

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 ¹³¹I 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 ¹³¹I. Cellular growth was measured by MTT. During exposure to ¹³¹I, the morphological changes were observed daily with or without Hoechst33342 staining. Cell cycle analysis was performed by FCM using PI staining.

Results: According to MTT results, BCPAP cells treated with ¹³¹I at 5- to 30- μ Ci doses for 72 hours showed similar death rates as the control group, but doses of more than 30 μ Ci showed toxicity. With the extension of ¹³¹I 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 ¹³¹I 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 ¹³¹I.

Discussion & Conclusion: Multinucleation of BCPAP cells is induced by exposure to ¹³¹I. This aberrant mitotic arrest may lead to abnormal cell proliferation and apoptosis.