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SYNOPSIS/MINI-REVIEW

Tumour Lysis Syndrome in Paediatric Oncology: Pathophysiology, Risk Assessment, Prevention and Management

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E-mail: adekunle.motunrayo@lagosstate.gov.ng<https://orcid.org/0000-0003-2820-3945>**Introduction**

Tumour Lysis Syndrome (TLS) is a life-threatening metabolic emergency caused by the rapid destruction of tumour cells, either spontaneously or following cancer treatment. It is mostly seen in malignancies with a high tumour burden, rapid cell proliferation, and high sensitivity to treatment. TLS may occur after chemotherapy, immunotherapy, targeted therapy, and, less commonly, radiotherapy. Mortality rates can be as high as 21%, particularly in resource-limited settings.

Pathophysiology

TLS results from the rapid lysis of tumour cells, leading to the release of large quantities of intracellular contents into the bloodstream. These include nucleic acids, potassium, and phosphate. The breakdown of nucleic acids (DNA and RNA) produces uric acid, causing hyperuricaemia, while the release of intracellular potassium and phosphate results in hyperkalaemia and hyperphosphataemia, respectively. Elevated phosphate levels bind to calcium, forming calcium-phosphate complexes and leading to hypocalcaemia. Uric acid is poorly soluble in water and can precipitate within the renal tubules, causing urate nephropathy. In addition, calcium-phosphate crystal deposition within the kidneys contributes to the development of acute kidney injury (AKI).

The major clinical consequences of TLS include:

- Hyperkalaemia, which may cause life-threatening cardiac arrhythmias and sudden death.
- Hypocalcaemia, which can result in tetany and seizures.
- Acute kidney injury, due to uric acid and calcium-phosphate crystal deposition in the renal tubules.
- Acute respiratory distress syndrome (ARDS), which may develop secondary to

the systemic inflammatory response and renal failure associated with severe TLS.

Risk Factors

Malignancies associated with a high tumour burden, rapid cellular proliferation, and high treatment sensitivity are at greatest risk of TLS. These include:

- Burkitt lymphoma/leukaemia
- Haematologic malignancies (AML and ALL), especially with hyperleukocytosis.
- Diffuse large B-cell lymphoma (DLBCL)
- Chronic myeloid leukaemia (CML), especially during blast phase.
- Other high-grade lymphomas and rapidly proliferating malignancies
- Other risk factors include pre-existing renal impairment, renal disease or tumour infiltrating the kidneys, elevated baseline uric acid and hypovolaemia.

Risk Assessment

Risk assessment is essential in the prevention and management of TLS, particularly for identifying patients at high risk who require intensive prophylaxis and monitoring. Patients are generally stratified into low-, intermediate-, or high-risk categories based on tumour- and patient-related factors.

The key parameters considered during risk assessment include:

- Underlying malignancy and tumour burden
- White blood cell count
- Serum lactate dehydrogenase (LDH) level
- Renal function
- Baseline serum uric acid and phosphate levels
- Planned anticancer therapy and its expected efficacy

Risk scores can then be used to guide management, with scores greater than 5 indicating high risk and

scores less than 1 indicating low risk of TLS. The CancerCalc TLS Risk Assessment Tool is a readily available online calculator that stratifies patients into low-, intermediate-, or high-risk groups and can assist clinicians in determining appropriate prophylactic and monitoring strategies. It is used to predict the occurrence of TLS.

Grading

The Cairo-Bishop classification is a common tool used in diagnosing and grading TLS. It is used after TLS develops. It includes clinical and laboratory parameters. TLS can then be graded according to the severity of the clinical manifestations, from Grade 0 to Grade 5, with Grade 5 representing death.

Laboratory

Laboratory TLS is diagnosed when two or more of the following abnormalities occur within three days

before or seven days after initiation of anticancer therapy:

Parameters	Cairo-Bishop Threshold
Uric acid	≥ 8 mg/dL (476 μmol/L) or 25% increase from baseline
Potassium	≥ 6.0 mmol/L or 25% increase from baseline
Phosphate	≥ 6.5 mg/dL or 25% increase
Calcium	≤ 7mg/dl (1.75 mmol/L) or 25% decrease from baseline

Clinical

Clinical TLS is defined as Laboratory TLS plus one or more of the following clinical complications:

- Acute kidney injury/renal insufficiency
- Cardiac arrhythmia or sudden death
- Seizure

Cairo Bishop TLS Grading

Grade	Renal/Creatinine	Cardiac	Seizure
0	No TLS	No arrhythmia	No seizure
1	Creatinine (Cr) 1.5 x ULN	No intervention required	No seizure
2	Cr > 1.5 -3 X ULN	Asymptomatic: Drug intervention required	Short/generalized or controlled seizure
3	Cr > 3-6 X ULN	Symptomatic, not controlled by medication, controlled with defibrillator	Seizure with impaired consciousness/generalized seizure
4	Cr > 6 X ULN	Life-threatening arrhythmia/cardiac decompensation	Prolonged or repetitive seizures
5	Death due to TLS	Death	Death

