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.

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, hypokalemia, 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,