Oncologic Emergency Update: Tumor Lysis Syndrome

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ONS Congress
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Oncologic Emergency Update: Tumor Lysis Syndrome

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Objectives

- Describe Tumor Lysis Syndrome (TLS)
- Assess for Complications of TLS
- Describe Medical and Nursing Management of TLS
Tumor Lysis Syndrome (TLS) is a term applied to a group of metabolic abnormalities that can be a potentially life threatening oncologic emergency.
TLS Pathology

- Result of rapid breakdown of malignant cells
  - Cell membrane is disturbed
  - Lysis of tumor cells
  - Release of intracellular ions, nucleic acids, proteins and their metabolites into circulation
  - Lead to multi-system organ failure
  - Hallmark sign is electrolyte abnormalities
Tumor Cell Lysis

Steroids

cytotoxics

immunotherapy

K+

Cardiac arrhythmia

Uric acid

Calcium phosphate precipitation in soft tissues

PO₄

Precipitate in renal tubules

Hypocalcemia

Renal failure

Timing

- Occurs 24-48 hours into treatment
- May persist up to one week
Risk for TLS by Tumor Type

- Burkitt’s lymphoma
- Lymphoblastic lymphoma
- Acute leukemia

- Low-grade lymphoma treated with chemotherapy, radiotherapy, or steroids
- Breast carcinoma treated with chemotherapy or hormonal therapy
- Small-cell lung carcinoma
- Seminoma

- Low-grade lymphoma treated with interferon
- Merkel’s cell carcinoma
- Medulloblastoma, neuroblastoma
- Adenocarcinoma of the GI tract

Tumor-Related Risk Factors

- Highly proliferative tumors
- Chemosensitive tumors
- Bulky tumors (>8–10 cm)

Ezzone SA. *Semin Oncol Nurs* 1999.
Rheingold SR. In: *Principles and Practice of Pediatric Oncology*, 2002.
Clinical Risk Factors for TLS

- Bone marrow involvement
- Elevated LDH
  - Hyperuricemia (uric acid >8 mg/dl)
  - Dehydration
  - Elevated WBC
- Renal insufficiency
  - Elevated BUN and Serum Cr
  - Decreased GFR
  - Decreased of absent urine output
  - Acidic urine

Ezzone SA., Semin Oncol Nurs 1993
Rheingold SR. In: Principles and Practice of Pediatric Oncology, 2002
How do we define TLS?

- Difficult to determine the incidence
- No clear definition
- Two classification systems have been developed
  - Hande & Garrow (1993)
  - Cairo & Bishop (2004)
Developed a classification system that distinguishes between lab and clinical TLS

- Limitations
  - Only a few patients with LTLS develop CTLS
  - LTLS requires a 25% increase in baseline labs. Does not account for abnormal pre-existing values
  - Requires changes within 4 days of beginning treatment. Narrow window-excludes patients with TLS before day 1 or after day 4 of chemotherapy.
Cairo & Bishop

- Attempted to develop a modified version of the classification system.
  - Goal to be clinically relevant
  - Suggested grading No TLS/LTLS/CTLS
  - 25% increase in lab value from baseline
  - True TLS: 1 of 3 most significant clinical complications must be present

Cairo MS et al. Br J Haematol 2004
## Cairo-Bishop Grading System for TLS

<table>
<thead>
<tr>
<th>Grade</th>
<th>LTLS</th>
<th>Creatinine</th>
<th>Cardiac arrhythmia</th>
<th>Seizure</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>−</td>
<td>≤1.5 × ULN</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>I</td>
<td>+</td>
<td>1.5 × ULN</td>
<td>Intervention not indicated</td>
<td>None</td>
</tr>
<tr>
<td>III</td>
<td>+</td>
<td>&gt;1.5–3.0 × ULN</td>
<td>Nonurgent medical intervention indicated</td>
<td>One brief generalized seizure; seizure(s) well controlled or infrequent; focal motor seizures not interfering with ADL</td>
</tr>
<tr>
<td>III</td>
<td>+</td>
<td>&gt;3.0–6.0 × ULN</td>
<td>Symptomatic and incompletely controlled medically or controlled with device</td>
<td>Seizure in which consciousness is altered; poorly controlled seizure disorder; breakthrough generalized seizures despite medical intervention</td>
</tr>
<tr>
<td>IV</td>
<td>+</td>
<td>&gt;6.0 × ULN</td>
<td>Life-threatening</td>
<td>Seizures of any kind that are prolonged, repetitive, or difficult to control</td>
</tr>
<tr>
<td>V</td>
<td>+</td>
<td>Death*</td>
<td>Death*</td>
<td>Death*</td>
</tr>
</tbody>
</table>

LTLS, laboratory tumor lysis syndrome; ULN, upper limit of normal; ADL, activities of daily living.

