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Tumor lysis syndrome: review article

Síndrome de lisis tumoral: artículo de revisión

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ABSTRACT

Introduction: Tumor lysis syndrome (TLS) is a potentially lethal complication that originates after the start of cytotoxic chemotherapy. It triggers multiple metabolic alterations due to the rapid lysis of tumor cells and is characterized by symptoms of hyperuricemia, Hyperkalemia, hyperphosphatemia, Hypocalcemia, uremia, and acute kidney injury. **Purpose of the review:** To present the available evidence on tumor lysis syndrome—highlighting those relevant aspects of the topic—to broaden the focus on recognizing it and the guidelines for its prevention and therapeutic management. A bibliographic review was carried out in the electronic databases PubMed, SciELO, and Elsevier; 42 studies and one oncology text published in English and Spanish from 2019-2024 were analyzed. **Relevance:** Early recognition is essential to prevent progression to multiple organ failure. Therapeutic management includes hydration, hypouricemia-lowering agents, and correction of electrolyte imbalance supervised by a multidisciplinary team in a hospital unit equipped for effective patient monitoring. Hemodialysis is the auxiliary therapy in a patient's refractory to medical treatment. **Conclusions:** Given the high mortality due to tumor lysis syndrome, it is essential to identify patients at risk and implement preventive therapeutic measures early, avoiding organic damage.

Keywords: Tumor lysis syndrome, Cancer chemotherapy, Oncology, Cancer.

RESUMEN

Introducción: El síndrome de lisis tumoral es una complicación potencialmente letal que se origina tras el inicio de la quimioterapia citotóxica y desencadena múltiples alteraciones metabólicas por la rápida lisis de las células tumorales. Está representada por cuadros de hiperuricemia, hiperpotasemia, hiperfosfatemia, hipocalcemia, uremia y lesión renal aguda. **Propósito de la revisión:** Presentar la evidencia disponible sobre el síndrome de lisis tumoral —resaltando aquellos aspectos relevantes con relación al tema— para ampliar el enfoque de cómo reconocerlo y las directrices para su prevención y manejo terapéutico. Se realizó una revisión bibliográfica en las bases de datos electrónicas PubMed, SciELO y Elsevier; se analizaron 42 estudios y un texto de oncología, en idiomas inglés y español, publicados en el periodo 2019-2024. **Relevancia:** El reconocimiento temprano es fundamental para evitar el progreso hacia falla multiorgánica. El manejo terapéutico incluye hidratación, hipouricemiantes y corrección del desequilibrio electrolítico, supervisado por un equipo multidisciplinario en una unidad hospitalaria equipada para una monitorización eficaz del paciente. La hemodiálisis es la terapia auxiliar en pacientes refractarios al tratamiento médico. **Conclusiones:** Dada la alta mortalidad por síndrome de lisis tumoral, resulta muy importante la identificación de pacientes en riesgo para iniciar de manera temprana las medidas terapéuticas preventivas y evitar el daño orgánico.

Palabras Clave: Síndrome de lisis tumoral, Quimioterapia, Oncología, Cáncer.

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1. Introduction

The Tumor Lysis Syndrome (TLS) represents a metabolic alteration set. It was first described decades ago after the invention of cytoreductive therapy for the management of neoplastic diseases. It constitutes a true once-metabolic emergency that is seen quite frequently in clinical practice in adult and pediatric cancer patients who undergo therapeutic management with chemotherapy [1, 2].

Cancer constitutes one of the leading causes of morbidity and mortality worldwide, with a variable primary origin, which, added to the mysterious gap in the life cycle of neoplastic cells, generates a wide range of manifestations in the body as a metabolic response [3, 4]. TLS usually appears after the initiation of chemotherapy, although there are reports of spontaneous occurrences in patients with high-grade malignant hematological neoplasms [5, 6].

Since it is a potentially fatal entity, it is relevant to identify those patients with a high risk of presenting TLS. In this way, early recognition of the alterations associated with the timely initiation of therapeutic management is the fundamental pillar to preserve the patient.

This article aims to highlight those relevant aspects of the topic to provide a clearer vision of how to recognize TLS and, with that, the foundations for its prevention and therapeutic management.

