Preventing Tumor Lysis Syndrome: Two Case Studies of Unexpected Outcomes

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Background: Tumor lysis syndrome (TLS) is a potentially fatal complication in patients with large, rapidly proliferating tumor cell cancers that may occur after chemotherapy. Patients with TLS are complicated to treat and often have an unpredictable trajectory.

Objectives: The purpose of this article is to report two cases with unusual clinical manifestations and unexpected outcomes during cancer treatment and to share best practices for this situation.

Methods: The authors described details from two unusual cases and outlined lessons learned. The authors described a newly developed clinical order set (protocol) to support optimal care for patients at risk for TLS.

Findings: Implementing best practices, the order set prompts early identification of TLS risk and provides step-by-step guidance to eliminate or control TLS.

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Key words: tumor lysis syndrome; nursing care; rasburicase; methemoglobinemia

Digital Object Identifier: 10.1188/16.CJON.195-200

Tumor lysis syndrome (TLS) is caused by rapid cell lysis releasing intracellular contents and by-products (e.g., potassium, phosphate, calcium, nucleic acids) into the bloodstream, which the liver metabolizes into uric acid. Drugs used to treat lymphoid leukemias, non-Hodgkin lymphoma, and acute myelogenous leukemia often lead to the accumulation of toxic plasma levels of purine metabolites (i.e., uric acid). These high levels of uric acid and other electrolytes can cause acute renal failure, requiring dialysis, and lead to numerous metabolic disturbances (King, 2008; Wilson & Berns, 2014).

The incidence of TLS is unknown and can vary among different malignancies, with higher frequencies of TLS associated with bulky, aggressive, treatment-sensitive tumors (Fleming & Doukas, 1992). As advances are made in cancer treatment and high-dose regimens become more commonplace, TLS incidence may emerge in more malignancies (Ikeda, Krishnan, & Jaishankar, 2016; Wang et al., 2015). TLS occurs in all age groups; however, advanced age combined with impaired renal function may predispose a patient to TLS because of a decreased ability to eliminate the by-products of tumor lysis (Ikeda et al., 2016). Signs and symptoms of TLS may occur as early as a few hours after the start of chemotherapy, but they typically occur within the first 24–48 hours and may persist for five to seven days (Lewis, Dirksen, Heitkemper, & Bucher, 2013). TLS occasionally may be present prior to the start of therapy in patients with high-grade (grade 3 or 4) hematologic malignancies (Wilson & Berns, 2014). Early symptoms include weakness, muscle cramps, diarrhea, nausea, and vomiting. The primary treatment is to increase urine production using (hyper) hydration therapy and decrease uric acid concentrations (Lewis et al., 2013) using allopurinol (Zyloprim®) and rasburicase (Elitek®) (Brant, 2002; Lehne, 2013).

Diagnosis

TLS is diagnosed by laboratory tests and clinical signs and symptoms. Some healthcare teams follow the Cairo-Bishop
grading system (Cairo & Bishop, 2004) to classify and define laboratory abnormalities in serum uric acid, potassium, and phosphorus levels. This system considers laboratory value changes that occur in patients from three days before to seven days after chemotherapy begins. The clinical abnormalities classified by this system are graded based on severity and include age-adjusted serum creatinine, cardiac dysrhythmias, and seizures (Cairo & Bishop, 2004).

Outcomes and Prognosis

The prognosis for patients at risk for TLS depends on early recognition of signs and symptoms of patients, including early identification of abnormal clinical laboratory values to prevent otherwise life-threatening complications of the condition (Ikeda et al., 2016). Potential complications of TLS include uremia and oliguric renal failure because of tubule precipitation of uric acid, calcium phosphate, or hypoxanthine. Severe electrolyte disturbances (e.g., hyperkalemia, hypocalcemia) predispose patients to cardiac arrhythmias and seizures. Iatrogenic complications (e.g., pulmonary edema from overly vigorous hydration, metabolic alkalosis from excess exogenous administration of bicarbonate) also can occur and may be life-threatening if not immediately addressed. Methemoglobinemia is a rare side effect of rasburicase, a medication used to treat TLS in patients with cancer (Bucklin & Groth, 2013).

