

3. Presence of *JAK2* V617F mutation or other clonal markers

or

in the absence of a clonal marker, no evidence that the bone marrow fibrosis or other changes are secondary to infection, autoimmune disorder or other chronic inflammatory condition, hairy cell leukemia or other lymphoid neoplasm, metastatic malignancy, or toxic myelopathies¹

Minor PMF criteria

1. Leukoerythroblastosis
2. Increased LDH
3. Anemia
4. Splenomegaly

Diagnosis requires all three major criteria and two minor criteria.

Diagnostic, Prognostic, and Treatment Significance of Molecular Markers in MPNs

The *JAK2* V617F mutation has quickly become an important diagnostic tool for Philadelphia chromosome-negative MPNs. Testing for this mutation has become a routine part of diagnostic testing for patients suspected of having one of these disorders. The most effective testing method is real time polymerase chain reaction (RT-PCR), which assesses the allele load of the mutation and tracks the effectiveness of treatment. While MPNs tend to have an indolent and prolonged course, living with their effects makes it beneficial to find approaches that alleviate the

symptoms and prevent progression to either myelofibrosis or leukemia. Current treatment for Philadelphia chromosome-negative myeloproliferative neoplasms is directed toward preventing thrombosis using low-dose aspirin therapy, cytoreduction using phlebotomy, and cytoreduction using pharmaceuticals such as hydroxyurea or interferons.³ The success of imatinib in the treatment of CML by inhibition of tyrosine kinase has led to attempts to develop drugs that target the *JAK2* mutations. Currently, there are drugs in phase II and III clinical trials for targeted *JAK2* positive mutations.⁶

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ERRATA: In the Spring 2011 Volume 24 of Clinical Laboratory Science the figure on Page 80 should have read “Arrows point to sickle cells. Note NRBC on right as well as Howell-Jolley Body and polychromasia.”