*Case Series*

Challenges in Managing Adult Acute Lymphoblastic Leukaemia: A Case Series from a Resource-Constrained Environment

# ABSTRACT

# Background: Acute lymphoblastic leukaemia (ALL) is a clonal haematological malignancy characterized by the rapid proliferation of lymphoblasts which mostly affects children however, it also occurs in adults. The pathogenesis of ALL is multifactorial involving genetic and environmental components. The clinical presentation includes anaemia, fever, bleeding and organomegaly. Diagnosis requires ≥20% lymphoblasts in the peripheral blood and / or bone marrow. Immunophenotyping, cytogenetics and molecular assays are important to determine specific mutations involved. Definitive treatments include chemotherapy, targeted therapy, haematopoietic stem cell transplantation and chimeric antigen receptor T-cell therapy. This study aimed to report the few cases of adult ALL encountered in our practice over a ten-year probationary period.

# Methods: This was a retrospective observational study. The case files of all patients aged ≥18 years of age diagnosed with ALL, either by peripheral blood or bone marrow findings, within the period of January 2014 to December 2024. Overall survival was calculated using the Kaplan-Meier curve.

# Case Presentation: The median age was 30 years, with a male to female ratio of 8.3:1. Lymphadenopathy and splenomegaly were present in 66.7% and 50% cases respectively. The mean haemoglobin concentration (HB) was 6.9 (±2.4) g/dL, median WBC 27.8 X 109/L (range 1.24 – 153.8 X 109/L), mean platelet 51.0 (±33.8) X109/L. Average peripheral and bone marrow blast counts were 59.2% and 74.8% respectively. FAB subtype L3 was the commonest subtype. Immunophenotyping was done for only 2 patients who were the only patients who received chemotherapy. The median duration of treatment was 10 days (range 1 – 186 days) with a median survival of 6.9 days.

# Conclusion: This was a retrospective case series which showcased the multifaceted challenges in the diagnosis and management of adult ALL within resource-limited settings in the areas of clinical presentations, laboratory investigations, treatment options and outcomes.

# Keywords: Acute lymphoblastic leukaemia, Bone marrow aspiration, haematology, CAR-T cell therapy, platelet-rich plasma

# INTRODUCTION

Acute lymphoblastic leukaemia (ALL) is the most common disease in pediatric oncology. The history of developmental therapeutics for ALL began in the 1960s with the repetition of “unreliable” medical interventions against this lethal disease.[[1]](#endnote-1) It is a clonal haematological malignancy characterized by the rapid proliferation of immature lymphoid cells in the bone marrow, peripheral blood, or extramedullary sites. It is a rare malignant disorder arising from an abnormal proliferation of hematopoietic precursor cells.[[2]](#endnote-2) It mostly affects children however, it also occurs in adults. ALL is the second most common acute leukaemia in adults.[[3]](#endnote-3) In adults, ALL may have distinct biological behaviour compared to paediatric cases; the prognosis and response to therapy may also differ in adults. In the United States, approximately 6,000 new cases are seen per year, with about 60% of the diagnosed in patients <20 years old.[[4]](#endnote-4) [[5]](#endnote-5) [[6]](#endnote-6) Although the exact incidence of adult ALL in our environment has not been recorded, the peak age of occurrence of ALL in adults was recorded in the 20 – 40 years age group.[[7]](#endnote-7)

The pathogenesis of adult ALL includes a combination of genetic predisposition and mutations, environmental exposures, and dysregulation of the immune system. Over 90% of adult ALL cases have at least one genetic mutation present.[[8]](#endnote-8) The BCR-ABL mutation is present in about 25-50% of cases of adult ALL and initially conferred a poor prognosis; however, with the advent of tyrosine kinase inhibitors, this has significantly improved the outcome in BCR-ABL adult ALL. Other mutations in adult ALL include Ph-like mutations, TCF3::PBX1 rearrangements, TAL and LMO rearrangements, TP53 and NOTCH1 gene mutations. These mutations make adult ALL a heterogeneous and complex disease.[[9]](#endnote-9),[[10]](#endnote-10) The clinical presentation is usually acute, although some patients have symptoms that evolve over months. Symptoms include features of anaemia such as easy fatiguability, weakness and loss of appetite, recurrent fever, bleeding episodes and organomegaly such as lymphadenopathy or hepatosplenomegaly.