Care Goals

The goals of care for patients with cancer receiving chemotherapy include (a) identifying patients at high risk for TLS and initiating preventive therapies; (b) identifying metabolic and renal complications if they occur; and (c) quickly providing supportive care to reduce adverse outcomes (Ikeda et al., 2016; Lewis et al., 2013). Nurses can provide the life-saving care needed by patients with TLS (or those at high risk) by becoming familiar with clinical cases describing patient scenarios with TLS and lessons learned by the healthcare team members. This article examines the clues that led one healthcare team to solve the mystery of why two patients with cancer and TLS became hypoxic, and what the team did to prevent occurrences in similar patients in the future. Because of the two case studies described in this article, healthcare team members developed a multidisciplinary clinical order set (protocol) to support optimal care for patients at risk for TLS. This order set prompts early identification of TLS risk and steps to follow when TLS clinical manifestations are present to meet the goals of care and to eliminate or control TLS.

Case Study 1

A.A. was a 73-year-old man admitted to the oncology unit with fatigue, severe pain radiating into both legs, anorexia, nighttime fever, and bleeding under the fingernails. His laboratory work was notable for elevated uric acid, lactate dehydrogenase (LDH), and white blood cell (WBC) count (see Table 1). The consulting oncologist suspected acute leukemia (later confirmed by bone marrow biopsy and aspiration), with spontaneous TLS. Twelve hours after admission, A.A. received rasburicase 0.2 mg/kg IV over 30 minutes, along with allopurinol 300 mg orally daily, followed by chemotherapy. Allopurinol blocks uric acid production, and rasburicase accelerates uric acid removal, preventing crystal formation that damages kidney tubules and causes renal failure (Lehne, 2013; Lopez-Olivo, Pratt, Palla, & Salahudeen, 2013) (see Table 2). At this time, A.A. was hypoxic, believed to be because of hyperleukocytosis, which is not uncommon in severe acute myelogenous leukemia (Mauro, 2003) and TLS.

TABLE 1. Case Study 1 Laboratory Values

<table>
<thead>
<tr>
<th>Variable</th>
<th>On Admission</th>
<th>36 Hours Later (After Rasburicase)</th>
<th>2.5 Days After Admission</th>
<th>Reference Ranges</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatinine (mg/dl)</td>
<td>1.6</td>
<td>1.3–2.2</td>
<td>–</td>
<td>0.7–1.3</td>
</tr>
<tr>
<td>Lactate dehydrogenase (IU/L)</td>
<td>636</td>
<td>–</td>
<td>–</td>
<td>125–243</td>
</tr>
<tr>
<td>Methemoglobin (%)</td>
<td>–</td>
<td>–</td>
<td>16.2</td>
<td>0–1.5</td>
</tr>
<tr>
<td>Phosphorus (mg/dl)</td>
<td>–</td>
<td>3.1</td>
<td>–</td>
<td>2.3–4.7</td>
</tr>
<tr>
<td>Potassium (meq/L)</td>
<td>4.7</td>
<td>5.4</td>
<td>–</td>
<td>3.5–5.1</td>
</tr>
<tr>
<td>Uric acid (mg/dl)</td>
<td>10.2</td>
<td>&lt;0.3</td>
<td>–</td>
<td>3.5–7.2</td>
</tr>
<tr>
<td>White blood cells (k/uL)</td>
<td>161.1</td>
<td>103.4</td>
<td>–</td>
<td>5–10</td>
</tr>
</tbody>
</table>

Note. Reference ranges are from the laboratories of the authors’ institutions.
Enzymes, oncology

Clinical Journal of Oncology Nursing  •  Volume 20, Number 2  •  Preventing Tumor Lysis Syndrome

2013). Methemoglobinemia occurs in patients with a rare inherited mutation of a gene that controls enzyme activity for glucose-6-phosphate dehydrogenase, or G6PD. The deficiency is more common in patients of African or Mediterranean descent (Luzzatto & Seneca, 2014). G6PD deficiency is inherited as an x-linked trait and causes excess peroxide by-product when the peroxide molecule is not converted to water and oxygen as it should be (Luzzatto & Seneca, 2014). Methemoglobin, an oxidized hemoglobin, is a very poor transporter of oxygen, and, as levels increase, the patient will experience significant shortness of breath. The excess peroxide triggers red blood cell (RBC) hemolysis and methemoglobinemia (Browning & Kruse, 2005). Methemoglobinemia can be treated with methylene blue or blood transfusions (Percy, McFerran, & Lappin, 2005).