*Probably or definitely attributable to clinical TLS.

Cairo-Bishop

Definition of Clinical TLS

The presence of laboratory TLS and one or more of the following criteria:

1. Creatinine: $\chi \geq 1.5$ ULN (age $>12$ yr or age adjusted)*
2. Cardiac arrhythmia/sudden death*
3. Seizure*

ULN, upper limit of normal.

*Not directly attributable to a therapeutic agent.

Cairo-Bishop Definition of Laboratory TLS

Two or more of the following within 3 days before or 7 days after chemotherapy initiation:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uric acid</td>
<td>$\chi \geq 8 \text{ mg/dL} (476 \mu\text{mol/L})$ or 25% increase from baseline</td>
</tr>
<tr>
<td>Potassium</td>
<td>$\chi \geq 6.0 \text{ mEq/L} (6.0 \text{ mmol/L})$ or 25% increase from baseline</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>$\chi \geq 6.5 \text{ mg/dL} (2.1 \text{ mmol/L})$ (children), $\chi \geq 6.5 \text{ mg/dL} (2.1 \text{ mmol/L})$ (adults), or 25% increase from baseline</td>
</tr>
<tr>
<td>Calcium</td>
<td>$\chi \leq 7.0 \text{ mg/dL} (1.75 \text{ mmol/L})$ or 25% decrease from baseline</td>
</tr>
</tbody>
</table>

Assessment assumes hydration (± alkalinization) and UA-lowering agent will be started

Spontaneous TLS

- Release of purine metabolites that result in metabolic abnormalities
- Highly proliferative, poorly differentiated malignancies
  - Burkitt's lymphoma/leukemia
  - Acute lymphoblastic lymphoma/leukemia
Treatment Induced

- Chemotherapy/Biotherapy
- Radiation therapy
- Corticosteroids
Spontaneous vs Treatment Induced

- Usually no elevation in phosphorous in spontaneous tumor lysis syndrome.
- Postulated: rapid proliferation rates of tumor cells can increase uric acid levels through rapid nucleoprotein turnover.
- Tumor then utilizes released phosphorus for synthesis of new tumor cells.
TLS Associated With Treatment

- Acute rise in Uric Acid
  - Result of cell destruction
  - No re-utilization of PO4 released from cell lysis
  - Lack of urate oxidase to convert uric acid to allantoin for excretion
Metabolic Abnormalities
Can lead to life threatening complications

- Acute renal failure (ARF)/renal insufficiency
  - Most common

- Cardiac arrhythmias
Acute Renal Failure

- ↑ UA- rapid release & breakdown of intracellular nucleic acids
- ↑ PO4- rapid release from malignant cells which contain 4x the amount of organic/inorganic PO₄ as compared to normal cells
- Leads to ARF
Acute Renal Failure

- ↑K+ result in the kidneys inability to clear K+ released by tumor cells
  - Leads to uremia- ARF
- ↓Ca++ result of ↑PO4
  - Symptomatic or asymptomatic
  - May also be a/w a low albumin
  - Check ICA to determine if ↓Ca++ is true
Uric Acid Nephropathy

- Direct result of ↑ UA crystals forming in renal tubules and distal collecting system

- When associated with TLS it is more likely to see oliguria (<100 ml/d) or anuria

- In this patient, normalization of PO4 is necessary for quick recovery of renal function
Hyperuricemia and Hyperphosphatemia

- Most frequent electrolyte abnormality associated with ARF
  - Deposition of CaPO₄ into renal parenchyma
- ARF can then be exacerbated by intravascular volume depletion
Uremia

- CaPO4 deposition
- Tumor infiltration into the kidney
- Tumor associated obstructive uropathy
- Drug toxicity
Tumor Lysis

- ↑ Ca++
- ↑ K+
- ↑ PO₄
- ↑ Uric Acid

↓ Ca++
Metabolic Abnormalities

cause \( \uparrow \) tissue catabolism

- **Hyperkalemia** - occur within 6-72 hrs
  - Destruction of cell releases K+ extracellular
    - Cardiac
    - Numbness/tingling
    - Nausea/diarrhea
    - Muscle cramps
Hypocalcemia

