Original Article

Epidemiology, Pathogenesis And Treatment Of Chronic Lymphocytic Leukaemia: An Update

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ABSTRACT
Chronic lymphocytic leukaemia (CLL) is a neoplastic disease characterised by the accumulation of small, mature-looking lymphocytes in the blood and bone marrow (BM). It is an indolent leukaemia characterised by the progressive accumulation of CD5-positive B cells in the blood and bone marrow because of a failure in programmed cell death or apoptosis. Peripheral blood immunophenotyping is required to confirm CLL diagnosis. The treatment of CLL currently consists of two main approaches — continuous therapy with Bruton's tyrosine kinase inhibitors and fixed-duration regimens combining venetoclax with CD20 antibodies and/or Bruton's tyrosine kinase inhibitors. Chemotherapy and chemo-immunotherapy are gradually becoming obsolete.

Keywords: -B-cells, Binet, Bruton tyrosine kinase inhibitors, Chemoimmunotherapy, Chronic Lymphocytic Leukaemia, Immunophenotype, Lymphocytes, Rai.

CHRONIC LYMPHOCYTIC LEUKAEMIA (CLL)
CLL is a neoplastic disease characterised by the accumulation of small, mature-looking lymphocytes in the blood and bone marrow (BM)[1]. It is the commonest leukaemia among Caucasians, and runs a variable clinical course. The tissue variant of CLL is called small lymphocytic lymphoma (SLL), a predominantly lymph node disease. A pre-leukaemic phase, monoclonal B cell lymphocytosis (MBL), which is defined as peripheral blood finding of monoclonal B-cell of <5000/µl without lymphadenopathy, has also been described.[2,3]

Epidemiology
The incidence of CLL is highest among Caucasians and low among Asian/Pacific Islanders[3, 4]. The highest incidence rates in 2004 were found to be in Australia, United States of America (USA), Ireland and Italy, with incidence ranging between 4 to 6 cases per 100,000 people per year[5, 6,77]. The median age of diagnosis in USA, Europe and Australia is approximately 70 years. The incidence increases with age, with about one quarter of patients being <65 years, and approximately 6% less than 50 years, with a male to female ratio of 1.5-2:1[7]. The incidence in Nigeria is not known but studies by Nwannadi et al.,[4] and Omoti et al.,[8] revealed that it is the most prevalent leukaemia in adults in South-South Nigeria, with a median age of 50 years and a female preponderance.[8]

Aetiology / Risk Factors
The aetiology of CLL is unknown, but some risk factors to the disease have been identified. The strongest risk to developing CLL is seen among relatives of CLL patients.[9] There is a 3-8 times higher predilection among relatives than in the normal population. Other risk factors include; occupational exposure (ionising radiation, petrochemicals, herbicides, pesticides, fertiliser etc). A higher incidence of CLL has been noted in hepatitis C (HCV) infection, though not being CLL-specific, as HCV infection is associated with a wide variety of lymphoproliferative disorders.[4,10]

Pathogenesis
The key feature in CLL pathogenesis is the progressive accumulation of CD5-positive B cells because of a failure in programmed cell death or apoptosis.[10,11] The site of origin of the neoplastic cells is still being debated, but studies using gene microarray analyses have provided evidence that CLL cells are derived from antigen-experienced, memory-type B cells.1 This could be germinal centre-derived [with evidence of somatic hypermutation in the variable regions of their immunoglobulin heavy chain gene (IGHV)], or marginal zone-derived (without somatic mutations of IGHV).[2, 3] In contrast with the model suggesting a B cell origin of the entire CLL pathogenesis, recent data suggest that the initiating event may occur in a haematopoietic stem cell.[1]

Most CLL cells in peripheral blood are in G0 phase of the cell cycle, and are long-lived. Studies, however, revealed

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that the leukaemic cells have a proliferation rate ranging from 0.1 per cent to greater than 1.0 per cent of the entire clone per day.1 Such high leukaemia-cell proliferation were noted even in patients with apparently stable blood lymphocyte counts.[12] The stimulus for proliferation, survival and death of the neoplastic cell is transmitted through the B-Cell antigen receptors (BCR). These BCRs have been found to be structurally similar and polyreactive in many CLL cases. Though the stimulating antigens are unknown, it is possible that latent viruses, commensal bacteria, environmental antigen or auto-antigens could provoke clonal expansion[13]. This antigenic stimulation, along with interactions with the microenvironment, is the promoting factor that stimulates proliferation of CLL cells and allows them to avoid apoptosis.

