Nancy Campbell, 31, has recently been admitted to your unit for her first chemotherapy treatment of acute leukemia. She’s complaining of cramps, nausea, and an irregular heartbeat. As you draw an electrolyte panel, you suspect tumor lysis syndrome (TLS).

In this article, I’ll discuss the patient population at the greatest risk for TLS and show you how to identify its warning signs.

**TLS in a nutshell**

TLS is a rapidly occurring metabolic emergency that can cause renal failure, cardiac dysrhythmias, and even asystole. Although TLS may occur spontaneously, it’s usually a complication of effective treatment of cancers with high cell turnover. Most chemotherapy works during specific periods of the cell cycle, and it’s at these times that most cells are vulnerable. With a large number of cells vulnerable at the same time, high kill rates are possible within a short period of time. The release of byproducts from this rapid cell death makes up a constellation of problems. TLS has also been reported in conjunction with surgery, biotherapy, hormone therapy, radiation therapy, and even steroid therapy in those tumors that are sensitive to treatment.

When the tumor cells die and disintegrate, the intracellular contents become extracellular. As a susceptible cancer cell is killed, you can expect the contents of the cell to spill out; as these byproducts are released, the serum levels of potassium, phosphate, uric acid, and calcium change. As the intracellular potassium and phosphate are released, nucleic acids also spill out, causing a rise in uric acid levels. Because of the attraction between phosphate and calcium, as phosphate is released, serum calcium levels fall. Multiply this by millions of cells dying at the same time and the imbalances can become life-threatening.

Certain types of cancers can increase this risk even more because of their abnormal cell contents. For example, patients with leukemia may have as much as four times the intracellular phosphate content of normal cells. When these cells are destroyed, phosphate is released and calcium levels can fall very quickly. When plasma concentrations of uric acid rise, crystals can form in the kidneys. Because potassium is the most abundant intracellular electrolyte, there’s a danger of potassium dumping from the lysed cells and rising to deadly levels within hours of the first cell deaths.

**Who’s at risk?**

Patients with cancers that have produced large numbers of abnormal cells, have a short turnover rate, and are very sensitive to treatment are the most at risk. These include hematological cancers such as Burkitt’s lymphoma, acute leukemias, and other diffuse cancers such as small-cell lung cancer. The rapid destruction of some bulky tumors will rarely cause TLS.

**Make an ID**

Prevention is the number one priority. Patients who are at risk should be identified and you should be aware of these patients before therapy is started. Remember that the danger of a patient developing TLS is usually with the first treatments. Preexisting conditions, such as congestive heart failure, renal failure, and elevated uric acid levels, can increase the likelihood of TLS.

Baseline lab tests should include a metabolic panel (electrolytes) with blood urea nitrogen and creatinine, uric acid levels, and urine pH. Lab tests should be drawn...
Manifestation and management of TLS

Clinical manifestations
- **Neurologic:** Fatigue, weakness, memory loss, altered mental status, muscle cramps, tetany, paresthesias (numbness and tingling), seizures
- **Cardiac:** Elevated BP, shortened QT complexes, widened QRS waves, dysrhythmias, cardiac arrest
- **Gastrointestinal:** Anorexia, nausea, vomiting, abdominal cramps, diarrhea
- **Renal:** Flank pain, oliguria, anuria, renal failure, acidic urine pH

Diagnostics
- Electrolyte imbalances identified by lab test results

Medical management
- To prevent renal failure and restore electrolyte balance, aggressive fluid hydration is initiated 48 hours before and after the initiation of cytotoxic therapy to increase urine volume and eliminate uric acid and electrolytes. Urine is alkalinized by adding sodium bicarbonate to I.V. fluid to maintain a urine pH of 7 or higher; this prevents renal failure secondary to uric acid precipitation in the kidneys.
- Diuretic therapy, with a carbonic anhydrase inhibitor or acetazolamide, is administered to alkalinize the urine.
- Allopurinol therapy is administered to inhibit the conversion of nucleic acids to uric acid.
- A cation-exchange resin, such as sodium polystyrene sulfonate, is administered to treat hyperkalemia by binding and eliminating potassium through the bowel.
- Hypertonic dextrose and regular insulin are administered to temporarily shift potassium into cells and lower serum potassium levels.
- Phosphate-binding gels, such as aluminum hydroxide, are administered to treat hyperphosphatemia by promoting phosphate excretion in the feces.
- Hemodialysis may be needed when patients are unresponsive to the standard approaches for managing uric acid and electrolyte abnormalities.

Nursing management
- Identify at-risk patients, including those in whom TLS may develop up to 1 week after therapy has been completed.
- Institute essential preventive measures, such as fluid hydration and allopurinol.
- Assess patients for signs and symptoms of electrolyte imbalances.
- Assess urine pH to confirm alkalinization.
- Monitor serum electrolyte and uric acid levels for evidence of fluid volume overload secondary to aggressive hydration.
- Instruct patients to report symptoms indicating electrolyte disturbances.

Adequate hydration must be maintained to ensure optimum renal function and avoid concentrated serum levels. I.V. hydration should begin the day before treatment and be maintained at such a rate that urinary output is at least 100 mL/hour. Diuretics may be added to encourage excretion of potassium and uric acid and avoid fluid overload. Allopurinol or rasburicase are added to prevent formation of uric acid crystals. Urine pH may also be altered due to the increased excretion of uric acid.

For your consideration
Because an elevated potassium level is often the first sign of trouble, watch your patient for signs of elevated potassium such as cramping, nausea, vomiting, and an irregular heart rate. Assess medications for those that can alter levels of potassium, phosphate, uric acid, or calcium. Monitor input and output to ensure adequate hydration and notify the healthcare provider if urine output falls below 100 mL/hour, or as ordered. I.V. fluids are generally ordered to ensure adequate hydration. Monitor lab values and notify the healthcare provider if levels are above baseline. For a potassium level of greater than 6, prepare for an ECG and immediately notify the healthcare provider.

For suspected TLS, prepare to treat the imbalances. Sodium polystyrene sulfonate, insulin glucose therapy, or diuretics may be ordered to treat hyperkalemia. Calcium gluconate and phosphate binding agents, such as aluminum hydroxide, may be given for low calcium levels. Urine alkalization may also be ordered. If levels aren’t controlled via these measures, renal dialysis may be required, so a nephrologist should also be made aware of patients who are at risk for TLS.

Learn more about it