

THERAPEUTIC STRATEGIES IN CHRONIC MYELOPROLIFERATIVE SYNDROMES Ph NEGATIVE

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According to World Health Organization classification (WHO) in 2008 in chronic myeloproliferative neoplasms include: polycythemia vera (PV), essential thrombocythemia (ET), primary myelofibrosis (MFP), chronic myelogenous leukemia (CML), chronic neutrophilic leukemia and mastocytosis.

After the discovery of JAK mutation (Janus Kinase) in 2005, JAK 2 mutant myeloproliferative neoplasms were classified into chronic myeloproliferative neoplasms BCR-ABL negative. JAK2V617F mutation - the more frequent abnormality present in PV (96%), PMF (65%) and TE (55%).

Understanding the pathophysiological mechanisms and the crucial role of JAK 2 in the differentiation and proliferation of hematopoietic progenitor cells (erythrocytes, platelets, leukocytes) have opened a new era in the treatment of these hematologic disease.

Classic treatment options for PV, TE and MFP were focused on palliative symptoms and prevent complications intrinsic disease.

With the discovery of the mutation JAK (Janus Kinase) in 2005 and JAK-STAT signaling pathway, it was opened new opportunities for therapeutic interventions, using JAK inhibitors.

Most studies have tracked the response to therapy in patients with MPN JAK2 inhibitors in advanced or progressive disease while their effectiveness in early stage has not yet been investigated.