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Spontaneous Tumor Lysis Syndrome in Undifferentiated Pelvic Solid Tumor with Associated New Onset Atrial Flutter: A Case Report

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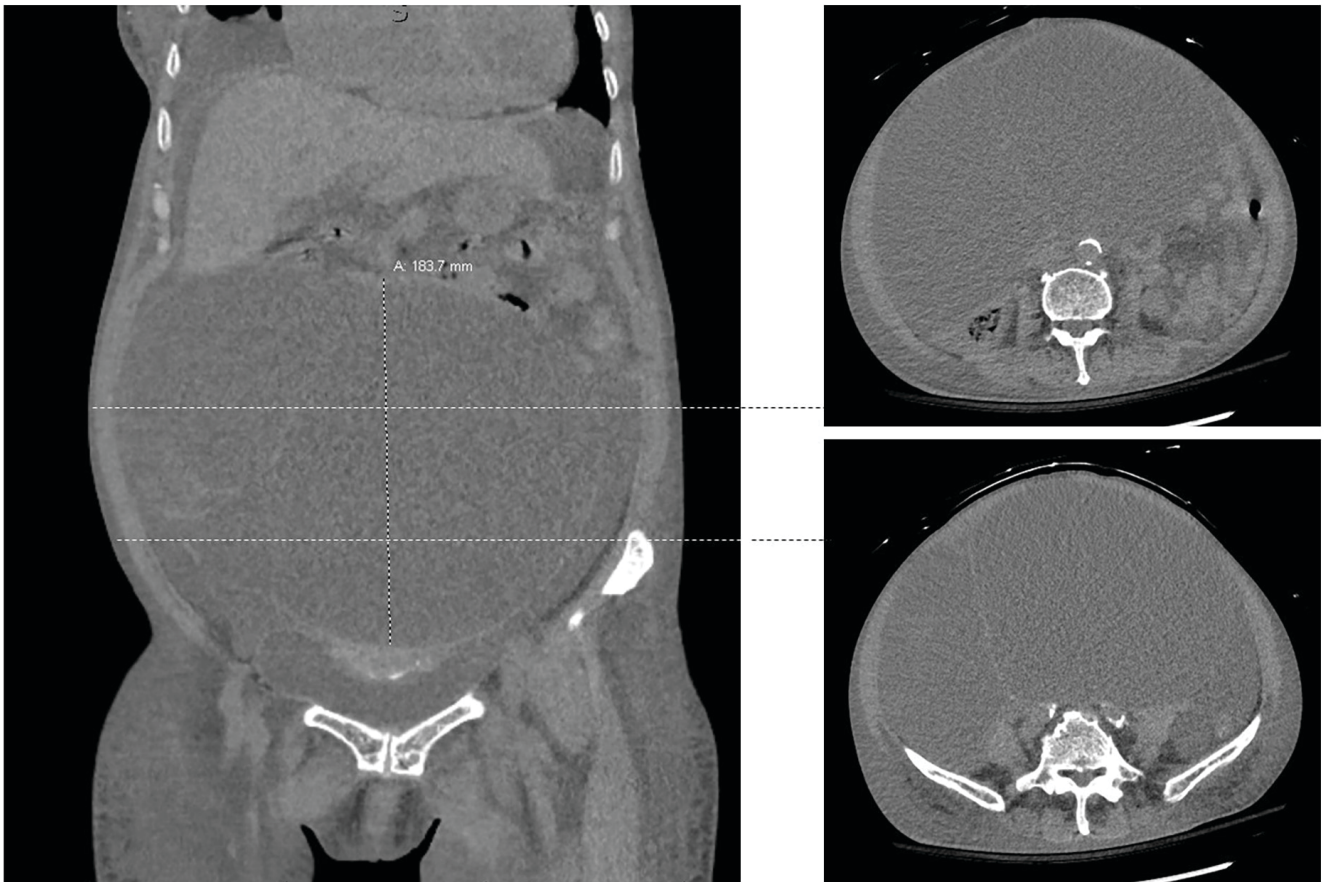