A.A.’s methemoglobin level declined to 7.8 on day 1 of treatment and to 2.3 on day 2. He became asymptomatic, and treatment with methylene blue was not necessary. Fortunately, his uric acid levels remained low, likely because of the initial rasburicase administration, the effect of dialysis, and the smaller effect of allopurinol administration. Eight days later, A.A. was transferred from the ICU to the oncology unit and discharged home in two weeks. He subsequently returned for additional chemotherapy treatments without consequence.

### Case Study 2

B.B. was a 26-year-old African American man with history of non-Hodgkin lymphoma. He received chemotherapy three days prior to admission. B.B. presented to the emergency department three days after his first cycle with shortness of breath, hypovolemia, tachycardia, general weakness, syncope, and 84% oxygen saturation. After being placed on supplemental oxygen, he was transferred to the ICU and referred to pulmonology services for medical care.

With oxygen at 3 L via nasal cannula, B.B.’s oxygen saturation rose to 98%, with vital signs of blood pressure 125/76, pulse 92, respirations 22, and temperature 100.3°F. B.B.’s computed tomography angiogram was negative, indicating no pulmonary infiltrates or emboli. B.B.’s healthcare team consulted the nephrologist because of elevated levels of blood urea nitrogen in B.B.’s case, the outpatient setting for chemotherapy and non-Hodgkin lymphoma. He received chemotherapy three days prior to admission. B.B. presented to the emergency department three days after his first cycle with shortness of breath, hypovolemia, tachycardia, general weakness, syncope, and 84% oxygen saturation. After being placed on supplemental oxygen, he was transferred to the ICU and referred to pulmonology services for medical care.

On day 2 after admission, B.B. was transferred out of the ICU to the oncology unit. The nephrologist noted acute hemolysis with renal injury of unknown etiology. The search for causes of the hemolysis included the following:

• A chemotherapy adverse reaction causing hemorrhagic cystitis (However, B.B. received recommended pretreatment of mesna [Mesnex®] and hydration to avoid this complication.)
• Renal lymphoma infiltration or tumor-related glomerulonephritis (not known to occur with lymphoma)
• TLS (although B.B. showed no evidence of it)

During the next three days, numerous specialists noted low uric acid levels and no evidence of TLS. B.B. received an additional two units of packed RBCs for continued low RBC counts. His creatinine level increased to 2.3 mL/dl as renal function continued to deteriorate. A sonogram evaluation of kidneys continued to deteriorate. A sonogram evaluation of kidneys detected no abnormalities.

The next day, B.B.’s primary oncologist returned from vacation and reported that B.B. received rasburicase as an outpatient prior to outpatient chemotherapy for non-Hodgkin lymphoma. The healthcare team came to new conclusions based on this revelation. The team identified that B.B., as an African American, possibly had a higher risk for an enzyme G6PD deficiency, a known association with hemolysis and methemoglobinemia. B.B.’s primary oncologist listed the following diagnoses:

• Acute tubular necrosis secondary to hemoglobinuria
• Methemoglobinemia secondary to rasburicase
• Hemolysis secondary to rasburicase

In B.B.’s case, the outpatient setting for chemotherapy and preventive rasburicase treatment obscured physicians from making an immediate identification of rasburicase as the likely cause for B.B.’s hemolysis and methemoglobinemia. Another piece of the puzzle fell into place with knowledge of B.B.’s prehospitalization treatments. The healthcare team did not know why B.B.’s uric acid levels remained so low during the hospitalization, but prior rasburicase treatment also could account for

