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Tumor Lysis Syndrome

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Tumor lysis syndrome (TLS) is an oncologic emergency characterized by electrolyte and metabolic disturbances that most commonly are associated with lymphoproliferative malignancies and chemotherapy administration. It is manifested by hyperkalemia, hyperphosphatemia, hypocalcemia, and hyperuricemia. Without immediate treatment, TLS can lead to severe cardiac arrhythmias, acute renal failure, and death. The exact incidence of TLS in the cancer population is unknown; however, 5%–25% of patients with leukemia and lymphoma will develop TLS (Doane, 2002). Oncology nurses are in a key position to prevent the life-threatening sequelae of TLS and should be knowledgeable about the predisposing factors, signs, and symptoms and prevention and treatment strategies.

TLS is caused by three sequential processes. Rapid death of predominantly malignant cells occurs following chemotherapy administration. Cell lysis releases intracellular components, including potassium and phosphorous, into the circulation. If not promptly corrected, fatal arrhythmias can occur. Nucleic acids, also released into the circulation, are degraded further into uric acid. Compromised renal function can cause an inability to adequately excrete uric acid, resulting in hyperuricemia, precipitation of urate crystals, and acute renal failure.

Patients at risk for TLS include those with leukemias, lymphoproliferative malignancies, and bulky, chemosensitive disease such as small cell carcinoma (Gobel, 2002). Those with Burkitt's lymphoma, high-grade lymphomas, and acute and chronic leukemias are at greatest risk for developing TLS. The syndrome can occur in patients with solid tumors; however, this is less frequent than in those with lymphoproliferative malignancies. Other risk factors include preexisting hyperuricemia and elevated lactate dehydrogenase (LDH) that can be seen with some lymphomas, renal dysfunction, and the presence of volume depletion (Brant, 2002). Concomitant therapies that may predispose patients to TLS include potassium and phosphorous oral supplements, enteral and parenteral nutrition, potassium-sparing diuretics, and concurrent nephrotoxic medications (Gobel).

Case Study

Mr. F is a 72-year-old man who presents with complaints of abdominal pain, nausea, vomiting, and a 30-pound weight loss over a two-month period. A computed tomography (CT) scan of the abdomen showed a large 8 cm periaortic abdominal mass. A CT needle biopsy was completed with pathology revealing large cell lymphoma. All other staging examinations, including a CT of the chest and a bone marrow biopsy, were negative for additional sites of disease. Initial laboratory studies showed hemoglobin 11.2 g/dl, a platelet count of 245,000/mm^3, LDH 932 mcg/l, uric acid 12.6 mg/dl, sodium 136 mmol/l, potassium 4.1 mmol/l, glucose 107 mg/dl, blood urea nitrogen (BUN) 36 mg/dl, and creatinine 1.8 mg/dl. Mr. F was started on oral allopurinol 300 mg daily and received six cycles of cyclophosphamide, doxorubicin, vincristine, and prednisone chemotherapy. Following completion of chemotherapy, Mr. F underwent restaging examinations and laboratory studies. His repeat abdominal CT scan showed no change in the periaortic abdominal mass. Laboratory results showed hemoglobin 9.5 g/dl, LDH 743 mcg/l, uric acid 3.4 mg/dl, BUN 45 mg/dl, and creatinine 2.2 mg/dl. Mr. F was fatigued but had no further nausea and vomiting, and his weight was stable. The decision was made to hold further chemotherapy and observe the patient closely.

A repeat CT scan of the abdomen was performed three months later that found no change in the abdominal mass; however, an increasing retroperitoneal adenopathy existed. Mr. F was sent for a repeat needle biopsy of the abdominal mass, but the procedure was not performed because the patient was found to have BUN 52 mg/dl and creatinine 5.0 mg/dl. Mr. F underwent urologic evaluation and had bilateral ureteral stents placed for hydronephrosis as a result of obstruction from the abdominal disease. Follow-up laboratory studies after the stent placement showed BUN 30 mg/dl and creatinine 2.0 mg/dl.

Mr. F continued to be weak and fatigued and had a dramatic decrease in his hemoglobin from 11.4 g/dl to 9.3 g/dl. He was admitted for further workup and found to have a gastrointestinal bleed, BUN 132 mg/dl, and creatinine 8.0 mg/dl. Mr. F was dialyzed, underwent stent replacement, and was treated for his gastrointestinal bleeding. Once stabilized, a biopsy and pathology of his abdominal mass again revealed large cell lymphoma. Upon discharge, Mr. F's laboratory studies were hemoglobin 12.6, BUN 48 mg/dl, and creatinine 3.4 mg/dl. Mr. F then was started on a new chemotherapy regimen of rituximab.

Two days after his first rituximab therapy, Mr. F presented to the clinic with complaints of weakness, light-headedness, muscle cramping, numbness and tingling of his extremities, nausea and vomiting, diarrhea, decreased urine output, and bilateral pedal edema. Vital signs showed blood pressure 190/100 mm/Hg, pulse 64 beats per minute, temperature 100°F, and respiration 24

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