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Gene Polymorphisms and Chemotherapy in Non-small Cell Lung Cancer

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摘要

The phamacogenetics is being used to predict whether the selected chemotherapy will be really effective and tolerable to the patient. Irinotecan, oxidized by CYP3A4 to produce inactive compounds, is used for treatment of various cancers including advanced non small cell lung cancer (NSCLC) patients. CYP3A4*16B polymorphism was associated with decreased metabolism of irrinotecan. Irinotecan is also metabolized by carboxylesterase to its principal active metabolite, SN-38, which is subsequently glucuronidated by UGT1As to form the inactive compound SN-38G. UGT1A1*28 and UGT1A1*6 polymorphisms were useful for predicting severe toxicity with NSCLC patients treated with irinotecan-based chemotherapy. Platinum-based compounds (cisplatin, carboplatin) are being used in combination with new cytotoxic drugs such as gemcitabine, paclitaxel, docetaxel, or vinorelbine in the treatment of advanced NSCLC. Cisplatin activity is mediated through the formation of cisplatin-DNA adducts. Gene polymorphisms of DNA repair factors are therefore obvious candidates for determinants of repair capacity and chemotherapy efficacy. ERCC1, XRCC1 and XRCC3 gene polymorphisms were a useful marker for predicting better survival in advanced NSCLC patients treated with platinum-based chemotherapy. XPA and XPD polymorphisms significantly increased response to platinum-based chemotherapy. These DNA repair gene polymorphisms were useful as a predictor of clinical outcome to the platinum-based chemotherapy. EGFR kinase inhibitors induce dramatic clinical responses in NSCLC patients with advanced disease. EGFR gene polymorphism in intron 1 contains a polymorphic single sequence dinucleotide repeat (CA-SSR) showed a statistically significant correlation with the gefitinib response and was appeared to be a useful predictive marker of the development of clinical outcome containing skin rashes with gefitinib treatment. The other polymorphisms of EGFR were also associated with increased EGFR promoter activity. EGFR gene mutations and polymorphisms were also associated with EGFR kinase inhibitors response and toxicity.

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