Spontaneous Tumor Lysis Syndrome in a Patient with Metastatic Small Cell Carcinoma of the Lung

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Abstract

Tumor lysis syndrome (TLS) is a life-threatening condition which could result in electrolyte imbalances, acute renal failure, seizures, arrhythmias and sudden death. It is mostly seen after the initiation of chemotherapy in hematological malignancies such as Burkitt’s lymphoma and other Non-Hodgkin’s Lymphoma. Solid tumors are rarely associated with Tumor Lysis Syndrome. TLS prior to the initiation of chemotherapy is known as spontaneous TLS, of which most of the literature involves hematological malignancies. There have been only a handful of case reports of spontaneous TLS occurring in solid tumors. We are presenting a patient who was newly diagnosed with metastatic small cell cancer of lung who developed spontaneous TLS. To our knowledge this is the first reported case of spontaneous TLS in small cell cancer of the lung. We will also review the literature on spontaneous TLS in solid tumors and whether as clinicians we can identify high-risk patients. Prompt diagnosis is essential in order to initiate necessary prophylactic precautions. After reviewing the few case reports it appears bulky tumors, site of metastases, prior renal functions may predict risk of suffering from TLS in solid tumors.

Keywords: Spontaneous tumor lysis syndrome; Small cell cancer of lung; Acute renal failure; Chemotherapy; Solid tumors

Introduction

Tumor Lysis Syndrome is characterized by an array of metabolic derangements such as hyperuricemia, hyperphosphatemia, hyperkalemia and hypocalcemia. The rapid release of cellular components into the cells after the lysis of malignant cells [1] leads to the above metabolic abnormalities. It is predominantly seen after the initiation of chemotherapy in hematological malignancies and is seldomly seen in solid tumors [1]. We present a case of a patient diagnosed with metastatic small cell carcinoma who developed tumor lysis syndrome prior to the initiation of chemotherapy, which is a rare manifestation of this disease. With a thorough search of Medline, Cochrane and CINAHL databases, to our knowledge this is the first reported case of spontaneous tumor lysis syndrome in small cell carcinoma of the lung.

Case Presentation

We present a 73 year old Caucasian lady who presented to our hospital with complaints of abdominal distention and bilateral lower extremity swelling that had been ongoing for 2 weeks. At her primary care physicians’ clinic she was found to have an ALT of 315, AST of 689, ALP of 294, GGTP of 465 and Lipase of 92 and was sent to our hospital for further evaluation. On further questioning she complained of non-bloody diarrhea that started a week prior to her visit to her family physician. She denied suffering from any recent infections, fever, chills, vomiting, abdominal pain, or ingestion of any new medications. She denies having been diagnosed with any medical illnesses or malignancies in the past. She has never been diagnosed with any renal disease in the past. Apart from a multivitamin she takes no prescribed medications. She had a 50 pack year smoking history and denies abusing IV drugs. Her mother died of a pulmonary embolism and her sister has been diagnosed with lung cancer, the details of which she was not aware of.

On physical examination her vitals were within normal limits. She appeared cachectic. There were no palpable lymph nodes. Her liver was enlarged, hard and nodular. She had a palpable spleen. A moderate amount of ascites and a positive fluid thrill was elicited. Her abdomen was tender to palpate and was encompassing her entire abdomen. There were no signs of guarding or rigidity. Her blood pressure was 135/70 mmHg. She had deranged liver enzymes. Her salicylate levels, ethanol levels and hepatitis panel were all within normal limits. Her WBC count on presentation was 9910/mcL, her hematocrit was 35.1% and Platelet count was 214,000/mcl. The pertinent initial labs are all revealed in Table 1.

A CT scan of her Thorax and Abdomen revealed a right upper lobe mass of approximately 4cm with right hilar and mediastinal lymphadenopathy, and bronchial wall thickening. Her liver was enlarged measuring 19 cms in the cranio-caudal dimension with innumerable subcentimeter, hypodense, non-enhancing lesions with no arterial phase enhancement. There was a high suspicion that she was suffering from a metastatic lung malignancy. Once the patient was aware of her findings she requested outpatient follow up. Appointments were made with our Oncology, Pulmonology and Gastroenterology departments.