- Caused by extravascular deposition PO₄ (ARF/TLS) causes acute ↓Ca++. Ca++ deposited mostly in bone is now in extraskeletal tissue

- Cardiac
- Mental Status changes
- Muscle cramping/twitching/tetany
- Carpopedal spasm
Hyperphosphotemia

- Azotemia: retention of nitrogenous waste products excreted by the kidneys
  - Oliguria (<400 ml/d)
  - Anuria
Hyperuricemia

- Nausea/vomiting/diarrhea
- Anuria
- Oliguria
- Flank pain
- Cloudy urine
Cytokine Release Syndrome

- Side effect of chemotherapy
  - Results from a massive release of cellular cytokines
  - Similar to the effects seen with Monoclonal antibody infusions
Symptoms

- Hypotension
- Tachycardia
- Fevers
- Respiratory distress
  - Hypoxia
  - Dyspnea
  - Audible wheezing
Obtaining and Assessing Labs

- CBC
- Phosphorus
- Potassium
- Uric acid
- Calcium
- Bicarbonate
- BUN
- Creatinine
- LDH
- Urinalysis
Radiology Exams

- CXR
- Renal ultrasound

Avoid IV contrast!!
# Obtaining Patient History

<table>
<thead>
<tr>
<th>Obstructive Nephropathy</th>
<th>Hyperuricemia</th>
<th>Hypocalcemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flank Pain</td>
<td>Lethargy</td>
<td>Muscle Cramps/ Twitching</td>
</tr>
<tr>
<td>Hematuria</td>
<td>N/ V</td>
<td>Numbness/ Tingling</td>
</tr>
<tr>
<td>↓ Urine Output</td>
<td>Edema</td>
<td>Carpopedal Spasms</td>
</tr>
<tr>
<td></td>
<td>Oliguria</td>
<td>Seizures</td>
</tr>
</tbody>
</table>
Obtaining Patient History

Hyperkalemia

- Diarrhea
- Nausea
- Muscle cramps
- Muscle weakness
- Irregular heart beat

- Peaked T waves
- QRS widening
- Heart block
- Ventricular arrhythmias
- Cardiac arrest
Completing Physical Assessment

- Blood pressure
- Cardiac rate and rhythm
- Respiratory:
  - Distress
  - Wheezing
  - Rales
  - ↓ Breath sounds
- Lymphadenopathy
- Abdominal masses
- Ascites
- Edema
- Weight changes
Traditional Management of TLS: Overview

- Maintain optimal hydration
- Consider alkalinization
- Correct electrolyte abnormalities
- Administer uric acid-lowering therapy
  - Historically, allopurinol, a xanthine oxidase inhibitor, has been used for prevention and treatment
- Consider hemodialysis as appropriate

Rheingold SR. In: Principles and Practice of Pediatric Oncology, 2002.
Traditional Management of TLS: Hydration

- IV hydration is the most important intervention in patients with TLS because it maintains renal blood flow and promotes urinary excretion of uric acid and phosphate.
  - Begin 24 to 48 hr before induction chemotherapy
  - 3–6 L/m²/day or 125–250 mL/m²/hr
  - Maintain urine output at 100 mL/m²/hr and specific gravity <1.010
  - No potassium in IV solution
  - Adjust sodium load per age and clinical status

Rheingold SR. In: Principles and Practice of Pediatric Oncology, 2002.
Traditional Management of TLS: Alkalinization

- Add sodium bicarbonate 20–40 mEq/L IV fluid; bolus with 0.5–1.0 mEq/kg for pH <6.5.
- Maintain urine pH 6.5–7.5 to enhance UA solubility and to promote excretion.
- Controversy: Alkalinization may increase precipitation of calcium phosphate in renal tubules; it also interferes with renal tubular reabsorption of phosphorus.
- Alkalinization is recommended during allopurinol treatment, but is not required with rasburicase.

