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Observation on histological changes and expression of proliferative cell nuclear antigen and oncogene p53 in patients with lung carcinoma after radiotherapy or chemotherapy

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Abstract Alterations of proliferative cell nuclear antigen (PCNA) and cancer-inhibitory gene p53 were determined in 37 patients with lung carcinoma after radiotherapy or chemotherapy with ABC immunohistochemical staining. Some lung tissues were observed with electron microscopy. The patients before therapy were observed likewise to serve as the control. It was found that: (1) The cytoplasm and nuclei of the cancerous tissue were destroyed to various degrees by radiotherapy or chemotherapy; (2) PCNA was significantly decreased after treatment ($P < 0.01$ or $P < 0.05$); (3) The expression rate of p53 in cancerous tissue was decreased after radiotherapy and slightly changed after chemotherapy.

Key words lung carcinoma; proliferative cell nuclear antigen; p53; radiotherapy; chemotherapy

IGF-Ⅱ受体(IGF-Ⅱ R)反义基因对肝癌细胞癌基因转录及生物学行为的影响

Effect on oncogenes transcription and biological behavior of human hepatoma cells by IGF-Ⅱ receptor antisense gene transfection

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IGF-Ⅱ和 IGF-Ⅱ R过量表达是肝癌细胞自分泌和/或邻分泌系统的分子基础,自分泌生长刺激机理是肝癌细胞恶性增殖的重要原因。应用反义技术的原理,构建 IGF-Ⅱ R反义基因真核表达系统,经磷酸钙-DNA共沉淀的方法,导入 SMMC-7721人肝癌细胞株,有效地抑制了7721细胞内源性 IGF-Ⅱ R的表达,阻断 IGF-Ⅱ→IGF-Ⅱ R的自泌、邻泌作用途径。结果发现 c-myc基因 mRNA转录发生一定程度下调,而 IGF-Ⅰ R基因 mRNA转录明显上调。p53 TTR N-ras基因转录无明显变化。IGF-Ⅱ R反义基因抑制细胞转染克隆的形成,但转染细胞生长速度无明显变化。同时,这些细胞进入 S期比例及细胞凋亡发生率均明显增多,细胞不能在软琼脂培养基中生长,在裸鼠体内的致瘤能力降低。而 IGF-Ⅱ R正义基因及 pCMVneo空载体转染细胞对照组无上述改变。本研究说明: c-myc转录下调可能促进肝癌细胞的分化,而 IGF-Ⅰ R转录上调可能为代偿机理或为 IGF-Ⅱ R反义基因对其的激活作用所致。IGF-Ⅱ R反义基因可能抑制肝癌细胞的生长,但当 IGF-Ⅰ R表达增加时可产生对抗作用,从而使这些细胞生长不表现明显的抑制,同时可能促进细胞由 $G_1 \rightarrow S$期。IGF-Ⅱ R反义基因还可诱导肝癌细胞发生凋亡,一定程度地逆转其恶性表型。我们的研究结果表明,采用反义技术干预肝癌细胞生长因子受体基因表达可能给肝癌基因治疗提供新的途径,有进一步探索的价值。

关键词 IGF-Ⅱ R;反义基因;肝癌
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