Clinical Features

In developed countries 70 to 80% of patients are diagnosed incidentally during a routine blood count and will have early-stage (Binet A) disease[10]. In Nigeria most patients present with late stage (Binet B or C) or due to complications of disease9. Common clinical features include; generalised lymphadenopathy, splenomegaly, fatigue or malaise (due to anaemia), bruising or bleeding (due to thrombocytopaenia), fever and recurrent infection (due to immuno-suppression) and weight loss[1].

Investigations in CLL

The diagnosis of CLL requires the presence of more than or equal to 5 × 109/L (5000/μL) monoclonal B lymphocytes in the peripheral blood for the duration of at least 3 months, demonstration of the clonality of the population (kappa/lambda analysis) and a characteristic immunophenotype: SmIg weak, CD5+, CD19+, CD20 weak, CD23+[1].

Full Blood Count finding

This shows lymphocytosis which may be marked (>100 x109/L). There may be anaemia (packed cell volume <30%), usually with normal red cell indices or thrombocytopaenia (platelet count of <100 x109/L).

Peripheral Blood Film finding

The peripheral blood smears in CLL cases show a high number of small lymphocytes with scanty cytoplasm and clumped chromatin, with presence of smudge cells or Gumprecht nuclear shadows (i.e., ruptured CLL cells). Prolymphocytes can be admixed with small lymphocytes in variable proportions, but usually represent less than 10% of the cells[13].

Immunophenotyping

Peripheral blood immunophenotyping is required to confirm CLL diagnosis. Flow cytometry or immunohistochemistry is used to demonstrate the clonality of B-cell expansion, showing the evidence of light chain restriction (i.e. either kappa or lambda). Chronic lymphocytic leukaemia cells express B-cell markers, like CD19, along with low levels of CD20, and are positive for CD5 and CD23. Recently CD200 and ROR1 have been demonstrated to be instrumental in differentiating CLL from other lymphoproliferative disorders[6, 10]. Matutes established a scoring system based on five indicators such as CD5, CD23, FMC7, CD22 or CD79b, and SmIg for diagnosis and differential diagnosis of typical CLL/SLL, atypical CLL/SLL as well as MCL[14, 15]. Newer scoring systems include Moreau score system (MSS) and New Score System (NSS)[15].

Bone Marrow Examination

In CLL, characteristically more than 30% of the nucleated cells in the aspirate are matured lymphocytes. A marrow aspirate and biopsy generally are not required for the diagnosis of CLL. However, they can be used to evaluate other causes of cytopaenia that may or may not be related to leukaemia-cell infiltration of the marrow[16].

Molecular Genetics

Using interphase Fluorescent In-Situ Hybridisation (FISH), cytogenetic lesions can be identified in more than 80% of all CLL cases[6]. The most common deletions are in the long arm of chromosome 13 [del(13q12.1)]. Other frequent chromosomal aberrations include deletions and/or trisomy of chromosome 12, deletions in the long arm of chromosomes 11 [del(11q)] and 6 [del(6q)], and in the short arm of chromosome 17 [del(17p)][2,17].

Serum Markers

Several studies have found that certain serum markers e.g. CD23, thymidine kinase, and β2-microglobulin may predict survival or progression-free survival in CLL[18].

Other Investigations

The following ancillary investigations should be done; Direct antiglobulin test (DAT) to rule out autoimmune anaemia, routine biochemistry (Fasting blood sugar, Lactate dehydrogenase, Liver function test, Serum electrolyte urea and creatinine), Hepatitis B and C infection screening, Human immunodeficiency virus (HIV) screening[19].

Clinical Staging of CLL

Two staging systems are currently applied in CLL patients to define disease burden and treatment indication: These systems are the Rai and the Binet staging system.

Binet staging system[1]

Stage	Clinical features at diagnosis	Median survival (years)
A	Blood and marrow lymphocytosis and less than 3 areas of palpable lymphoid-tissue enlargement	12
B	Blood and marrow lymphocytosis and 3 or more areas of palpable lymphoid-tissue enlargement	9
C	Same as B with anaemia (haemoglobin below 11 g/dL in men or 10 g/dL in women) or thrombocytopaenia (platelets less than 100,000/L)	7

Kipps et al., 2015

Rai classification of CLL[2]

Rai Stage	Clinical Features	Risk Category	Median Survival (Historical)
Stage 0	Lymphocytosis only in blood and bone marrow	Low risk	>10 years
Stage I	Lymphocytosis + lymphadenopathy	Intermediate risk	7-9 years
Stage II	Lymphocytosis + splenomegaly and/or hepatomegaly (with or without lymphadenopathy)	Intermediate risk	7-9 years
Stage III	Lymphocytosis + anaemia (Hb <11 g/dL) with or without organ enlargement	High risk	1.5-5 years
Stage IV	Lymphocytosis + thrombocytopaenia (platelets <100 × 10⁹/L) with or without anaemia, lymphadenopathy, or organ enlargement	High risk	1.5-5 years