Diagnosis relies on the demonstration of blasts in the peripheral blood and or bone marrow via morphological and immunophenotypic methods. The French-American-British classification classifies ALL on a morphological basis into 3 subtypes,[[11]](#endnote-11) however, the more recent World Health Organisation classification is based on cytogenetic and molecular mutations.[[12]](#endnote-12) These recent advances have led to improved risk stratification of patients. Treatment of ALL is complex and in phases using multi-agent chemotherapeutic agents. In recent times, the use of immunotherapy and targeted therapy has improved outcomes in adult ALL. Treatment of ALL in young adults is largely adapted from paediatric protocols.[[13]](#endnote-13) Tyrosine kinase inhibitors (TKIs) used in the treatment of BCR-ABL-positive ALL have revolutionised treatment, leading to better remission rates and overall survival.[[14]](#endnote-14) Haematopoietic stem cell transplantation and chimeric antigen receptor (CAR) T-cell therapy for select patients offer a curative option for patients with adult ALL.[[15]](#endnote-15) [[16]](#endnote-16) Despite these, the treatment of adult ALL remains challenging, with generally shorter survival rates and poor outcomes, unlike paediatric ALL.6 The aim of this case series is to report the few cases of adult ALL managed in our facility located in a resource-constrained environment.

# METHODS:

This was a retrospective observational study. The case files of all patients aged ≥18 years of age diagnosed with ALL, either by peripheral blood or bone marrow findings, within the period of January 2014 to December 2024. Details including biodata, presenting complaints, clinical findings, laboratory and other investigations, treatment and outcome were extracted from the folders to summarise each case. Thereafter, the extracted data was input into an Excel spreadsheet and formatted into Tables. Frequencies, means, and medians were calculated for the cohort. Overall survival was calculated using the Kaplan-Meier curve. Data obtained from patients were treated with utmost confidentiality in accordance with the ethical requirements of our hospital.

# CASE PRESENTATIONS:

## CASE 1

A 60-year-old male, working in the petroleum industry, presented to the haematology clinic with a 6-month history of abdominal swelling, progressive weight loss, weakness and anorexia. He gave a history of receiving multiple blood transfusions, a total of 3 units prior to admission. On examination, he was found to be moderately pale, anicteric, afebrile, with nil pedal oedema, with generalised bulky lymphadenopathy involving the submandibular, cervical, axillary and inguinal nodes (all bilaterally). His pulse rate was 108 beats per minute (BPM), and blood pressure was 138/80 mmHg. Examination of the abdomen revealed splenomegaly of 16cm below the costal margin.

A preliminary diagnosis of lymphoma was made. Significant investigation results were full blood count (FBC), which showed pancytopenia; haemoglobin concentration (Hb) 8.2g/dL, white blood cell count (WBC) 1.24 X109/L, platelets 80 X109/L. The peripheral blood film revealed a hypochromic, microcytic anaemia with leucopenia and reduced platelets on film. Uric acid was 512umol/L. He was admitted to the ward, and a bone marrow aspiration (BMA) was performed. The BMA findings were that of a hypocellular marrow with reduced erythropoiesis, myelopoiesis and few megakaryocytes, while there was predominance of moderate to large-sized granular blasts with high nuclear cytoplasmic ratio and deeply basophilic cytoplasm containing multiple vacuoles. The impression of the BMA was ALL, French African British (FAB)- L3 phenotype. Further tests done included immunophenotyping: CD20+, Pax5+, CD34+, TdT+, CD3-, MPO-, CD117-, ckit. The echocardiogram done for him was normal. On the 19th day of admission, he commenced chemotherapy using Cyclophosphamide, weekly Vincristine and Prednisolone for a duration of one cycle of 28 days. While on admission, he received 3 more units of fresh whole blood (FWB) and 8 platelet-rich plasma (PRP). He received on triplet antimicrobial prophylaxis of Ciprofloxacin, Fluconazole and Acyclovir. He was also placed on Allopurinol 300mg daily. While on admission, thrombocytopenia continued to worsen despite platelet and whole blood transfusions. On the 64th day, he succumbed to the disease.

## CASE 2

A 28-year-old male who presented to the accident and emergency (A&E) with a 5-day history of cough, fever and haematuria, with generalized body weakness associated and excessive sweating of one month duration. He had received 3 units of blood at a peripheral centre prior to presenting

at the A & E. Significant findings on general examination were pallor and fever (temperature 39.8 °C). There was no peripheral lymphadenopathy. The abdominal examination showed the spleen was enlarged 12 cm below the costal margin.

