The Pathophysiology of Tumor Lysis Syndrome in Oncology Patients

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Pathophysiology of Tumor Lysis Syndrome in Oncology Patients

Jessica Richardson RN, BSN, PCCN
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Introduction

Tumor Lysis Syndrome (TLS) is a potentially life-threatening emergency that frequently lands patients in the emergency department. TLS occurs more frequently in patients with acute leukemia than in solid tumor patients, particularly upon the initiation of chemotherapy or radiotherapy. TLS results from the rapid destruction of malignant cells and the release of intracellular contents into the extracellular space, causing a variety of complications from hyperuricemia to hyperkalemia. The pathophysiology of TLS involves a disruption in the renal mechanisms that normally maintain extracellular fluid electrolyte and acid-base balance. Extreme hyperuricemia can result in uric acid crystallization in the joints and kidneys, with hypocalcemia and hyperphosphatemia leading to calcium phosphate deposition, resulting in a potentially fatal syndrome. TLS can be a major cause of morbidity and mortality in cancer patients [1]. TLS is particularly associated with acute lymphocytic leukemia, acute myelocytic leukemia, lymphoma, and multiple myeloma [2].

Signs and Symptoms

• Flank pain
• Irregular heart rhythms
• Life-threatening arrhythmias
• Paralysis
• Gross hematuria
• Intestinal cramping
• Muscle weakness

Poster is to provide information to common reasons for patients to receive ICU. 
A higher risk of developing TLS then [8].

Signs and symptoms include: 
- Hyperkalemia will cause characteristic electrolyte abnormalities in the blood.
- Hyperuricemia may result in uric acid crystallization in the joints and kidneys, with hypocalcemia and hyperphosphatemia leading to calcium phosphate deposition, resulting in a potentially fatal syndrome.
- The key to TLS is to recognize the clinical symptoms and to institute immediate intervention and escalation of care when necessary.

References
[5] Byrnes, M., & Byrnes, A. (2010). The key to TLS is to recognize the clinical symptoms and to institute immediate intervention and escalation of care when necessary.

Pathophysiology

Table 2: Metabolic Abnormalities Associated With Tumor Lysis Syndrome

<table>
<thead>
<tr>
<th>Abnormality</th>
<th>Associated Signs/Symptoms</th>
<th>Clinical Action</th>
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Conclusion

TLS is an oncological and a major cause of mortality and morbidity in cancer patients [1]. TLS causes cell lysis before chemotherapy can be administered to tumor, potassium, phosphate, and nucleic acid in the central circulation [5]. This can cause a threat to the heart, brain, and other organs. TLS is associated with high mortality within the blood that leads to complications such as renal dysfunctions, pre-treatment, and death. Medical management of TLS is prevention, with prompt recognition of the signs and symptoms of TLS leading to early intervention, and escalation of care when necessary.

Pathology

Tumor Lysis Syndrome (TLS) results from the rapid destruction of malignant cells by chemotherapy or radiotherapy, releasing nucleic acids, which act as a calorie source for leukemic cells. TLS occurs as the result of tumor cell lysis, usually after exposure to chemotherapy or radiotherapy. When cancer cells are destroyed, they release high levels of nucleic acids into the extracellular space, causing a variety of complications from hyperuricemia to hyperkalemia. The pathophysiology of TLS involves a disruption in the renal mechanisms that normally maintain extracellular fluid electrolyte and acid-base balance. Extreme hyperuricemia can result in uric acid crystallization in the joints and kidneys, with hypocalcemia and hyperphosphatemia leading to calcium phosphate deposition, resulting in a potentially fatal syndrome.

Significance of Pathophysiology

Understanding the pathophysiology of TLS allows practitioners to predict complications associated with cancer treatments in moderate to high-risk individuals. All patients who are at high risk should receive intravenous hydration beginning 2 days before treatment and continuing for 2–3 days after chemotherapy [3]. Fluids are administered at a rate of 2–3 L/day with a goal urine output of 100–200 mL/hour [4]. This helps to maintain renal perfusion and minimize uric acid crystal formation in the renal tubules [5]. Nurses should pay particular attention to urine output; if output remains low despite aggressive fluid therapy, loop diuretics are recommended to achieve urine output [6].

Table: 2 Metabolic Abnormalities Associated With Tumor Lysis Syndrome

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Significance of Cont. (12)

Patients should also be pre-treated with allopurinol or rasburicase. Both these medications help to reduce tumor cell death levels by interfering with purine catabolism. However, allopurinol can also disrupt the xanthine oxidase alterations by increasing the conversion of enzymes that form uric acid [5].

Patients undergoing TLS should be closely monitored and receive aggressive fluid support. If TLS occurs, intravenous hydration and allopurinol should be started immediately. In addition, blood uric acid levels should be monitored daily.

Patients and family education is also important. Patients should be educated about complications of chemotherapy and the potential of TLS as early as possible. All patients should be educated for the nurse to recognize these problems and for early intervention. Especially if the patient is pre-medicating at home with allopurinol or rasburicase before treatment.

Information of correct medication administration may be key in preventing complications associated with TLS. Keeping the patient and family informed about the patient's condition and ensuring all family members to feel more in control and take a more active role in their treatment.