She failed to make her scheduled appointments and returned to the hospital after 10 days. At this time she was suffering from worsening dyspnea, non-productive cough and abdominal distention. Her liver was tender to palpate and was encompassing her entire abdomen. A repeat CT scan of her Thorax and Abdomen revealed a rapidly progressing right upper lobe mass with post-obstructive atelectasis.

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Table 1: The pertinent initial labs.

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<th>Laboratory Tumor Lysis Syndrome</th>
<th>Cardiac Tumor Lysis Syndrome</th>
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<td>Hyperuricemia: Uric acid &gt;8 mg/dL (475.8 μmol/l) in adults or above the upper limit of the normal range in children</td>
<td>Cardiac dysrhythmia or sudden death probably of definitely caused by hyperkalemia</td>
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<td>Hyperphosphatemia: Phosphorus &gt;4.5 mg/dL (1.5 mmol/L) in adults or &gt;6.5 mg/dL (2.1 mmol/L) in children</td>
<td>Cardiac dysrhythmia, sudden death, seizure, neuromuscular irritability</td>
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<td>Hyperkalemia: Potassium &gt; 6 mmol/L</td>
<td>Eletany, paresthesias, muscle twitching, carpopedal spasm, Trousseau's sign, Chvostek's sign, laryngospasm, or bronchospasm, hypotension, or heart failure probably or definitely caused by hypocalcemia</td>
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<td>Hypocalcemia: Corrected calcium &lt;7.0 mg/dL (1.75 mmol/liter) or ionized calcium &lt;1.2 (0.3 mmol/liter)</td>
<td>Increase in the serum creatinine level of 0.3 mg/dL (26.5 μmol/liter) or a single value &gt;1.5 times the upper limit of the ageappropriate normal range if no baseline creatinine measurement is available</td>
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<td>or the presence of oliguria, no baseline creatinine measurement is available</td>
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Table 2: Laboratory Tumor Lysis Syndrome and Clinical Tumor Lysis Syndrome [21].
of Bisphosphonates [22] too have been reported. It is unclear as to whether TACE or Bisphosphonates puts a patient at higher risk as there have been no further reports since.

Our patient had a uric acid >8 mg/dL, phosphorus of >4.5 mg/dL. She did develop acute kidney injury as her creatinine increased by more than 0.3 mg/dL [23]. As she had two of the metabolic derangements and acute kidney injury she had clinical tumor lysis syndrome [24]. Our patient had a metastatic, highly chemosensitive tumor with a high tumor burden. She also had an elevated LDH and liver metastases. All these findings put her at a higher risk of developing TLS. Prophylactically starting her on Allopurinol and aggressive hydration [7] may have prevented her from developing TLS. It may not have prevented her mortality as she died of respiratory failure secondary to extrinsic bronchial obstruction from her tumor rather than from the TLS.

Need the presence of two metabolic derangements for diagnosis of Laboratory TLS and for a diagnosis of Clinical TLS one needs Laboratory TLS plus one of the three clinical criteria.

Conclusion

In conclusion, TLS is an oncological emergency that should be promptly identified and requires aggressive management as it is reversible if treated early. It’s incidence is higher in hematological malignancies but bulky tumors or chemosensitive tumors have a high predilection towards developing TLS after initiation of chemotherapy. Although there are studies in patients with AML or other leukemias and lymphomas, it is difficult to risk stratify patients to develop acute or spontaneous TLS. By reviewing the limited case reports available it appears that patients suffering from solid tumors who present with azotemia, significantly elevated LDH (>1000 IU/L), bulky tumors, hepatic metastases, bony metastases or rapidly growing primary tumor are at greater risk. Since its incidence is low, the benefits of TLS prophylaxis in this population are unknown. Its incidence may be rising and as clinicians we should be vigilant in identifying the higher risk patients firsthand. After identification we should keep a close monitor on their electrolytes, renal function, LDH and uric acid levels. A low threshold should be kept for initiation of TLS prophylaxis in the high-risk population.

References