He was admitted and investigations done revealed: FBC (Hb - 9g/dL, WBC- 26 X10^9/L, Platelets- 63). Peripheral blood smear showed the presence of large agranular blasts with cytoplasmic vacuoles, blast count 64%. Urinalysis confirmed haematuria. Uric acid- 611umol/L. He was transfused with a unit of FWB and commenced on antimicrobials (IV Ceftriaxone, oral fluconazole and acyclovir. On the 3rd day of admission, BMA was done, which revealed a hypercellular marrow with lymphoid hyperplasia associated with maturation arrest of the lymphoid cells. Blasts were large-sized cells with a high nuclear cytoplasmic ratio and vacuolated basophilic cytoplasm; marrow blast count was 97%. Immunophenotyping was not done due to financial constraints.

He received a total of 4 units of FWB and 3 units of PRP while on admission as cytopenias worsened. Attempts to improve cytopenias before commencing chemotherapy failed, and he later died on the 13th day of admission after complaints of a severe headache.

## CASE 3

A 25-year-old female presented to the A&E with a menorrhagia of over two weeks duration, requiring her to be transfused with three units of blood prior to presentation at our centre. She was first reviewed at the A&E by the Gynaecology Team, who had made an initial impression of menorrhagia secondary to uterine leiomyoma for which they requested some preliminary investigations, including abdomino-pelvic ultrasound scan and bedside PCV. The ultrasound scan did not show the presence of leiomyoma, and the bedside PCV was 10%. For this reason, the Haematology Team was called to review her, where they immediately ordered a full blood count, which revealed Hb- 2.6g/dL, WBC – 61.1 X 109/L, Platelet- 15 X 109/L. This warranted a blood film, which showed the presence of blasts in 90%. The patient was immediately booked for a bone marrow aspiration and biopsy; however, she succumbed to the disease within an hour of review. However, from the peripheral blood film, a diagnosis of ALL, FAB type L1 was made.

## CASE 4

A 32-year-old male, working in an oil refinery, presented with weakness, breathlessness and swelling in the neck and groin of 2 months duration. He gave a history of being treated for malaria severally times and receiving a total of eight units of red cell transfusions in a different facility prior to presentation. On the day of presentation, he had an episode of haematuria, which warranted his coming to the hospital. He was found to be markedly pale and febrile on examination, with generalised lymphadenopathy and splenomegaly of 10cm below the costal margin. His FBC showed Hb- 7.6g/dL, WBC- 153 X 109/L, Platelets 29 X 109/L and a peripheral blood film blast count of 95%. The blasts were moderate to large-sized cells with a high nuclear-cytoplasmic ratio and multiple prominent nucleoli. He was rehydrated, placed on intravenous antibiotics and given a unit of fresh whole blood. The next day, a bone marrow aspiration was performed, and he received another FWB. The bone marrow findings were that of a markedly hypercellular marrow with maturation arrest of the lymphoid cell line and a blast count of 95% with associated reduced erythropoiesis, granulopoiesis and megakaryopoiesis. He and his relatives were counselled on the disease, and the plan was to commence chemotherapy the next day. Unfortunately, he died early hours of the next morning.

## CASE 5

A 73-year-old retiree who presented with chronic anaemia of about 6 months duration, for which he had received a total of four units of red cell transfusions at two monthly intervals prior to presentation. His symptoms at first review were weakness, dizziness and persistent headaches. On examination, he was found to be markedly pale, anicteric, afebrile, with no pedal oedema (however, he was wearing a compression sock on the right leg as his past medical history revealed that he had been managed for a deep vein thrombosis some months before). He had no palpable abdominal organomegaly, nor were his peripheral lymph nodes enlarged. Urgent PCV done showed 15%, based on this, he was admitted, and further investigations were ordered. His FBC revealed Hb- 5.9g/dL, WBC- 5.8, the differential count showed monocytosis of 26.1%, platelets – 97 X 109/L. The significant finding on the peripheral blood film was monocytosis (see figure). He had a bone marrow aspiration done the next day, which showed a hypoplastic marrow with the presence of about 20% lymphoblasts with cytoplasmic vacuoles. A diagnosis of ALL-FAB subtype L3 was made. Further tests done included immunophenotyping; CD19+, CD10, HLA-DR++, CD38+/++, CD66c+, TdT+. He received a total of 16 units of red cells and 12 units of PRP while at our facility, however, he and his family later requested to be referred to a centre outside of the country for further care where a medical update letter sent to the haematology team stated that he had been placed on Blinatumomab therapy. Unfortunately, he succumbed to the disease after receiving 2 cycles of Blinatumomab.

