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Case report

A case report on spontaneous tumour lysis syndrome: a perioperative challenge

Informe de un caso de síndrome de lisis tumoral espontáneo: un desafío perioperatorio

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Abstract

Spontaneous Tumour Lysis Syndrome is an uncommon but critical medical phenomenon where cancer cells break down on their own before any treatment begins. The sudden release of the products of the internal cell components into the blood creates a dangerous metabolic imbalance that can cause severe kidney damage or death if not managed immediately. We present a case of a 61-year-old female patient who presented to the "Immediate Care" department accompanied by her family member who referred altered mental status, intense pain and increased growth of the tumour on her right elbow, diagnosed as cutaneous epidermoid carcinoma. Laboratory evaluation showed a classic pattern of metabolic imbalances: elevated levels of uric acid, potassium, and phosphate, alongside low calcium. These findings were accompanied by met criteria of acute kidney injury, prompting immediate diagnosis and intervention. This case highlights the importance of rapidly identifying risk factors and the criteria and classification for the diagnosis of Tumour Lysis Syndrome and the necessity for a multidisciplinary team for successful management.

Keywords

Tumour Lysis Syndrome, Acid-Base Imbalance, Acute Kidney Injury.

Resumen

El Síndrome de Lisis Tumoral Espontánea es un fenómeno poco común, pero es una urgencia oncológica grave en la cual las células cancerígenas se destruyen por si solas antes de que comience cualquier tratamiento. La liberación súbita de los productos de las células en la sangre resulta en un desequilibrio metabólico peligroso que puede causar daño renal grave o la muerte si no se maneja de inmediato. Presentamos un caso de una paciente de 61 años que se presentó al servicio de Atención Inmediata acompañada por su familiar quien refirió cambios en el estado mental, dolor intenso y aumento del crecimiento del tumor en el codo derecho de la paciente, diagnosticado como carcinoma epidermoide cutáneo. Los estudios de laboratorio mostraron un patrón clásico de desequilibrios metabólicos: niveles elevados de ácido úrico, potasio y fosfato junto con niveles bajos de calcio. Estos hallazgos juntos con criterios confirmando lesión renal aguda resultaron en un diagnóstico e intervención inmediatos. Este caso resalta la importancia de identificar en tiempo oportuno los factores de riesgo, los criterios y la clasificación del Síndrome de Lisis Tumoral y la necesidad de un equipo multidisciplinario para un adecuado manejo.

Palabras claves

Síndrome de Lisis Tumoral, Desequilibrio Ácido-Básico, Lesión Renal Aguda.

Abbreviations

TLS: Tumour Lysis Syndrome
mcg: Micrograms
kg: Kilograms
min: Minute
ICU: Intensive Care Unit
L: Litres
VCV: Volume controlled ventilation
IBW: Ideal Body Weight
Pinsp: Inspiratory Pressure
PS: Pressure Support
PEEP: Positive End Expiratory Pressure

Introduction

Tumour lysis syndrome is a pathophysiological condition that results when there has been an immense destruction of malignant cells and release of intracellular substances into the blood, causing electrolyte disturbances and effects of the cardiovascular system, skeletal muscles, central nervous system, and kidneys due to administration of antineoplastic therapy, such as conventional chemotherapy, corticosteroids, molecular-targeted therapy, immunotherapy, and even after radiotherapy and chemoembolization or by spontaneous occurrence. To be able to diagnose, classify and manage this syndrome there are pertinent risk factors, diagnostic criteria and treatment options in relation to risk stratification and diagnosis.

In proposed classification systems, patients diagnosed with haematological (Burkitt lymphoma and acute lymphoblastic leukaemia), bulky solid tumours (lung and hepatic tumours) and having renal dysfunction are at high risk of developing tumour lysis syndrome. The current risk stratifications class groups of cancers into their respective risk and provide recommended management.

Ultimately, an event of spontaneous lysis syndrome is exceedingly rare and more so in a patient with cutaneous epidermoid carcinoma which has not been previously reported.

Currently, the proposed classification for this phenomenon is spontaneous cutaneous tumour lysis syndrome.

