Mashhad University of Medical Sciences (MUMS)

Reviews in Clinical Medicine

Clinical Research Development Center Ghaem Hospital

Nucleophosmin 1 expression in acute myeloid leukemia

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ARTICLE INFO

Article type
Review article

Article history
Received: 6 Dec 2014
Revised: 22 Jan 2015
Accepted: 1 Feb 2015

Keywords
Acute myeloid leukemia
Immunohistochemistary
Nucleophosmin1 mutation

ABSTRACT

Nucleophosmin1 is a multifunctional protein that shuttles between nucleus and cytoplasm in some subtypes of acute myeloid leukemias. Mutated Nucleophosmin1 expresses aberrantly in the cytoplasm of the cell and transports from nucleolus to the cytoplasm. It is diagnosed by immunohistochemical techniques, flow cytometry assay and mutational analysis.

The aim of this study is to evaluate the effects of Nucleophosmin1 mutation on the clinical presentations, prognosis, diagnosis and the treatment of acute myeloid leukemia. Thirteen articles were extracted from PubMed, Google scholar and Scopus in which the Nucleophosmin1 mutation correlated with gingival hyperplasia, high white blood cell count, lymphadenopathy, high platelet count and other signs and symptoms of myelomonocytic and monocytic acute myeloid leukemias.

This mutation is a provisional entity in the classification of acute myeloid leukemia, which influences on the prognosis, clinical course and the treatment of some subtypes of acute myeloid leukemias. Nucleophosmin1 mutation has favorable prognostic value in the absence of other concomitant mutations.

Please cite this paper as:
Davoudi M, Davoudi P. Nucleophosmin 1 expression in acute myeloid leukemia . Rev Clin Med. 2015;2(4):209-211.

Introduction

Nucleophosmin 1 (NPM1) is a multifunctional phosphoprotein that contains several functional domains, which the molecule binds partners in different cellular compartments. NPM1 is located in nucleus and constantly shuttles between the nucleus and the cytoplasm (1). NPM1 gene corporates in leukemogenesis as a mutant protein with oncogenic effect, through loss of one functional allele. This mutation translocates the product from nucleus to the cytoplasm of blasts, alters through loss or gain of functions on different proteins (1). NPM1 leukemogenesis is a result of two major mechanisms including the production of a mutated protein and the decreasing level of wild type protein expression. NPM1 gene alterations generates a mutated protein that is associated with hematopoietic malignancies in the case of acute myeloid leukemia (AML) with NPM-cytoplasmic positive (NPMc+) mutant. This loss of one functional allele is stable in AML and NPM1 mutation, represents a founder genetic lesion. Myeloid related protein 8 is the promoter of NPMc+, and controls the overexpression of NPM1c+ in the monocytic progenitors, myeloid progenitors, and mature granulocytes (1). NPM1 affects ribosomal protein transportation and assembly, and prevents protein aggregation in the nucleolus (2). NPM1 binds to alternate-reading-frame protein (ARF) and is used for p53-independent cell cycle regulation; via cyclin E/cyclin dependent kinase 2 phosphorylation. NPM1 initiates

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