## CASE 6

A 23-year-old graduate who presented to the haematology clinic with complaints of easy fatiguability and bleeding gums on brushing his teeth, which had started about 3 weeks prior to presentation. History revealed that he had been transfused with a total of five units of red cells at a peripheral centre before presenting in the clinic. On examination, he was found to be moderately pale, with petechiae and purpura on both upper and lower limbs and the anterior abdominal wall. FBC done showed Hb- 8.3g/dL, WBC- 29.5 X109/L, platelets 22 X109/L. Peripheral blood film showed the presence of lymphoblasts of heterogeneous sizes with prominent nucleoli, making a working diagnosis of ALL- FAB phenotype L2. He received two units of FWB in the subsequent days; however, due to financial constraints, he was not able to afford the bone marrow aspiration/ biopsy procedure, so that was not done. Several other investigations were also not done due to financial constraints. Seven days after he was admitted, he developed a severe headache and died a few hours later. Table 1 gives the clinical features, important laboratory parameters and management of the cases.

TABLE : Summary of clinical features, laboratory parameters and treatment for all six cases with adult acute lymphoblastic leukaemia

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| Parameter (Reference Range) | Case 1 | Case 2 | Case 3 | Case 4 | Case 5 | Case 6 | MEAN (MN) / MEDIAN (MD) |
| Age (Years) | 60 | 28 | 25 | 32 | 73 | 23 | 30.0 (MD) |
| Sex | M | M | F | M | M | M |  |
| Lymphadenopathy | Yes | No | Yes | Yes | No | Yes |  |
| Spleen Size (cm) | 16 | 12 | Not  palpable | 10 | Not palpable | Not  palpable | 12.7 (MN) |
| HB (>10g/dL) | 8.2 | 9 | 2.6 | 7.6 | 5.9 | 8.3 | 6.9 (MN) |
| WBC (3.0 – 11.0 X109/) | 1.24 | 26 | 61.1 | 153.8 | 5.8 | 29.5 | 27.8 (MD) |
| Platelet(100 – 400 X 109/L) | 80 | 63 | 15 | 29 | 97 | 22 | 51.0 (MN) |
| PBF Blasts % | 0 | 64 | 90 | 95 | 26 | 80 | 59.2 (MN) |
| BM BLAST % | 85 | 97 | Not  done | 95 | 22 | Not  done | 74.8 (MN) |
| ALL (FAB Subtype) | ALL-L3 | ALL-L3 | ALL-L1 | ALL-L2 | ALL-L3 | ALL-L2 |  |
| IMPT/ IHC | CD20+,Pax5+  ,CD34+,TdT+, CD3-,MPO-  ,CD117-,ckit | Not done | Not done | Not done | CD19+, CD10, HLA- DR++, CD38+/++, CD66c+, TdT | Not done |  |
| Number of Blood transfusions before  Presentation | 3 | 3 | 3 | 8 | 4 | 5 | 4.3 (MN) |
| No of Blood Transfusion during  management | 3 | 4 | 1 | 3 | 16 | 2 | 3.0 (MD) |
| Platelet  transfusions | 8 | 3 | 0 | 0 | 12 | 0 | 3.8 (MN) |
| Chemo received? | Yes | No | No | No | No\*\* | No |  |
| Chemotherapy Regimen | Vincristine, prednisolone | None | None | None | Blinatumomab  (REFERRED) | None |  |
| Number of Cycles | 1 | 0 | 0 | 0 | 1 | 0 |  |
| Total Duration of  RX / Survival (Days) | 64 | 13 | 1 | 3 | 186 | 7 | 10 (MD) |

M- male, F- female, Rx- treatment, HB- haemoglobin concentration, WbC- white blood cells, IMPT- immunophenotyping, IHC- immunohistochemistry, MD- Median, MN- Mean, BM- bone marrow, ALL- acute lymphoblastic leukaemia



*FIGURE 1: Peripheral blood film and bone marrow micrographs from the six cases.*

*Note- Cases 3 and 6 succumbed to the disease before BMA could be done*

*CASE 1: 60-year-old (M), pancytopenia, no circulating blasts on PBF; BMA with 85% L3 blasts (IMPT done)*

*CASE 2: 28-year-old (M), 64% and 97% L3 type blasts on PBF and BMA.*

*CASE 3: 25-year-old (F), morphological diagnosis of L1; BMA not done due to early mortality*

*CASE 4: 32-year-old (M), morphological diagnosis of L2 from both PBF and BMA*

*Case 5: 73-year-old (M), had frequent monocytes on PBF, BMA was hypocellular with presence of L3 blasts* *(IMPT done)*

*Case 6: 23-year-old (M), blast count of 80% on PBF; BMA not done due to patient’s early demise*