The *Cairo & Bishop* definition and criteria facilitate the diagnosis of tumour lysis syndrome, however, there is no established guideline for its treatment or management in the perioperative setting.

Narrative

A 61-year-old woman with a history of Cutaneous Epidermoid Carcinoma diagnosed 4 months prior to her current visit presented with altered mental status, intense pain and increased growth of the tumour on her right elbow. Notable laboratory data included leucocytosis, anaemia, thrombocytopenia, elevated serum creatinine, hyperkalaemia, hypochloraemia, hyperphosphatemia, hyperlacticaemia, and metabolic acidosis.

Laboratory results: Leucocytes 24.17 thous/cu.mm3, Haemoglobin 8.9g/dL, Platelets 80 thous/cu.mm3, Creatinine 2.54 mg/dL, Potassium 5.5meq/L, Chloride 102meq/L, Phosphate 9.77mg/dL, Lactate 2.74, pH 7.2, pCO_2 35.8, pO_2 48.2, HCO_3 14.5, PaO_2/FiO_2 96

Antibiotic therapy: Linezolid 600mg was administered in "Immediate Care" Service.

Surgical management was decided and the patient was programmed for an emergency transhumeral amputation of the upper right limb and was immediately taken to the operating theatre where she was somnolent but cooperative with medical personal.

Physical Examination: a fetid, ulcerated exophytic lesion approximately 15 x 12cm in size with redness and oedema extending to the shoulder and forearm and the presence of a small bore peripheral intravenous catheter (22G) in her inferior right limb due to unsuccessful attempts at venous cannulation in other sites.

Adhering to standard basic monitoring, the patient was monitored.

Vital signs: BP: 121/68mmHg, MAP: 86mmHg, HR: 78bpm, RR: 14 bpm, SpO_2: 91%, T: 36.5°C

Premedication started with 1gram Paracetamol and 200 milligrams Hydrocortisone while being preoxygenated with supplemental oxygen at 10 L/min delivered by a facial mask.

Due to reduced level of consciousness and unknown fasting status, Rapid Sequence Induction with fentanyl 200 micrograms, lidocaine 60 milligrams, rocuronium 100 milligrams and propofol 70 milligrams, and subsequently orotracheal intubation with a video-laryngoscope, hyperangulated blade, and a 7.5 Murphy endotracheal tube were performed and the patient was connected to the ventilation machine.

Ventilator settings: VCV, TV: 450, RR: 16rpm, FiO_2: 50%, Flow: 2L, PEEP: 5cmH_2O for IBW 53kg.

Hypnosis was achieved and maintained with Sevoflurane at an end-tidal concentration of 0.8 minimum alveolar concentration.

A 14Fr foley catheter was inserted to monitor diuresis.

After induction, a mean arterial pressure of 58mmHg was observed, during which time a three-lumen central line was being placed in the right jugular vein using ultrasound guidance. Continuous Norepinephrine infusion was established

using a volumetric infusion pump at a rate of 0.05mcg/kg/min, gradually increasing to 0.1mcg/kg/min to maintain a mean arterial pressure of 70 mmHg throughout the surgery.

Fluid therapy was initiated with a volumetric perfusion pump, administering Lactated Ringer`s solution at 2ml/kg/hour at a rate of 130ml/hour.

Using volumetric infusion pumps, separate infusions of fentanyl at a rate of 0.02 mcg/kg/min, maintaining a plasmatic concentration of 0.0016mcg/ml and lidocaine 2% at a rate of 1.5mg/kg/hour were being perfused to assure analgesia and potential anti-tumour effects respectively. This was done using the medial lumen of the central line.

To ameliorate acute kidney injury and compensate for blood loss during amputation, liberal hydration was started with the administration of Lactated Ringer`s solution at 8ml/kg/hour; a rate of 600ml/hour.

The patient maintained hemodynamically stable but in critical condition. Despite metabolic derangements the patient did not present with alterations in the constant electrocardiogram monitoring.

Immediately after amputation of the limb, albumin 3% with 0.9% normal saline, total volume of 250 mL was administered within 30minutes. Followed by a transfusion of 256 mL of packed red blood cells within a 45 minute period.