2. Materials and methods

2.1 Study design

Descriptive study. A search of scientific literature on the topic was conducted in digital journals published in the last five years in English and Spanish.

2.2 Databases analyzed

To search for information, we used the electronic databases PubMed, SciELO, and Elsevier. These databases allowed us to collect information from scientific articles, systematic reviews, descriptive studies, and literature reviews focused on the topic.

2.3 Search terminology

The search was conducted in English and Spanish using the descriptors: “Tumor lysis syndrome,” “Oncology,” “Cytotoxic chemotherapy,” “Cancer,” obtained from the Descriptors in Health Sciences (DeCS) and “Tumor lysis syndrome,” “Oncology,” “Cytotoxic chemotherapy,” “Cancer” obtained from Medical Subject Headings (MeSH), linked with the Boolean operator AND.

2.4 Inclusion criteria

Scientific articles published in the period 2019-2024 in English and Spanish.

2.5 Exclusion criteria

Duplicate articles or articles unrelated to the topic in languages other than those indicated or published outside the established period.

3. Results

From the total number of studies yielded by the search, 42 articles and one oncology text were selected and analyzed.

3.1 Definition

TLS is defined as an acute, potentially fatal condition in both adults and infants associated with the initiation of cytotoxic therapy in neoplastic treatment [2]. It is characterized by a pattern of metabolic alterations resulting from the massive release of intracellular contents from cancer cells into the systemic circulation. These specific findings include conditions such as uremia, hyperphosphatemia, Hyperkalemia, hyperuricemia, and Hypocalcemia, which together can lead to severe complications such as cardiac arrhythmias, seizures, kidney failure, and even death due to multiple organ failure [2, 8].

3.2 Etiology and risk factors

The presence of TLS is generally recognized in patients suffering from hematological malignancies. It is also common for it to occur in high-grade lymphomas after having started treatment with aggressive chemotherapy. However, solid tumors can also cause TLS, although to a lesser extent. Among them, the most frequent are neuroblastoma and hepatoblastoma. Some data mention that a clinical picture of TLS can spontaneously exist without starting chemotherapy [8, 9].

It has also been considered that the risk of TLS increases in the presence of leukocyte counts greater than 100,000 cells/mm, lactate dehydrogenase twice the normal level, bulky disease (greater than 10 cm), hepatomegaly, splenomegaly, bone marrow involvement, and preexisting kidney disease [10,11].

3.3 Epidemiology

The incidence of TLS is not known with certainty; multiple factors influence its occurrence and development, e.g., a high tumor burden, the presence of neoplasms with a high rate of cell proliferation, and greater sensitivity to cytoreductive therapy. Apart from preexisting kidney disease or rapid deterioration of the patient during their illness, there is no predisposition based on race or sex [10].

3.4 Pathophysiology

TLS is a set of clinical manifestations resulting from metabolic alterations due to the massive release of intracellular ions such as potassium, phosphorus, and uric acid, which in large quantities exceed the kidney's capacity to excrete them [6]. The degradation of nucleic acids produces xanthine as a final product, a compound that, when oxidized, leads to the formation of uric acid. This substantial metabolic load causes the deposition of calcium phosphate crystals, xanthine, and uric acid in the distal renal tubules, collectively resulting in obstructive uropathy and a subsequent decrease in the glomerular filtration rate (GFR), which in turn leads to acute kidney injury (AKI). Factors such as cellular sensitivity to cytotoxic therapy, the efficacy of chemotherapy, underlying renal system dysfunction, dehydration, and urinary acidity create an environment that contributes to the clinical development of TLS [10,11].

Furthermore, there is a premise that uric acid stimulates AKI through mechanisms beyond crystal formation, like renal vasoconstriction, which leads to reduced blood flow to the organ, inflammation, and deregulation of the internal environment. This also results in the overexpression of C-reactive protein and nitric oxide [10, 12].

3.5 Electrolyte Imbalance

3.5.1 Hyperkalemia

Tumor cell lysis causes a massive release of potassium. The liver and skeletal muscles absorb excess potassium, and the remainder is excreted through the renal and gastrointestinal systems. In obstructive uropathy, uric acid salts significantly limit potassium excretion, leading to dangerously high levels and an increased risk of arrhythmias and cardiac arrest [13, 14].

