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Bone marrow failure in the elderly**Gustavo Rivero**

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Pathogenic mechanisms that account for marrow failure in aging population are complex. With a worldwide population achieving successful chronologic aging, an increase in these conditions and cost for management are expected to increase substantially. It is possible that decline in stem cell physiology, acquired somatic mutation and autoimmunity interface to dictate individual propensity for hemopoietic failure. Aplastic anemia (AA) paroxysmal nocturnal hemoglobinuria (PNH), myelodysplastic syndrome (MDS) and large granular lymphocytic (LGL) leukemia are commonly observed. Among them, MDS is cytogenetically, epigenetically and immunologically heterogenous. Treatments are limited with growth factor support and transfusion representing frontline therapy for low-risk MDS. For high risk cases, allogeneic stem-cell-transplantation for suitable candidates or epigenetic therapy is the only treatment modalities. In recent years, the role of innate immunity in elderly marrow failure has ignited interest. Here, we discuss current scientific advances that highlight possibility for development of interventions aimed at targeting pathogenic deregulated innate immunity in MDS initiation.

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