Adult ALL affected younger adults (23 - 32 years) and older adults (60 -73) years, the median age for all cases was 30 years. Of the 6 cases, 5 were males with one female, giving a male-to-female ratio of 8.3:1. Lymphadenopathy was present in 4 of the 6 cases (66.7%). Half of the patients had splenomegaly of with a mean size of the spleen was 12.7cm below the costal margin. The mean haemoglobin concentration (HB) was 6.9 (±2.4) g/dL, median WBC 27.8 X 109/L (range 1.24 – 153.8 X 109/L), mean platelet 51.0 (±33.8) X109/L. All 6 cases presented with anaemia and thrombocytopenia. With regards to the WBC, one patient had leucopenia, four had leukocytosis, while one had a normal total WBC. Five of the six cases (83.3%) had the presence of peripheral blood film (PBF) blasts, the mean blast count was 59.2 (±38.2)%. Bone marrow aspirate was performed for four of the six cases, of which there was an average BM blast count of 74.8 (35.6)%.

All 6 patients received blood transfusions at peripheral centres prior to presentation at our centre, with a mean transfusion of about 4.3 (±1.9) units of blood. Median number of blood transfusions while on admission in our facility was 3 units (range 1 – 16). Three patients received platelet transfusions. Of the six cases, three (50%) had FAB subtype L3, two (33.3%) had L2 and one (16.7%) patient had L1 phenotype. Immunophenotyping was done for only 2 patients, these 2 patients had FAB type L3.

With regards to chemotherapy, one patient received treatment with cyclophosphamide, vincristine and prednisolone, while one was referred and received Blinatumomab, and the other patients died prior to therapy. The median duration of treatment was 10 days (range 1 – 186 days) with a median survival of 6.9 days (see figure 2).

A screen shot of a graph

AI-generated content may be incorrect.

*FIGURE 2: Survival in Adult ALL patients*

## DISCUSSION

## The demographics showed that there were 2 peak ages of occurrence. ALL tended to occur in young adults, mostly in their twenties and older adults well above their fifties, which is in keeping with epidemiological data from our locality and other climes.2,4,6–8 The male predominance in our cases aligns with global and local trends indicating a higher incidence of ALL among males,[[17]](#endnote-17) although the male to female ratio in this study is much higher than commonly reported figures; this skew may be due to the small number of cases we had. However, it has been reported that male predominance may increase with increasing age in ALL.[[18]](#endnote-18)

## It is well established that ALL occurs more frequently in children, therefore, it was not surprising that within the 10-year period in review, only 6 cases were reported. Although the number of patients was few, the sex distribution showed males were markedly more affected than females, this may be reflective of the fact that more males are likely to have acute lymphoblastic leukaemia compared with females.2,4,6,7,14

## All the cases had cytopenias at the time of presentation, with varying degrees of anaemia and thrombocytopenia seen in all the cases; this is consistent with the bone marrow failure caused by the infiltration of malignant lymphoblasts. Organs such as the lymph nodes and spleen could also be infiltrated and are a common clinical feature of ALL.[[19]](#endnote-19) In this series, lymphadenopathy and splenomegaly were prominent clinical features. Lymphadenopathy is a common manifestation of ALL. The mean splenic enlargement of 12.7cm below the costal margin suggests significant disease burden at presentation, potentially due to delayed diagnosis and referral. The uniform presence of anaemia and thrombocytopenia across all patients further indicates advanced disease stages upon initial evaluation. No patient manifested with central nervous system (CNS) disease.

## Although more patients presented with leukocytosis on the FBC, one patient presented with pancytopenia, while another had a normal WBC count (despite having a normal WBC, the patient had circulating blasts in the PBF). The patient with leucopenia did not show any blasts on PBF. All patients who had BMA done had high blast counts. These findings indicate the importance of a peripheral blood film as blasts may circulate in the blood despite a normal total white cell count, ; it also buttresses the fact that malignant blood cells could be present in the marrow without spilling into the blood, therefore emphasizing the need for both PBF and BMA in the diagnostic workup and follow-up of ALL cases, irrespective of WBC count .7, [[20]](#endnote-20) Incidentally, all patients with FAB L3 had lower white cell counts in comparison to the other FAB subtypes.

## Good blood support is an integral part of the management of the leukaemias. It has been estimated that one patient with leukaemia may need up to 100 units of blood in total supportive care for such cases.[[21]](#endnote-21) Our patients presented to the haematology department after receiving a mean of 4.3 units of blood transfusions at various peripheral centres. For most of them, they were transfused on account of low Hb, and it is likely that they would have been transfused with sedimented cells from stored blood. This may dilute the available platelets,[[22]](#endnote-22) causing further reduction and delaying diagnosis of the disease. A FBC (if done) before transfusion in these peripheral facilities would have shown the need for a quick referral to haematologists; there is therefore for training and re-training of healthcare providers about the importance of FBC check as against bedside packed cell volume, especially in patients who present with symptoms of haematological disease.

