Tumor Lysis Syndrome

Tumor lysis syndrome (TLS) occurs most often in patients with leukemia or lymphoma who are starting chemotherapy with cytotoxic agents. These agents destroy tumor cells, causing them to release their cellular contents into the blood. The electrolyte imbalances can be life-threatening. As such, TLS is an oncologic emergency. Clinical features include GI distress, fatigue, arrhythmias, and acute kidney injury. Laboratory findings include hyperkalemia, hyperphosphatemia, hyperuricemia, and hypocalcemia. Management starts with aggressive hydration and correction of electrolyte disturbances. This may include allopurinol or rasburicase.



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Characteristics

Leukemia/Lymphoma Treatment
Leukemia-Luke and Lime-foam Treats
Tumor lysis syndrome commonly presents in patients who are undergoing treatment for leukemia or lymphoma. The cytotoxic treatment may trigger TLS.

Oncologic Emergency
Orc Emergency-lights
Tumor lysis syndrome is an oncologic emergency. That means it can result in death if not treated immediately. This is due to all the electrolyte imbalances.

Clinical Features

GI Distress
GI Flare-gun
Gastrointestinal distress is common in TLS patients, and may be due to hyperkalemia. The high serum potassium levels can result in nausea, vomiting, and diarrhea.

Fatigue
Sleepy-guy
Fatigue is most often due to hypovolemia from acute kidney injury.

Arrhythmias
Broken Arrhythmia-drum
Arrhythmias may occur if serum potassium levels get too high. Serious life-threatening arrhythmias can develop rapidly if serum potassium is not corrected.

Acute Kidney Injury
Acute-angle Kidney-injured
Catabolism of nucleic acids released from the dying tumor cells results in uric acid buildup in the blood. This travels to the kidneys and may result in urate crystal deposition in the renal tubules which can impair urine flow and glomerular filtration rate. Acute kidney injury can also occur from phosphate buildup in the blood as it binds with free calcium to precipitate calcium phosphate crystals.

Lab Findings

Hyperkalemia
Hiker-banana
Hyperkalemia remains the most dangerous component of TLS because it can cause sudden death due to cardiac arrhythmia. Patients should limit potassium and phosphorus intake during the risk period for TLS.

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Hyperphosphatemia
Hiker-phosphate-P
Hyperphosphatemia can cause secondary hypocalcemia, leading to neuromuscular irritability (tetany), dysrhythmia, and seizure, and can also precipitate as calcium phosphate crystals in the kidneys, where these crystals can cause acute kidney injury.

Hyperuricemia
Hiker-unicorn
Hyperuricemia occurs as a result of release of nucleic acids from dying tumor cells. The nucleic acids are catabolized to uric acid.

Hypocalcemia
Hippo-calcified-cow
Signs and symptoms of hypocalcemia include neuromuscular irritability, nephrolithiasis, and seizures. Although calcium is released from dying tumor cells, it is not substantial and the released phosphate actually binds free calcium ions in the blood, lowering serum calcium levels.

Management

Aggressive Hydration
Aggressive Water-bottle
Hydration is the most effective preventative measure for tumor lysis syndrome.

Electrolyte Correction
Electric-lights
Electrolytes should be monitored and corrected. Hyperkalemia can be corrected with glucose and insulin while hyperphosphatemia can be corrected with PO phosphate binders/chelators like sevelamer. Hypocalcemia should only be treated if symptomatic. Calcium gluconate is a common option in these cases.

Allopurinol
Aloe-piranha
Allopurinol should be used in patients who are at low to intermediate risk for tumor lysis syndrome. Allopurinol works by blocking xanthine oxidase, thus lowering production of uric acid.

Rasburicase
Raspberry-case
Rasburicase is a recombinant version of urate oxidase that aids in decreasing the levels of uric acid in the blood system. Rasburicase should be used in individuals who are at high risk of developing tumor lysis syndrome and in patients whose baseline uric acid is higher than 7.5 mg/dL.

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