CLINICAL VIGNETTE

Spontaneous Tumor Lysis Syndrome in Mantle Cell Lymphoma

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Case

An 83-year-old male was referred to the ED for abnormal labs. He had multiple problems including paroxysmal Atrial Fibrillation, non-obstructive CAD, Chronic Kidney Disease and Heart Failure with a recovered EF. Routine bloodwork drawn earlier that day revealed multiple derangements including a new leukocytosis, anemia and thrombocytopenia. The most notable lab abnormality was a white cell count of 40 with a lymphocyte predominance and smudge cells on peripheral smear and Hbg of 6.7. Other abnormal labs included hyperkalemia and a new acute kidney injury with new elevated Creatinine of 3.7 from baseline 1.5. Potassium was 5.0 mmol/L and LDH and uric acid were elevated at 380 and 15.4. Pertinent physical findings including 1+ distal tibial and mild forearm edema. EKG did not demonstrate any T wave changes. Upon further discussion with the patient, he reported a new mild fatigue and decreased appetite over the past 2-3 weeks. Vitals signs on admission were BP of 107/52, HR 64, RR 16 and temp 97.1°F. His presentation raised concern for spontaneous tumor lysis in the setting of an undiagnosed hematologic malignancy. Hematology/Oncology was consulted in the ED who recommended aggressive IV hydration and a single dose of rasburicase. He was admitted to the MICU and transfused 1 unit of PRBCs. CT imaging confirmed the presence of mediastinal and intraabdominal adenopathy as well as splenomegaly. Labs were monitored q6 hours and the patient was transitioned to allopurinol. Over the next three days, his creatinine returned to near baseline and electrolyte abnormalities had stabilized and he was subsequently discharged. An outpatient bone marrow biopsy favored mantle cell lymphoma. Due to severity of disease, the recommendation was to initiate chemoimmunotherapy with rituximab and bendamustine. Final diagnosis is pending FISH analysis and cytogenetic studies.

Discussion

Spontaneous tumor lysis syndrome (TLS) is an oncologic emergency caused by tumor cell lysis resulting in the release of large amounts of potassium, phosphate, and intracellular contents into the circulation.[1] Although most commonly a complication after initiating cytotoxic therapy in lymphoid malignancies, it can occur spontaneously with other tumor types as in this case, Mantle Cell Lymphoma. TLS is considered spontaneous when it occurs prior to cytotoxic or definite treatment. A widely used diagnostic criteria system proposed by Cairo and Bishop classifies TLS based on laboratory findings and clinical manifestations.[2] Lab findings reveal hyperkalemia, hypocalcemia, hyperphosphatemia, hyperuricemia, and acute kidney injury.[3] Associated symptoms may include lethargy, edema, seizures, cardiac dysrhythmias and potentially sudden death. Treatment is centered around aggressive hydration, correction of electrolyte abnormalities and uric acid lowering therapy.[4] In oliguric individuals, renal replacement therapy may also be warranted. With regards to hyperuricemia, administration of uricostatic agents (allopurinol), and/or recombinant urate oxidase (rasburicase) should be initiated. Rasburicase is often indicated in cases of severe hyperuricemia[5] and has a more rapid onset than allopurinol. Some literature suggests screening for G6PD deficiency prior to administering the medication. In the case of this patient, he was deemed low risker due to his Mexican heritage but nonetheless, a screen was completed.

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