Johnson et al., 2014

Prognosis in CLL

Some clinical, biological and genetic features of CLL have been shown to determine the clinical outcome of the disease. These prognostic markers in CLL are listed in the table below;

Prognostic factors in CLL[2]

Prognostic Marker	Better Prognosis	Worse Prognosis
Sex	Female	Male
Age	**<70 years**	**>70 years**
Plasma vitamin D level	High	Low
Rai stage	**0, I, and II**	**III and IV**
Lymphocyte count	<12 × 109/L	≥12 × 109/L
Lymphocyte doubling time	**>12 months**	**< 12 months**
Number of "smudge cells"	≥30%	<30%
β2-microglobulin level	**Low**	**High**
Flow cytometry: B-cell count	<11 × 109/L	≥11 × 109/L
CD38a	**<20% cells positive**	**≥20% cells positive**
ZAP-70	<20% cells positive	≥20% cells positive
FISH Deletion	**13q Deletion**	**11q22-23 or 17p13**
Tp 53 gene	Unmutated	Mutated
IgVH mutation status	**Mutated**	**Unmutated**

Johnson et al., 2014

Indications for Treatment[2, 3, 17]

At diagnosis, many CLL patients will not require treatment. However, patients with the following features should be treated;

- Evidence of progressive marrow failure as manifested by the development of, or worsening of, anaemia and/or thrombocytopaenia.
- Massive or progressive or symptomatic splenomegaly.
- Bulky nodes or progressive or symptomatic lymphadenopathy.
- Progressive lymphocytosis with an increase of more than 50% over a 2-month period or lymphocyte doubling time (LDT) of <6 months.
- Autoimmune anaemia and/or thrombocytopaenia that is poorly responsive to corticosteroids or other standard therapy.
- Constitutional symptoms, defined as any one or more of the following disease-related symptoms or signs:
  - Unintentional weight loss of 10% or more within the previous 6 months;
  - Significant fatigue (i.e., Eastern Cooperative Oncology Group Performance Score 2 or worse; inability to work or perform usual activities);
  - Fever higher than 38°C for two or more weeks without other evidence of infection; or
  - Night sweats for more than 1 month without evidence of infection.

Treatment of CLL

The clinical course of CLL is variable. Some patients may not need treatment for their disease at the time of diagnosis. Patients in this category are put on the watch and wait list and monitored periodically. For those that have an indication for treatment, the choice of treatment will depend on the following[2]:

- Patient's fitness status based on comprehensive geriatric assessment
- The genetic profile (i.e., the presence vs. absence of TP53 alterations, detected by FISH and/or by mutation analysis)
- The disease status (first-line treatment vs. ≥2 line of treatment; relapse vs. refractoriness to last treatment)[2]

Chemoimmunotherapy, particularly the combination of fludarabine, cyclophosphamide, and rituximab (FCR), was historically the standard of care for fit patients with CLL. Clinical studies demonstrated high response rates and prolonged progression-free survival, especially in patients with mutated IGHV20.

However, the use of chemoimmunotherapy has declined due to significant toxicities such as myelosuppression, infections, and secondary malignancies. Furthermore, outcomes are poor in patients with high-risk cytogenetics such as TP53 mutations, limiting its applicability in modern practice.[3,20] Consequently, current guidelines increasingly favour targeted therapies over chemotherapy in most patient populations.

TARGETED THERAPIES IN CLL

Bruton Tyrosine Kinase (BTK) Inhibitors

BTK inhibitors have revolutionised the treatment of CLL by targeting B-cell receptor signalling, a critical pathway for CLL cell survival. Agents such as ibrutinib, acalabrutinib, and zanubrutinib have demonstrated superior progression-free survival compared to chemoimmunotherapy and are now considered first-line treatment options [3,17].

Recent studies show that continuous BTK inhibitor therapy results in durable disease control, particularly in patients with high-risk cytogenetic abnormalities20. Second-generation BTK inhibitors such as acalabrutinib and zanubrutinib offer improved safety profiles with reduced cardiovascular toxicity compared to ibrutinib [21].

BCL-2 Inhibitors

Venetoclax, a selective inhibitor of the anti-apoptotic protein BCL-2, induces programmed cell death in CLL cells and has demonstrated high efficacy in both treatment-naïve and relapsed disease. The CLL14 trial showed that venetoclax combined with obinutuzumab significantly improved progression-free survival compared with chlorambucil-based therapy [21].

Importantly, venetoclax-based regimens are administered for a fixed duration and are associated with high rates of minimal residual disease (MRD) negativity, representing a major shift towards time-limited therapy in CLL[22]. This contrasts with continuous therapy required for BTK inhibitors and provides an alternative treatment strategy.

