

A Rare Occurrence of Tumor Lysis Syndrome in Non-Small Cell Lung Cancer

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INTRODUCTION:

Tumor lysis syndrome (TLS) is an adverse complication cytotoxic therapy that results from the breakdown of tumor cells causing catastrophic metabolic derangements. It is common in hematologic cancers like leukemia and lymphoma. It is also seen in proliferating solid tumors, such as testicular, breast, and small cell lung cancer (SCLC), but is not commonly associated with non-small cell lung cancer(NSCLC)¹.

We present to you a case report of an extremely rare occurrence of TLS in NSCLC, undergoing chemotherapy with Pembrolizumab and Paclitaxel.

CASE:

A 59 year old male with a recent diagnosis of non-small cell lung cancer 3 months back, presented with sudden onset, gradually worsening lethargy and reduced urine output for 1-2 weeks. He received whole brain radiation for the metastatic lesions 10 days following his diagnosis. He was on chemotherapy following the radiation therapy with Pembrolizumab once every week and Paclitaxel once every 3 weeks. He got his last dose of chemotherapy 1 week prior to presentation.

On arrival, he was tachycardic with heart rate of 116/min and hypotensive with blood pressure ranging between 90-100s/60s. He also had slow responses to commands. He was alert, awake and oriented to place and person.

His labs were significant for acute kidney injury with an elevated creatinine (baseline between 0.8 -1 during his last chemotherapy 1 week prior), hyperkalemia, hypocalcemia, hyperuricemia and hyperphosphatemia. Comprehensive labs are shown in *Table No. 1*.

He had oliguria with a urine output of 130 cc in 12 hours. TLS was indicated by the hypocalcemia, hyperphosphatemia and hyperuricemia and he was admitted in the Intensive Care Unit. Chest Xray showed a small right pleural effusion. CT Chest and CT abdomen /pelvis was performed, which showed right hilar mass, right pleural effusion and atelectasis as shown in *Figure 1*.

Our patient's complete metabolic panel was strongly suggestive of Tumor Lysis Syndrome with anion gap metabolic acidosis. After 2 liters of normal saline bolus, the patient was started on sodium bicarb drip 150 mEq at 200 cc/hour. Patient was also started on rasburicase and oral kayexalate was given. The patient remained in the ICU for 6 days and was downgraded to general-med with telemetry monitoring upon stabilization.

| Test | Result | Normal Value |
|-----------------|------------------------------|-------------------------------------|
| RBC | 3.13 x 10 ⁶ /cumm | 4.40 - 6.20 x 10 ⁶ /cumm |
| Hemoglobin | 9.1 g/dL | 13.0 - 17.0 g/dL |
| Neutrophils | 82.0% | 37.0 - 75.0% |
| Lymphocytes | 7.0% | 12.0 - 50.0% |
| BUN | 148 mg/dL | 6 - 20 mg/dL |
| Creatinine | 9.82 mg/dL | 0.66 - 1.25 mg/dL |
| Calcium | 7.3 mg/dL | 8.4 - 10.0 mg/dL |
| Sodium | 131 mmol/L | 136 - 145 mmol/L |
| Potassium | 6.2 mmol/L | 3.5 - 5.1 mmol/L |
| Chloride | 87 mmol/L | 99 - 112 mmol/L |
| CO ₂ | 12 mmol/L | 21 - 33 mmol/L |
| Anion Gap | 32 mEq/L | |

Table. 1 showing relevant lab values



Figure 1 showing pleural effusion and mass

DISCUSSION:

Tumor Lysis Syndrome is a potentially fatal oncologic emergency that is preceded by anti-neoplastic therapy. It occurs when a large number of tumor cells breakdown rapidly and cause electrolyte derangement. The massive release of metabolites is at a more rapid rate than the excretion capacity of the kidneys. This triggers a cascade of life threatening complications in response to the disrupted homeostasis. The released potassium can lead to dangerous dysrhythmias and the hyperphosphatemia can cause secondary hypocalcemia. This can in turn precipitate arrhythmias, seizures, an episode of tetany, as well as deposition of calcium phosphate crystals in various organs, especially kidneys, ultimately driving the acute kidney injury in TLS. Hyperuricemia can also accelerate kidney injury by both crystal dependent and independent processes. The cytokine storm causes further deterioration of clinical status and can lead to multi-organ failure².

TLS is commonly associated with high grade rapidly proliferating hematologic malignancies after chemotherapy. It is seldom associated with solid tumors. Even though less common, it is seen with small cell lung cancers relative more frequently, but its presence in non-small cell cancers is very seldom^{1,3,4}.

The low proliferative rate and chemo-resistant nature of NSCLC is thought to be the reason why TLS is rarely reported in NSCLC^{1,4,5}. To our knowledge, this is will only be the 10th case report in literature of TLS in association with NSCLC.

CONCLUSION:

With this case report, our aim is to emphasize on the close clinical suspicion needed to diagnose TLS in solid tumors and initiate treatment promptly to avoid a fatal outcome.

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