

Spontaneous tumor lysis syndrome in a patient of chronic lymphocytic leukemia

Sir,

Spontaneous tumor lysis syndrome (TLS) describes electrolyte abnormalities and renal dysfunction occurring in the presence of rapid cellular turnover before the administration of cytoreductive therapies.^[1] Spontaneous TLS is well-described in Burkitt's lymphoma, acute lymphoid leukemia, diffuse large B-cell lymphoma and in solid malignancies such as breast cancer, lung cancer, prostate cancer and malignant melanoma.^[2-4] Spontaneous TLS is exceedingly rare in patients with the chronic lymphocytic leukemia (CLL).

A previously healthy 57-year-old male patient was admitted to the emergency with 2 days history of nausea, vomiting and anuria. Physical examination revealed generalized lymphadenopathy and splenomegaly (4 cm below costal margin). Laboratory finding revealed hemoglobin 12 g/dL, total leucocyte $152 \times 10^9/L$ (90% lymphocyte), platelet count $150 \times 10^9/L$, urea 142 mg/dL (normal 10-50), creatinine 8.1 mg/dL (normal 0.5-1.8), calcium 6.2 mg/dL (normal 8.1-10.4), phosphate 4.0 mg/dL (normal 2.5-4.5), uric acid 14 mg/dL (normal 2.0-7.4), sodium 139 mEq/L (normal 130-149), potassium 6.4 mEq/L (normal 3.5-5.0) and metabolic acidosis (pH-7.29). Liver function test were within normal limit. We started aggressive hydration with lasix, urinary alkalization with sodium bicarbonate and allopurinol. Urine output was subsequently increased and serum creatinine was decreased from 8.1 to 1.1 and other abnormal parameters were within the normal limit over a period of 7 days. Further flow cytometric analysis of peripheral blood revealed 90% CD19+, CD5+ abnormal lymphoid cell with lambda chain restriction, which are positive for CD20, CD23, CD45 and negative for FMC 7 and CD3. Coombs test was negative and lactate dehydrogenase was 521 U/L. Further staging work-up consisted with diagnosis of CLL Rai Stage II. After recovery of acute renal failure, he has given fludarabine (25 mg/m²) d1-3, cyclophosphamide d1-3 (250 mg/m²) and rituximab (375 mg/m²) d1. After six cycle, the disease was in complete remission.

TLS is characterized by elevated serum levels of uric acid, potassium, phosphate, and serum creatinine due to release of intracellular contents of tumor cells and lead to acute renal failure and fatal arrhythmias. TLS most often occurs during chemotherapy for high grade lymphoma and leukemia. TLS in CLL is rare, it has been observed after treatment with either chlorambucil, fludarabine, rituximab

or steroid.^[5] Spontaneous TLS has not been reported in CLL. It is characterized by hypocalcemia, hyperkalemia, hyperurecemia, acute renal failure, but hyperphosphatemia is not the usual feature. The reason for normal phosphate level might be due to reutilization of phosphorus by rapidly proliferating tumor prior starting of chemotherapy, but in post chemotherapy TLS, rapid cell death secondary to treatment produces increased uric acid and phosphorous levels but the phosphorous is not used for synthesis of new cells. It is reasonable to do all routine lab parameters in all hematological and solid malignancies to initiate treatment of TLS as early as possible when suspected. Prophylactic hydration and administration of allopurinol are initiated prior to giving chemotherapy to reduce the risk of developing renal failure and fatal arrhythmias secondary to hyperkalemia. In the case presented, the patient had been diagnosed with CLL at the time of development of the syndrome.

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