**TABLE 2. Overview of Rasburicase (Elitek®)**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Classification</td>
<td>Enzymes, oncology</td>
</tr>
<tr>
<td>Indication</td>
<td>To prevent or correct hyperuricemia associated with cancer treatments; catalyzes the oxidation of uric acid into allantoin, a soluble metabolite of uric acid to promote uric acid removal; pregnancy category: C</td>
</tr>
<tr>
<td>Preparation considerations</td>
<td>Administer IV, reconstitute with provided diluent, infuse over 30 minutes. Start infusion 4–24 hours prior to first dose of chemotherapy. Warning: screen patients for G6PD deficiency (e.g., patients of African or Mediterranean ancestry) prior to first dose to avoid severe hemolysis with methemoglobinemia.</td>
</tr>
<tr>
<td>Adverse effects</td>
<td>Greater than 10%; mucositis, vomiting, nausea, abdominal pain, diarrhea, constipation, fever, headache, and rash; 1%–10%; neutropenia, respiratory distress, hyperphosphatemia, sepsis, and neutropenic fever; less than 1%: anaphylaxis, hemolysis, methemoglobinemia, severe rash, dehydration, acute renal failure, pancytopenia, myocardial infarction, cellulitis, cardiac failure, and hot flashes</td>
</tr>
<tr>
<td>Nursing considerations</td>
<td>Can cause anaphylactic reaction, so have diphenhydramine (Benadryl®) and epiinephrine (EpiPen®) at the bedside. Immediately and permanently discontinue for patients exhibiting hypersensitivity reactions, hemolysis, or methemoglobinemia. Keep patient well hydrated to manage uric acid. Subsequent dose efficacy may be reduced because of antibody formation. Immediately chill patient blood samples and transport them to the laboratory for testing completion within four hours.</td>
</tr>
</tbody>
</table>

**Note.** Based on information from Lehne, 2013.
the cause (Dinnel, Moore, Skiver, & Bose, 2015). The hemolysis and methemoglobinemia explain the renal function deterioration and the oxygen saturation gap with symptoms of shortness of breath and fatigue on exertion.

On day 7, B.B.'s G6PD level was 9.6 (normal range = 7–20.5 U/g hemoglobin). Unfortunately, this level is not reliable because this test must be drawn prior to the administration of rasburicase. In addition, patients of African descent exhibit a higher incidence of the G6PD enzyme deficiency; therefore, B.B.'s true G6PD levels were likely lower (more deficient) than the laboratory values reflected. With improving laboratory results, B.B. was discharged home on day 7 (see Table 3).

**Discussion**

These two patient cases describe how, within two months at one acute care hospital, 2 of 13 patients treated with rasburicase experienced methemoglobinemia. This is a higher incidence of methemoglobinemia than the reported rate of less than 1% (Sanofi-Aventis, 2009). In both cases, the patients required care in an ICU before recovering from hypoxia. These two situations prompted the team to identify a need for more stringent monitoring of potential patients with TLS and rasburicase outcomes to maximize therapeutic benefits and minimize risk for adverse reactions.

**New Order Set**

The healthcare team created a hospital order set to guide nurses, pharmacists, laboratory personnel, and physicians working with patients receiving cancer chemotherapy. The pharmacy and therapeutics council and the medical executive team council approved the order set for use with all patients with cancer (see Appendix A). To facilitate care providers' knowledge of new processes, the oncology pharmacist spearheaded an extensive educational campaign. The pharmacist ensured that the order set was placed with the admission documents for all patients with cancer.

**Screening for Tumor Lysis Syndrome and Methemoglobinemia**

When rasburicase is ordered, oncology nurses do a thorough history assessment on patients with African or Mediterranean backgrounds to identify which patients may have the enzyme deficiency. This step is critical because medical record software systems may not adequately address this aspect of ethnicity documentation. Nurses then notify the pharmacist and the physician ordering rasburicase of the screening results.

**Risk–Benefit Analysis**

Based on ethnicity, the physician decides whether to order a simple blood test for genetic testing for the G6PD deficiency. This test may take 2–3 days to provide results, so in the interim, the oncologist evaluates the risk versus benefit to determine if rasburicase should be administered.

