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Spontaneous Tumor Lysis Syndrome: Case Report from UMAE No.71 and Review of the Literature

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Abstract: Tumor lysis (TLS) is an oncologic emergency resulting from the massive release of intracellular metabolites after rapid tumor cell destruction. Although it usually occurs after cytotoxic chemotherapy, spontaneous TLS may develop before treatment, especially in high-grade hematologic malignancies such as Burkitt lymphoma. We report the case of a 42-year-old male patient with constitutional symptoms, progressive abdominal distension, dyspnea, bulky abdominal and retroperitoneal disease, and histopathological findings compatible with non-Hodgkin lymphoma. On initial hematologic evaluation, laboratory test showed acute kidney injury, severe hyperuricemia, hyperkalemia and hyperphosphatemia, consistent with TLS before chemotherapy. HIV infection was subsequently confirmed and bone marrow aspiration was compatible with Burkitt lymphoma/leukemia. Despite intravenous hydration, allopurinol, phosphate binders, and urgent hemodialysis, the patient developed refractory metabolic abnormalities. Following induction chemotherapy with HyperCVAD, TLS worsened, leading to severe hyperkalemia with a sinusoidal electrocardiographic pattern and death. Spontaneous TLS is a rare but life-threatening complication requiring early recognition, risk stratification, and prompt multidisciplinary management..

Keywords: Tumor lysis syndrome, spontaneous tumor lysis syndrome, Burkitt lymphoma, HIV, acute kidney injury, hyperkalemia.

INTRODUCTION

Tumor lysis syndrome is one of the most common oncohematologic metabolic emergencies. It may occur either at the time of cancer diagnosis or during the initiation or course of antineoplastic treatment. (1).

Although it was first mentioned in 1929 by Bedrna and Polka, the complete clinical syndrome was not described until 1980, mainly in patients with Burkitt Lymphoma. (2)

TLS usually develops after the initiation of cytotoxic chemotherapy. However, cases of spontaneous tumor lysis have been documented, particularly in hematologic malignancies with high cellular proliferation rate.

The spontaneous form of the syndrome, occurring in the absence of an identifiable triggering agent, represents an uncommon complication in solid tumors. It may b e associated with intrinsic tumor-related factors and is characterized by high mortality, even when managed according to established international clinical guidelines.

From a pathophysiological standpoint, TLS is characterized by an acute metabolic imbalance secondary to the massive release of intracellular contents (mainly potassium,

phosphorus, and nucleic acids) into the systemic vascular compartment following the accelerated destruction of malignant cells. (3)

This condition may lead to tubular obstruction, resulting in increased pressure within both the proximal and distal tubules, with the subsequent elevation of peritubular capillary pressure and vascular resistance. In addition, uric acid may promote crystal formation and hemodynamic alterations, including renal vasoconstriction and impaired autoregulation due to decreased nitric oxide availability.

Hyperkalemia is the first hydroelectrolytic disturbance to appear, secondary to the cellular lysis. It is often asymptomatic; however, when symptoms occur, they may be nonspecific, including nausea, vomiting, and muscle weakness. Electrocardiographic abnormalities may also be present, such as widening of the QRS complex, absence of P waves, and marked T-wave elevation. These changes may progress to ventricular fibrillation and sudden cardiac death. (4)

Given its potentially fatal nature, early identification of patients at high risk for TLS is essential in order to implement timely prophylactic and therapeutic measures. Early recognition of metabolic abnormalities and prompt initiation of targeted management are the fundamental pillars for reducing associated morbidity and mortality and preserving the patient´s life.

CASE REPORT

A 42-year-old male patient presented with a clinical course that began in November 2024, characterized by profuse diaphoresis, an unintentional weight loss of approximately 14 kg over three months, and, three weeks prior to his initial evaluation progressive abdominal distension associated with worsening night diaphoresis and dyspnea on minimal exertion.

Due to persistence of symptoms, the patient sought medical attention at his regional general hospital, where a diagnostic workup was initiated.

