Letter to the Editor

Succesful Management of Tumour Lysis Syndrome in Burkitt's Lymphoma

Sir,

Tumour Lysis Syndrome (TLS) is a life threatening metabolic complication occurring in malignancies with high tumour load and rapidly proliferating cells. These generally include - leukemias and lympho-proliferative disorders especially aggressive lymphomas like Burkitt's lymphoma. It is associated with significant morbidity and mortality. TLS is characterized by the increased release of intracellular contents into the blood stream leading to hyperuricemia hyperkalemia and hyperphosphatemia with accompanying renal compromise. TLS usually occurs following chemotherapy; however, it may also occur spontaneously.[1]

CASE: An 11-year-old boy presented with complaints of pain in abdomen, loss of appetite and progressive weakness of 3 months duration. There was no history of fever, bladder / bowel dysfunction. Prior to hospitalization he was investigated outside by a general practitioner and received treatment for colitis. Ultrasound & CT scan were done which revealed matted bowel loops, dilated large gut with interloop fluid, swollen pancreas, thickened bowel loops with infarction and fistulization, midgut volvulus, mild ascites and bilateral pleural effusions. Further barium meal follow through showed dilated jejunal loops and malrotation of the gut. Pleural tap was exudative in nature and patient was put on ATT empirically. He also underwent FNAC of intestinal wall, which was suggestive of lympho-proliferative disorder. Physical examination revealed an average nourished boy in mild respiratory discomfort. He was conscious, oriented, afebrile, respiratory rate of 26/min, pulse rate 84/min, BP 110/70mmHg. Patient had mild pallor, bilateral pedal edema, no icterus, cyanosis, no clubbing or peripheral lymphadenopathy and JVP was normal. Systemic examination: CVS was normal. There were absent breath sound at right base. Patient had distended abdomen with hepato-splenomegaly and ascites. CNS examination was essentially normal.

Investigations: Hb 11.5 gm%, TLC 8700/ cumm, DLC – P74, L19, E4, M3, platelets 7.03 lac/cumm, ESR 05 mm in 1st hour, random blood sugar 90mg%, BUN 6.8 mg%, creatinine 0.5 mg%, uric acid 3.5 mg%, calcium 9.0 mg%, phosphorus 3.5mg%, sodium 139 mEq/L, potassium 4.6 mEq/L, serum bilirubin total 1.0 mg% SGOT 132 IU/L, SGPT 200 IU/L, serum protein 6.4 gm%, albumin 4.0gm%. Chest X-ray showed right pleural effusion. ECG was within normal limits. Further ascitic tap was done which was suggestive of acute lymphoblastic lymphoma / leukemia. Bone marrow aspiration showed 7% atypical, lymphomatous cells suggestive of Burkitt's lymphoma. Anti tubercular treatment was discontinued. Patient was started on Allopurinol and hydration. He received modified chemotherapy because of deranged liver functions (Low dose cyclophosphamide + prednisolone). Within 48 hours of starting chemotherapy patient developed deranged renal parameters – BUN 107.8-mg%, creatinine 3.2-mg%, sodium 133 mEq/L, potassium 5.3 mEq/L, phosphorus 12.4mg%, uric acid 11.5-mg%, calcium 8.0 mg%. Next day patient became more short of breath, his urine output dropped and he developed facial puffiness and tachycardia (138/min). Chest showed bilateral crepitations with absent air entry in right lung. ABG analysis showed metabolic acidosis, so patient was transferred to ICU and was put on BiPAP mask. Patient became drowsy, his urine output dropped further. Repeat investigations showed, Hb 11.4 gm%, TLC 10,000/cumm, BUN 127.8 mg%, creatinine 4.0 mg%, potassium 5.6 mEq/L, sodium 138 mEq/L, uric acid 18.1 mg%, calcium

7.6 mg%, phosphorus 14.7 mg%. Urine output 10-15 ml/hour. Patient was put on ventilator because of poor respiratory drive. Patient became comatose on 5th days in ICU. Urine output was around 10-15 ml/hour. So he was put on peritoneal dialysis. His TLC increased to 45000/cumm. Patient was started on broad-spectrum antibiotics. Blood and urine cultures were sterile. CT head and CSF examination were normal.

On 7th day in ICU, patient was put on hemodialysis. Chest X-ray showed bilateral fluffy opacities suggestive of ARDS. Antibiotics, hemodialysis and ventilation were continued. He remained anuric for 10 days. Patient's urine output and renal parameters improved on 14th day of ICU. Slowly he regained consciousness and his urine output improved to 70-100 ml/ hour. Patient was extubated and hemodialysis was stopped. Chemotherapy was repeated during 4th week of ICU stay to which patient tolerated well. His parameters were Hb 11.6 gm%, TLC 5900/cumm, BUN 22.5 mg%, creatinine 0.6 mg%, sodium 133 mEq/L, potassium 4.3 mEq/L, uric acid 5.9 mg%, calcium 8.6 mg%, phosphorus 4.0 mg% and urine output of 2500 ml/day. Subsequent repeat CT Scan was consistent with complete remission and no evidence of disease.

Present case was diagnosed with Burkitt's lymphoma and developed tumour lysis following chemotherapy and developed renal failure secondary to tumour lysis syndrome as suggested by raised serum creatinine of 4 mg% along with hyperphosphatemia, hyperkalemia and hyperuricemia. Clinicians should be aware of this condition. Early management (hydration, alklization, allopurinol) could prevent the life threatening complications and improve the outcome in such patients. Special attention is necessary in patients with high tumour burden as these features predispose to tumour lysis syndrome.

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