

Tumour lysis syndrome

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1 Tumour lysis syndrome (TLS) is an emergency

Tumour lysis syndrome is caused by a rapid breakdown of malignant cells releasing cellular contents into the blood.¹ Most often, TLS occurs early after the start of chemotherapy, although it can spontaneously occur before treatment, or with radiation, steroid or targeted treatments in children and adults.^{1,2} Mortality may be as high as 20%.³⁻⁵

2 Patients with hematological malignancies are at a higher risk than those with solid tumours^{1,3}

Patients with highly proliferative hematological malignancies — characterized by elevated leukocyte count, lactate dehydrogenase levels and bulky lymphadenopathy (e.g., Burkitt lymphoma, acute lymphocytic leukemia) — and those with renal dysfunction are at an increased risk of TLS.^{1,4} Incidence ranges from 3% to 40% among patients with high-risk hematological malignancies.⁴ Patients at risk for TLS usually receive prophylactic strategies by their oncologist, such as hydration, allopurinol or rasburicase and frequent electrolyte monitoring.^{1,2,4}

3 Clinical presentation is nonspecific

Symptoms and signs of TLS may include anorexia, nausea and vomiting, diarrhea, pain, cramps, tetany, oliguria, paresthesias, weakness, seizures and dysrhythmias.

4 Diagnosis is based on both clinical and laboratory findings

Clinicians should watch for changes in laboratory values from baseline, particularly hyperuricemia, hyperkalemia, hyperphosphatemia, hypocalcemia and an increase in creatinine.^{1,2} A laboratory diagnosis of TLS is made with 2 or more of the following: elevated uric acid ($\geq 476 \mu\text{mol/L}$ or 25% increase from baseline), elevated potassium ($\geq 6.0 \text{ mmol/L}$ or 25% increase), elevated phosphate ($\geq 1.45 \text{ mmol/L}$ [adults] or 25% increase) and lowered calcium ($\leq 1.75 \text{ mmol/L}$ or 25% decrease).^{1,2} A clinical diagnosis requires a laboratory diagnosis of TLS with either elevated creatinine (≥ 1.5 times the upper limit of normal), seizure or arrhythmia.^{1,2} Both laboratory and clinical TLS should be treated.

5 Management should involve nephrologists and oncologists

Management of TLS includes aggressive fluid hydration to improve renal function (urine output $100 \text{ mL/m}^2/\text{h}$),^{4,5} rasburicase (oxidizes uric acid) to manage uric acid ($> 450 \text{ mmol/L}$) and phosphate binders (e.g., aluminum hydroxide) to treat hyperphosphatemia.⁵ Hyperkalemia should be treated as usual. Seizures or arrhythmias secondary to hypocalcemia should be treated with calcium.⁵ Asymptomatic hypocalcemia typically does not require treatment.^{2,5} Dialysis may be required.¹

References

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