Hydration therapy was ongoing with Lactated Ringer`s solution.

Throughout the perioperative period, resuscitation with fluid and blood therapy resulted in the gradual decrease of the dose of norepinephrine to 0.01mcg/kg/min maintaining mean arterial pressure of 70-80mmHg ensuring organ-tissue perfusion.

Vital signs maintained constant: FC: 72-81bpm, RR, 16-20bpm, SO2 97-99%, CO_2: 33-35 (Figure 1).

The procedure was completed within 120 minutes.

Infusions of fentanyl and lidocaine were stopped and the total used throughout the surgery were 300 mcg and 160 mg respectively.

There was an estimated blood loss of 80 millilitres in gauzes and approximately 170 millilitres of sequestrated blood in the limb; a total of 250ml blood loss. Quantified diuresis of 120 millilitres; urine output 0.47ml/kg/hour. Intravenous fluids: Lactated Ringer`s solution**:** 1000mL, 0.9% Sodium Chloride Solution: 250mL.

To determine weaning, Pressure-support ventilation Pro (PSV-Pro), a partial ventilatory support mode was programmed: Pinsp 6cmH_2O, PS: 6cmH_2O, PEEP 5cmH_2O with a TV: 386, RR: 8bpm, CO_2: 42 but due to the presence of asynchronous ventilation the ventilator automatically changed to Synchronized Intermittent Mandatory Ventilation (SIMV).

Following this automated transition to a mode that would not be effective in liberating the patient from mechanical ventilation, the clinical decision was made to revert the patient to Volume-controlled ventilation (Figure 2).

The **arterial blood gas** at the end of surgery: Hb 7.8 Hct 24 Lactate 1.3, pH 7.1, pCO_2 37, pO_2 44.2, HCO_3 14.3, PO_2/FiO_2 88.4 Cl 103, K 5.57, Ca 1.46, Glucose 105.