## The median and mean number of blood and platelets transfused after diagnosis was made in our patients were 3.0 and 3.8 units, respectively; this is far from optimum. Of these transfusions, the bulk were received by the two patients who could afford immunophenotyping to confirm the diagnosis of ALL, and these two patients were also the ones who had longer duration of treatment days (64 days and 186 days), they were also the two patients who were exposed to chemotherapy before their demise. These findings suggest that patients who have a better financial situation are more likely to have a longer duration of treatment and survival. Blood component therapy is a major management modality for ALL patients for clinical stabilization before chemotherapy, but even with blood support majority of the patients died before chemotherapy could be commenced.

## A significant limitation in this cohort was the underutilization of immunophenotyping, performed in only two patients. Immunophenotyping is crucial for accurate classification of ALL subtypes, guiding treatment decisions, and prognostication.7 The reliance on morphological classification alone, particularly the FAB system, may lead to misclassification and suboptimal treatment strategies. The predominance of the FAB L3 subtype in half of our patients is atypical for adult ALL and may reflect diagnostic inaccuracies due to limited access to advanced diagnostic tools.[[23]](#endnote-23)

## Treatment was received by one patient in our centre who had conventional chemotherapy, while the other patient, who received treatment, had immunotherapy with the bi-specific T-cell engager (Blinatumomab) because he was referred to a facility abroad where he had access to the drug. Although both patients still succumbed to their disease, they were the two patients with the longer survival. The other patients died before chemotherapy could be instituted.

## Our patients paid out of pocket for their care, posing a financial burden and limiting the type of investigations, supportive care and specific treatment they received;[[24]](#endnote-24) this may have impacted on the outcome of our patients. The two patients who had a longer survival were the ones who were able to afford not just platelet transfusions but also Immunophenotyping, and they were the two cases who went ahead to receive treatment. Because the patients all had thrombocytopenia, they would have all benefited from PRP, and this may have impacted on outcome as platelet transfusions play an important role in the supportive management of patients with ALL. Diagnosis by morphology alone has its limitations, but due to the cost of Immunophenotyping, most of the patients could not afford it. BCR-ABL mutations are common in adult ALL, found in up to a third of patients, and treatment with TKIs significantly improves outcome,[[25]](#endnote-25) yet this was not done due to a combination of financial constraints and unavailability in our locality. Cytogenetic analysis and molecular studies are also important in investigating ALL due to the presence of mutations, however, this could not be done for any of our patients due to the unavailability in our locality.

## Prognosis in ALL is poor in adults, especially in males.9 Our patients had a poor survival rate with an abysmal median survival of only 6.9 days. Most of them did not survive long enough to commence chemotherapy. The two patients who received chemotherapy still succumbed to the disease.

## CONCLUSION

## This was a retrospective case series which showcases the multifaceted challenges in the diagnosis and management of adult ALL within resource-limited settings in the areas of clinical presentations, laboratory investigations, treatment options and outcomes. Morphology of the peripheral blood and bone marrow was our main mode of diagnosis. Immunophenotyping was done in only those who could afford it. Only a few patients received chemotherapy, as the majority died prior to treatment. Our patients had a poor outcome with an abysmal median survival.

## DECLARATION

## Due to retrospective nature and anonymization of patient data, this manuscript fulfills all the guidelines of the IEC of our institutions.

Disclaimer (Artificial intelligence)

Option 1:

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

## REFERENCES

1. Hayashi, H., Makimoto, A., & Yuza, Y. (2024). Treatment of pediatric acute lymphoblastic leukemia: a historical perspective. Cancers, 16(4), 723. [↑](#endnote-ref-1)
2. 24. Mwizabi, S. A., Musuka, B., Mwape, C., Mulenga, A., Mweene, M., Maliko, C., ... & Nyirenda, C. (2023). Acute Leukemia in A Young Female Adult Presenting for Care in A Resource Constrained Setting. Clinical Medicine And Health Research Journal, 3(6), 709-712. [↑](#endnote-ref-2)
3. Radhakrishnan, V. S., Agrawal, N., Bagal, B., & Patel, I. (2021). Systematic review of the burden and treatment patterns of adult and adolescent acute lymphoblastic leukemia in India: comprehending the challenges in an emerging economy. Clinical Lymphoma Myeloma and Leukemia, 21(1), e85-e98. [↑](#endnote-ref-3)
4. Jabbour E, O'Brien S, Konopleva M, Kantarjian H. New insights into the pathophysiology and therapy of adult acute lymphoblastic leukemia. Cancer. 2015 Aug 1;121(15):2517-28. doi: 10.1002/cncr.29383. Epub 2015 Apr 17. PMID: 25891003; PMCID: PMC11726371 [↑](#endnote-ref-4)
5. Peters C, Locatelli F, Bader P. Acute Lymphoblastic Leukaemia in Children and Adolescents. 2024 Apr 11. In: Sureda A, Corbacioglu S, Greco R, et al., editors. The EBMT Handbook: Hematopoietic Cell Transplantation and Cellular Therapies [Internet]. 8th edition. Cham (CH): Springer; 2024. Chapter 73. Available from: https://www.ncbi.nlm.nih.gov/books/NBK608333/ doi: 10.1007/978-3-031-44080-9\_73 [↑](#endnote-ref-5)
6. Liu YF, Wang BY, Zhang WN, Huang JY, Li BS, Zhang M, Jiang L, Li JF, Wang MJ, Dai YJ, Zhang ZG, Wang Q, Kong J, Chen B, Zhu YM, Weng XQ, Shen ZX, Li JM, Wang J, Yan XJ, Li Y, Liang YM, Liu L, Chen XQ, Zhang WG, Yan JS, Hu JD, Shen SH, Chen J, Gu LJ, Pei D, Li Y, Wu G, Zhou X, Ren RB, Cheng C, Yang JJ, Wang KK, Wang SY, Zhang J, Mi JQ, Pui CH, Tang JY, Chen Z, Chen SJ. Genomic Profiling of Adult and Pediatric B-cell Acute Lymphoblastic Leukemia. EBioMedicine. 2016 Jun;8:173-183. doi: 10.1016/j.ebiom.2016.04.038. Epub 2016 May 13. PMID: 27428428; PMCID: PMC4919728 [↑](#endnote-ref-6)
7. Damulak Obadiah Dapus\*, Egesie OJ, Jatau ED, Ogbenna AA and Adediran AA. The Pattern of Leukaemias among Adults in Jos, North Central Nigeria. World J Blood. 2017; 1(1): 1001 [↑](#endnote-ref-7)
8. Sun X, Liu X, Li Y, Shi X, Li Y, Tan R, Jiang Y, Sui X, Ge X, Xu H, Wang X, Fang X. Characteristics of Molecular Genetic Mutations and Their Correlation with Prognosis in Adolescent and Adult Patients with Acute Lymphoblastic Leukemia. Oncology. 2024;102(1):85-98. doi: 10.1159/000531522. Epub 2023 Jul 12. PMID: 37437551 [↑](#endnote-ref-8)
9. Roberts KG. Genetics and prognosis of ALL in children vs adults. Hematology Am Soc Hematol Educ Program. 2018 Nov 30;2018(1):137-145. doi: 10.1182/asheducation-2018.1.137. PMID: 30504302; PMCID: PMC6245970 [↑](#endnote-ref-9)
10. Gökbuget N, Boissel N, Chiaretti S, Dombret H, Doubek M, Fielding A, Foà R, Giebel S, Hoelzer D, Hunault M, Marks DI, Martinelli G, Ottmann O, Rijneveld A, Rousselot P, Ribera J, Bassan R. Diagnosis, prognostic factors, and assessment of ALL in adults: 2024 ELN recommendations from a European expert panel. Blood. 2024 May 9;143(19):1891-1902. doi: 10.1182/blood.2023020794. PMID: 38295337. [↑](#endnote-ref-10)
11. Marinescu C, Vlădăreanu AM, Mihai F. Acute Lymphocytic Leukemia in Adults. Pathologic Features and Prognosis. Rom J Intern Med. 2015 Jan-Mar;53(1):31-6. doi: 10.1515/rjim-2015-0004. PMID: 26076558. [↑](#endnote-ref-11)
