Case Report

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Spontaneous Tumor Lysis Syndrome in a 62-Year-Old Male with Acute Myeloid Leukaemia: A Case Report

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Abstract

Spontaneous tumor lysis syndrome (sTLS) is a rare but severe oncological emergency that occurs without the initiation of chemotherapy, characterized by the rapid release of intracellular components leading to metabolic disturbances. This case report details a 62-year-old male with acute myeloid leukemia (AML) who presented with fatigue, weakness, and metabolic abnormalities indicative of TLS. Laboratory investigations revealed elevated serum uric acid, potassium, phosphate, and creatinine levels, along with hypocalcemia, confirming sTLS. The patient was managed with aggressive hydration, allopurinol, rasburicase, and correction of electrolyte imbalances. Hemodialysis was initiated due to worsening renal function. The patient showed significant improvement with normalization of metabolic parameters and was subsequently started on induction chemotherapy for AML. This case highlights the importance of early recognition and prompt management of sTLS in AML patients to prevent severe complications and improve outcomes.

Keywords:- Tumor Lysis Syndrome, Acute Myeloid, Leukemia, Hyperuricemia, Metabolic Complications

INTRODUCTION

Tumor lysis syndrome (TLS) is an oncological emergency resulting from the rapid release of intracellular components into the bloodstream following the lysis of malignant cells.[1] It is typically associated with the initiation of cytotoxic therapy in high-grade hematologic malignancies, such as acute myeloid leukemia (AML) and high-grade lymphomas. However, spontaneous tumor lysis syndrome (sTLS) occurs without the initiation of chemotherapy, making it a rare and particularly severe manifestation.[2]

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AML is a hematopoietic malignancy characterized by the clonal proliferation of myeloid precursors with a diminished capacity for differentiation. It is the most common acute leukemia in adults, with an incidence increasing with age. The pathophysiology of AML involves genetic mutations and chromosomal abnormalities that lead to the uncontrolled growth of myeloid progenitor cells. These leukemic blasts can proliferate rapidly, leading to high tumor burden and increased risk of complications like TLS.

The clinical presentation of TLS includes symptoms resulting from the metabolic disturbances caused by the release of intracellular ions and metabolic byproducts. These include hyperuricemia, hyperkalemia, hyperphosphatemia, and secondary hypocalcemia, which can lead to acute kidney injury, cardiac arrhythmias, seizures, and multi-organ failure. Diagnosis is based on clinical and laboratory criteria, including elevated serum levels of uric acid, potassium, and phosphate, and reduced calcium levels. Early recognition and treatment are crucial to prevent severe complications and mortality.

An important finding in sTLS is its occurrence without chemotherapy, which requires a high index of suspicion in patients with high tumor burden or rapidly proliferating tumors, such as in untreated AML. This case highlights the need for awareness of sTLS as a potential initial presentation of AML and the importance of rapid intervention.

CASE REPORT

A 62-year-old male with no significant past medical history presented to the emergency department with complaints of fatigue, weakness, and generalized body aches over the past week. He also reported decreased urine output and nausea. Physical examination revealed pallor, hepatosplenomegaly, and diffuse tenderness on palpation.

Initial laboratory investigations revealed severe metabolic abnormalities: serum uric acid of 15.2 mg/dL, potassium of 6.8 mmol/L, phosphate of 7.5 mg/dL, and calcium of 6.2 mg/dL. Renal function tests showed elevated creatinine at 3.1 mg/dL and blood urea nitrogen (BUN) at 45 mg/dL. A complete blood count revealed a white blood cell count of 85,000/µL, hemoglobin of 7.8 g/dL, and platelets of 45,000/µL. Peripheral blood smear showed numerous blasts, prompting further hematologic evaluation.

Bone marrow aspiration and biopsy confirmed the diagnosis of acute myeloid leukemia with a high burden of leukemic blasts. Given the laboratory findings and clinical presentation, a diagnosis of spontaneous tumor lysis syndrome was made.

Management included aggressive hydration with intravenous fluids, administration of allopurinol and rasburicase to reduce uric acid levels, and correction of electrolyte imbalances with calcium gluconate for hypocalcemia, insulin and dextrose for hyperkalemia, and phosphate binders for hyperphosphatemia. Hemodialysis was initiated due to worsening renal function and refractory electrolyte abnormalities.

Over the course of the next few days, the patient's metabolic parameters gradually improved with the treatment. Serum uric acid decreased to 7.0 mg/dL, potassium to 4.5 mmol/L, phosphate to 4.2 mg/dL, and calcium normalized to 8.5 mg/dL. Renal function also improved with creatinine levels decreasing to 1.8 mg/dL.

The patient was stabilized and subsequently started on induction chemotherapy for AML. He tolerated the treatment well, and follow-up showed no recurrence of TLS. The patient was monitored closely for any signs of metabolic derangement during the initial phase of chemotherapy.

Parameter	Initial Value	Post-treatment Value
Serum Uric Acid	15.2 mg/dL	7.0 mg/dL
Potassium	6.8 mmol/L	4.5 mmol/L
Phosphate	7.5 mg/dL	4.2 mg/dL
Calcium	6.2 mg/dL	8.5 mg/dL
Creatinine	3.1 mg/dL	1.8 mg/dL
Blood Urea Nitrogen (BUN)	45 mg/dL	30 mg/dL

Table 1 :- Metabolic profile of the case.

DISCUSSION

Spontaneous tumor lysis syndrome is an uncommon but life-threatening complication that can precede the diagnosis of high-grade hematologic malignancies, including AML. Several cases have been reported in the literature, underscoring the importance of recognizing sTLS even in the absence of chemotherapy. For instance, a review paper by puri et al describes the clinical features and outcomes of sTLS in a cohort of AML patients, highlighting the necessity of early intervention to prevent renal failure and other severe complications.[3]

In this case, the patient presented with severe metabolic abnormalities indicative of TLS, which necessitated aggressive management to prevent further complications.[4] The presence of high uric acid, potassium, and phosphate levels with concurrent hypocalcemia and renal impairment required a multidisciplinary approach, including oncologists, nephrologists, and intensivists.[5,6]

The crucial aspect of management of sTLS remains early recognition of sTLS, the role of prophylactic measures in high-risk patients, and the need for rapid correction of metabolic disturbances.[7,8] The use of rasburicase in addition to allopurinol was crucial in reducing uric acid levels effectively. Hemodialysis played a significant role in managing refractory hyperkalemia and renal dysfunction.[9,10]

This case contributes to the existing literature by highlighting the need for heightened vigilance in patients presenting with high tumor burden and metabolic abnormalities suggestive of TLS. It underscores the necessity of prompt intervention and continuous monitoring to ensure optimal outcomes.

CONCLUSION

Spontaneous tumor lysis syndrome in patients with acute myeloid leukemia is a rare but critical condition requiring immediate medical attention. Early recognition and aggressive management are essential to prevent severe complications and improve patient outcomes. This case underscores the importance of considering sTLS in the differential diagnosis of patients with high tumor burden and metabolic abnormalities, even before the initiation of chemotherapy.

Conflict of interest
None

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