**Assessing Tumor Lysis Syndrome Risk**

The team evaluates TLS risk level to guide future actions, including preventive treatment for TLS. The order set also guides laboratory testing and monitoring for TLS. The values that form the Cairo-Bishop grading system are the same values used to help form the treatment and monitoring approach (except seizure/arrhythmia, which is a very late finding of TLS). The patients' assessment is based on laboratory values, including G6PD; potential for TLS based on type of cancer, tumor burden, and pretreatment with allopurinol, hydration, and steroids (lessens risk); antineoplastics; and, very importantly, oncologist experience and input.

**Determining Rasburicase Dose**

The decision to use rasburicase is a joint decision involving pharmacy staff, nursing staff, and the oncologist, but the oncologist provides the ultimate decision. Dosing and re-dosing are tailored to patient condition and tumor size and characteristics. At the authors' institution, the rasburicase doses are 3 mg IV piggyback (IVPB) for patients at medium risk for TLS and 6 mg IVPB for those at high risk (Cairo & Bishop, 2004). Additional doses of rasburicase are only needed in severe cases of prolonged TLS and

**TABLE 3. Case Study 2 Laboratory Values**

<table>
<thead>
<tr>
<th>Variable</th>
<th>On Admission</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 4</th>
<th>Day 7 (Discharge Day)</th>
<th>Reference Ranges</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood urea nitrogen (mg/dl)</td>
<td>21.8</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>8.4–25.7</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>1.4</td>
<td>–</td>
<td>–</td>
<td>2.3</td>
<td>1.9</td>
<td>0.7–1.3</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>15.5</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>39–48</td>
</tr>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>5</td>
<td>–</td>
<td>–</td>
<td>6.8</td>
<td>9.2</td>
<td>13–16</td>
</tr>
<tr>
<td>Methemoglobin (%)</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>6.7</td>
<td>0–1.5</td>
</tr>
<tr>
<td>Oxygen content (ml/dl)</td>
<td>5.6</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>15–23</td>
</tr>
<tr>
<td>Phosphorus (mg/dl)</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>3.4</td>
<td>2.3–4.7</td>
</tr>
<tr>
<td>Red blood cells (m/ul)</td>
<td>1.62</td>
<td>–</td>
<td>&lt;1</td>
<td>2.26</td>
<td>2.92</td>
<td>4–5.4</td>
</tr>
<tr>
<td>Uric acid (mg/dl)</td>
<td>–</td>
<td>&lt;1</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>3.5–7.2</td>
</tr>
<tr>
<td>White blood cells (k/ul)</td>
<td>17.8</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>5–10</td>
</tr>
</tbody>
</table>

Note. Reference ranges are from the laboratories of the authors’ institutions.
are based on maintaining safe levels of uric acid. Most patients do not require additional doses. This approach has proven to be effective and far less costly (Dinnel et al., 2015). Patients receiving rasburicase also receive allopurinol to decrease the need for additional doses of rasburicase. Uric acid levels are monitored, along with other laboratory values, noting the rate of increase. A faster rate of increase requires a quick response with redosing of rasburicase (usually 5 mg, unless the increase is extreme). Of note, raburicase only treats uric acid levels. The other elevated laboratory values associated with TLS usually require nephrologist intervention. Creatinine levels usually improve with uric acid control but do not always return to normal levels because of renal injury.

Close Monitoring

Regardless of ethnicity and G6PD test results, patients on rasburicase are closely monitored for the following:
- Shortness of breath or other breathing difficulties, including hypoxia; these symptoms usually arise 1–3 days after the rasburicase dose. If any evidence of hypoxia is detected, care providers order a methemoglobin level test.
- Clinical and laboratory levels of uric acid, potassium, phosphate, and calcium; care is then tailored as indicated by these laboratory values and the patient’s condition. If methemoglobinemia is present, treatment includes methylene blue or blood transfusion.

Implications for Practice

- Advocate for genetic testing for G6PD deficiency in all patients before administering rasburicase (Elitek®).
- Be vigilant in assessing for shortness of breath and other adverse effects of rasburicase, particularly in patients with an African or Mediterranean background.
- Use standardized tools to guide best practice and promote interdisciplinary collaboration in providing care of patients at risk for tumor lysis syndrome.