Truini-Pittman L. *Semin Oncol Nurs* 2002.
Correction of Electrolyte Abnormalities: Hyperkalemia

- Remove K from IV fluids, diet, and drugs
- Kayexalate
- Diuretics (furosemide)
- Insulin and glucose
- Sodium bicarbonate
- Theophylline
- Hemofiltration/dialysis
- Calcium gluconate (for life-threatening arrhythmia)

Correction of Electrolyte Abnormalities: Hyperphosphatemia, Hypocalcemia

- Administer oral phosphate binder
- Dietary phosphate restriction
- Avoid calcium, unless signs of tetany
- Forced diuresis (furosemide, mannitol)
- Hemofiltration/dialysis

Management of TLS: Uric Acid-Lowering Agents

- **PURINE**
- **HYPOXANTHINE**
- **XANTHINE**
- **URIC ACID** (urinary excretion)
- **ALLANTOIN** (highly soluble)
- **RASBURICASE**
- **XANTHINE OXIDASE**
- **ALLOPURINOL**

# Recommendations for Uric Acid-Lowering Therapy

<table>
<thead>
<tr>
<th></th>
<th>Allopurinol</th>
<th>Rasburicase</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>UA level</strong></td>
<td>Normal</td>
<td>Elevated</td>
</tr>
<tr>
<td><strong>Tumor type</strong></td>
<td>Nonhematologic; Hodgkin’s lymphoma, CML</td>
<td>Burkitt’s lymphoma, lymphoblastic lymphoma, ALL, AML</td>
</tr>
<tr>
<td><strong>Tumor burden</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WBC count</td>
<td>$&lt; 50 \times 10^9/\text{L}$</td>
<td>$&gt; 50 \times 10^9/\text{L}$</td>
</tr>
<tr>
<td>LDH</td>
<td>$&lt; 2 \times \text{normal}$</td>
<td>$&gt; 2 \times \text{normal}$</td>
</tr>
<tr>
<td><strong>Cytoreductive intensity</strong></td>
<td>Mild</td>
<td>Aggressive</td>
</tr>
<tr>
<td><strong>Kidney tumor infiltration</strong></td>
<td>Absent</td>
<td>Present</td>
</tr>
</tbody>
</table>

Allopurinol/ Aloprim™

- Blocks uric acid production by inhibiting xanthine oxidase
- Decreases/Prevents hyperuricemia
- Available in Oral and IV formulations (TID)
Allopurinol: Blocks Uric Acid Production

Nucleic Acids

Hypoxanthine
Xanthine

Uric Acid

Xanthine Oxidase

ALLOPURINOL

Urine
Guidelines for Allopurinol Administration

**Recommended dose:**

- PO: 100 mg/m²/dose TID (max 800 mg/day)
- IV: 200 mg/m²/day in 1-3 divided doses, each infused over 60 minutes
- Maintain aggressive IV hydration, without added potassium
- IV alkalinization is required
- Begin allopurinol 1-2 days prior to initiating induction chemotherapy

Management of TLS: Rasburicase

- Recombinant urate oxidase
  - Cloned from *Aspergillus flavus*
  - Expressed in a modified *Saccharomyces cerevisiae* strain

- Uricolytic/antihyperuricemic enzyme
  - Transforms UA into allantoin:
    - Very soluble compound
    - Excreted by the kidneys

- Higher purity and greater specificity compared with nonrecombinant form

Rasburicase: Converts Uric Acid to Allantoin & Promotes Excretion

- Hypoxanthine
- Xanthine
- Xanthine Oxidase
- Uric Acid
- RASBURICASE
- Allantoin
- Urine

Nucleic Acids

Tumor Cell Lysis
Rasburicase Dosage and Administration

- Recommended dose and schedule: 0.15 or 0.20 mg/kg IV as a single daily dose for 5 days
- Although most studies designed to administer 5–7 days of treatment, ultimately, 2–3 doses are used most frequently\(^1\)
- No alkalinization required\(^2\)
- Filters should not be used for the infusion\(^2\)
- Do not administer as a bolus infusion\(^2\)
- Dedicated infusion line should be used\(^2\)
- Reconstituted solution should be administered within 24 hr\(^2\)

\(^2\)Rasburicase (ELITEK\(^{TM}\)) Prescribing Information. Sanofi-Synthelabo Inc.; 2004.
Uric Acid Blood Sampling During Rasburicase Therapy

- Rasburicase causes enzymatic degradation of UA in blood samples at room temperature, resulting in falsely low levels of UA
- Collect blood in prechilled heparin tubes
- Immediately place tubes in ice
- Analyze samples within 4 hr of collection

Contraindications to Uric Acid-Lowering Therapy

- Allopurinol
  - History of allergy to allopurinol

- Rasburicase
  - Hypersensitivity to rasburicase or urate oxidase
  - Known history of clinically relevant G6PD deficiency
    - Defined by intermittent jaundice or anemia, usually precipitated by drugs or infection
    - Most common in African American males and males of Mediterranean ancestry