On February 23, 2025, a non-contrast and contrast- enhanced abdominopelvic computed tomography scan was performed. The study reported a poorly defined hypodense hepatic lesion in the left hepatic lobe measuring 12.7 x 11.2 mm, with an attenuation of 74 HU; a solid mass in the mesogastrium, anterior to the abdominal aorta, measuring 76 x 65 mm; and a retroperitoneal lymph node conglomerate measuring 179 x 85 x 59 mm, causing partial compression of both renal veins. The spleen measured 135 mm. The findings were interpreted as a mass of probable neoplastic origin with diffuse peritoneal involvement, retroperitoneal lymph node conglomerate, and hypodense hepatic lesion compatible with secondary infiltration.

During hospitalization, a palpable left supraclavicular lymphadenopathy was identified; therefore, an excisional biopsy was performed on February 28, 2025. The histopathological report, issued on March 5, 2025, described a lymphoproliferative process compatible with non-Hodgkin lymphoma, without complementary immunohistochemical analysis. Based on these findings, the patient was referred to the Hematology Department of UMAE No. 71 for further diagnostic evaluation and specialized management.

On March 7, 2025, during his initial evaluation by Hematology, urgent laboratory tests were requested, revealing a serum creatinine level of 4.2 mg/dL, uric acid of 27.2

mg/dL, potassium of 5.1 mEq/L, and phosphorus of 4.4 mg/dL, findings consistent with tumor lysis syndrome (TLS). Management was initiated with intravenous hyperhydration and allopurinol adjusted according to renal function. A Nephrology consultation was also requested; the Nephrology team placed a Mahurkar catheter and indicated urgent hemodialysis.

Additionally, phosphate binders were administered, initially aluminum hydroxide due to the unavailability of sevelamer, and subsequently sevelamer. Despite hemodialysis sessions, only a transient decrease in metabolic abnormalities was observed, followed by a progressive increase in the aforementioned serum values.

Table 1: Serial laboratory values during Hospitalization. Evolution showing persistent and progressive metabolic abnormalities consistent with severe TLS.

Date	Uric Acid (mg/dl)	Potassium (mmol/L)	Phosphorus (mg/dL)	Calcium (mg/dL)
08/03/2025	23.2	8.9	5.9	8.3
09/03/2025	23.8	4.9	8.7	8.3
10/03/2025	-	5.5	9.7	7.6
11/03/2025	>34	7.4	22.7	6.9
11/03/2025	>34	14.2	32.4	6.5

The patient developed grade II ascites with worsening dyspnea. A therapeutic large-volume paracentesis was proposed, with explanation of its risk and benefits; however, the patient refused the procedure and signed an informed refusal form.

During hospitalization, a viral panel was requested, which was reactive for HIV. Seropositivity was confirmed on March 10, 2025, by confirmatory testing.

A bone marrow aspiration was performed on March 10, 2025, which was compatible with Burkitt lymphoma / leukemia.

On March 11, 2025, induction chemotherapy with the HyperCVAD regimen was initiated, after the patient had received a detailed explanation regarding the inherent risks.

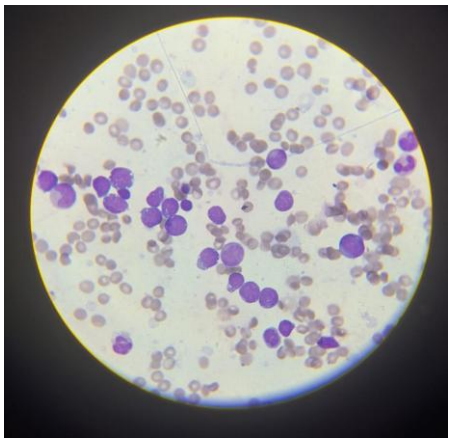

Figure 1: Bone marrow infiltration of 40% by large lymphoblast, L3 type, with vacuoles consistent with Burkitt morphology, preserved erythroid/myeloid ratio, with reactive bands and metamyelocytes.

