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临床研究

非小细胞肺癌人群中c-MET基因的扩增检测

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Detection of c-MET Gene Amplification in Non-small Cell Lung Cancer

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摘要 目的：c-MET基因扩增是非小细胞肺癌对EGFR TKIs（吉非替尼或厄罗替尼）产生耐药的主要机制之一。本研究探讨没有接受TKIs治疗与TKIs治疗后耐药的NSCLC中c-MET基因的扩增是否存在差异。方法:获得55例术后非小细胞肺癌（NSCLC）的肿瘤组织（基线组）以及23例对TKIs耐药的肿瘤组织（耐药组）后，通过激光显微切割筛选癌细胞后提取基因组DNA，实时荧光定量PCR TaqMan探针法检测所有标本的c-MET基因的拷贝数。 结果:1.基线组和耐药组的临床病理特征均与c-MET基因的扩增无关。2.基线组中c-MET基因扩增阳性率为5.5% (3/55)；耐药组的c-MET基因扩增阳性率为21.7% (5/23)。两组之间有统计学差异（Fisher精确概率法，P=0.045）。3.在7例获得TKI治疗前后肿瘤组织的NSCLC中，TKI治疗前没有出现c-MET的基因扩增，TKI治疗后有2例患者出现了c-MET的基因扩增（2/7）。TKI治疗前后的c-MET基因扩增差异无统计学意义。结论:NSCLC的临床病理特征不能预测c-MET基因扩增；在没有接受EGFR TKIs治疗的NSCLC中，c-MET基因扩增仅为少见事件。但经过吉非替尼或厄罗替尼治疗后出现耐药情况NSCLC中，部分患者的c-MET基因出现扩增。

关键词： NSCLC c-MET 基因扩增 耐药

Abstract： Objective：c-MET gene amplification was regarded as one of the main resistance mechanisms in non-small-cell lung cancer (NSCLC) treated with tyrosine kinase inhibitors (TKIs, gefitinib and erlotinib).The study was to evaluate the difference of c-MET gene amplification between TKI-na ve NSCLC cohort and patients resistant to EGFR TKIs. Methods：The baseline group included 55 TKI-na ve NSCLC patients and their tumor specimens were obtained via the lung resection.The resistant group included 23 TKIs resistant patients after first-line TKIs monotherapy.Five tumor samples were collected from the primary cancer via the resection, and nine samples were from metastatic sites via the resection, and the rest were obtained by core needle biopsy of the primary tumors.All the tumor tissues were stored at -80℃ for use.Genomic DNA was extracted from tumor tissues after selection by laser microdissection.Copy numbers of c-MET gene were assessed by quantitative real-time PCR.Results：c-MET gene amplification was not related to clinicopathologic characteristics both in the baseline group and the resistant group.The prevalence of c-MET amplification in baseline group was 5.5% (3/55), which was lower than the rate in TKIs resistant group (P=0.045, Fisher s exact test).In seven pairs of the tumor samples before and after TKIs treatment, c-MET gene was not amplified in pre-treatment samples, whereas the c-MET gene amplification was found in two cases after TKIs treatment.Conclusion：

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Clinicopathologic characteristics may not predict c-MET amplification.In the TKI-na ve NSCLC patients, c-MET amplification is accidental.But thegene is amplified in part of NSCLC patients when treated with EGFR TKIs.

Key words： NSCLC c-MET Gene amplification Resistance

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没有本文参考文献

[1] 王耕;黄韬;薛家鹏;王明华;惠震 . 三羟异黄酮对人乳腺癌MCF-7/ADM细胞体外抑瘤效应、细胞周期及凋亡的影响 [J]. 肿瘤防治研究, 2011, 38(8): 886-890.

[2] 许淑茹;马军;袁志刚;黄勇奇;苏上贵;胡启平 . 蛇毒精氨酸酯酶Agkihpin对人鼻咽癌CNE-2细胞系MRP1表达的影响 [J]. 肿瘤防治研究, 2011, 38(7): 731-735.

[3] 贾文韫;李宝兰;岳文涛. c-Met/ErbB3/PI3K信号通路在肺癌EGFR-TKI获得性耐药中的研究进展 [J]. 肿瘤防治研究, 2011, 38(6): 721-723.

[4] 陶丹;龙志雄;王斌;刘艳屏;高霞;黄娅丽;吴晶;黄慧;万欢 . 直线加速器联合γ刀放疗同步DP方案化疗治疗局部晚期非小细胞肺癌的临床研究[J]. 肿瘤防治研究, 2011, 38(5): 575-578.

[5] 张雪玉;张咏梅;杨彩虹;刘莉莉;吴蔚 . HGF、c-Met和VEGF-C在宫颈癌中的表达[J]. 肿瘤防治研究, 2011, 38(4): 411-415.

[6] 马向涛. STAT3信号转导通路与肿瘤耐药研究进展 [J]. 肿瘤防治研究, 2011, 38(4): 474-475.

[7] 肖玉洁;王红梅;韩正祥;高向阳;裴冬生;曾令宇;杜秀平. 靶向stathmin和mdr1基因逆转卵巢癌细胞 紫杉醇耐药的研究[J]. 肿瘤防治研究, 2011, 38(3): 243-246.

[8] 陈漫霞;姚振江;陈思东;王漫云;许雅;蔡旭玲 . 原发性肝细胞癌中P-gp、TopoⅡα和P53的 表达及意义 [J]. 肿瘤防治研究, 2011, 38(3): 278-280.

[9] 李焘综述;王雄伟审校 . EGFR及其下游通路在胶质瘤治疗中的研究进展[J]. 肿瘤防治研究, 2011, 38(10): 1204-1206.

[10] 李伟忠;王晓燕;霍秋菊. 环氧合酶-2抑制剂对人舌鳞癌Tca8113/BLM 细胞MDR1/P-gp表达的影响[J]. 肿瘤防治研究, 2011, 38(1): 9-12.

[11] 魏 玲;宋现让;孙菊杰;王兴武;宋 宝;郑 燕. MDR1基因下调逆转人白血病阿霉素耐药细胞株K562/ADM的耐药性[J]. 肿瘤防治研究, 2010, 37(5): 515-518.

[12] 卢红;樊青霞;王瑞林. 人食管鳞癌紫杉醇耐药细胞株Ec9706/P-1 凋亡的变化[J]. 肿瘤防治研究, 2010, 37(12): 1356-1359.

[13] 万光升;孙 珏;李 琦. 中医药逆转大肠癌耐药的实验研究进展[J]. 肿瘤防治研究, 2010, 37(08): 959-962.

[14] 赵珍珍;罗 庆;宿玉玺;郑改焕;刘 伟;金先庆. Ad-EGFP-MDR1转染对小鼠骨髓细胞的影响[J]. 肿瘤防治研究, 2010, 37(08): 855-858.

[15] 杜 芹;魏 玲;姜 超;杨锡贵. 人恶性淋巴瘤细胞Raji长春新碱耐药株Raji/VCR的建立及其生物学特性[J]. 肿瘤防治研究, 2010, 37(07): 751-753.