IODINE 131 INDUCED MULTINUCLEATION OF PAPILLARY THYROID CARCINOMA BCPAP CELLS

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Background/Purpose: Radioiodine 131 therapy is commonly used to treat thyroid cancer exhibiting high ^{131}I uptake, but the cellular and molecular mechanisms among radiation-induced-apoptosis are not fully understood.
Methods: Cultured BCPAP cells grown to confluence were treated with ^{131}I. Cellular growth was measured by MTT. During exposure to ^{131}I, the morphological changes were observed daily with or without Hochest33342 staining. Cell cycle analysis was performed by FCM using PI staining.
Results: According to MTT results, BCPAP cells treated with ^{131}I at 5- to 30-μCi doses for 72 hours showed similar death rates as the control group, but doses of more than 30 μCi showed toxicity. With the extension of ^{131}I exposure time, morphologic modifications appeared, similar to EMT-like changes, including enlargement of cell volume, spindle-shaped morphology, appearance of pseudopodia, and reduced adhesion. One week later, multinucleated cells were found. After another three or four weeks, multinucleated cells accounted for approximately 50% of all cells, some of these cells with more than 20 nuclei. Then the multinucleated cells exhibited characteristic features of senescence and apoptosis, the multinucleated cell proportion gradually reduced, and disappeared about 8 weeks later. Cell cycle analysis indicated that ^{131}I induced apparent octaploidy-like peak besides diploidy and tetraploidy. In addition, other thyroid carcinoma cell lines, including K1, FTC-133 and 8505C, also appeared enlargement volume and spindle-shaped morphology but not multinucleation occurrence during exposure to ^{131}I.
Discussion & Conclusion: Multinucleation of BCPAP cells is induced by exposure to ^{131}I. This aberrant mitotic arrest may lead to abnormal cell proliferation and apoptosis.