3.5.2 Hyperphosphatemia

Tumor lysis causes the nucleic acids within neoplastic cells to release phosphate groups, which in turn release a higher-than-normal amount into the bloodstream. Since phosphorus is primarily excreted via the kidneys, AKI inhibits the kidney's ability to filter this element. Hyperphosphatemia is more commonly observed in chemotherapy-induced TLS than in spontaneous TLS, and as a result of hyperphosphatemia, calcium chelation occurs, thus leading to Hypocalcemia [15, 16].

3.5.3 Hypocalcemia

It represents a potentially fatal condition that occurs as a secondary effect of hyperphosphatemia. It also leads to complications that require immediate attention due to the risk of mortality, e.g., cardiac arrhythmias, tetany, and seizures [17, 18].

3.6 Histopathology

Findings consistent with deposits of uric acid, xanthine, and calcium phosphate crystals can be found in renal tissue. The factors associated with and contributing to crystal formation include low urine flow, a higher concentration of solutes, and their low solubility in the urinary environment [19, 20].

3.7 Clinical Assessment

Symptoms reveal metabolic alterations that arise approximately 12 to 72 hours after starting chemotherapy or spontaneously without having initiated cytoreductive therapy [21]. It is necessary to consider the primary cause that triggered the clinical manifestations, as well as to identify common symptoms such as anorexia and weight loss. In this case, digestive symptoms include nausea, vomiting, and diarrhea; urinary symptoms include dysuria and hematuria. Notable signs of fluid overload include pitting edema, marked abdominal distension, and facial edema. The presence of vomiting, spasms, muscle cramps, convulsive episodes, tetany, or altered mental status suggests Hypocalcemia. Uremia may manifest as a metallic taste in the mouth, pruritus, abnormal lung sounds due to volume overload, joint pain, uremic pericarditis that muffles heart sounds, and renal colic-like pain. Multiple pathophysiological events may lead to extensive TLS, resulting in multiorgan failure and sudden death [22, 24].

3.8 Diagnosis

The diagnosis can be classified under clinical and laboratory criteria. In this case, it was based on the Cairo-Bishop classification criteria, which include a 25% change from the initial values of uric acid, potassium, phosphorus, and calcium. The diagnosis is determined when two or more criteria are identified within the period from three days before to seven days after the start of cytotoxic therapy [25, 26].

3.8.1 Laboratory Diagnosis

Two or more of the following criteria within 24 hours [10, 25]:

- Uric acid elevation of 25% or ≥ 8.0 mg/dL
- Potassium elevation of 25% or ≥ 6.0 mg/dL
- Phosphate elevation of 25% or ≥ 4.5 mg/dL
- Calcium decrease of 25% or ≤ 7.0 mg/dL

3.8.2 Clinical Diagnosis

Includes a positive laboratory diagnosis plus the presence of at least one clinical criterion [10, 25]:

- Elevated serum creatinine or ≥ 1.5 times the upper standard limit.
- Cardiac arrhythmias or sudden death
- Seizures

When performing imaging studies, it is necessary to be cautious when administering intravenous contrast media due to the presence of AKI in TLS. The electrocardiogram (ECG) is a fundamental part of the patient's evaluation, as it helps detect Hypocalcemia and Hyperkalemia. A complete blood analysis assists in assessing the cellular and metabolic components in the blood; an elevated blood chemistry value could indicate TLS. In urinalysis, analyzing the pH is advisable, as it determines the effectiveness of the treatment regarding urinary alkalinization [2, 10].

3.9 Treatment

Laboratory values should be monitored every 4 to 6 hours initially. Additionally, urine output should be closely observed. Given the need for close monitoring, the patient's admission to the intensive care unit (ICU) should be considered for optimal management [27].

3.9.1 Hydration

The fundamental part of treatment is rapidly expanding intravascular volume with isotonic solutions; crystalloid solutions help increase the glomerular filtration rate (GFR). Intravenous hydration should begin 48 hours before chemotherapy and continue for 48 hours after completion. Approximately 3 to 3.5 liters/m^2 per day are necessary to achieve a diuresis of 80 to 100 ml/hour, or approximately 3 liters daily; this can be supported by using potassium-reducing loop diuretics [27, 28]. It is essential to consider factors such as a history of heart failure [29].