PI3K Inhibitors

PI3K inhibitors such as idelalisib and duvelisib have demonstrated clinical efficacy in relapsed or refractory CLL; however, their use is limited by significant immune-mediated toxicities, including colitis, pneumonitis, and opportunistic infections [20]. As a result, these agents are typically reserved for patients who are unsuitable for BTK or BCL-2 inhibitor therapy.

Combination and Sequential Therapies

Emerging evidence suggests that combining targeted therapies may enhance treatment efficacy and achieve deeper remissions. Studies evaluating combinations of BTK inhibitors and venetoclax have demonstrated promising results, including higher rates of MRD negativity and prolonged remission durations [22].

Additionally, treatment sequencing has become an important consideration, particularly in the context of resistance. Venetoclax-based therapies have shown high response rates even after failure of BTK inhibitors, highlighting their role in subsequent lines of therapy[23].

Immunotherapy and Novel Approaches

Monoclonal antibodies targeting CD20, such as rituximab and obinutuzumab, remain integral components of CLL therapy, particularly in combination regimens. Novel immunotherapeutic approaches, including chimeric

antigen receptor (CAR) T-cell therapy, are under investigation and have demonstrated efficacy in relapsed or refractory disease, although their use is currently limited to specialised centres.

In addition, emerging therapies such as non-covalent BTK inhibitors and BTK degraders are being evaluated in clinical trials and may overcome resistance to existing therapies [23].

Complications in CLL [1,2,24]

The complications of CLL include:

- **Immunosuppression:** there is reduced innate and adaptive immunity.
- **Cytopaenia:** this could be anaemia, thrombocytopaenia or neutropaenia.
- **Transformation:** the commonest transformation in CLL is Richter's transformation (this is the development of diffuse large B-cell lymphoma in a patient with CLL or SLL). Other rarer transformations include prolymphocytic leukaemia, acute leukaemia, multiple myeloma.
- **Second malignancy:** the incidence of a second malignancy is higher in patients with CLL compared to other lymphoid malignancies. Squamous and basal cell carcinoma of the skin are the most common second malignancies. Others include cancer of the respiratory tract, gastrointestinal tract, breast and prostate.

African and Nigerian Context

Data on CLL treatment in Africa remain limited, highlighting a significant gap in the literature. Studies from South Africa suggest that targeted therapies such as BTK inhibitors are effective and may be cost-effective within public healthcare systems, although access remains a major challenge[25].

In Nigeria, limited awareness, delayed diagnosis, and restricted access to novel therapies contribute to suboptimal outcomes. Recent reports emphasise the need for improved healthcare infrastructure, increased research, and equitable access to modern treatments in African populations [26]. Furthermore, most existing clinical trials are conducted in high-income countries, limiting the generalisability of findings to African settings.

CONCLUSION

Chronic lymphocytic leukaemia is a relatively common haematological malignancy affecting older adults. Advances in genomic studies have profoundly impacted the understanding of CLL pathogenesis, revealing specific genetic mutations and chromosomal abnormalities that influence disease progression and treatment response. Key prognostic markers, such as the deletion of chromosome 17p (del(17p)), mutations in the TP53 gene, and the mutational status of the immunoglobulin heavy chain variable region (IGHV), determine the prognosis. The treatment of CLL has evolved dramatically from conventional chemotherapy to targeted and immune-based therapies. BTK inhibitors and venetoclax-based regimens now form the cornerstone of modern treatment, offering improved survival and better tolerability.

RECOMMENDATIONS

1. **Increased Awareness and Early Diagnosis:** there is a need for increased awareness of chronic lymphocytic leukaemia among healthcare providers and the general population in order to promote early diagnosis and prompt referral, to reduce the incidence of patients presenting with advanced disease.
2. **Healthcare Policy and Funding Support:** Governments and healthcare agencies should provide increased funding for cancer care, expansion of diagnostic laboratory services (such as flow cytometry, immunophenotyping, fluorescence in-situ hybridisation (FISH), and molecular testing), as well as improving the availability and affordability of targeted therapies such as Bruton tyrosine kinase (BTK) inhibitors and venetoclax-based regimens in developing countries and access to novel therapies in order to improve outcomes in patients with CLL.
3. **Development of Local Treatment Guidelines:** there is a need to develop local treatment guidelines useful for the diagnosis and management of CLL in African populations, taking into account the peculiar issues such as the sub-par healthcare realities and resource limitations.
4. **Strengthening Research in African Populations:** more multi-centre studies are needed in Africa to better understand the epidemiology, genetic profile, treatment outcomes, and survival patterns of CLL among African patients.
5. **Increased Participation in Clinical Trials:** efforts should be made to improve the inclusion of African populations in international clinical trials to enhance the applicability of emerging treatment data.

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