INTRODUCTION

Tumor lysis syndrome (TLS) is an oncologic emergency that is caused by electrolyte derangements from the lysis of malignant tumor cells. The syndrome consists of several laboratory abnormalities including hyperuricemia, hyperkalemia, hyperphosphatemia, and hypocalcemia[1,2]. When these lab findings are associated with end-organ damage such as acute renal failure, seizures, or cardiac dysrhythmias amongst others, it is known as clinical TLS[3]. TLS is more commonly associated with hematological malignancies given their tendency of rapid cellular turnover. The most common culprits include acute lymphocytic leukemia and Burkitt's lymphoma. It is, however, quite rare for TLS to occur secondary to a solid malignancy[4]. In fact, only 74 cases of solid-tumor TLS have been reported between 1977-2011[5]. Furthermore, in case of solid tumors, they are almost always related to administration of cytotoxic chemotherapy leading to rapid cell death. Therefore, the case described here of spontaneous TLS leading to atrial flutter in an 89-year-old female with large pelvic mass is a rare presentation.

CASE REPORT

An 89-year-old female arrived at the ED on the recommendation of her outpatient provider after a lab test showed hyperkalemia. For a few months before this, she had been noticing fatigue, bloating, and increased abdominal girth. She was referred to gynecologic oncology about a month prior to her presentation who performed an ultrasound which showed a very large complex multiloculated, complex, cystic mass that measured 24 x 13 x 28 cm overtaking the entire pelvic and abdominal region which prompted the decision to carry out an exploratory laparotomy. The preoperative lab work was significant for a potassium level of 6.0, for which she was asked to seek care immediately.

At presentation to the emergency department, her heart rate was 145, blood pressure 127/79, respiratory rate 20, and oxygen saturation of 99%. She was uncomfortable with diffuse wheezing, profound abdominal distention, and 2+ bilateral lower edema. An EKG carried out at the time exhibited atrial flutter with a right bundle branch block. Prior EKGs had been normal sinus rhythm and the patient denied any history of rhythm disorders. Initial labs were significant for potassium 5.7, BUN 105, creatinine 4.23, calcium 8.0, LDH 526, urate 15.7, and phosphate of 8.2. She was given diltiazem, which decreased her rate to the low 100's. CT abdomen and pelvic mass showed that the mass was significantly displacing the abdominal structures including vasculature, but no hydronephrosis was present (**Figure 1**). She was admitted to the inpatient service for further management.

Patient's hyperkalemia was temporized with administration of insulin and dextrose along with albuterol and multiple doses of calcium gluconate. Upon admission, she was started on bicarbonate drip at 80ml/hr and was dosed several times with intravenous Lasix. However, her urine output remained poor with only 200 ml over the course of the first 24 hours of admission. Her acute renal failure was likely precipitated by a combination of TLS and mass effect. Given the significant elevation in her uric acid level 3 mg IV rasburicase was also administered on admission and was redosed 24 hours later. Despite the above interventions, the patient's electrolytes abnormalities and her renal function worsened. Dialysis was considered as a possible management option and nephrology was consulted, however, given the overall poor prognosis the decision was made to abort this effort in discussion with the family. At this point her mental status acutely worsened, and she became lethargic, only intermittently opening her eyes during discussions. Goals of care discussions were initiated with her family, who ultimately decided on comfort measures following which she was discharged to inpatient hospice.

