TPM Vol. 32, No. S3, 2025
ISSN: 1972-6325
https://www.tpmap.org/

Open Access

A CASE REPORT OF CHILD WITH ACUTE LYMPHOBLASTIC LEUKEMIA WITH TUMOR LYSIS SYNDROME.

DR. ASHWINI AMBALAVANAN, DR. KOUSALYA KUMAR, DR. DINESH, DR. RADHA KUMAR, DR. DEVANAND GULAB CHAUDHARY,

SAVEETHA MEDICAL COLLEGE, SAVEETHA INSTITUTE OF MEDICAL AND TECHNICAL SCIENCES

CORRESPONDING AUTHOR: DR. DEVANAND GULAB CHAUDHARY

ABSTRACT:

INTRODUCTION:

Acute Lymphoblastic leukemia (ALL) is considered the most common childhood cancer. The clinical presentation of B-ALL is varied, with symptoms ranging from fatigue, fever and easy bruising to more severe complications. The standard treatment for B-ALL includes intensive chemotherapy regimens, and the prognosis and response to therapy can vary widely based on factors such as age, genetic abnormalities, and initial response to induction therapy.

OBJECTIVE:

The aim of this case report is to contribute to the existing literature, raise awareness about the varied manifestations of B-ALL, and stimulate further discussion and research in the field of paediatric and adult hematologic malignancies

KEYWORDS: B-ALL, Tumour Lysis Syndrome, Flow Cytometry, Chemotherapy.

INTRODUCTION:

Acute Lymphoblastic leukemia is said to be the most prevalent cancer among children. The clinical presentation of B-ALL is diverse, with symptoms ranging from fatigue, fever and easy bruising to more severe complications. Lymphoid lineage precursors gave rise to the heterogeneous neoplasm known as ALL [1].

To diagnosis ALL, a combination of clinical presentation, laboratory tests, bone marrow biopsy, immunophenotypic analysis and genetic tests are employed. Currently, the prognosis and classification for appropriate treatment for ALL and its associated complications are greatly influenced by cytogenetics and molecular testing [2,3].

One such complication is Tumor lysis syndrome, a life-threatening condition resulting from the rapid disintegration of malignant cells that can happen either spontaneously or as a result of chemotherapy. It is an oncological emergency characterized by the presence of two or more of the following abnormalities of hyperkalemia, hyperuricemia, hyperphosphatemia and hypocalcemia. Tumor lysis syndrome may cause acute kidney injury, arrhythmias and seizures [4]. This case report discusses a paediatric patient diagnosed with B-ALL who developed tumour lysis syndrome spontaneously, highlighting the clinical presentation, diagnostic challenges, and management strategies.

CASE REPORT:

11 year old female child presented with complaints of giddiness and vomiting associated with generalized myalgia and bilateral lower limb pain at night since 1 week. There was no history of fever, abdominal pain, diarrhea, no history of cough, shortness of breath. There was no history of weight loss or drug ingestion. There was no history

of hematological disorders or cancer among family members. The child is first born of a nonconsanguineous marriage with a birth weight of 3kg and was not vaccinated till 10 years of age.

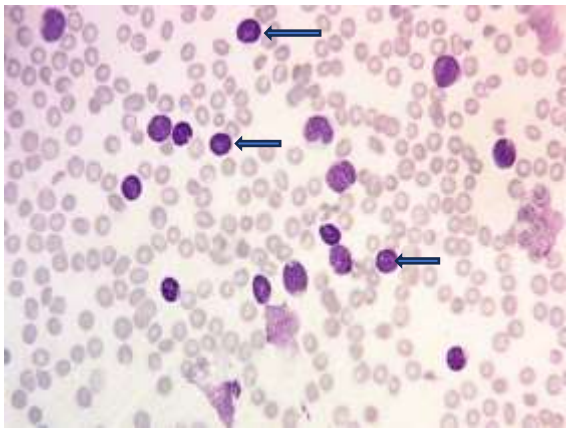
Physical examination revealed child had severe pallor, facial puffiness, bilateral cervical lymph node of 2*2cm size, 4-5 in number in the anterior and posterior triangle and bilateral axillary lymph nodes palpable of 2*1cm in size 4-5 in number, firm and nontender. Multiple ecchymotic lesions over abdomen and lower limbs of varying sizes with presence of sternal tenderness. Respiratory System examination showed normal bilateral air entry with no added sounds. Cardiovascular examination showed hyperdynamic precordium, normal S1 and S2 heart sounds and no murmurs. Abdominal examination revealed presence of hepatosplenomegaly with liver palpable 7.5 cm below right costal margin and a liver span of 14cm. Spleen was enlarged 8 cm below left costal margin, firm and nontender. A provisional diagnosis of acute leukemia was considered in view of the history and examination findings.

INVESTIGATIONS:

Laboratory investigation of the child showed Haemoglobin of 2.4gm/dl, platelet of count 12,000 / cu mm, total leucocyte counts 74,750 cells /cu mm with differential count showing 3% neutrophils and 46% lymphocytes along with 50% blasts and 1% myelocytes. Peripheral smear showed presence of 50% blasts with markedly reduced numbers of RBC's and platelets. There was no mediastinal mass or pulmonary infiltrates on Chest X-ray. Serum LDH was elevated with a value of 3702U/L. Flow cytometry was consistent with PRECURSOR B-ALL with aberrant CD117 and CD7. Cytogenetic reports revealed KMT2A (MLL) rearrangement (abnormal FISH). Biochemical parameters showed serum calcium of 6.4mg/dl, serum phosphorus of 1.9mg/dl, serum potassium of 4.2mg/dl. Renal function tests was within normal range.