Management of TLS: Overview of Nursing Role

- Identify patients at risk for development of TLS
- Monitor carefully to prevent potentially life-threatening complications or delays in chemotherapy administration
- Frequently communicate relevant laboratory values, physical findings, and overall patient status with clinicians
- Support patients and families through education and reassurance

Ezzone SA. Semin Oncol Nurs 1999.
Ongoing Monitoring

- Strict I/O q 4 hours
- Maintain UOP ≥3–5 ml/kg/hour
- Urine SG q 4 hours (keep ≤1.010)
- Urine pH q 4 hours (keep 7.0 – 7.5)
- Weight q 12 hours
- VS at least q 4 hours
Ongoing Monitoring: Labs

- Obtain lysis labs q 6–12 hours
- Send to lab “STAT”
- Evaluate results “STAT”
Ongoing Nursing Assessment

- **Respiratory** - rate, breath sounds
- **Edema** - periorbital, scrotal, pedal
- **Neuromuscular** - lethargy, weakness, numbness/tingling, seizures
- **Gastrointestinal** - nausea, vomiting, diarrhea
Ongoing Monitoring: Cardiac

- Potassium $\geq 6.5$
- Calcium $\leq 7.0$
- Abnormal rate or rhythm
Tumor Lysis

- ↑ K+ > 5.5
- ↑ PO₄ > 6.5
- ↑ Uric Acid > 7
- ↓ Ca²⁺ < 8.0
Management of Complications: Hyperkalemia ($K^+ > 5.5$)

- Stop potassium intake
- IV Lasix®
- Kayexalate®
- Glucose/insulin infusion
- Sodium bicarb IV push
- IV calcium gluconate
- Dialysis
Management of Complications: Hyperuricemia (Uric Acid > 7)

- Vigorous Hydration
- Alkalization/Allopurinol
- Rasburicase/Elitek
- Forced diuresis
  - Lasix®
  - Mannitol
Management of Complications: Hyperphosphatemia (Phos > 6.5)

- Oral phosphate binder (aluminum hydroxide)
- Dietary phosphate restriction
- D/C phosphate-containing meds
- IV glucose/insulin infusion
Management of Complications: Hypocalcemia (Ca^{++} \leq 8 \text{ or } \text{Ionized Ca}^{++} \leq 1.5)

- Treat hyperphosphatemia first!
- Keep serum bicarb \leq 30
- IV calcium gluconate if severely symptomatic
- Seizure precautions
Management of Complications: Acute Renal Failure

- Hemofiltration
- Dialysis
Summary

- TLS is a potentially life-threatening metabolic complication for patients with hematologic malignancies.
- Further complicating the clinical picture is the risk of renal deterioration and the need for hemodialysis support.
- These events delay chemotherapy, increase patient LOS, and significantly increase the cost of care.
Summary

- Supportive care and frequent monitoring are essential in TLS management.

- Safe and rapid lowering of UA levels:
  - Facilitates timely chemotherapy administration
  - Improves patient outcomes
  - Reduces associated health care costs

- Allopurinol and rasburicase are both effective; however, rasburicase provides advantages because of its mechanism of action and rapid onset:
  - Avoids delay in chemotherapy
  - Effectively reduces uric acid exposure
  - Good safety profile
  - Reduces potential for UA crystallization, which leads to renal failure and the need for dialysis
Case Study: Alex
10 Year Old Male

- WBC 79,000
- Lymphadenopathy
- Splenomegaly
- Mediastinal mass

What labs should you check?
Case Study: Alex

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium</td>
<td>4.1 mEq/L</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>4.0 mg/dl</td>
</tr>
<tr>
<td>Uric acid</td>
<td>16.1 mg/dl</td>
</tr>
<tr>
<td>Calcium</td>
<td>9.6 mg/dl</td>
</tr>
<tr>
<td>BUN</td>
<td>12 mg/dl</td>
</tr>
<tr>
<td>Creatinine</td>
<td>1.0 mg/dl</td>
</tr>
<tr>
<td>LDH</td>
<td>6323 U/L</td>
</tr>
</tbody>
</table>
Case Study: Alex

You receive the following IV fluid order:

D5\(\frac{1}{2}\) NS + 20mEq KCl/ L at 3000 cc/ m\(^2\)/ 24 hours

Is this order correct?
Case Study: Alex

What med(s) should Alex be on?

How often should you check his urine output & pH?

