, chemotherapy-induced thrombocytopenia, and chronic kidney disease (CKD) stage IV. The timely identification and intervention for hypovolemic shock in this patient were crucial for preventing further deterioration and improving clinical outcomes.

The patient presented with hypotension (63/45 mmHg), tachycardia (119 bpm), and cold extremities, indicative of peripheral hypoperfusion. These clinical signs, along with a total urine output of 800 mL of dark red urine over the 24 hours following PCN, confirmed the diagnosis of hypovolemic shock. The fluid challenge test, conducted alongside the Trendelenburg position and laboratory investigations, corroborated the presence of significant hypovolemia. Showing in figure 1 that the inferior vena cava (IVC) ultrasound the diameter was 1.19 cm with a collapse of 51%. These findings align with the diagnostic criteria for hypovolemic shock, particularly in patients with active bleeding.

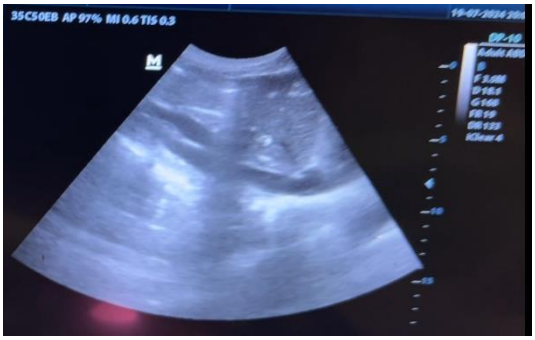

Figure 1. Inferior Vena Cava

Fluid resuscitation led to an improvement in blood pressure (74/58 mmHg) and a reduction in heart rate (106 bpm), demonstrating a positive response to initial treatment. However, subsequent laboratory results indicated a significant drop in hemoglobin from 11.6 g/dL to 9.0 g/dL, confirming the presence of acute blood loss. Immediate blood transfusion with packed red cells (PRC) was essential to restore oxygen-carrying capacity and stabilize the patient's hemodynamics. The decision to transfuse two units of PRC was based on the estimated blood loss of 800 mL, in accordance with guidelines for managing hypovolemic shock with blood loss.

Thrombocytopenia, with platelet counts dropping from 104,000/mm^3 to 36,000/mm^3, further complicated the patient's condition, likely contributing to the prolonged hematuria and increased bleeding risk. The decision to transfuse platelets

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aimed to rapidly increase platelet counts and mitigate the bleeding, in line with standard protocols for active hemorrhage in thrombocytopenic patients.

Chronic kidney disease (CKD) also played a critical role in this patient's management, with elevated urea (126 mg/dL) and creatinine (3.5 mg/dL) levels reflecting compromised renal function. The patient's glomerular filtration rate (GFR) of 15 mL/min/1.73 m² placed her in CKD stage IV, a known risk factor for both exacerbated fluid imbalance and complicating the clearance of nephrotoxic chemotherapy agents.

The management of hypovolemic shock secondary to acute bleeding, especially in oncology patients, remains challenging due to the interaction between cancer therapies and their effects on hematological parameters. Studies have shown that chemotherapy agents such as paclitaxel and carboplatin are associated with significant hematological toxicity, including thrombocytopenia, which can worsen post-surgical bleeding.[14] In this case, the patient's progressive drop in platelet counts following chemotherapy supports the hypothesis that chemotherapy-induced thrombocytopenia was a significant contributor to the prolonged bleeding post-PCN.

Other studies on PCN complications in cancer patients highlight the increased risk of bleeding, particularly in those receiving chemotherapy.[15] The patient's clinical course aligns with findings from these studies, where aggressive management of both the hypovolemia and thrombocytopenia was essential in improving outcomes. Fluid resuscitation, blood product replacement, and careful monitoring of renal function have been proven effective in similar clinical contexts.

However, the literature also indicates that patients with advanced cancer and CKD often have poorer outcomes due to the compounded effect of impaired renal function on fluid management and drug clearance.[16] The patient's CKD likely exacerbated the challenge of managing her shock, particularly in balancing fluid resuscitation without overloading the kidneys.

One limitation of this case study is the inability to definitively isolate the exact cause of thrombocytopenia. Although chemotherapy-induced thrombocytopenia is a plausible explanation, other factors such as underlying malignancy and prior surgical interventions may have contributed to the patient's low platelet count. Further studies with larger sample sizes are necessary to confirm the specific role of chemotherapy in exacerbating bleeding risks in cervical cancer patients post-PCN.

Additionally, the short follow-up period post-discharge limited the assessment of long-term outcomes related to thrombocytopenia and renal function recovery. The influence of chronic kidney disease on chemotherapy clearance and the potential nephrotoxicity of the treatment regimen requires longer-term monitoring and evaluation.

Finally, the absence of advanced imaging techniques such as contrast-enhanced CT scans during the acute phase of shock limits the understanding of the full extent of the hemorrhage and its precise source. Future studies could incorporate more detailed imaging to provide better insights into the mechanisms of bleeding in similar cases.

CONCLUSION

This case illustrates that hypovolemic shock in oncology patients can result from multifactorial causes, such as procedural bleeding compounded by chemotherapy-induced thrombocytopenia. Prompt fluid resuscitation, blood and platelet transfusions, and close hemodynamic monitoring were key to successful management. Clinically, this case reinforces the need for early recognition and individualized interventions in high-risk cancer patients, especially those with hematological complications and comorbidities like CKD.

ETHICAL APPROVAL

There is no ethical approval

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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AUTHOR CONTRIBUTIONS

Conceptualization, NF and R; methodology, NF and R; software, NF and PA; validation, R, F, and RM; formal analysis, NF and F; investigation, NF and F; resources, NF; data curation, NF; writing—original draft preparation, NF; writing—review and editing,

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PA; visualization, PA; supervision, R; project administration, PA; funding acquisition, NF..

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