12. Choi JK, Xiao W, Chen X, Loghavi S, Elenitoba-Johnson KS, Naresh KN, Medeiros LJ, Czader M; WHO 5th Edition Classification Project. Fifth Edition of the World Health Organization Classification of Tumors of the Hematopoietic and Lymphoid Tissues: Acute Lymphoblastic Leukemias, Mixed-Phenotype Acute Leukemias, Myeloid/Lymphoid Neoplasms With Eosinophilia, Dendritic/Histiocytic Neoplasms, and Genetic Tumor Syndromes. Mod Pathol. 2024 May;37(5):100466. doi: 10.1016/j.modpat.2024.100466. Epub 2024 Mar 7. PMID: 38460674. [↑](#endnote-ref-12)
13. Carobolante F, Chiaretti S, Skert C, Bassan R. Practical guidance for the management of acute lymphoblastic leukemia in the adolescent and young adult population. Ther Adv Hematol. 2020 Feb 3;11:2040620720903531. doi: 10.1177/2040620720903531. PMID: 32071710; PMCID: PMC6997963. [↑](#endnote-ref-13)
14. Byun JM, Koh Y, Shin DY, Kim I, Yoon SS, Lee JO, Bang SM, Kim KH, Jung SH, Lee WS, Park Y, Jang JH, Han JJ, Yhim HY, Kim DS, Lee YJ, Lee H, Choi YS, Lee S; Korean Adult ALL Working Party, Korean Society of Hematology. BCR-ABL translocation as a favorable prognostic factor in elderly patients with acute lymphoblastic leukemia in the era of potent tyrosine kinase inhibitors. Haematologica. 2017 May;102(5):e187-e190. doi: 10.3324/haematol.2016.159988. Epub 2017 Jan 12. PMID: 28082339; PMCID: PMC5477621. [↑](#endnote-ref-14)
15. Gökbuget N, Boissel N, Chiaretti S, Dombret H, Doubek M, Fielding A, Foà R, Giebel S, Hoelzer D, Hunault M, Marks DI, Martinelli G, Ottmann O, Rijneveld A, Rousselot P, Ribera J, Bassan R. Management of ALL in adults: 2024 ELN recommendations from a European expert panel. Blood. 2024 May 9;143(19):1903-1930. doi: 10.1182/blood.2023023568. PMID: 38306595. [↑](#endnote-ref-15)
16. Agrawal V, Murphy L, Pourhassan H, Pullarkat V, Aldoss I. Optimizing CAR-T cell therapy in adults with B-cell acute lymphoblastic leukemia. Eur J Haematol. 2024 Feb;112(2):236-247. doi: 10.1111/ejh.14109. Epub 2023 Sep 29. PMID: 37772976. [↑](#endnote-ref-16)
17. Dachi RA, Mustapha FG, Mahdi M, Abbas H. Acute Leukaemias in Bauchi State, Northeastern Nigeria: Pattern of Presentations and Clinical Entities. West Afr J Med. 2022 May 27;39(5):497-500. PMID: 35633629. [↑](#endnote-ref-17)
18. Jaime-Pérez JC, Hernández-De Los Santos JA, Fernández LT, Padilla-Medina JR, Gómez-Almaguer D. Sexual Dimorphism in Children and Adolescents With Acute Lymphoblastic Leukemia: Influence on Incidence and Survival. J Pediatr Hematol Oncol. 2020 Jul;42(5):e293-e298. doi: 10.1097/MPH.0000000000001665. PMID: 31725540. [↑](#endnote-ref-18)
19. Puckett Y, Chan O. Acute Lymphocytic Leukemia. [Updated 2023 Aug 26]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK459149/> [↑](#endnote-ref-19)
20. Ladikou EE, Ashworth I, Seviar D, Chevassut T. Acute leukaemia: no reason to panic. Clin Med (Lond). 2022 May;22(3):221-224. doi: 10.7861/clinmed.2022-0149. PMID: 35584840; PMCID: PMC9135077. [↑](#endnote-ref-20)
21. Cannas, G., & Thomas, X. (2015). Supportive care in patients with acute leukaemia: historical perspectives. Blood transfusion = Trasfusione del sangue, 13(2), 205–220. <https://doi.org/10.2450/2014.0080-14> [↑](#endnote-ref-21)
22. Ishida A, Handa M. [Examination and treatment for dilutional thrombocytopenia]. Rinsho Byori. 2005 Jul;53(7):654-7. Japanese. PMID: 16104535. [↑](#endnote-ref-22)
23. Tripathi AK, Chuda R. Laboratory Evaluation of Acute Leukemia. [Updated 2025 Jan 5]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK611988/> [↑](#endnote-ref-23)
24. Korubo KI, Okoye HC, Efobi CC. The economic burden of malignant and premalignant hematological diseases in Southern Nigeria. Niger J Clin Pract 2018;21:1396-402 [↑](#endnote-ref-24)
25. Saleh K, Fernandez A, Pasquier F. Treatment of Philadelphia Chromosome-Positive Acute Lymphoblastic Leukemia in Adults. Cancers (Basel). 2022 Apr 1;14(7):1805. doi: 10.3390/cancers14071805. PMID: 35406576; PMCID: PMC8997772 [↑](#endnote-ref-25)