3.9.2 Reduction of uric acid levels:

Allopurinol is a xanthine oxidase inhibitor that can reduce uric acid levels and prevent the development of TLS. Therefore, it is a practical choice for managing patients at risk of TLS and can be used at a dose of 300 mg, with a maximum dose of 800 mg, as long as renal function is preserved. Its effect is achieved after 48 to 72 hours of treatment, which should be administered 2 to 3 days before starting chemotherapy and continued for 10 to 14 days [27, -30]. In cases of hypersensitivity to the drug, febuxostat has shown effective control of TLS-related hyperuricemia in studies with a good safety profile without causing the hypersensitivity reactions associated with allopurinol (eosinophilia, hepatitis, and interstitial nephritis). It is usually administered at a dose of 120 mg [30, -31]

Derived from Aspergillus, recombinant urate oxidase or rasburicase is a drug that, after its metabolism in the body, converts uric acid into allantoin, carbon dioxide, and hydrogen peroxide; allantoin is a metabolite up to 10 times more soluble in urine compared to uric acid [32]. It should be administered between 4 and 24 hours before the start of chemotherapy at a dose of 0.2 mg/kg/day in a 30-minute intravenous infusion for five days and in patients with low risk of TLS, a dose of 0.1 to 0.15 mg/kg [33]. Studies revealed that administering single doses of rasburicase of 6 mg in adults and 0.15 to 1.15 mg in children provides normalization of serum uric acid levels [34]. In contrast, Yaman et al. [35] mention in

their study that a single dose of 7.5 mg effectively controls clinical TLS. It is contraindicated in patients with glucose-6-phosphate dehydrogenase deficiency (risk of methemoglobinemia or hemolytic anemia), pregnant women, and lactating women [36, 37]. It is important to emphasize that its use is limited by its high economic cost [37].

Urinary alkalinization with sodium bicarbonate was a previously recommended method to facilitate uric acid excretion; however, it is no longer recommended due to its ability to promote the precipitation of phosphate and calcium in the kidney tubules, further aggravating AKI in TLS [38, 39].

3.9.3 Correction of Electrolyte Abnormalities

Hyperkalemia, particularly with values above six mmol/L, can be addressed using bicarbonate, polarizing solutions, calcium gluconate, or furosemide [40, 41]. If phosphate levels exceed 6 mg/dL and the patient is stable, treatment can be administered orally with 300-600 mg of aluminum hydroxide [16]. Asymptomatic Hypocalcemia is not treated due to the risk of calcium crystal precipitation in the renal tubules; however, if symptoms are present, it can be managed with 1 gram of calcium gluconate orally. This dose may be repeated until symptoms resolve. Managing hyperphosphatemia helps prevent secondary Hypocalcemia [42]. Hemodialysis is indicated in cases with significantly elevated and potentially life-threatening potassium and phosphorus levels. Continuous renal replacement therapy (CRRT) can prevent rebound hyperkalemia [42, 43].

4. Conclusion

TLS is a potentially fatal hematological-oncological emergency best managed by a multidisciplinary team of professionals, including specialists in oncology, nephrology, internal medicine, intensive care, and the ICU nursing team. Given the delicate nature of this clinical environment, it is essential to identify patients at risk for TLS so that therapeutic management can begin as early as possible. Early recognition of metabolic and renal alterations and timely treatment initiation are crucial for saving the patient's life.

5. Abbreviations

CRRT: Continuous Renal Replacement Therapy

ECG: Electrocardiogram

AKI: Acute Kidney Injury

TLS: Tumor Lysis Syndrome

GFR: Glomerular Filtration Rate

ICU: Intensive Care Unit

6. Administrative information

6.1 Additional files

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6.3 Authors contributions

The author is responsible for conceptualizing the idea, analyzing it, searching it, and writing the manuscript.

6.4 Funding

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6.5 Declarations

6.5.1 Ethics Committee Approval

Not applicable.

6.5.2 Conflicts of Interest

The author declares that there are no conflicts of interest**.**

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