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Editorial

The Clinical Characteristics and Causes of Plasma Cell Leukemia

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EDITORIAL NOTE

CML (Chronic Myelogenous Leukaemia), often called chronic myeloid leukaemia, is a type of white blood cell malignancy. It's a type of leukaemia marked by a rise in uncontrolled myeloid cell proliferation in the bone marrow, as well as an accumulation of these cells in the blood. CML is a clonal bone marrow stem cell condition in which mature granulocytes (neutrophils, eosinophils, and basophils) and their precursors proliferate in the bone marrow. It's a form of myeloproliferative neoplasm marked by a chromosomal rearrangement known as the Philadelphia chromosome.

Since 2001, targeted medications known as Tyrosine-Kinase Inhibitors (TKIs) have drastically improved long-term survival rates in CML patients. When compared to previous chemotherapy medicines, these drugs have transformed the treatment of this disease and allow most patients to enjoy a good quality of life.

CML accounts for 15-25 percent of all adult leukemias and 14% of all leukemias in Western countries (including the paediatric population, where CML is less common). The way CML manifests is determined by the stage of the disease at the time of diagnosis, although it has been known for CML to skip stages in some cases.

The majority of patients (90%) are diagnosed during the chronic stage, which is usually asymptomatic. In some circumstances, a raised white blood cell count on a regular laboratory test may be used to diagnose it. It can also show signs of hepatosplenomegaly and the pain that comes with it in the left upper quadrant. The spleen's enlargement may put pressure on the stomach, resulting in a decrease of appetite and weight loss. Due to a raised basal level of metabolism, it might also cause a minor temperature and nocturnal sweats.

Some (about 10%) are diagnosed during the advanced stage, which is characterised by bleeding, petechiae, and ecchymosis. Fever is most usually caused by opportunistic infections in these patients.

Some patients are diagnosed during the blast phase, which is characterised by fever, bone pain, and increased bone marrow fibrosis. Males are more likely than females to have CML (male to female ratio of 1.4:1), and the elderly are more likely to develop it, with a median age at diagnosis of 65 years. Based on a 50-fold greater prevalence of CML in Hiroshima and Nagasaki nuclear bombing survivors, ionising radiation appears to be a risk factor. The rate of CML in these people appears to peak around ten years following exposure.

CML was the first malignancy to be linked to a specific genetic defect, the so-called Philadelphia chromosome translocation. This chromosomal anomaly was named after two doctors from Philadelphia, Pennsylvania, who discovered and characterised it in 1960: Peter Nowell of the University of Pennsylvania and David Hungerford of Fox Chase Cancer Center.

Parts of two chromosomes (the 9th and 22nd) swap positions in this translocation. As a result, a portion of chromosome 22's BCR ("Breakpoint Cluster Region") gene is fused to chromosome 9's ABL gene. This aberrant "fusion" gene produces a protein with a mass of p210 or p185 (p210 is short for 210 kDa protein, a shorthand used for characterising proteins based solely on size). The bcr-abl fusion gene product is also a tyrosine kinase since abl contains a region that may add phosphate groups to tyrosine residues (a tyrosine kinase).

The interleukin 3beta(c) receptor component interacts with the fused BCR-ABL protein. The BCR-ABL transcript is always active and does not require other cellular messaging proteins to activate it. BCR-ABL then activates a cascade of proteins that control the cell cycle, causing cell division to speed up. Furthermore, the BCR-ABL protein hinders DNA repair, resulting in genomic instability and increasing the risk of subsequent genetic abnormalities in the cell. The pathophysiologic cause of chronic myelogenous leukaemia is the action of the BCR-ABL protein. Targeted medicines (the first of which was imatinib) that specifically block the activity of the BCR-ABL protein have been created as a result of greater understanding of the nature of the BCR-ABL protein and its action as a tyrosine kinase. These tyrosine kinase inhibitors can elicit full remissions in CML patients, indicating that bcr-abl is the primary cause of CML.

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