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Sickle Cell Anemia: From Genetic Epidemiology to New Therapeutic Strategies

Guest Editor:

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Message from the Guest Editor

Sickle cell anemia is a monogenic disease associated with recurrent painful episodes, inflammation, hemolytic anemia, and progressive multiorgan damage. However, its clinical expression is highly variable and dependent on other genes. The most significant genetic factors are the HbF levels, alpha gene number, and beta-haplotypes. The polymerization of deoxygenated HbS is responsible for red cell sickling, causing impaired blood rheology, intra- and extravascular hemolysis and vaso-occlusion. Hemolytic anemia is associated with cerebral macrovasculopathy, priapism, leg ulcers, and pulmonary hypertension. Blood transfusions remain the mainstay of therapy. Hydroxyurea has been the first drug treatment able to reduce the rate of vaso-occlusive crises and acute chest syndromes. New molecules have emerged, such as crizanlizumab, or voxelotor (GBT-440). However, to date, hematopoietic stem cell transplantation is the only disease-reversing treatment modality and highly successful results obtained with matched-sibling donors support using other donors, such as haplo-identical and unrelated donors, and exploring autologous transplantation modified by gene-therapy.

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