



Myelodysplastic Syndromes: From Inflammatory to Therapeutic Approaches

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Message from the Collection Editor

Impaired function of immune cells and aberrant inflammatory response of the immune system is frequently observed for patients with MDS and CMML. Increased secretion of inflammatory cytokines, including tumor necrosis factor-(TNF) and interferon (IF), contributes to cell death of myeloid precursor cells and triggers transformation to AML. Autoimmune disorders (AD) observed for MDS and CMML patients include vasculitis, seronegative polyarthritis, relapsing polychondritis, or neutrophilic dermatosis. In a subset of patients, cytopenias are the consequence of autoreactive cytotoxic T cells and respond to immunosuppressive treatment (IST). Interestingly, despite IST, other drugs are of interest by treating MDS and CMML-associated AD and inflammatory response as the hypomethylating agent azacytidine improves cytopenias, delays disease progression, and controls AD by sparing corticosteroids and other immunosuppressive drugs. The immunomodulatory drug lenalidomide improves anemia in specific subtypes of MDS and inhibitors of the JAK-STAT pathway should be evaluated for the treatment of selected MDS and CMML patients.





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