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Perspective

Pathophysiology of Myeloid Leukemia and the Role of Hematopoietic Stem Cells

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DESCRIPTION

Myeloid leukemia is a form of cancer that originates in the bone marrow, the soft, spongy tissue at the center of bones where blood cells are produced. In this disease, immature blood cells, particularly myeloid cells, grow uncontrollably, leading to various health complications. Myeloid leukemia is essential first to comprehend the role of myeloid cells in the body. The bone marrow produces three types of blood cells: Red blood cells (which carry oxygen), platelets (which help in blood clotting), and white blood cells (which fight infections). White blood cells are further divided into lymphoid and myeloid cells. Myeloid cells, such as neutrophils, eosinophils, and monocytes, are vital in the body's immune response. In myeloid leukemia, these cells undergo malignant transformation, proliferating abnormally and inhibiting the production of normal, healthy blood cells.

Acute Myeloid Leukemia (AML)

AML is characterized by the rapid growth of abnormal cells that accumulate in the bone marrow and blood. These cancerous cells interfere with the production of normal blood cells, leading to symptoms such as anemia, fatigue, increased susceptibility to infections, and easy bruising or bleeding. The onset of AML is sudden and requires immediate medical intervention.

Pathophysiology of AML

In AML, mutations in the DNA of myeloid progenitor cells cause them to develop into immature, dysfunctional cells known as blasts. Normally, these progenitor cells mature into functional white blood cells; however, in AML, this maturation process is disrupted. Blasts continue to replicate uncontrollably, crowding the bone marrow and spilling into the bloodstream, disrupting normal hematopoiesis (blood cell production). AML is highly heterogeneous, meaning it has several subtypes, each defined by specific genetic mutations or chromosomal abnormalities, which can affect prognosis and treatment strategies.

Chronic Myeloid Leukemia (CML)

In contrast to AML, Chronic Myeloid Leukemia (CML) progresses more slowly. It is characterized by the overproduction of mature white blood cells. CML is strongly associated with a specific genetic abnormality known as the Philadelphia chromosome, where parts of chromosomes 9 and 22 swap places, creating an abnormal fusion gene called *BCR-ABL*. This gene produces a protein that promotes the uncontrolled growth of myeloid cells.

Pathophysiology of CML

The *BCR-ABL* fusion gene leads to the production of a tyrosine kinase enzyme that is always driving the proliferation of abnormal white blood cells. Over time, the number of these cells increases in the bloodstream, leading to symptoms like fatigue, night sweats, weight loss, and an enlarged spleen. CML progresses through three phases: The chronic phase, where the disease is usually asymptomatic; the accelerated phase, where symptoms become more apparent; and the blast crisis phase, resembling acute leukemia and requiring aggressive treatment.

Immunotherapy

Immunotherapy, which harnesses the body's immune system to fight cancer, is an emerging area in myeloid leukemia research. CAR-T cell therapy, where T cells are genetically engineered to target cancer cells, has shown promise in treating other types of leukemia and is being investigated for myeloid leukemia.

Epigenetic therapies

Researchers are increasingly focusing on the role of epigenetic changes, such as DNA methylation and histone modification, in the development of leukemia. Drugs that reverse these changes, known as epigenetic modifiers, are being tested in clinical trials. For example, azacitidine and decitabine, which inhibit DNA methylation, are already used to treat certain subtypes of AML,

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particularly in older patients who cannot tolerate high-dose chemotherapy.

CONCLUSION

Myeloid leukemia is a complex and multifaceted disease that has significant challenges to patients and healthcare providers. However, advances in our understanding of the disease at the genetic and molecular levels have led to the development of more effective treatments, particularly targeted therapies and immunotherapies. While challenges such as drug resistance, relapse, and treatment toxicity remain, ongoing research offers hope for improved outcomes and the eventual cure of this devastating disease.