



Authors and institutions

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Introduction

Chronic Myeloid Leukemia (CML) is marked by an unregulated growth of myeloid cells, making up 15% of all leukemia cases. This ailment is predominantly observed in individuals aged 40-60, with a minor male preponderance, and is relatively rare in childhood. Patients may present in three disease phases: chronic phase (CP), accelerated phase (AP), and blast phase (BP), or blast crisis (BC), which is characterized by an increased percentage of blasts from the myeloid, lymphoid, or mixed/undifferentiated lineage. Myeloid BC occurs about twice as frequently as lymphoid BC, accounting for approximately 70%. Patients receiving modern therapies tend to have a life expectancy similar to that of aged-matched controls. The symptoms of CML are nonspecific and may include fever, fatigue and weight loss, often as a result of anemia and splenomegaly. When the disease progresses to BC, symptoms may worsen and include bone pain and bleeding. However, approximately 50% of CP CML patients do not show any symptoms and might only be diagnosed during routine blood tests. **Objective:** Conduct an in-depth and comprehensive examination and characterization of scientific literature pertaining to the clinical aspects of Chronic Myeloid Leukemia, including but not limited to risk factors, signs and symptoms, prevalence, management strategies, and pathophysiology.

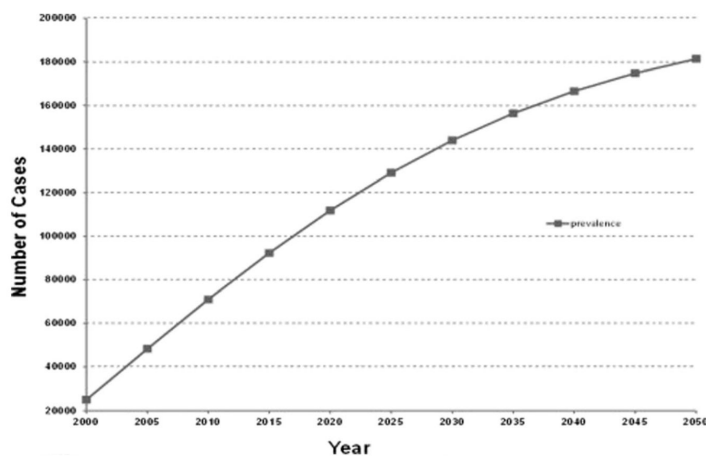
Methods

We conducted a thorough analysis and exploration of the scientific literature regarding Chronic Myeloid Leukemia (CML) by scrutinizing articles published between 2007 and 2021 in prominent platforms such as PUBMED, The Lancet and The New England Journal of Medicine. Our search included articles in both Portuguese and English languages using a range of relevant search terms, including "CML," "Chronic Myeloid Leukemia," "clinical diagnosis," "prognosis," "epidemiology," and "treatment."

Chronic myeloid Leukaemia was the first neoplastic disease for which knowledge of the genotype led to a rationally designed therapy. As a result of its well known pathophysiology, straightforward diagnosis, well established prognostic factors, and treatment for the cause of disease. The importance of research discovered the translocation between chromosomes 9 and 22 causing this abnormality, along with the specific genes and implications of this rearrangement. CML has been studied to an extent that far exceeds that expected from its frequency, and serves as a model disease for other cancers. This knowledge led to an extraordinary therapy. Using tyrosine kinase inhibitors improved survival rates so patients now have life expectancies similar to the general population. Nonetheless, there remain unanswered questions and challenges that hinder progress and the ultimate cure of some patients. As a result, CML prevalence in the United States is projected to rise to 180,000 by 2050, making it the most widespread myeloid neoplasm. **References:** Hehlmann R, Hochhaus A, Baccarani M; European LeukemiaNet. Chronic myeloid leukaemia. Lancet. 2007 Jul 28;370(9584):342-50. doi: 10.1016/S0140-6736(07)61165-9. PMID: 17662883. / Rohrbacher M, Hasford J. Epidemiology of chronic myeloid leukaemia (CML). Best Pract Res Clin Haematol. 2009 Sep;22(3):295-302. doi: 10.1016/j.beha.2009.07.007. PMID: 19959081. / Thompson PA, Kantarjian HM, Cortes JE. Diagnosis and Treatment of Chronic Myeloid Leukemia in 2015. Mayo Clin Proc. 2015 Oct;90(10):1440-54. doi: 10.1016/j.mayocp.2015.08.010. PMID: 26434969; PMCID: PMC5656269.

Results

The broad use of tyrosine kinase inhibitors has resulted in an improvement in the overall survival to the point where the life expectancy of patients today is nearly equal to that of the general population. Still, there are challenges and unanswered questions that define the reasons why the progress still escapes many patients, and the details that separate patients from ultimate cure. The variations of incidences amongst published CML reports might indicate geographic or ethnic variability beyond technical artefacts. In most cases, the diagnosis can be made on the basis of characteristic blood count and differential (excessive granulocytosis with typical left shift of granulopoiesis) if myelofibrosis and myelodysplasia have been excluded. The recognition of prognostic factors at diagnosis is essential. Most important is accurate identification of the disease stage (or phase), but in early chronic phase important prognostic information is derived from clinical and laboratory features. The scores differentiate between patients with low, intermediate, and high risk of short survival. Cytogenetic changes such as deletions of the derivative chromosome 9 also affect prognosis.



Conclusion and References

Contact