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Phosphorylated H2AX (Gamma-H2AX), Activated Natural Killer (NK) Cells and Poly ADP-Ribose Polymerase (PARP) in Acute Leukemia

Yetti Hernaningsih[1*], Diane Lukito Setiawan[2] and Ami Ashariati[3]

*[*1]Department of Clinical Pathology, Faculty of Medicine, Universitas Airlangga, Dr. Soetomo General Academic Hospital, Surabaya, East Java-60285, Indonesia*

[2]Faculty of Medicine, Universitas Surabaya, Surabaya, East Java-60292, Indonesia

[3]Department of Internal Medicine, Faculty of Medicine Universitas Airlangga, Dr. Soetomo General Academic Hospital, Surabaya, East Java-60285, Indonesia.

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ABSTRACT

Molecule of H2AX is a member of H2A histone which plays a role in chromatin formation. H2AX will be phosphorylated at the serine-139 as a response of DNA damage.γH2AX itself will induce apoptosis through 2 mechanisms, i.e., induce p53 suppressor gene and activation of natural killer (NK) cells. Activated NK cells will release perforin and granzyme B then lead to apoptosis of cell target. Meanwhile, the result of apoptosis is breakdown of DNA repair enzyme, including poly ADP-ribose polymerase (PARP). The aim of this study is to investigate the role of γH2AX and activated NK cells to PARP as a product of apoptosis in adult acute leukemia.

This study was conducted on 21 adult patients with diagnosis of acute leukemia in Dr. Soetomo General Academic Hospital in Surabaya, Indonesia. Bone marrow aspirate and peripheral blood were collected at diagnosis. Peripheral bloods from 10 healthy donors were used as a control group. Phosphorylated H2AX and 89 kDa fragment of PARP were tested from peripheral blood mononuclear cells (PBMC) specimens. Activated NK cells were determined using antibody of CD56 FITC/CD69PE/CD45PerCP from whole blood-EDTA specimens. All of these tests were performed by flow cytometry. Statistical analysis was used independent sample t-test and linear regression analysis

The level of γH2AX, activated NK cells and PARP of leukemic patients revealed significant higher than control with p=0.013, p=0.000 and p=0.000, respectively. The γH2AX and activated NK cell did not have an influence on PARP with p=0.591 and p=0.181, respectively.

In conclusion, γH2AX, activated NK cells and PARP increase in leukemic patients, however H2AX and activated NK cells independently do not cause increasing of PARP as an apoptosis product.

Keywords: γH2AX, Activated NK, PARP, Leukemia

Corresponding author: Yetti Hernaningsih, Department of Clinical Pathology, Faculty of Medicine, Universitas Airlangga, Dr. Soetomo General Academic Hospital, Surabaya, East Java-60285, Indonesia, E-mail: yettihernaningsih@gmail.com

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