References


Luzzatto, L., & Seneca, E. (2014). G6PD deficiency: A classic example of rasburicase, detailed data should be collected for each patient and the order set should be used to guide care. The order set standardizes processes and provides a thorough treatment and monitoring approach to quickly identify TLS and rasburicase complications and intervene as necessary. This multidisciplinary approach minimizes patient risk for life-threatening complications from chemotheraphy.

Implications for Nursing

These two cases exemplify how keeping patients safe requires multidisciplinary teamwork. Communication and collaboration among all team members are essential in patients at risk for TLS and form a foundation for the proactive attention needed to prevent negative outcomes. In these cases, nurses and physicians added valuable assessment data, and pharmacists guided order set development and implementation. Nurses often are considered the last line of defense in medication administration. Their advocacy role includes asking targeted questions, providing patient education, and carefully examining patients’ backgrounds and medication histories before administering rasburicase. As genetic aspects of disorders and medication responses are becoming more widely understood, healthcare providers of all disciplines must recognize and appreciate how genetic variations and family background, such as G6PD deficiency, influence health outcomes.

Conclusion

TLS can cause a life-threatening complication of hyperuricemia and is often treated with rasburicase. Rasburicase can lead to methemoglobinemia, requiring that physicians, nurses, and pharmacists provide a multidisciplinary approach for its safe and effective use. Although the reported incidence of methemoglobinemia after rasburicase is less than 1%, the potentially catastrophic nature of the side effect warrants heightened processes to protect patients. The authors’ healthcare team developed an order set aimed to improve outcomes in patients with TLS who may be treated with rasburicase. With increasing use of rasburicase, detailed data should be collected for each patient and the order set should be used to guide care. The order set standardizes processes and provides a thorough treatment and monitoring approach to quickly identify TLS and rasburicase complications and intervene as necessary. This multidisciplinary approach minimizes patient risk for life-threatening complications from chemotheraphy.
of pharmacogenetics with on-going clinical implications. 
*British Journal of Haematology*, 164, 469–480. doi:10.1111/bjh.12665


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**Tumor Lysis Syndrome Treatment and Prevention Order Set Laboratory Tests**

Obtaining baseline laboratory values is the first step for any patient at risk for tumor lysis syndrome. The following are drawn for all patients at risk for tumor lysis syndrome and prior to rasburicase administration:

- ✔ Lactate dehydrogenase x 1 if not already done
- ✔ Potassium level x 1 if not already done
- ✔ Phosphorus level x 1 if not already done
- ✔ Uric acid level x 1 if not already done
- □ Uric acid level every 12 hours for high-risk patients
- □ Uric acid level daily with morning laboratories for low- and medium-risk patients
- ✔ Nurse to order methemoglobin level after rasburicase (Elitek®) administered if patient exhibits new-onset shortness of breath
- □ G6PD level (Consider prior to rasburicase initiation if patient has a high risk for deficiency, including patients of African or Mediterranean ancestry. Please note that results take 2–3 days.)

**Medications**

These medications are given to patients at risk for tumor lysis syndrome and continued until risk subsides or has ended.

- **Low Risk**
  - □ Normal saline IV 100 ml per hour
  - □ Allopurinol (Zyloprim®) 300 mg orally daily (Dose may need to be adjusted based on renal function.)

- **Medium Risk**
  - □ Normal saline IV 100 ml per hour (not recommended to use bicarbonate with rasburicase)
  - □ Rasburicase 3 mg IV x 1
  - □ Allopurinol 300 mg orally twice a day

- **High Risk**
  - □ Normal saline IV 100 ml per hour (not recommended to use bicarbonate with rasburicase)
  - □ Rasburicase 6 mg IV x 1
  - □ Allopurinol 300 mg orally twice a day

*Note.* Boxes that are prechecked are required on all patients.

**APPENDIX A. Rasburicase (Elitek®) Order Set**

*Note.* Prepared September 2014 by Jaclyn K. Priest, PharmD, Christopher J. Norris, MD, and William W. Brown, RPh. Reviewed by Rex A. Schimpf, RPh, MS, and Frederick L. Bishop, MD. Reprinted with permission from Texas Health Resources at Texas Health Arlington Memorial Hospital.