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A CASE REPORT ON TUMOR LYSIS SYNDROME ASSOCIATED WITH NEUROENDOCRINE TUMOR

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ABSTRACT

Tumor lysis syndrome is a life-threatening emergency. Hence rapid diagnosis is necessary to prevent the progression of the disease and development of acute kidney injury. Prophylaxis and early identification are key factors in managing the condition. This case focuses on the early management of Tumor lysis syndrome and the rare occurrence of TLS in Neuro endocrine tumors.

INTRODUCTION

Tumor lysis syndrome is a clinical condition that can occur spontaneously or after the initiation of chemotherapy. TLS is usually seen in lymphomas or leukemia's with high white cell count. TLS is associated with the following metabolic derangements: hyperkalemia, hyperphosphatemia, hypocalcemia, and hyperuricemia leading to end-organ damage. This can lead to acute kidney injury (AKI), fatal arrhythmias, and even death. Due to the rapid turnover of tumor cells, there is an overwhelming production of uric acid, which then crystallizes along with calcium phosphate in the renal tubules causing obstructive uropathy and decreased glomerular filtration rate. [1] The incidence and severity of the tumor lysis syndrome depends on the cancer mass, the potential for lysis of tumor cells, the characteristics of the patient and supportive care. [2] Cairo Bishop criteria is used for diagnosing TLS.

Laboratory Tumor Lysis Syndrome

Abnormality in 2 or more of the following, occurring within 3days before or 7days after chemotherapy:

- Uric acid ≥ 8 mg/dL or 25% increase from baseline
- Potassium ≥ 6mEq/L or 25% increase from baseline

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- Phosphate ≥ 4.5mg/dL or 25% increase from baseline (≥ 6.5 for children)
- Calcium ≤ 7 mg/dL or 25% decrease from baseline

Clinical Tumor Lysis Syndrome

Laboratory tumor lysis syndrome plus 1 or more of the following:

- Creatinine > 1.5 times upper limit of age-adjusted reference range
- Cardiac dysrhythmia or sudden death
- Seizure [7]

Neuro endocrine tumors commonly involve GI tract, lungs, pancreas, and adrenal glands. [5] NETs are usually detected in the late stages when the disease has already progressed. Tumor lysis syndrome occurring in solid tumors is rare, even more rare in NETs.

CASE STUDY

A 71-year-old lady with a history of systemic hypertension and dyslipidemia as comorbidities, presented to the oncology department with complaints of generalized weakness, loss of appetite, nausea, abdominal distension and back pain of one-month duration. Her ultrasound abdomen showed abdominal lymphadenopathy with enlarged lymph nodes in peri pancreatic and pre-aortic and para-aortic region with compression of distal common bile duct (CBD) leading to ectasia of CBD and intrahepatic biliary radical dilatation (IHBRD). The biopsy from Para aortic lymph node showed morphological features and immunohistochemistry profile consistent with neuroendocrine tumor, with unknown primary. Her plasma chromogranin level was also elevated (2217 ng/ml). Her serum bilirubin steadily worsened due to biliary obstruction from the nodal mass. She was started on Chemotherapy with Carboplatin. She also received Inj. Octreotide 30 mg depot in view of neuroendocrine carcinoma with high serum Chromogranin A level. [8] The next day after chemotherapy her serum potassium was 6.1 meq/l, phosphorus 13.3 mg/dL, uric acid 12.11 mg/dL and calcium level was 6.76mg/dL. She fulfilled the lab criteria for TLS with hyperkalemia, hyperphosphatemia, hyperuricemia and hypocalcemia. She was started on T. Allopurinol 100 mg once daily. Her serum creatinine started to increase with decrease in the urine output, fulfilling the clinical criteria for TLS as well. She was managed with IV fluids, Inj. Rasburicase 1.5 mg, T. Febuxostat 40 mg BD, phosphate binder (T. Sevelamer 800 mg TDS) and anti-hyperkalemic measures. Her peak Creatinine went up to 5.49 mg/dl. Her renal function improved gradually with medical management and did not require dialysis support.

DISCUSSION

Quick and early recognition of the renal and metabolic derangements associated with tumor lysis syndrome and initiation of treatment can save a patient's life. [2] Prophylactic measures are needed to prevent tumor lysis syndrome in high-risk patients before initiating therapy. [3] Patients with TLS should be treated timely with proper fluid management, urinary alkalization, correction of acidosis and electrolyte imbalance, inhibition of the formation of uric acid or destruction of the already circulating uric acid molecules, and a reduction of chemotherapy intensity. [4] They should also be provided with dietary restrictions for potassium and phosphate. Patients with solid tumors who have a greater risk for TLS should get intravenous hydration and allopurinol before initiating the chemotherapy. Once the patient is diagnosed with TLS they should be provided with higher amounts of fluids, allopurinol or febuxostat, phosphate binders and calcium supplements. Using high doses of calcium supplements can cause calcium phosphate deposition in urinary tubules and blood vessels due to high calcium phosphorus product, hence priority should be given to correction of hyperphosphatemia first. Continuous cardiac monitoring during hyperkalemia correction and careful correction of other electrolyte imbalances is cornerstone for the treatment. Hence awareness should be created among health professionals regarding possibility of TLS even in low-risk tumors like NET and when to start anti TLS treatment. [6]

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CONCLUSION

Occurrence of Tumor lysis syndrome in neuro endocrine tumors is rare. Increase in tumor burden from metastatic cancer is a contributing risk factor for the development of TLS in solid tumors. Prophylactic measures and early diagnosis of TLS is important in saving the life of a patient. A delay in diagnosis or treatment of TLS can put the patient at risk of acute kidney injury and the need for dialysis. A multidisciplinary approach involving the oncologist, nephrologist, clinical pharmacist, and dietitian is crucial in the management of tumor lysis syndrome.

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CONFLICTS OF INTEREST

There are no conflicts of interest.

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