7. Kongres Hrvatskog torakalnog društva

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RESPONSE TO THIRD-LINE ERLOTINIB IN AN EGFR MUTATION-**NEGATIVE PATIENT WITH NON-SMALL-CELL LUNG CANCER**

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Introduction

Lung cancer is the leading cause of death among patients with malignant cancer. About 80% of lung cancer has characteristics of NSCLC in whose group we include lung adenocarcinoma , which is present acc 40 % with an average 5-year survival of about 18%. Cytostatic effects of standard chemotherapy regimens treatment of NSCLC in recent years have reached a plateau, but the discovery of new drugs non-cyostatic as "targeted" therapies, brought new improvements to the quality of life and survival of these patients. Target, molecular, therapy directed at specific molecules in inhibiting tumor growth and progression. One example of this therapy is erlotinb which is a tyrosine kinase receptor for epidermal growth factor, and indicated for the treatment of local advanced or metastatic non-small cell lung.

Methods and results

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We present a 71 year old female patient, active smoker, whose symptomes were mild dyspnea and cough which lasted over a period of 2-3 months. The patient was diagnosed adenocarcinoma of right upper lobe with metastatis of mediastinum, EGFR negative, ALK negative, III B stage disease. Given that it was inoperable process, systemic concomitant chemoradiotherapy has been indicated. The first-line chemotherapy in combination pacitaksel and platinum has been conducted. After first cycle od chemotherapy and begginig od radiotherapy controled diagnostic investigation has shown progression of the tumor in the right lower lobe and propagation in mediastinal lymph nodes. Radiotherapy of the primary tumor and mediastinum and then the second line with pemetreksed and carboplatinum (IV cycles) have been conducted. During the regular control diagnostic treatment shown further progressive dynamics of the primary process and therapy with erlotiniom in the third line has been started. Reevaluation after two cycles showen sever hepatic leasion and acute renal insufficiency dute to hepatorenal syndrome probably caused by erlotinib. Therapy has been stopped for 4 months in which period pharmacological testing has been conducted and it has been indicated to continue with half of the dose with erlotinib. Reevaluation of the disease after 37 cycles, including PET/CT shows primary tm without metabolic activity and without metabolic activity in the mediastinal lymph nodes.

Conclusion

Application of tyrosine kinase inhibitors of epidermal growth factor in the multimodal treatment of locally advanced lung adenocarcinoma has shown good response to therapy with regression to perhapse chronic stable disease.