Subsequently, exacerbation of tumor lysis parameters was documented, accompanied by progressive dyspnea and hypoxemia, requiring increased oxygen support. Follow-up laboratory tests revealed a serum creatinine level of 5 mg/dL, potassium of 14 mEq/L and uric acid greater than 34 mg/dL; therefore, urgent hemodialysis was performed. Despite treatment no significant clinical improvement or reduction in dyspnea was observed. The patient's family was informed about the critical condition and poor prognosis.

On March 13, 2025, an electrocardiogram was performed, showing a sinusoidal pattern compatible with severe hyperkalemia. The patient died the same day as a result of refractory metabolic complications secondary to severe tumor lysis syndrome.

DISCUSSION

In the present case, the patient was diagnosed with Burkitt Lymphoma, probably associated with HIV infection, a diagnosis that will be discussed in greater detail in subsequent sections. This subtype of high-grade lymphoproliferative neoplasm is characterized by a high proliferative rate and, consequently, an increased risk of developing tumor lysis syndrome (TLS).

In addition to this intrinsic risk, the patient presented with bulky disease and a significant elevation of lactate dehydrogenase (LDH), parameters that placed him in the high-risk category for TLS according to established clinical criteria.

Upon admission to the unit, the patient already had acute kidney injury and metabolic abnormalities compatible with TLS, including refractory electrolyte disturbances. These abnormalities persisted despite renal replacement therapy with intermittent hemodialysis, without achieving complete correction of the metabolic imbalances.

Epidemiology

The incidence of tumor lysis syndrome (TLS) has not been clearly established, as it depends on multiple factors related to tumor burden and the biological characteristics of the neoplasm. The main determinants include serum lactate dehydrogenase (LHD) levels, histologic subtype, the presence of bulky disease, leukocytosis at diagnosis, and the degree of tumor chemosensitivity. (5)

Estimated Incidence rates vary widely, reaching up to 40% in certain high-grade hematologic malignancies, with an associated mortality rate close to 20% despite the implementation of therapeutic strategies. (6)

Pathophysiology

TLS results from the massive release of intracellular metabolites- mainly potassium ions, phosphorus, and products of nucleic acid degradation- that exceed the renal excretory capacity. During nucleic acid catabolism, xanthine acts as a metabolic intermediate which, when oxidized by xanthine oxidase, produces uric acid. This metabolic overload leads to intratubular precipitation of calcium phosphate, xanthine and uric acid crystals in the distal renal tubules, resulting in obstructive uropathy with subsequent decrease in glomerular filtration rate (GFR) and development of acute kidney injury (AKI). (7)

Predisposing factors for the development of TLS include high tumor sensitivity to cytotoxic chemotherapy, effectiveness of antineoplastic treatment, preexisting renal dysfunction, dehydration, and urinary acidity, all of which together create a favorable metabolic environment for the development of the syndrome.

Hyperkalemia is the earliest electrolyte abnormality generally observed between 12 and 24 hours after the onset of cellular lysis. It is a potentially life-threatening complication that may present with asthenia, electrocardiographic changes, severe arrhythmias and even sudden cardiorespiratory arrest.

Neoplastic cells contain up to four times more phosphorus than normal cells; therefore, its massive release into the bloodstream disrupts the calcium-phosphorus balance, promoting calcium phosphate precipitation with secondary renal tubular obstruction. Hyperphosphatemia usually becomes clinically evident between 24 and 48 hours after the initiation of chemotherapy.

Risk Classification

According to the risk associated with the underlying neoplasm, TLS, is classified into three main pronostic categories: (8)

High Risk

This category corresponds to a probability greater than 5% of developing TLS. It is most frequently observed in acute leukemias-particularly acute lymphoblastic leukemia (ALL) and acute myeloid leukemia (AML)- associated with elevated serum lactate dehydrogenase (LDH) levels and marked leukocytosis. This group also includes Burkitt lymphoma, both in advanced stages and in localized forms with increased LDH, as well as diffuse large B-cell with bulky disease and elevated LDH levels.

Intermediate Risk

This category includes a 1% to 5% probability of developing TLS. It comprises highly chemosensitive solid tumors, such as neuroblastoma and germ cell tumors. in which a rapid response to cytotoxic treatment may induce significant cellular lysis.

