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Tumor Lysis Syndrome

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Abstract:
Tumor Lysis syndrome refers to the constellation of deranged metabolic state, characterized by hyperkalemia, hyperuricemia, hypocalcemia, and/ or, secondary to rapid breakdown of tumor cells. It is life threatening emergency that typically follows administration of chemotherapy or may be spontaneous. Malignancies, which have a large tumor burden, rapid turnover, as well as a speedy breakdown following chemotherapy, are susceptible. Acute lymphoblastic leukemia and non-Hodgkins lymphoma are typically predisposed. TLS is best managed by early anticipation and preventive measures than the complicated task of treating an established TLS. Vigorous intravenous hydration is the cornerstone of prevention as well as treatment. Rasburicase has revolutionized the management. It is available in India for the past 1 1/2yr, although the cost is a limiting factor. Children with acute leukemia in developing countries may reach a health facility late, with severe anemia and hyperleukocytosis and enable safe administration of fluids. Dialysis may be required when the metabolic trash overwhelms the renal excretion, resulting in renal failure. Chemotherapeutic drugs are often administered in phase manner in susceptible patients, in an attempt to prevent precipitous lysis of tumor cells. Presentation and management of TLS in relevance to the pediatric emergency room is outlined

Keywords: *Lysis, metabolic, hyperkalemia, hyperuricemia, hypocalcemia, hyperleukocytosis*

1. Introduction

Tumor lysis syndrome (TLS) refers to the constellation of deranged metabolic state, characterized by hyperkalemia, hyperphosphatemia, hyperuricemia, and /or azotemia secondary to rapid breakdown of tumor cells.

2. Definition

Tumor lysis syndrome (TLS, alternative spelling tumour lysis syndrome) is a group of metabolic complications that can occur after treatment of cancer usually lymphomas and leukemias, and sometimes even without treatment. These complications are caused by the breakdown products of dying cancer cells and include hyperkalemia, hyperphosphatemia and hyperuricemia, hyperuricosuria, hypocalcemia and consequent acute uric acid nephropathy and acute renal failure

3. Cause and Risk Factors

The most common tumors associated with this syndrome are poorly differentiated lymphomas, such as Burkitt's lymphoma, and leukemias, such (ALL) and acute myeloid leukemia (AML). Other cancers (such as melanoma) have also been associated with TLS but are less common.

Usually, the precipitating medication regimen includes combination chemotherapy, but TLS can be triggered in cancer patients by steroid treatment alone, and sometimes without any treatment—in this case the condition is referred to as "spontaneous tumor lysis syndrome".

4. Symptoms and Pathogenesis

Potassium is mainly an intracellular ion. High turnover of tumor cells leads to spill of potassium into the blood. Symptoms usually do not manifest until levels are high (> 7 mmol/L) [normal 3.5-5.0 mmol/L] and they include

- cardiac conduction abnormalities (can be fatal)
- severe muscle weakness or paralysis
- Hyperphosphatemia like potassium, phosphates are also predominantly intracellular. Hyperphosphatemia causes acute renal failure in tumor lysis syndrome, because of deposition of calcium phosphate crystals in the renal parenchyma.

- Hypocalcemia. Because of the hyperphosphatemia, calcium is precipitated to form calcium phosphate, leading to hypocalcemia. Symptoms of hypocalcemia include (but are not limited to):
- tetany
- sudden mental incapacity, including emotional lability
- parkinsonian (extrapyramidal) movement disorders
- papilledema
- myopathy

Hyperuricemia and Hyperuricosuria. Acute Uric Acid Nephropathy (AUAN) due to hyperuricosuria has been a dominant cause of acute renal failure, but with the advent of effective treatments for hyperuricosuria, AUAN has become a less common cause than hyperphosphatemia. Two common conditions related to are not features of tumor lysis syndrome.

5. Diagnosis

TLS should be suspected in patients with large tumour burdens, who develop acute renal failure along with hyperuricemia (> 15 mg/dL) or hyperphosphatemia (> 8 mg/dL). (Most other acute renal failure occurs with uric acid < 12 mg/dL and phosphate < 6 mg/dL). Acute uric acid nephropathy is associated with little or no urine output. The urine analysis may show uric acid crystals or amorphous urates. The hypersecretion of uric acid can be detected with a high urine uric acid - creatinine ratio > 1.0, compared to a value of 0.6-0.7 for most other causes of acute renal failure.

5.1. Laboratory tumor lysis syndrome: abnormality in two or more of the following, occurring within three days before or seven days after chemotherapy.

- uric acid > 8 mg/dL or 25% increase
- potassium > 6 meq/L or 25% increase
- phosphate > 4.5 mg/dL or 25% increase
- calcium < 7 mg/dL or 25% decrease

5.2. Clinical tumor lysis syndrome: laboratory tumor lysis syndrome, plus one or more of the following:

- increased serum creatinine (1.5 times upper limit of normal)
- cardiac arrhythmia or sudden death
- seizure

A grading scale (0-5) is used depending on the presence of lab TLS, serum creatinine, arrhythmias, or seizures.

6. Prevention

Patients about to receive chemotherapy for a cancer with a high cell turnover rate, especially lymphomas and leukemias, should receive prophylactic oral or IV allopurinol (axanthineoxidase inhibitor, which inhibits uric acid production) as well as adequate IV hydration to maintain high urine output (> 2.5 L/day).

Uricase is an alternative to allopurinol and is reserved for patients who are high-risk in developing TLS. It is a synthetic urate oxidase enzyme and acts by degrading uric acid.

7. Treatment

Treatment is first targeted at the specific metabolic disorder.

7.1. Acute renal failure prior to chemotherapy.

Since the major cause of acute renal failure in this setting is uric acid build-up, therapy consists of rasburicase to wash out excessive uric acid crystals as well as a loop diuretic and fluids. Sodium bicarbonate should not be given at this time. If the patient does not respond, hemodialysis may be instituted, which is very efficient in removing uric acid, with plasma uric acid levels falling about 50% with each six hour treatment.

7.2. Acute renal failure after chemotherapy.

The major cause of acute renal failure in this setting is hyperphosphatemia, and the main therapeutic means is hemodialysis. Forms of hemodialysis used include continuous arteriovenous hemodialysis (CAVHD), continuous venovenous hemofiltration (CVVH), or continuous venovenous hemodialysis (CVVHD).

8. References

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4. A. R. Moossa, Stephen C. Schimpff, Martin C. Robson (1991). Comprehensive text book of oncology volume 2 lippincott williams&wilkins. Retrieved 2 May 2012. "... result in severe metabolic derangements (e.g., hyperuricemia, hypocalcemia, lactic aci- dosis, and the acute tumor lysis syndrome) which require expeditious management. Hyperuricemia Uric acid is the end product of purine catabolism.