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Opinion Article

Assessment of the Treatment to Chronic Myeloid Leukemia

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DESCRIPTION

A monoclonal proliferation at the expense of mature lymphocytes is what is meant by Chronic Myeloid Leukemia (CML). It accounts for 25% of all cases of leukemia in adults and is the most prevalent kind in Western nations. It is challenging to estimate the true incidence because it might exist in people with chronic lymphocytic leukemia. Before diagnosis, it was estimated that 30% of cases were asymptomatic. However, in the last 20 years, the percentage of CML cases that present in the asymptomatic stages has increased from 30% to 60%, most likely as a result of the increased use of blood tests for other medical or surgical purposes. Moreover, sensitive methods for identifying CML and separating it from other chronic lymphoproliferative diseases have only lately become widely used. The exact cause is unknown, but there is a lot of evidence that suggests a genetic component, including the fact that CML is more common in first-degree relatives, the anticipation phenomenon, which shows that severity and age increase with each generation, and a higher prevalence of autoimmune diseases in people who have relatives with CML. Ionizing radiation, chemicals (such as petrol and solvents from the rubber industry), and medications were not linked to the condition. Chronic lymphocytic leukemia is a model of failed apoptosis BCL-2 family proteins are expressed (which are essential regulators of apoptosis) are overexpressed in 90% of B-CLL (B-Cell Chronic Lymphocytic Leukemia) cells, however in the vast majority of cases (96%–99%) there is no translocation involving BCL-2.

Slow growing B-CLL cells accumulate in the body, mostly in the G0 phase of the cell cycle one consequence is acquired resistance to agents active in the cell cycle. An unbalanced ratio of certain essential proteins, including BAX (Bcl-2-Associated X protein) and BAK (Bcl-2 Antagonist Killer 1) which induce apoptosis, BCL-2 (which inhibits it), BAD, BIK, and HRK (inhibitors of apoptosis) They appear to be crucial in how CML behaves and responds to treatment, though there isn't yet strong clinical proof of this. Increasing expression of the cyclin-dependent p27 kinase inhibitor and mutations of the p53 tumors suppressor gene have both been linked to disease progression and a generally bad prognosis. Tumor Necrosis Factor (TNF), Interleukin 8 (IL-8), and IL-2, which is produced by T lymphocytes and taken up by CML cells through particular receptors, are cytokines that are produced and released directly by CML cells and have an impact on the survival and growth of CML cells.

CD30 expression is more prevalent when IL-4 is produced. Since most CML cells appear to express CD30, this interaction may have an impact on the surroundings of CML cells and their immunological capabilities. The crosslinking expression of CD40 or CD154, which is made by activated T cells, is one of the crucial stages of the immunological response to an antigen. Severe immune deficiencies are caused by the overexpression of this ligand that is brought on by CML leukemic cells.

In patients with Richter syndrome, a chromosome 17 aberration has been linked to a p53 mutation, fludarabine resistance, and therapy failure. Uncertainty surrounds the primary or secondary nature of certain cytogenetic anomalies, including unusual CML morphology. In a group of individuals with superior clinical outcomes and increased survival, *IgV* gene mutations have been linked to lower CD38 expression. As a result, CD38 may be helpful as a prognostic indicator and a stand-in for *IgV* genetic alterations. Chronic lymphocytic leukemia frequently develops infections, autoimmune leukopenia, and high-grade lymphoma, among other consequences. In roughly 1% of chronic lymphocytic leukemia patients each year, the disease can progress from chronic lymphocytic leukemia to diffuse large B-cell lymphoma or Hodgkin's lymphoma, which may impact up to 16% of individuals. When primary chronic lymphocytic leukemia is present along with diffuse large B-cell lymphoma, this is known as Richter's transition, and the prognosis is typically quite bad. Richter's transformation is more likely in people with chronic lymphocytic leukemia who have *NOTCH1* mutations.

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