MORPHOLOGICAL AND BIOCHEMICAL CHANGES INDUCED BY ACLARUBICIN IN BREAST CANCER MCF-7 CELLS

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Breast cancer, the most common malignancy among women is moderately sensitive to several cytotoxic agents. It has been demonstrated that development of multidrug resistance to the first anthracyclines doxorubicin and daunorubicin limits the success of chemotherapy in this disease. The present study was designed to assess the ability of a new anthracycline derivative, aclarubicin (ACL) to induce apoptosis in MCF-7 human breast cancer cells. Aclarubicin is a trisaccharide anthracycline drug active against a wide variety of solid tumors and hematological malignancies. This drug displays different toxic properties from the classical monosaccharide anthracyclines. ACL is less cardiotoxic and to act as catalytic inhibitor of topoisomerase II and I. The MCF-7 breast cancer cell line was cultured in Dulbecco's Modified Eagle's Medium (DMEM). The sensitivity of the cells to ACL was measured by trypan blue dye exclusion and by 3-(4,5-dimethyl-2-thiazolyl)-2,5-diphenyl-2H-tetrazolium bromide (MTT assay). Apoptosis was detected in cells using terminal deoxynucleotidyl transferase-mediated nick-end labeling (TUNEL) assay and by measuring the activation of caspase-3. The results demonstrate that aclarubicin is more cytotoxic to MCF-7 cells than doxorubicin. Aclarubicin induced both apoptosis and necrosis in cultured breast cancer cells. ACL-induced changes were dependent on the drug concentration and time of incubation.