ORIGINAL RESEARCH PAPER

General Medicine

A CASE OF ACUTE LYMPHOBLASTIC LEUKAEMIA WITH LEUKAEMIC RETINOPATHY

KEY WORDS: ALL, Blast cells, leukaemic retinopathy

Dr. Harsh Deo Singh* Resident Doctor *Corresponding Author

Dr. Prashant Purkar Professor and HOU MD medicine

Dr. Tejas Bhosle AP MD medicine

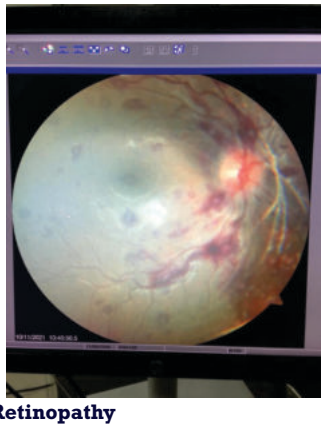
ABSTRACT

A case of 13 year female who developed fatigue and lethargy is being reported. she had generalised lymphadenopathy and had 92% blast cell on Peripheral smear. Acute Lymphoblastic leukaemia is the most common cause of childhood leukaemia (90-95%)and early diagnosis leads to better prognostic results

INTRODUCTION

Acute lymphoblastic leukaemia (ALL) is a type of cancer that affects the blood and bone marrow. ALL is characterised by an overproduction of immature white blood cells, called lymphoblasts or leukaemic blasts. Because the bone marrow is unable to make adequate numbers of red cells, normal white cells and platelets, people with ALL become more susceptible to anaemia, recurrent infections, and to bruising and bleeding easily.

The blast cells can then spill out of the bone marrow into the bloodstream and accumulate in various organs including the lymph nodes (glands), spleen, liver and central nervous system (brain and spinal cord).



Leukaemic Retinopathy

Case Report

13yrs old female was admitted in D Y Patil hospital with cbc report suggesting bicytopenia with leucocytosis and complaints of fatigue and lethargy since 21 days, she also had generalised lymphadenopathy (including cervical, pre auricular , axillary ,sub mental lymph nodes) Patient had no other comorbidities.

On examination Pulse 110 bpm ,BP 100/70mmhg , SpO2-99% on Room air, Afebrile. Patient had pallor but no icterus. On S/E abdomen was soft non tender, patient had splenomegaly with generalised lymphadenopathy Respiratory examination- breath sounds were equal with no added sounds

On CNS examination she was conscious and oriented in time place and person with no focal neurological deficit A clinical diagnosis of acute leukaemia was made and patient was posted for bone marrow examination to confirm the diagnosis

Investigations

- CBC-Hb-5.1,WBC- 150,000, platelets-42
- Sr. Uric Acid- 15.3
- Sr Calcium - 6.3
- LDH -247
- Peripheral smear - had 92% blast cells suggestive of acute leukaemia
- Usg Neck-multiple non necrotic lymph nodes seen seen(1a,1b,2,3,4,5a,5b)
- CSF study - No cells Seen

Bone Marrow

1. Aspirate - normal hematopoetic elements are replaced by blast swith 93% promyeolocye blast
2. Biopsy - Hypercellular Bone marrow with replacement of myelopoesis and erythropoiesis by sheets of blasts with vesicular nucleus with scant to moderate cytoplasm
3. Immunophenotyping - CD8, CD2, CD38, CD99, CD10, CD1a positive
4. Cytogenetics - t(12,21) seen

Treatment

- Inj. Ceftriaxone 1gm bd (prophylactically)
- Inj rasburicase 12mg stat dose iv
- T. Allopurinol 100 TDS
- T.valcyclovir 500 OD
- T.Voriconazole 200 BD
- Total prbc transfused - 2
- Total SDP transfused - 2

Chemotherapy

- BFM-90 PROTOCOL
- T. Dexa 8mg-0-4mg (mon -sat)
- T. Dexa 8mg-0-0 (Sunday)

Induction Phase 1 (33 Days)

- vincristine 1.5mg/m2 (day 8,15,22,29)
- Daunorubicine - 30mg/m2(day 8,15,22,29)
- L- asparginase 10000IU/m2 (day9,12,16,19,23,26,30,33)
- Intrathecal methotrexate 12mg (day 1, 15, 29)

Induction Phase 2 (28 Days)

- Cyclophosphamide 1000mg/m2 (day 36,64)
- Ara - C 75mg/m2 (day 38 - 62)
- 6 -MP 60mg/m2 (day 36 - 62)

2Weeks Rest After Phase 2

Consolidation (45 Days)

- High dose methotrexate 4 doses
- 5mg/m2 over 24 hours (day 1,15,30,45)

Leucoverine rescue total 6 doses 6 hours apart according to blood methotrexate level at 24 and 48 hours

2 Weeks Rest After High Dose Therapy

Maintenance (2 Years)

6MP + methotrexate once weekly oral Intrathecal methotrexate every 3 months

CONCLUSION

Several recent clinical trials in B- and T-lineage ALL have compared dexamethasone vs prednisone during the induction phase of treatment. The rationale for using dexamethasone includes greater potency and CNS penetration, which is appealing in T-ALL, given the higher rates of CNS disease. This has been counterbalanced, however, by higher rates of infectious toxicity with dexamethasone-based treatment regimens.Several recent trials have supported the use of dexamethasone in T-ALL.Most TALL disease recurrences occur within 2 years of diagnosis, and relapsed disease remains very difficult to salvage, with survival rates lower than 25%.At this time, hematopoietic cell transplantation is the only curative treatment, but successful remission reinduction is a prerequisite, and this has remained another significant challenge.

With recent advances in treatment, including refinement in corticosteroid, asparaginase, and CNS-directed therapy, outcomes for T-ALL have improved significantly and now approach those observed in B-lineage disease. The therapy needed to achieve cure, however, is intensive, with risks for acute and late toxicities, and there has been limited success in treating relapsed disease. In recent years, there have been remarkable discoveries in underlying T-ALL biology, which are envisioned to lead to further advances in therapy in the near future.

REFERENCES

1. M철ricke A, Zimmermann M, Valsecchi MG, et al.. Dexamethasone vs prednisone in induction treatment of pediatric ALL: results of the randomized trial AIEOP-BFM ALL 2000
2. Patrick K, Wade R, Goulden N, et al.. Improved outcome for children and young people with T-acute lymphoblastic leukaemia: Results of the UKALL 2003 Trial. *Blood*. 2014;124(21):3703
3. Place AE, Stevenson KE, Harris MH, et al. Outcome of childhood Tcell acute lymphoblastic leukemia (T-ALL)