Cloroquina cessa o ciclo celular em G2/M, aumenta a apoptose e altera o citoesqueleto sendo eficaz no câncer de mama

Cell growth inhibition, G2/M cell cycle arrest, and apoptosis induced by chloroquine in human breast cancer cell line Bcap-37.

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Abstract

Chloroquine is an antimalarial drug that has been used in the treatment and prophylaxis of malaria since the 1950s. The present study was undertaken to examine the effects of chloroquine on Bcap-37 human breast cancer cells' growth, cell cycle modulation, apoptosis induction, and associated molecular alterations in vitro. The chloroquine treatment decreased the viability of Bcap-37 cells in a concentration- and time-dependent manner, which correlated with G(2)/M phase cell cycle arrest. The chloroquine-mediated cell cycle arrest was associated with a decrease in protein levels/activity of polo-like kinase 1 (Plk1), phosphorylated cell division cycle 25C (Cdc25C), phosphorylated extracellular signal-regulated kinase 1/2 (ERK1/2), phosphorylated Akt. The chloroquine-treated Bcap-37 cells exhibited a marked decrease in the level of mitochondrial transmembrane potential (DeltaPsi_m), which was accompanied by the activation of caspase-3 and cleaved poly(ADP-ribose) polymerase (PARP). Exposure of Bcap-37 cells to chloroquine also resulted in the induction of spindle abnormalities. In conclusion, the findings in this study suggested that chloroquine might have potential anticancer efficacy, which could be attributed, in part, to its proliferation inhibition and apoptosis induction of cancer cells through modulation of apoptosis and cell cycle-related proteins expressions, down-regulation of mitochondrial transmembrane potential (DeltaPsi_m), and induction of spindle abnormalities.

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