Oxidative stress as an underling mechanism of anticancer drugs cytotoxicity on human red blood cells' membrane

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Description:

The aim of this study is to investigate the direct *in vitro*effects of anticancer drugs on red blood cells (RBCs) and to explore the underlying mechanism, mainly by measuring RBCs oxidative stress (OS) status. After RBCs direct contact with fourteen (14) anticancer drugs, several parameters were assessed including: cellular turbidity, methemoglobin (metHb) generation, released Hb and Hb stability. Moreover, intracellular Hb, considered as new molecular target of anticancer drugs, was quantified inside RBCs. MDA level, the main biomarker of OS, was simultaneously measured. The cellular turbidity reveled severe (docetaxel “TXT”, 0.03 ± 0.002), moderate (methotrexate “MTX”, 0.49 ± 0.009), or none (5-fluorouracil “5-FU”, 0.76 ± 0.029) membrane cytotoxicity (MC). An inverse relationship between cell concentration, released Hb and metHb content was obtained. High metHb generation, revealing intense OS, was also mostly expressed in paclitaxel “TXL” and etoposide “VP16”. Further, epirubicin “EPI” and “TXT” induced important oxidation of membrane lipids with 0.32 ± 0.014 and 0.26 ± 0.004, respectively. Also, MTX (0.17 ± 0.006) and doxorubicin “DOX” (0.32 ± 0.034) affected significantly Hb stability by a direct contact with molecule. These findings demonstrated that anticancer drugs have the ability to induce membrane damages by the exacerbation of OS through membrane lipid peroxidation and Hb oxidation even inside RBCs.

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