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Original Article

GRANULOCYTIC EXPRESSION OF CD11B/CD18 AND THROMBOTIC RISK IN PATIENTS WITH MYELOPROLIFERATIVE NEOPLASMS

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Summary

Myeloproliferative neoplasms (MPN) are clonal hematological conditions characterized by excessive production of one or more cell lines in the bone marrow. The blood cells produced are often hyperactive in their functions, which could lead to complications in the disorder's clinical course. We aimed to define the role of granulocytic CD11b/CD18 expression for the thrombotic risk in MPN patients. We investigated 110 patients with a histologically confirmed diagnosis of a myeloproliferative disease and a control group of 46 healthy volunteers. In the patient group, we found an average expression 4.59 times higher than in the control group. The highest expression was found in a subgroup of patients with polycythemia vera – 71.55% of the patients' neutrophils. In each subgroup with essential thrombocythemia, myelofibrosis, and chronic myeloid leukemia, the patients with a history of thrombotic complication had a higher expression than the patients without such complications.

Keywords: CD11b/CD18 granulocytic expression, myeloproliferative neoplasms, thrombotic complications

Introduction

Myeloproliferative neoplasms (MPN) are hematological diseases with clonal hematopoiesis and increased cell line proliferation. Classical MPNs include chronic myeloid leukemia (CML), polycythemia vera (PV), essential thrombocythemia (ET), and myelofibrosis (MF). These can be divided into two main groups, depending on the presence of Philadelphia chromosome t(9;22)(q34;q11) – Philadelphia-positive CML, and the rest of the Philadelphia-negative diseases. A point mutation V617F (G -> T, leading to dispositioning of phenylalanine with valine on the 617 position) of the JAK2 gene (encoding Janus kinase) is typical for the second group. Its prevalence is about 95% in PV and about 50-60% in ET and MF [1-9]. Mutation can be found in hematopoietic cells, leading to excessive cell proliferation and survival benefit by activating Janus kinase and increasing signal transduction in the JAK-STAT signal pathway [8]. Often, MPNs are associated with a higher thrombotic risk with multifactorial genesis: patient-related factors, genetic factors, abnormal coagulation, blood cell variations, and