What else should you be monitoring?
Case Study: Alex

Bone marrow is positive for T-cell ALL

*When should chemo be started?*
Case Study: Alex

You check a urine:
S.G. = 1.017
pH = 8.0

What should you do?
Case Study: Alex

Chemotherapy is initiated with daunomycin, vincristine, prednisone, and L-asparaginase.

8 hours later you note that he has had only 120 cc of urine/ 4 hours, and you get the following lysis lab results:
Case Study: Alex

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>31.8</td>
</tr>
<tr>
<td>K⁺</td>
<td>5.4</td>
</tr>
<tr>
<td>Phos</td>
<td>14.5</td>
</tr>
<tr>
<td>U.A.</td>
<td>11</td>
</tr>
<tr>
<td>Ca⁺⁺</td>
<td>5.1</td>
</tr>
<tr>
<td>Creat</td>
<td>1.7</td>
</tr>
<tr>
<td>BUN</td>
<td>38</td>
</tr>
</tbody>
</table>

What should you do now?
Case Study: Alex

Interventions Available

- Increase IV Hydration
- Strict I & O
- Strict Urine specific gravity and PH monitoring
- Strict tumor lysis labs monitoring
- Remove all sources of Potassium
- Oral Phosphate binder
- IV Glucose/Insulin
- Kayexalate®
- IV Calcium Gluconate
- Forced Diuresis
- Seizure Precautions
- Renal/ICU Consult
- Hemofiltration/Dialysis
Case Study: Alex

- After 4 days in the ICU with intensive monitoring and excellent nursing care...

- Alex is transferred to the Hem/Onc Unit...

- WBC 1.2

- Tumor Lysis labs within normal limits
Case Study

- SP 55 yo dx with CLL 96’. Received multiple treatments with fludarabine, HU1D10, and decadron over a 4 year period. Achieved minimal response to standard chemotherapy.
Case Study

- January 2005 enrolled on OSU 0055
- Single agent Flavopiridol
  - Cyclin dependent kinase inhibitor that induces apoptosis
  - Increase risk of hyperacute TLS
Data

- Labs pre treatment
  - WBC 57
  - Hgb 9.0
  - Plts 117
  - K+ 4.0 mmol/L
  - Cr 1.01 mg/dL
  - IP 4.1 mg/dL
  - UA 5.8 mg/dL
  - LD 207 U/L

- CT chest
  - Bulky bilateral LAD
  - Largest 3.3x4.3 cm

- CT abdomen
  - Bulky and diffuse LAD
  - Largest 12x14 cm
  - Splenomegaly

- Bone marrow
  - Hypercellular >95 with 17p abnormality
Tumor Lysis Syndrome

- SP had TLS in all treatments as defined by lab values
  - Rise in serum Phosphorus
  - Elevated LDH
  - Hyperkalemia present in 4/6 treatments despite interventions
<table>
<thead>
<tr>
<th>Interventions</th>
<th>Treatment 1</th>
<th>Treatment 2</th>
<th>Treatment 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>30 g Kayexalate</td>
<td>60g Kayexalate</td>
<td>60g Kayexalate</td>
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<tr>
<td></td>
<td></td>
<td>20 mg IV lasix</td>
<td>1 amp D50</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 amp D50</td>
<td>10 units regular insulin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10 units regular insulin</td>
<td></td>
</tr>
<tr>
<td>Treatment 4</td>
<td>90g Kayexalate</td>
<td>150g Kayexalate</td>
<td>60g Kayexalate</td>
</tr>
<tr>
<td></td>
<td>20 mg IV lasix</td>
<td>80 mg IV lasix</td>
<td>20 mg IV lasix</td>
</tr>
<tr>
<td></td>
<td>1 amp D50</td>
<td>Rasburicase</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10 units regular insulin</td>
<td>2 amps D50</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>10 units regular insulin</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>10 mg Albuterol nebulizer</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>20 mg IV dexamethasone</td>
<td></td>
</tr>
<tr>
<td>Treatment 5</td>
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<tr>
<td>Treatment 6</td>
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Treatment 5

- Labs at 4.5 hours
  - K+ 5.5 mmol/dL
  - Cr 1.24 mg/dL
  - IP 6.2 mg/dL
  - UA 4.2 mg/dL
  - LD 262 U/L

- Morning labs
  - K+ 4.0 mmol/dL
  - Cr 1.29 mg/dL
  - IP 6.5 mg/dL
  - UA 1.1 mg/dL
  - LD 2362 U/L