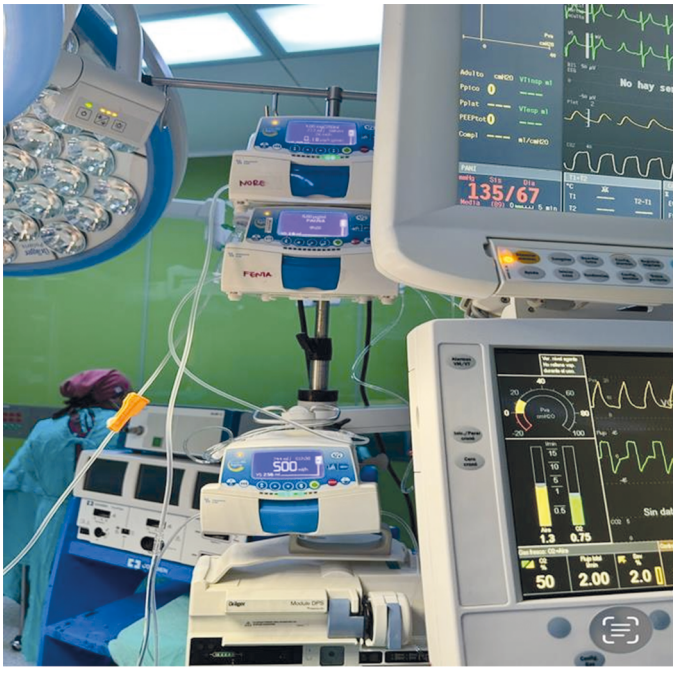

Figure 1. Intraoperative Management

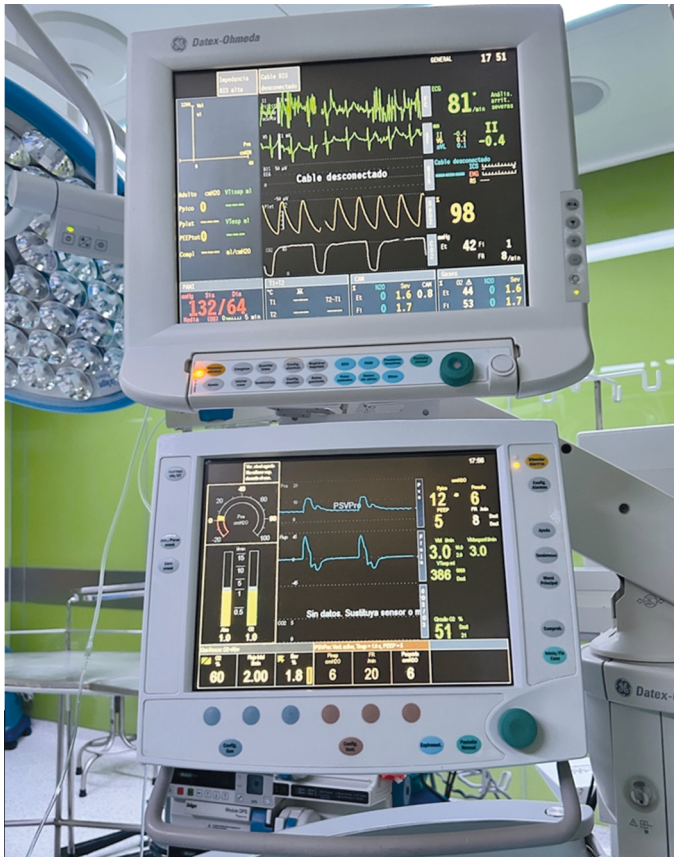

Figure 2. Ventilation Parameters at the end of surgery.

With the results of arterial blood gas above showing metabolic acidosis and a low Horowitz index it was decided to continue fluid therapy at 3.5mL/kg/hour a rate of 250mL/hour with Lactated Ringer`s solution and to maintain continued ventilatory support in VCV, TV: 450, RR: 16rpm, FiO_2: 50%, Flow: 2L, PEEP: 5cmH_2O.

The case was presented to the Intensive Care Unit.

Due to the patients critical condition and requirement for specialized management she was transferred to the Intensive Care Unit, intubated and with norepinephrine at 0.01mcg/kg/min with stable vital signs: BP: 100/56mmHg, TAM: 70mmHg, HR: 69bpm, RR: 18bpm, SpO_2: 96%.

The patient was extubated on the day of her admission to the ICU and remained in the unit for 15 days with the following diagnoses:

- Cutaneous Epidermoid Carcinoma
- Sepsis
- Cutaneous Abscess
- Postoperative Transhumeral Amputation
- Acute Kidney Injury: Stage 2
- Metabolic Acidosis
- Chronic Hyponatraemia
- Hypercalcaemia
- Hyperphosphataemia
- Hyperuricaemia
- Anaemia
- Thrombocytopenia
- Phantom Limb Syndrome

During her admission, electrolyte imbalances were corrected and the acute kidney injury began to resolve. She was later diagnosed with Pneumonia and despite these improvements, her condition fluctuated significantly. She eventually succumbed to cardiac arrest due to "severe sepsis with septic shock and acute respiratory failure."

Timeline

2024-12-30

- Diagnosis
  Patient is diagnosed with Cutaneous Epidermoid Carcinoma.

2025-04-30

- Immediate Care Unit
  Patient presents to the "Immediate Care" Unit with altered mental status, intense pain and a mass on her right elbow. After physical examination, revision of current laboratory tests and patient history she is admitted for emergency surgery.

2025-05-01

- Intensive Care Unit
  Patient is admitted to the Intensive Care Unit for specialized medical attention and monitoring.

2025-05-16

- Patient succumbs to cardiac arrest due to "severe sepsis with septic shock and acute respiratory failure."

