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Commentary

Erythropoietin and Blood Transfusions for Disease Related Anemia

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DESCRIPTION

Chronic Myelomonocytic Leukemia (CMML) is a type of leukemia that affects the bone marrow's blood-forming cells. In adults, blood cells are generated in the bone marrow through a process known as hematopoiesis. Increased levels of monocytes and immature blood cells (blasts) in the bone marrow and peripheral blood, as well as irregular cells (dysplasia) in at least one type of blood cell, are all found in CMML. CMML has features of both a Myelodysplastic Syndrome (MDS), which creates abnormal-looking blood cells, and a Myeloproliferative Neoplasm (MPN), which causes an overproduction of blood cells. This led to the classification of CMML as an MDS/MPN overlap disorder in 2002. According to the World Health Organization (WHO), the blood monocyte count must be greater than 1×10^9/L, that a must be no Philadelphia Chromosome or PDGFRA or PDGFRB gene mutations, the blast count must be less than 20%, so that must be dysplasia of at least one lineage of myeloid blood cells. The Food and Drug Administration (FDA) and the European Medicines Agency both have approved the drug azacitidine to be used in the treatment of CMML. Stem cell transplantation, which involves the transfer of donor hematopoietic stem cells into the recipient, is also used to treat CMML. Treatment options for anemia carried on by an illness include blood transfusions and erythropoietin.

Splenomegaly is one of the most prevalent symptoms of CMML, appearing in roughly half of all cases. Anemia, fever, weight loss, night sweats, infection, bleeding, synovitis, lymphadenopathy, skin rashes, pleural effusion, pericardial effusion, and peritoneal effusion are a few less typical signs and symptoms. Although the etiology of CMML is unknown, environmental carcinogens, ionizing radiation, and cytotoxic agents both may have a role. Approximately one-third of MDS cases with a monocyte count of >10% and 1×10^9/L will advance to CMML. Deregulation of this signaling system has been linked to the pathogenesis of the disease, that has a high rate of RAS mutation in CMML. TNF, GM-CSF, interleukin-3, interleukin-4, interleukin-6, and interleukin-10 may all have a role in hyper proliferative CMML cells. *In vitro*, these cytokines can promote CMML development. Many cancers experience hyper methylation of cytosine residues, which often occurs in the promoter regions of genes to control gene expression. The cell cycle regulation gene *p15INK4b* is one that is commonly hypermethylated in CMML.

While clonal genetic abnormalities are common in CMML, they do not ensure a right diagnosis. The 8+, 7/del (7q), and structural 12p anomalies are the most frequently discovered. 25-40% of CMML cases contain abnormalities in the KRAS and NRAS regions. In 10% of cases, the *Jak2 V617F* mutation is found. Up to 30% of cases have mutations in transcription factors such *RUNX1*, *CEBPA*, *NPM1*, and *WT1*. CBL mutations are identified in approximately 5-18% of cases. Around 40% to 50% of CMML had *TET2* gene mutations. Inactivating mutations in one of the two parental *GATA2* genes result in a decrease, or haploinsufficiency, in the cellular levels of the gene's product, the *GATA2* transcription factor, and hence in *GATA2* deficiency, a rare autosomal dominant genetic disease. A broad range of disorders, including the myelodysplastic syndrome, acute myeloid leukemia, and CMML, are linked to this illness. Like other forms of CMML, *GATA2*-deficiency induced CMML is usually preceded by monocytosis. A variety of anomalies can be seen in blood films. A monocyte count of more than 1×10^9/L is required to diagnose CMML. The presence of metamyelocytes, myelocytes, and promonocytes; monocytes with hypersegmented/abnormally shaped nuclei, increased cytoplasmic basophilia, and or the presence of cytoplasmic granules; eosinophilia (in cases of CMML with eosinophilia); and spherocytosis are extra features that may be prevalent. Leuko (in cases of direct Coombs test, DCT, positive hemolytic anemia). Platelet counts can be low, high, or normal. Red blood cells that are normocytic and normochromic generally have lower hemoglobin levels. Cold agglutinins and autoantibodies may be present, and 10% of CMML shows positive DCT results.

Aspirates of bone marrow will have hypercellularity and higher levels of granulocytic and monocytic cells. A preponderance of myelocytic and monocytic cells, aberrant localization of immature precursors, and dysplastic megakaryocytes may be seen in bone marrow core biopsies. Biopsies often include monocytic nodules. The succeeding phenotypical characteristics of CMML are present in 100% of cases: CD11b, CD11c, CD14, CD33, CD45,

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and CD64; CD13 in 95% of cases: CD4 in 76% of cases; HLA-DR in 71% of cases; CD56 in 53% of cases; CD2 in 34% of cases; CD16 in 29% of cases; CD10 in 28% of cases; CD23 and CD7 in 9% of cases; and CD117.

The treatment of CMML remains difficult due to a lack of clinical trials investigating the disease as a diverse clinical entity. Since it is frequently included along with MDS in clinical studies, the treatment of CMML is very similar to that of MDS. Because most therapies do not even actually improve survival, the majority of people are handled as supportive rather than curative. B symptoms, symptomatic organ involvement, raised blood counts, hyperleukocytosis, leukostasis, and/or worsening cytopenias all are indications for treatment. The U.S. Food and Drug Administration (FDA) for the treatment of high-risk non-proliferative CMML with 10-19% marrow blasts. It is a cytidine analogue that inhibits DNA methyltransferase to cause hypomethylation of DNA. The FDA has approved decitabine, a drug that really is similar to azacitidine and is used to treat all MDS subtypes, including CMML. Hydroxyurea is a treatment that is used to lower cell counts in the myeloproliferative form of CMML.

A fixed-dose combination drug called Decitabine/Cedazuridine (Inqovi) is used to treat adults with Chronic Myelomonocytic Leukemia (CMML) and Myelodysplastic Syndromes (MDS).