Vitiritti *BMC Geriatrics* 2010, **10**(Suppl 1):L74
http://www.biomedcentral.com/1471-2318/10/S1/L74

BMC Geriatrics

LECTURE PRESENTATION Open Access

Thrombocytosis

M Vitiritti

From de Senectute: Age and Health Forum
Catanzaro, Italy. 5-7 December 2009

Background

Thrombocytosis is defined as an increase in the number of circulating platelets than the normal value of between 150,000 and 250,000 platelets/uL. The limit beyond which the state of platelets is usually marked in platelets is defined as 400,000/uL.

Trombocytosis are divided into primary or autonomous, in the course of myeloproliferative disorders (essential thrombocythemia, polycythemia vera, chronic myelogenous leukemia) and secondary or reactive. The primitive forms having a common origin as stem cells of the bone marrow, and, although each has specific characteristics, often the clinical picture overlaps, making it difficult to secure a diagnosis between these four diseases. Indeed, essentially thrombocythemia is always an increase in platelets, but this can also occur in the other three diseases, also in idiopathic thrombocythemia, sooner or later, during the course of the disease, you may have increased white blood cells and, very rarely, even red blood cells. The duration of secondary thrombocytosis is variable and tied to the possibility of removing the underlying disease. The diagnosis is based on the finding, the examination emocromocitimetrico, a higher platelet rate than 400,000 items for UL with volume, morphology and platelet function is normal, and the search for possible pathological conditions that led the platelets. Mieloroliferative diseases should be excluded from the platelets, including essential thrombocythemia, which in addition to specific hematologic abnormalities, have higher rates with strong anisomacrocitosi platelet-platelet, and more frequent bleeding or thrombotic. Several studies have shown that in most patients with polycythemia vera and in about half of those with essential thrombocythemia or idiopathic myelofibrosis, it is a single nucleotide mutation that activates JAK2.

Conclusions

JAK2 mutations appear to confer hypersensitivity to hematopoietic growth factors and a selective advantage for growth marrow precursors carrying the mutated gene compared with normal or wild type precursors. Recently it was discovered a point mutation dependent kinase JAK2, which involves replacing a thymine with guanine nucleotide sequence, resulting in the appearance of a valine residue instead of phenylalanine in position 617 of the amino acid sequence or JAK2617V> F. This mutation appears to be myeloid-specific and can be used for the differential diagnosis between primitive myeloproliferative disorders and secondary forms of poliglobulia or thrombocytosis.

Published: 19 May 2010

Reference

1. Baxter EJ, Scott LM, Campbell PJ, East C, Fourouclas N, Swanton S, *et al*: Acquired mutation of the tyrosine kinase JAK2 in human myeloproliferative disorders. *Lancet* 2005, **365**:1054-61.

doi:10.1186/1471-2318-10-S1-L74
Cite this article as: Vitiritti: Thrombocytosis. *BMC Geriatrics* 2010 **10**(Suppl 1):L74.

Genetics Laboratory Ospedale "Mariano Santo" Cosenza, Italy