ULN - Upper limit of normal

Prevention

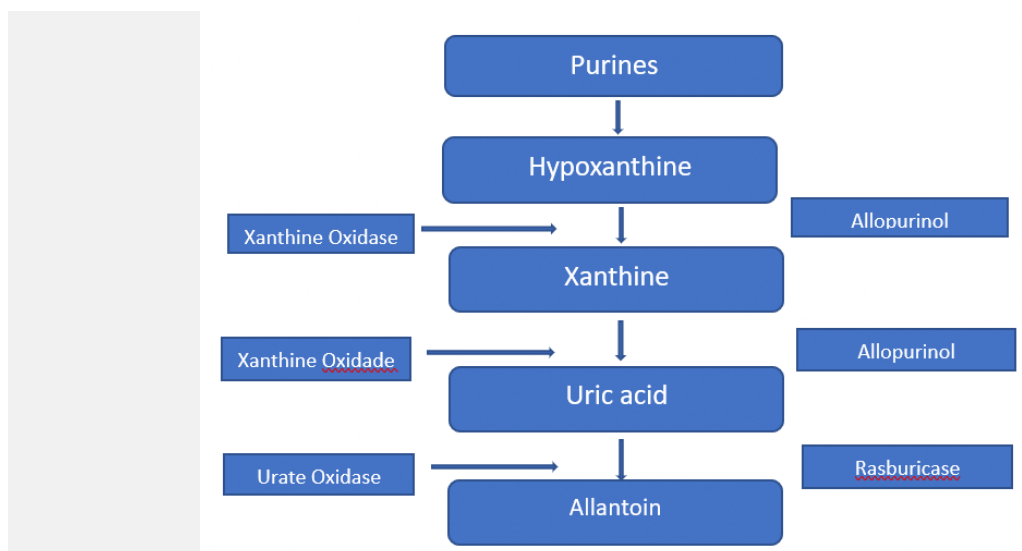
Prevention of TLS involves individualised risk assessment, adequate hydration, and appropriate prophylactic pharmacotherapy.

Prevention of TLS based on risk assessment:

Risk Strata	Treatment
Low	Hydration + Monitoring
Moderate	Hydration + allopurinol + closer biochemical monitoring; consider Rasburicase in selected patients
High	Intensive hydration + Rasburicase when appropriate + frequent laboratory monitoring + readiness for renal replacement therapy

Hydration: Adequate hydration is essential to increase renal perfusion, enhance urinary excretion of uric acid, phosphate, and potassium, and reduce the risk of uric acid and calcium phosphate crystal deposition in the renal tubules. Intravenous normal saline or half-normal saline is given at 2 to 3 L/m²/day.

Prophylactic pharmacotherapy: This includes the use of xanthine oxidase inhibitor (allopurinol) or recombinant urate enzyme (Rasburicase). The mechanism of action of both drugs is as shown below:



Allantoin is a highly water-soluble compound that the kidneys readily eliminate. Rasburicase rapidly converts pre-existing uric acid to allantoin, facilitating its renal excretion. In contrast, allopurinol inhibits xanthine oxidase and primarily prevents the formation of new uric acid rather than reducing existing uric acid levels. Rasburicase is contraindicated in individuals with glucose-6-phosphate dehydrogenase (G6PD) deficiency because it can cause oxidative stress and precipitate haemolytic anaemia and methaemoglobinemia.

Treatment steps

- Hydration and pharmacotherapy (rasburicase or allopurinol) are used in the management of TLS. Other treatment modalities include:
- Management of hyperkalaemia: Stop potassium-containing fluids, administer calcium gluconate for cardiac stabilisation, and implement stepwise management based on serum potassium levels.
- Management of hyperphosphataemia: Use phosphate binders such as aluminium hydroxide.
- Management of hypocalcaemia: Calcium therapy should be reserved for symptomatic patients to avoid further calcium-phosphate deposition.
- Management of metabolic acidosis: Urine alkalinisation is not necessary, as it may enhance calcium-phosphate precipitation.
- Dialysis: Initiate when indicated.

Conclusion

Tumour lysis syndrome (TLS) is an oncological emergency that requires prompt risk assessment and timely management. It may occur spontaneously or following initiation of anticancer therapy. Patients with a high tumour burden or highly proliferative malignancies are at increased risk of developing TLS. Appropriate intravenous fluid therapy, together with pharmacological agents to prevent uric acid formation and promote its excretion, should be promptly initiated.

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