DISCUSSION

Tumor lysis syndrome is one of the true oncologic emergencies precipitated by the release of intracellular components of malignant cells into the bloodstream often following initiation of chemotherapy. TLS is usually defined by the Cairo and Bishop's Classification System which requires at least two of the following metabolic derangement -- potassium ≥6 mmol/L or 25% increase from baseline; phosphorus ≥4.6 mg/dL or 25% increase from baseline; calcium ≤7.0 mg/dL or 25% decrease from baseline; uric acid ≥8.0 mg/dL or 25% increase from baseline-- for the diagnosis to be made. If the above-mentioned criteria is fulfilled, then the process is characterized as laboratory TLS[3]. If any end organ damage is present in addition to the electrolyte abnormalities, including but not limited to acute kidney injury and cardiac dysrhythmias, then diagnosis of clinical TLS can be made[1,3]. Our patient made the criteria for both laboratory and clinical TLS given her increased potassium, phosphate, uric acid along with acute kidney injury as evidenced by increased creatinine and oliguria along with new onset atrial flutter.

Given TLS is a consequence of rapid destruction of malignant cells, it is often associated with rapidly proliferating and chemosensitive hematological malignancies. Although they more commonly occur following chemotherapy, TLS can also develop in absence of any intervention. In such cases it is known as spontaneous TLS. This is often observed with particularly aggressive hematological cancers including acute lymphocytic leukemia, Burkitt's lymphoma, Diffuse large B-Cell lymphoma amongst others[4,6]. Although the exact reason for this is not clear, TLS is quite rare in solid tumors and cases of spontaneous solid tumor TLS are even more scarce[6]. As stated previously, only 74 cases of solid-tumor TLS have been reported between 1977-2011 with only a fraction of them being spontaneous[5]. Therefore, the presentation of severe TLS in this patient with gynecologic tumor in absence of chemotherapy is quite unique. It is likely this was precipitated by the heavy tumor bulk in the context of other risk factors including advanced age and overall poor health of the patient[4].

Another interesting part of this presentation is the development of new onset atrial flutter in the setting of hyperkalemia caused by her TLS. The ECG changes

associated with hyperkalemia have been well-described and include peaking of T waves, P-wave flattening, and QRS complex widening. Arrhythmias less commonly seen include atrial fibrillation and even asystole or ventricular fibrillation in severe cases. Atrial flutter, as seen in our patient, rarely occurs during hyperkalemia with only two previously described cases in our literature search[7]. In this patient's case, her risk for arrhythmia was likely increased by the presumed rapid onset of hyperkalemia along with her concurrent hypocalcemia, which can potentiate the effects of hyperkalemia.

Treatment of TLS is geared towards addressing the electrolytes derangements. The mainstay of treatment remains fluid administration with close monitoring of urine output. The rate of fluids is adjusted to maintain the urine output between 80 to 100 ml/hr[2,8]. If that level of output is not reached, diuretics are utilized to increase urine flow. Fluid administration ensures increased renal blood flow with subsequent increase in GFR and upregulation of elimination of potassium, phosphorous, and uric acid. In situations where, appropriate urinary output cannot be achieved, as might be the case in patients with chronic kidney disease, dialysis is indicated. Other management options are geared towards specific electrolyte derangement. Rasburicase, which upregulates transformation of uric acid into allantoin which is highly soluble in water thus facilitating urinary excretion is used to treat acute rise in uric acid. Allopurinol, a xanthine oxidase inhibitor, which block conversion of xanthine to uric acid can be used concurrently to prevent accumulation of uric acid[2,8]. Similarly, while hyperkalemia is more definitively addressed through urinary excretion or dialysis, intravascular potassium levels can be temporized through administration of insulin. Calcium, which is often depleted due to binding by phosphate is repleted through of addition of calcium gluconate.

CONCLUSION

Tumor lysis syndrome (TLS) is an oncologic emergency that is caused by electrolyte derangements from the lysis of malignant tumor cells resulting in laboratory abnormalities including hyperuricemia, hyperkalemia, hyperphosphatemia, and hypocalcemia. It is often associated with rapidly proliferating and chemosensitive hematological malignancies often following cytotoxic treatment. The case presented in this paper where TLS occurred not only with solid tumor but spontaneously in absence of any interventions is a rare presentation.

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"Healing" *– Heping Sheng, MD*

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