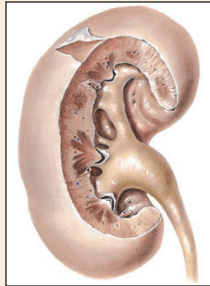


■ CNE Article

Prevention of Tumor Lysis Syndrome in an Outpatient Setting

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The prevention and identification of hyperuricemia are critical components in the management of tumor lysis syndrome (TLS), which is common in acute leukemias, large cell lymphomas, bulky tumors, large tumor burdens, and other cancers with high proliferation rates. Such cancers are very receptive to cytotoxic therapy because of increased mitotic rates. Lysis of their tumor cells results in metabolic abnormalities because of the release of their intracellular products. TLS is an oncologic emergency that could lead to renal failure and death; therefore, early identification of high-risk patients is vital for successful treatment outcomes. Treatment modalities include the use of allopurinol and hydration while implementing evidence-based practices for the prevention of TLS in the outpatient clinical center.

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Outpatient cancer care centers are the hub for many individuals diagnosed with cancer. In such centers, patients undergo multiple assessments and treatment modalities that are critical to determine the efficacy and safety of treatment. That setting can pose a multitude of challenges for oncology nurses when they are managing adverse outcomes associated with cytotoxic therapy; tumor lysis syndrome (TLS) is one such adverse outcome.

For that reason, oncology nurses need to intervene early with the proper tools and education to provide the most effective, preventive treatment of TLS. The main goals of TLS management are threefold: the identification of high-risk patients with initiation of preventive therapy, early recognition of metabolic and renal complications, and prompt supportive care with hydration and possibly hemodialysis if renal failure develops.

TLS is considered an oncologic emergency, with metabolic abnormalities that could lead to seizures, life-threatening arrhythmias, acute renal failure, and death (Ezzone, 1999). Acute renal failure in TLS is a result of volume depletion and hyperuricemia. Many patients with malignancies are dehydrated on diagnosis (Davidson et al., 2004). The causes are multifactorial and include nausea, vomiting, decreased oral intake, fasting for surgery, and fever (Davidson et al., 2004). According to Solh and Appel (2008), the incidence of TLS can range from 3%–22%, depending

on the type of malignancy, chemotherapeutic agents used, and other risk factors. For patients with TLS, the risk of renal failure (25%–38%) and death (5%–14%) is high.

Risk Factors

TLS is common in a variety of cancers with large tumor burdens, including acute leukemias, large cell lymphomas, and other cancers with high proliferation rates (Hande, Hixson, & Chabner, 1981). TLS occurs when large numbers of neoplastic cells are killed rapidly, leading to the emission of metabolic by-products (i.e., potassium phosphate and purine nucleic acids) and, subsequently, the release of those intracellular products into the bloodstream. That rarely occurs spontaneously; however, it is seen most commonly 48–72 hours after treatment (Krishnan, 2012). Signs of TLS include hyperkalemia, hyperphosphatemia, hypocalcemia, and hyperuricemia. Additional risk factors for TLS can be present in patients with solid tumors. Comorbidities such as dehydration, hyponatremia, preexisting renal damage, hyperuricemia, and elevated lactate dehydrogenase (LDH) may place patients at a higher risk of TLS. According to Tosi et al. (2008), advanced age, tumor infiltration of the kidney, and obstructive uropathy are other factors known to increase the risk of TLS.

Cytotoxic therapies that predispose patients to TLS are those with cycle-specific drugs such as cytosine, arabinoside,