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Commentary

Hematopoietic Stem Cells to Restore Proper Physiology to the Bone Marrow

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DESCRIPTION

Adult mammalian hematopoiesis begins in the bone marrow, where a small population of dormant stem cells gives rise to expanding populations of committed progenitors that replenish all blood cell lineages throughout the organism's lifetime. Hematopoietic Stem Cells (HSCs) have a high proliferation capacity because they have the unique ability to self-renew. Although the external signals that influence HSCs' decision to self-renew or differentiate are not totally understood, they are assumed to dwell in discrete micro environmental zones inside the bone marrow known as "niches."

Hematopoietic Stem Cells (HSCs) are responsible for the production of mature blood cells in the bone marrow; peripheral pancytopenia is a common clinical manifestation of a variety of conditions, including haematological or extra-haematological diseases (mostly cancers) that affect marrow function, as well as primary hematopoiesis failure. Primary bone marrow failure syndromes are a diverse collection of disorders that share a substantial impairment of the hematopoietic stem cell pool, resulting in universal or selective marrow aplasia.

Constitutional marrow failure syndromes are diseases caused by intrinsic deficiencies in HSCs. They are caused by inherited germline mutations that generate specific phenotypes, and they frequently affect organs and systems other than hematopoiesis. Hematopoietic stem cells in acquired marrow failure disorders, on the other hand, are assumed to be inherently normal but have extrinsic damage that affects their hematopoietic function. It's possible to show direct toxicity from chemicals or radiation, as well as links to viruses and other infectious agents. Aplastic Anaemia (AA) is a very instructive stem cell illness in that it reveals the function and amount of normal hematopoietic stem cells as well as their potential to regenerate. Understanding the pathophysiology of AA may reveal processes underlying the progression of other related bone marrow failure disorders such as paroxysmal nocturnal haemoglobinuria and myelodysplasia-clonal hematopoiesis diseases associated with faulty stem cells. Immunological mechanisms play a key role in idiopathic (AA), causing the hematopoietic compartment to be damaged and the hematopoietic stem cell pool to be depleted. Even if the target antigens are still unknown, clinical and experimental evidence supports the presence of a T cell-mediated immune assault, as proven by clonally expanded lymphocytes.

This simplistic model, however, must be combined with new results demonstrating that, even in the presence of extrinsic damage, pre-existing mutations or polymorphisms in genes might lead to a genetic tendency for marrow failure. Similar antigen-driven immune pathways may be involved in marrow failure associated with lymphoproliferative or autoimmune illnesses characterised by clonal growth of T cells, such as Large Granular Lymphocyte Leukemia, according to new research. Even when one or more HSCs and their progeny carry a somatic mutation of the PIG-A gene, the typical marrow failure in PNH is likely due to pathogenic mechanisms similar to those involved in AA, rather than the intrinsic abnormality conferred to the clonal population by the PIG-A mutation.

The investigation of hematopoietic stem cell function in marrow failure syndromes reveals particular molecular pathways disrupted in a variety of hematopoietic and non-hematopoietic stem cell illnesses. Marrow failure syndromes constitute a paradigm that provides intriguing insight into the quantity and function of normal hematopoietic stem cells, therefore advancing our understanding of stem cell biology.

CONFLICT OF INTEREST

Author has nothing to disclose.

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