Low Risk

This category includes patients with less than a 1% probability of developing TLS. This group includes most solid tumors with low chemosensitivity, multiple myeloma, indolent lymphomas, as well as chronic myeloid leukemia (CML) and chronic Lymphocytic leukemia (CLL).

Predisposing Factors

Several predisposing factors may increase the likelihood of developing TLS and its associated metabolic complications. These include preexisting hyperuricemia, previous renal

dysfunction or kidney injury, dehydration, recent exposure to nephrotoxic drugs, and obstruction at any level of the urinary tract. Together, these conditions impair renal clearance of metabolites and increase the risk of acute kidney injury secondary to TLS.

Diagnostic Criteria

Clinical Criteria:

Clinical TLS is considered when one or more of the following findings are present: (9)

- Acute kidney injury, evidenced by an increase in serum creatinine greater than 1.5 times the upper limit of normal.
- Cardiac rhythm disturbances attributable to electrolyte imbalance, including severe arrhythmias or sudden cardiac death.

Laboratory Criteria:

A biochemical or laboratory diagnosis is established when at least two of the following metabolic abnormalities are observed:

- An increase of ≥ 25% from basal serum phosphorus, potassium, uric acid or blood urea nitrogen (BUN) levels, exceeding the normal range.
- A decrease of ≥ 25% in serum calcium levels, occurring within three days before or up to seven days after the initiation of chemotherapy.

Table 2: Laboratory criteria for tumor lysis syndrome.

Uric Acid	Potassium	Phosphorus	Calcium
≥ 8 mg/dL or ≥ 25% increase from baseline.	≥ 6 mmol /L or ≥ 25% increase from baseline.	≥ 1.45 mmol/L or ≥ 25% increase from baseline	≤ 1.75 mmol/L or ≥ 25% decrease from baseline.

Management

In patients at high risk for TLS, therapeutic recommendations establish the need for intensive hydration at a rate of 3 liters per square meter of body surface area per day (3 L/m2/24 h), using isotonic or balanced solutions without added potassium, with the goal of maintaining a minimum urine output of 100 mL/m2/hour. (10) Urine output should be monitored every 3 hours, and a global fluid balance assessment must be performed at least every 6 hours. This is essential to ensure adequate diuresis, prevent volume overload, and optimize renal clearance of metabolites associated with tumor lysis.

Prevention

Continuous monitoring, adequate hydration, and the use of hypouricemic agents, constitute the cornerstone prophylactic measures in patients at risk of developing tumor lysis syndrome. (11)

Preventive interventions should be initiated 24 to 48 hours before the start of chemotherapy and continued for up to 72 hours afterward, depending on the patient's individual risk. These measures include oral hydration and administration of allopurinol in patients at low or intermediate risk, whereas in high-risk patients, intensive intravenous hydration combined with rasburicase is recommended.

The selection of the hypouricemic agent should be based on availability, pharmacological profile, and mechanism of action. Allopurinol acts by inhibiting the enzyme xanthine oxidase, thereby reducing the formation of new uric acid; for this reason, it is administered several days before the initiation of chemotherapy in patients at increased risk. In contrast, rasburicase catalyzes the oxidation of uric acid into allantoin, a highly soluble metabolite that is easily excreted by the kidneys, making it the agent of choice for both prophylaxis and treatment of established TLS.

CONCLUSION

Tumor lysis syndrome occurs most frecuently in hematologic malignancies characterized by high cellular proliferation rate and marked sensitivity to cytotoxic chemotherapeutic agents, which promotes the massive release of intracellular contents after treatment initiation.

It is also essential to consider the type of parenteral solution used for hydration therapy. The use of 0.9% normal saline is recommended because it contains no potassium, thereby reducing the risk of worsening hyperkalemia associated with cellular lysis. This is particularly relevant because neoplastic cells contain significantly higher concentrations of intracellular electrolytes compared with normal cells, which increases the metabolic risk of developing TLS during the treatment phase.

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