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[The effect of insulin on chemotherapeutic drug sensitivity in human esophageal and lung cancer cells]

[Article in Chinese]

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OBJECTIVE: To discuss the effect of insulin, as a metabolic promoter, on chemotherapeutic drug sensitivity in human esophageal and lung cancer cells. METHODS: Human esophageal cancer cells NEC and human lung adenocarcinoma cells GLC were cultured and then inoculated into the wells. MTT was added. Chemotherapeutic drugs, etopside, cisplatin, or 5-fluotouracilum was added to examine their cytotoxity activity. Then insulin of the final concentration of 5 mU/ml was added 8 hours before etopside (30 - 40 micro g/ml), cisplatin (2.5 micro g/ml), or 5-Fu (50 micro g/ml) was added. MTT A value was tested by colorimetry to evaluate the number of cancer cells, cell activity, and metabolism status so as to reflect the cytotoxity of the anti-tumor agent. Insulin was added into the suspension of cancer cells. Flow cytometry was used to detect the cell-cycle progresses. RESULTS: Insulin alone did not inhibit the cell growth and mildly promoted the cell metabolism with the concentration > 5 mU/ml. Insulin (2.0 - 15.0 mU/ml) enhanced the chemocytotoxity of etopside (30 micro g/ml) on human esophageal and lung cancer cells as indicated by MTT colorimetry. GLC cell cycle assay showed that the S phase block induced by etopside, cisplatin and 5-FU and the G(2)/M block induced by 5-FU were enhanced by insulin with the increased block rates of 80% and 90% respectively. The increased block rate induced by insulin in NEC cells was lower than in GLC cells. CONCLUSION: A reversible metabolic promoter, insulin enhances the cytotoxity of the chemotherapeutic agents. It is possible to increase the growth and metabolism of cancer cells first so as to enhance the chemosensibility, and then administer chemotherapeutic agents, thus improving their therapeutic effects.

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