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Case Report

A rare case of tumour lysis syndrome in multiple myeloma

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ABSTRACT

Tumour lysis syndrome (TLS) is a serious life-threatening complication of cancer chemotherapy which is a constellation of metabolic disturbances that typically occurs during therapy of bulky, rapidly proliferative tumours. TLS is an oncological emergency that causes a significant release of phosphate, potassium, uric acid, and nucleic acids into the blood stream. Incidence of TLS in multiple myeloma is rare. Here we present a case of multiple myeloma who later developed tumour lysis syndrome in its due course which was rare. A 67-year-old male was diagnosed with Multiple myeloma on the basis of M spike, osteolytic lesions and bone marrow analysis. First cycle of chemotherapy with lenalidomide, bortezomib and denosumab was initiated. During the course of follow up he presented to our casualty with complaints of chills and rigor. Based on the biochemical abnormalities a diagnosis of tumour lysis syndrome was made. He received I.V fluids, anti-hyperkalemic measures, allopurinol. He had neutropenic sepsis and went into septic shock and died after multiple resuscitative efforts.

Keywords: Mutiple myeloma, Tumour lysis syndrome, Bortezomib

INTRODUCTION

Tumour lysis syndrome (TLS), a major life-threatening complication of cancer chemotherapy, which is a confluence of metabolic abnormalities that frequently develops following treatment of large, quickly proliferating tumours. In the presence of tumours with a high proliferation index, it can also happen on its own.[1] Acute lymphoblastic leukaemia and high-grade non-Hodgkin lymphomas are the hematologic malignancies that TLS typically complicates. It is more common among hematologic malignancies due to the high rate of cell turnover and sensitivity to cytotoxic treatments.[2]

Multiple myeloma (MM) is usually a slowly proliferating tumour with a low incidence of TLS. TLS is seen to develop in only about 1% of patients receiving high dose chemotherapy for MM.[3] TLS is not common among MM as their plasma cells have low proliferation and the proportion of cells in the S phase at a particular time is low.[4,5]

We describe a case of TLS in multiple myeloma following first cycle of chemotherapy which is usually rare, with only a few cases reported.

CASE REPORT

67-year-old male presented with complaints of back pain and loss of appetite. On examination spinal and sternal tenderness was present. His initial evaluation revealed anemia (Hb – 3.2 gm/dl), thrombocytopenia (platelets–1.32 lakhs/cu.mm) and deranged renal parameters (urea – 63 mg/dl, creatinine – 3 mg/dl). High-resolution computed tomography (HRCT) thorax showed multiple osteolytic lesions in sternum, clavicle, ribs, vertebral bodies. Bone marrow aspiration revealed plasma cell dyscrasia (plasma cells – 56%) suggestive of multiple myeloma. He was diagnosed as multiple myeloma on the basis of M spike in serum protein electrophoresis, osteolytic lesions, bone marrow analysis, anemia and renal failure. Packed red blood cell transfusion was done for correction of anemia and initiated on steroids. First cycle of chemotherapy with

lenalidomide, bortezomib and denosumab was given and the patient was discharged.

After 1 week of discharge, patient presented to our casualty with complaints of chills and rigor. In view of the biochemical analysis of hyperkalemia, hyperuricemia, hyperphosphatemia and hypocalcemia, a diagnosis of tumour lysis syndrome was made. He received intravenous fluids for adequate hydration, antihyperkalemia measures, allopurinol and other measures for correction of his metabolic disturbances. In view of sudden desaturation, patient was intubated and kept on mechanical ventilation for 10 days. In due course of hospital stay, he developed neutropenic sepsis and was managed with broad spectrum antibiotics and antifungals. However, he deteriorated after developing septic shock and died after multiple resuscitative efforts.

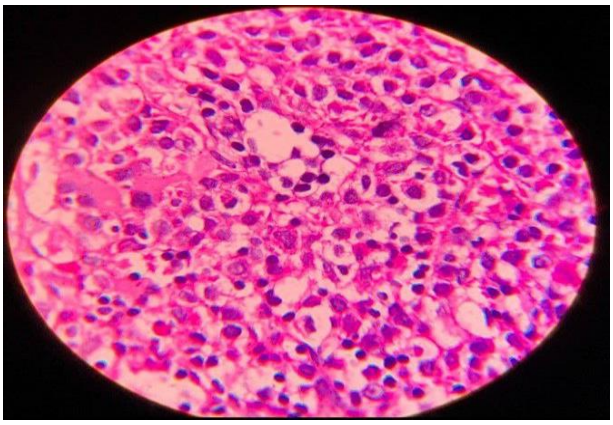

Figure 1: Bone marrow aspiration showing plasma cells.

DISCUSSION

The tumor lysis syndrome occurs as a result of rapid cellular death and release of the cellular contents into the bloodstream, either spontaneously or in response to therapy, leading to the classical laboratory findings of hyperuricemia, hyperkalemia, hyperphosphatemia, and hypocalcemia. These electrolyte and metabolic disturbances can progress to impairment of target organ functions.[6] TLS can be diagnosed on the basis of clinical or laboratory features as per the Cairo and Bishop classification.[7,8] It is a life threatening complication with high morbidity and mortality due to the metabolic manifestations as a result of tumour cell lysis.

Since potassium is the main intracellular cation, rapid tumour lysis causes hyperkalemia which is encountered as the first and most serious abnormality with potential to cause cardiac arrythmia and sudden death. Hyperphosphatemia leads to deposition of calcium phosphate complexes in renal tubules. The subsequent hypocalcemia which develops can give rise to hypotension, tetany and muscle cramps.

The clinical outcome is determined by acute kidney injury (AKI),which is the hallmark of TLS. Formation of uric acid crystals in the renal collecting system in patients with AKI was described as the likely pathophysiologic mechanism of AKI in TLS by Crittenden and Ackerman.[9]

TLS is a very rare occurence in MM and is encountered in approximately 1.4% in patients receiving bortezomib.[10,11] Identifying the risk factors by close laboratory and clinical monitoring can help to prevent the complication of TLS.

Treatment of TLS involves close monitoring of serum electrolytes, control of uric acid with allopurinol or rasburicase and hydration. Alkalinization of urine is also useful to increase the solubility of uric acid and help in its excretion. The use of sodium bicarbonate should be restricted to those patients with severe metabolic acidosis. Persistent hyperkalemia, hypocalcemia, hyperuricemia, or volume overload may require hemodialysis.[4,12]

CONCLUSION

Although rare, tumour lysis syndrome is a potentially severe complication in treatment of multiple myeloma particularly in those with high tumour burden receiving bortezomib. The rare incidence of TLS makes timely diagnosis challenging. Patients should be monitored carefully for the potential to develop TLS and supportive therapy to be instituted promptly in case of occurrence of TLS.

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