Special Issue

Therapeutic Strategies for Aplastic Anemia and Paroxysmal Nocturnal Hemoglobinuria

Message from the Guest Editors

Idiopathic Aplastic anemia and paroxysmal nocturnal hemoglobinuria belong to the spectrum of immune-mediated bone marrow failure disorders. The autoimmune attack by cytotoxic T lymphocytes to hematopoietic stem cells is the pathogenic hallmark of AA. The consequent reduction of the HSCs pool generates the classical AA clinical picture, dominated by peripheral cytopenias and an increased tendency for hemorrhagic and infectious complications. In the context of an AA immune attack, PIGA mutations may arise, ultimately generating the absence of glycosylphosphatidylinositol (GPI)-anchored protein on the membrane of all blood cells derived by the mutant clone. The absence of such GPI-linked proteins makes blood cells (particularly erythrocytes) vulnerable to complement-mediated lysis. In the last decade, new evidence on disease pathobiology furthered our understanding of AA and PNH, generating mechanistic clues on potential therapeutic opportunities. The addition of eltrombopag in AA and newer complement inhibitors in PNH is revolutionizing the treatment landscape of BMFs.

Guest Editors

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