Effect of Renmindevir on the Development of Oxidative Stress in HBL-100 Cells

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Abstract

The effect of Renmindevir on oxidative stress in HBL-100 cells was assessed using lipid peroxidation markers. Unlike cisplatin, Renmindevir did not induce oxidative stress, decreasing malondialdehyde levels and causing only minor changes in diene conjugates, suggesting a non-oxidative mechanism of cytotoxicity.

Keywords: Renmindevir; oxidative stress; lipid peroxidation; malondialdehyde; diene conjugates; cytotoxicity; HBL-100 cells.

Oxidative stress represents one of the key mechanisms of cellular damage and is considered an important component of the cytotoxic action of several anticancer drugs, including doxorubicin, cisplatin, and others [1]. Excessive generation of reactive oxygen species (ROS) and activation of lipid peroxidation (LPO) processes lead to disruption of cellular membrane integrity, damage to macromolecules, and the initiation of programmed cell death [2]. Cytotoxic activity that occurs independently of oxidative stress indicates the involvement of a specific molecular mechanism and reduces the likelihood of artifactual effects associated with ROS generation.

The aim of the present study was to evaluate the induction of oxidative stress by the antiviral drug Renmindevir in human breast adenocarcinoma HBL-100 cells. The intensity of lipid peroxidation was assessed by measuring the levels of primary (diene conjugates, DC) and secondary (malondialdehyde, MDA) LPO products. Cells were incubated with the tested compounds for 2 h at a concentration of 70 μg/mL, which resulted in 100% cell death after 24 h.

Treatment with cisplatin led to a 10.5% increase in MDA levels compared with the control and a 12% increase in diene conjugates, confirming the pronounced pro-oxidant nature of its cytotoxic effect. In contrast, dihydroquercetin reduced MDA levels to 91.6% of the control value (an 8.4% decrease) and did not affect diene conjugate levels, consistent with its known antioxidant properties. Under the same experimental conditions, Renmindevir did not induce oxidative stress: MDA levels decreased to 63% of the control (a 37% reduction), while the content of diene conjugates increased only slightly, by 6.3%.

Thus, the cytotoxic effect of Renmindevir in HBL-100 cells is not accompanied by oxidative stress or activation of lipid peroxidation processes. The absence of a pronounced increase in both primary and secondary LPO products suggests that Renmindevir–induced cell death is likely mediated by an alternative molecular mechanism.

Recent References

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