Diagnostics

Table 1. Preoperative and Postoperative Arterial Blood Gas Results

TEST	RESULT	UNIT	NOTES
Leucocytes	24.17	thous/cu.mm3	Preoperative
Haemoglobin	8.9	g/dL	Preoperative
Platelets	80	thous/cu.mm3	Preoperative
Creatinine	2.54	mg/dL	Preoperative
Potassium	5.5	mEq/L	Preoperative
Chloride	102	mEq/L	Preoperative
Phosphate	9.77	mg/dL	Preoperative
Lactate	2.74	mmol/L	Preoperative
pH	7.2		Preoperative
pCO_2	35.8	mmHg	Preoperative
pO_2	48.2	mEq/L	Preoperative
HCO3	14.5	mmHg	Preoperative
PaO_2/FiO_2	96	g/dL	Preoperative
Haemoglobin	7.8	g/dL	Postoperative
Haematocrit	24	%	Postoperative

TEST	RESULT	UNIT	NOTES
Lactate	1.3	mmol/L	Postoperative
pH	7.2		Postoperative
pCO_2	37	mmHg	Postoperative
PO_2	44.2	mmHg	Postoperative
HCO_3	14.3	mEq/L	Postoperative
PaO_2/FiO_2	88.4	mmHg	Postoperative
Chloride	103	mEq/L	Postoperative
Potassium	5.57	mEq/L	Postoperative
Calcium	1.46	mmol/L	Postoperative
Glucose	105	mg/dL	Postoperative

Patient Perspective

A formal perspective was not included as the patient`s cognitive status was declining and she was not able to provide a formal statement for publication.

Discussion

Tumour Lysis Syndrome is a life-threatening oncological emergency that results in several metabolic derangements and multisystem disorders. The current reported incidence is estimated as 5-70% which is an indication of its unpredictability[1].

The diagnosis of TLS is usually established based on the Cairo and Bishop criteria which considers both laboratory and clinical findings. It requires the presence of two or more of the following abnormalities: hyperuricemia, hyperkalaemia, hyperphosphatemia or hypocalcaemia and one of the following: acute renal failure, cardiac arrhythmia or seizure.

It is being proposed that there exists: Therapy-Associated Systemic Tumour Lysis Syndrome, Spontaneous (Idiopathic) Systemic Tumour Lysis Syndrome which takes into account patients with haematologic malignancies or solid tumours which have not yet received treatment and Cutaneous Tumour Lysis Syndrome[2].

In this case, it is important to note that the patient was diagnosed with Cutaneous Epidermoid Carcinoma or Cutaneous Squamous Cell Carcinoma which is a malignancy of epidermal keratinocytes and is the second most common type of nonmelanoma skin cancer[4]. This malignancy is not explicitly included in current risk stratification and management guidelines which emphasizes its status as a low-risk malignancy. This unique instance therefore highlights the clinical unpredictability of the syndrome regardless of established stratification and brings attention to the definition of cutaneous tumour lysis syndrome which refers to "the lysis of neoplastic cells located within the dermis of either hematologic malignancies or solid tumours." Clinically, cutaneous tumour lysis syndrome presents as ulcers within the tumour mass. The key feature of spontaneous cutaneous tumour lysis syndrome is necrosis and ulceration of skin tumours in the absence of therapy[2].

The initial assessment and management of Tumour Lysis Syndrome is carried out using a flowchart which correlates with the Caira and Bishop Criteria and the size of the cancer mass which then prompts the respective treatment pathway[5].

Negligible risk doesn't require prophylaxis or monitoring while low risk which involves the possibility of cell-lysis requires administration of intravenous fluids, allopurinol and daily laboratory tests. Patients that present with intermediate risk should be hospitalized and monitored and the same treatment scheme applies but with more frequent laboratory testing. In those who present with a high risk, it is necessary to hospitalize this population and assure cardiac monitoring and three times a day lab testing while administering intravenous fluids and rasburicase. Lastly, patients with an ongoing tumour lysis syndrome should be placed in an intensive care unit where constant monitoring and lab tests are performed. The frequency of these measurements is subsequently adjusted based on the degree of metabolic or electrolyte disruption and laboratory monitoring is eliminated as metabolic abnormalities resolve. Fluid therapy and rasburicase administration are vital[5].

Acute kidney injury is more often caused by hyperphosphataemia because of precipitation of calcium phosphate in the renal tissue leading to nephrocalcinosis. Phosphorus excretion is accomplished by aggressive administration of intravenous saline along with diuresis.

The greatest urgency is the correction of potassium levels exceeding 6.5 mmol per litre. Standard management for hyperkalaemia includes intravenous calcium for membrane stabilization, intracellular potassium redistribution via insulin-glucose infusion or beta-agonists, and enhanced renal excretion using loop diuretics. In extreme situations renal replacement therapy must be considered[1].

