

Case Report

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Tumor Lysis Syndrome in a Patient with Acute Lymphoblastic Leukemia Following Induction Chemotherapy: A Case Report

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Abstract

We report the case of a 35-year-old male with recently diagnosed acute lymphoblastic leukemia (ALL) who presented with confusion and dyspnea following his first cycle of induction chemotherapy. Clinical assessment revealed a constellation of findings including hyperkalemia, hyperuricemia, elevated lactate dehydrogenase (LDH), and metabolic acidosis consistent with tumor lysis syndrome (TLS). The patient also exhibited signs of acute kidney injury (AKI), septic shock, and severe anemia. This case highlights the importance of early recognition and multidisciplinary management of TLS and its complications in patients undergoing cytotoxic therapy for hematologic malignancies.

Keywords: Tumor lysis Syndrome, Acute Lymphoblastic Leukemia, Metabolic Emergencies, Chemotherapy Complications, Case Report

1. Introduction

Tumor lysis syndrome (TLS) is a potentially fatal oncological emergency that results from the rapid destruction of malignant cells, typically after initiation of chemotherapy. It is characterized by releasing intracellular ions and metabolites into the systemic circulation, leading to hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia, and renal dysfunction [1]. TLS most commonly occurs in hematologic malignancies such as acute lymphoblastic leukemia (ALL) and high-grade lymphomas, especially following cytotoxic treatment [2]. Prompt diagnosis and management are critical to prevent complications such as arrhythmias, seizures, renal failure, and death. Here, we present a case of TLS in a young male with ALL, illustrating the clinical complexity and need for early intervention.

2. Case Presentation

A 35-year-old male presented to the emergency department with acute confusion and shortness of breath. His past medical history was notable for recently diagnosed acute lymphoblastic leukemia (ALL), and he had completed his first induction chemotherapy

cycle five days prior. No allergies were known, and no current medications were documented aside from his chemotherapy regimen.

2.1 Clinical Examination

The patient appeared acutely ill, with an altered level of consciousness. An ABCDE assessment revealed:

- Airway: Patent
- **Breathing:** Tachypnea with increased work of breathing; oxygen saturation 89% on room air
- **Circulation:** Hypotension (BP: 90/60 mmHg), tachycardia (HR: 120 bpm), capillary refill time >3 seconds
- Disability: Confused (GCS 13/15), no focal neurological deficits
- Exposure: Febrile (T: 38.5°C), generalized pallor

2.2 Ultrasound Assessment

A point-of-care Rapid Ultrasound in Shock (RUSH) exam was performed and showed a collapsible inferior vena cava (figure 1), suggestive of hypovolemia, mildly increased cardiac contractility, and no pericardial effusion or pleural fluid accumulation.



Figure 1: Red Arrows Indicating Collapsed Inferior Vena Cava on Ultrasound Exam Laboratory Findings

Parameter	Value	Reference Range	Interpretation
Potassium (K ⁺)	6.5 mmol/L	3.5–5.0 mmol/L	Elevated
Uric Acid	12 mmol/L	0.2–0.4 mmol/L	Critically elevated
Phosphate (PO ₄ ³⁻)	2.1 mmol/L	0.8–1.5 mmol/L	Elevated
LDH	850 U/L	<250 U/L	Markedly elevated
Creatinine	150 μmol/L	45–90 μmol/L	Elevated (AKI)
BUN	Elevated		Suggestive of renal impairment
CRP	Elevated		Systemic inflammation
WBC	1 G/L	4–11 G/L	Pancytopenia
Platelets	20 G/L	150–450 G/L	Severe thrombocytopenia
Hemoglobin	6 g/dL	12–16 g/dL	Severe anemia
Arterial pH	7.25	7.35–7.45	Metabolic acidosis
HCO ₃ -	14 mmol/L	22–26 mmol/L	Low bicarbonate

2.3 Diagnosis

Based on the clinical and laboratory findings, the patient was diagnosed with:

• **Tumor Lysis Syndrome (TLS):** Evidenced by hyperkalemia, hyperphosphatemia, hyperuricemia, elevated LDH, and metabolic acidosis.

• Sepsis/Septic Shock: Suggested by febrile neutropenia, pancytopenia, and elevated inflammatory markers.

• Acute Kidney Injury (AKI): Likely secondary to TLS and/or sepsis.

• Severe Anemia: Contributing to symptoms of hypoxia and confusion.

2.4 Management

The patient was immediately transferred to the intensive care unit (ICU). Initial management included:

• Aggressive IV hydration with isotonic saline

• Rasburicase administration for uric acid reduction

• IV broad-spectrum antibiotics (piperacillin-tazobactam) for suspected neutropenic sepsis

• Packed red blood cell transfusion for severe anemia

• Electrolyte management (calcium gluconate for cardio protection, insulin-dextrose, and sodium polystyrene sulfonate for hyperkalemia)

• Continuous cardiac and renal monitoring

The patient's renal function and metabolic parameters gradually improved over the next 72 hours with supportive management. He remained in a critical yet stable condition, and he was later transitioned back to hematology for further oncologic treatment.

3. Discussion

This case illustrates the clinical urgency and diagnostic complexity of TLS, a well-documented oncologic emergency that is most observed following induction chemotherapy in hematologic malignancies [3]. The pathophysiology involves massive tumor cell lysis releasing potassium, phosphate, and nucleic acids into the bloodstream. Uric acid precipitates in renal tubules, contributing to AKI [4].

Pancytopenia following chemotherapy increases susceptibility to infections, which in this case likely progressed to septic shock, compounding the patient's clinical decline [5]. The metabolic derangements seen here, particularly hyperkalemia and metabolic acidosis, are potentially fatal if not rapidly corrected [6].

Management strategies must be multidisciplinary and include aggressive hydration, urate-lowering agents (e.g., rasburicase), renal support, and infection control [7]. Early risk stratification in leukemia patients before chemotherapy initiation can help guide prophylaxis and close monitoring.

4. Conclusion

Tumor lysis syndrome is a medical emergency that demands early recognition, especially in patients receiving chemotherapy for hematologic malignancies. This case highlights the overlapping burden of TLS, sepsis, and AKI in a post-chemotherapy setting and underscores the necessity of an integrated, rapid-response approach to optimize outcomes.

Declarations

Ethics Approval and Consent to Participate: This case report is exempt from IRB approval as it involves retrospective analysis of anonymized patient data and does not constitute human subject's research. Written informed consent was obtained from the patient.

Consent for Publication: Written informed consent was obtained from the patient for publication of this case report and accompanying details.

Availability of Data and Materials: Data sharing does not apply to this article as no datasets were generated or analyzed.

Competing Interests: The authors declare no competing interests.

Authors' Contributions: All authors contributed to the preparation and approval of the final manuscript.

Declaration of AI usage

During the preparation of this work, AI tools (ChatGPT, DeepSeek) were used for grammar and readability improvements. The authors reviewed and approved all content, ensuring its accuracy.

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