During the course of stay in the hospital, the biochemical parameters of the patient were elevated – hyperuricemia(26mg/dl), hypocalcemia(6.4mg/dl) indicating TLS which occurred spontaneously due the the high tumour burden on the body of the child. The child was managed consevatively with oral Allopurinol at 100mg TDS for 3 days and with intravenous fluids(DNS) at 100ml/hr. Hematologist and cardiologist opinion was obtained. Serial blood investiagations for TLS were repeated every 6th hour and vitals along with urine output was monitored. Treatment was continued till the biochemical parameters were stabilized . Supportive measures, including transfusion of PRBC and platelet for anemia and thrombocytopenia , were initiated as needed by the patient.

PERIPHERAL SMEAR: arrow marks showing blast cells, showing reduced RBC'S.



The child was classified under HIGH RISK STRATIFICATION considering age of the child being greater than 10 years , WBC count at the time of presentation >50,000 and elevated serum LDH. Child was then started on HIGH RISK induction chemotherapy regimen for B-ALL . Supportive measures, including transfusion for anemia and thrombocytopenia , were initiated as needed by the patient . Child was started on vincristine at a dose 1.5mg/m2, at 2mg in 100ml ns on day 1 and tab.prednisolone 60mg/m2 at 40mg bd given for 7days) .

DISCUSSION:

Tumour lysis syndrome (TLS) is a potentially life-threatening complication which results from the rapid destruction of malignant cells, that can occur spontaneously or as a result of chemotherapy causing the intracellular contents to be released into the bloodstream. This cascade of events can cause metabolic disturbance which can further lead to renal failure, cardiac arrhythmias, and seizures [5,6]. Clinically, sudden development of nausea and vomiting and sudden weakness can be indicators for early identification of tumour lysis syndrome.

A system was proposed by Hande and Garrow for identifying tumour lysis syndrome (TLS) as either laboratory or clinical TLS within the first four days of chemotherapy. However, individuals with abnormal laboratory results before beginning treatment or those who obtained abnormal results after the first four days of medication were not included in this method [8].

Based on the criteria set by CAIRO and BISOP, a) laboratory TLS, is present if > 2 of the following abnormalities are present within 3 days before or up to 7 days following chemotherapy – hyperuricemia (>8mg/dl), hyperphosphatemia (>6.5mg/dl), hyperkalaemia (>6mg/dl) signifying 25% increase of the above the baseline and hypocalcaemia <7mg/dl), signifying 25% decrease below the baseline

b) clinical TLS is present if one or more of the following is present: increased serum creatinine (more than 1.5times the upper limit of normal), cardiac arrhythmia/sudden death, or seizure. Hence it is important to identify high risk cases early, start on appropriate interventions and have strict monitoring for complications [5,7].

Some of the other conditions where there is a high risk of developing tumour lysis syndrome include ALL, AML, CLL, CML, Non-Hodgkin's lymphoma, Hodgkin's lymphoma, Multiple myeloma and solid tumours. The main strategies apart from hydration and use of allopurinol in managing TLS would include the use of rasburicase and febuxostat [8,9].

The cornerstone of TLS management involves both preventive and therapeutic measures. In our patient, aggressive hydration and allopurinol were initiated pre-emptively to reduce uric acid levels and promote renal excretion of metabolites. Our case highlights the importance of a multidisciplinary approach in managing TLS, involving oncologists, nephrologists, and critical care specialists.

CONCLUSION:

In conclusion, this case report emphasizes the critical importance of recognizing and managing TLS in paediatric patients with B-ALL. Through vigilant monitoring and a comprehensive, multidisciplinary treatment approach, the potentially fatal complications of TLS can be effectively managed, thereby improving the overall prognosis for these young patients.

BIBLIOGRAPHY:

1. Mroczkowska, A., Jaźwiec, B., Urbańska-Rakus, J. et al. A case report of pediatric acute lymphoblastic leukemia with e8a2 BCR/ABL1 fusion transcript. BMC Med Genomics 15, 20 (2022). https://doi.org/10.1186/s12920-022-01169-0
2. asian S, Loh M, Hunger S. Childhood acute lymphoblastic leukemia: integrating genomics into therapy. Cancer. 2015;121(20):3577–90. https://doi.org/10.1002/cncr.29573
3. Moorman A. The clinical relevance of chromosomal and genomic abnormalities in B-cell precursor acute lymphoblastic leukaemia. Blood Rev. 2012;26(3):123–35. https://doi.org/10.1016/j.blre.2012.01.001.
4. Naeem B, Moorani KN, Anjum M, Imam U. Tumor lysis syndrome in pediatric acute lymphoblastic leukemia at tertiary care center. Pak J Med Sci. 2019 Jul-Aug;35(4):899-904. doi: 10.12669/pjms.35.4.715. PMID: 31372114; PMCID: PMC6659073
5. Cairo MS, Bishop M. Tumor lysis syndrome:new therapeutic strategies and classification. *Br J Hematol.* 2004;127(1):3–11.
6. Tazi I, Nafil H, Elhoudzi J, Mahmol L, Harif M. Management of tumor lysis syndrome. *Arab J Nephrol & Transplant.* 2011;4:147–154.
7. Rajendra A, Bansal D, Marwaha RK, Singhi SC. Tumor Lysis Syndrome. *Indian J Pediatr.* 2013;80(1):50–54. doi:10.1007/s12098-012-0824-7.
8. Hande KR, Garrow GC. Acute tumor lysis syndrome in patients with high-grade non-Hodgkin's lymphoma. *Am J Med.* 1993;94:133–139. doi: 10.1016/0002-9343(93)90174-N.
9. Linck D, Basara N, Tran V, Vucinic V, Hermann S, Hoelzer D, Fauser AA
Bone Marrow Transplant. 2003;31(10):93