Special Issue

Thrombocythemia: Current Status, Challenges and Future Directions

Message from the Guest Editor

Essential Thrombocythemia (ET) is characterized by abnormal megakaryocytes and thrombocytosis. The ET is generally associated with mutations such as JAK2 exon 14, CALR exon 9, or MPL exon 10 (W515R/G/S). However, about 10% of patients with ET are triple negative (TN). TN patients can have "noncanonical" MPL mutations (T119I, S204F/P, E230G, Y252H and Y591D/N, R537W (exon 11), P453R and S505N (exon 9)). In addition, ET patient genes of platelet proliferation (ITGA2B and ITGB3) and DNA methylation profiles can characterize the ET and represent potential novel mechanisms of disease initiation. Thrombosis and myelofibrosis progression are a leading cause of morbidity and mortality in ET. ET is managed with aspirin and selected use of hydroxycarbamide (HC), interferon □ (IFN □), or anagrelide. However, therapy remains suboptimal with the ongoing risk for thrombosis and risk for transformation. An emerging therapy is represented by the JAK inhibitor (ruxolitinib).

Guest Editor

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