In this case setting, the clinical management could have been optimized. In order to mitigate the risk of crystalline nephropathy and acute tubular necrosis, the implementation of hyperhydration is paramount. A targeted fluid resuscitation rate of 3 L/m²/day with use of isotonic crystalloids is recommended to maintain renal perfusion. The clinical goal is to achieve a urine output of ≥100 mL/m²/hour to reduce the concentration of uric acid and calcium-phosphate precipitates within the renal tubules.

Since hyperkalaemia represents the most immediate life-threatening electrolyte derangement in these settings the administration of a "polarizing solution"—a co-infusion of regular insulin and concentrated dextrose (glucose) to maintain euglycemia and promote potassium redistribution.

While hypocalcaemia is a common secondary effect of hyperphosphatemia, routine supplementation is cautioned against due to the risk of calcification. When serum phosphate is elevated, exogenous calcium can bind with phosphate, precipitating as calcium-phosphate crystals in the heart, lungs, and kidneys—drastically worsening Acute Kidney Injury. Therefore, calcium administration is recommended in certain conditions: severe and symptomatic hypocalcaemia, changes of the electrocardiogram, and arrhythmia.

Ideally, rasburicase, a medication that converts uric acid to a soluble inactive metabolite that can be excreted by the kidneys is a first-line drug for the treatment of tumour lysis syndrome but this was not available during this event.

Another important point to consider is pain management. *Elsharkawy et al., 2025* indicate that regional anaesthesia reduces the incidence of chronic postsurgical pain by modulating the immunological and neuroplastic responses to surgery, however the scenario changes in a septic patient. Systemic sepsis results in lower pH of tissues throughout the body which would prevent the anaesthetic from entering the nerve cells effectively therefore affecting the duration of the medication of even the success rate.

Conclusion

Tumour Lysis Syndrome is an uncommon clinical emergency but when it does occur it is associated with high morbidity and mortality. The management of severe metabolic derangements in the setting of high-turnover malignancies requires a precise, multi-tiered clinical approach. In this case, the patient's significant hyperphosphatemia and hyperkalaemia, coupled with acute renal impairment, necessitate aggressive intervention to prevent multi-organ failure.

Risk stratification remains the primary driver of prophylaxis; however, once established, management must pivot to intensive volumetric expansion and pharmacologic mitigation which are the cornerstone of therapy to maintain an adequate urine output.

Furthermore, electrolyte correction must be handled with extreme caution to not exacerbate hemodynamic stability. Ultimately, the successful stabilization of such patients depends on the early recognition of laboratory and clinical findings and a proactive transition to intensive care for continuous monitoring and potential renal replacement therapy.

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Declaration of Conflict of Interest

The authors declare that they have no competing interests.

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Reference

1. Bociek RG, Lunning M. Tumor Lysis Syndrome. N Engl J Med. 2025 Sep 18;393(11):1104-1116. doi: 10.1056/NEJMra2300923.
2. Cohen PR, Prieto VG, Kurzrock R. Tumor Lysis Syndrome: Introduction of a Cutaneous Variant and a New Classification System. Cureus. 2021 Mar 11;13(3):e13816. doi: 10.7759/cureus.13816.
3. Cairo MS, Coiffier B, Reiter A, Younes A; TLS Expert Panel. Recommendations for the evaluation of risk and prophylaxis of tumour lysis syndrome (TLS) in adults and children with malignant diseases: an expert TLS panel consensus. Br J Haematol. 2010 May;149(4):578-86. doi: 10.1111/j.1365-2141.2010.08143.x.
4. Howard SC, Jones DP, Pui CH. The tumor lysis syndrome. N Engl J Med. 2011 May 12;364(19):1844-54. doi: 10.1056/NEJMra0904569. Erratum in: N Engl J Med. 2018 Sep 13;379(11):1094.
5. Yan, S. (2020). Squamous cell carcinoma. Pathology Outlines. https://www.pathologyoutlines.com/topic/skintumornonmelanocyticscc.html