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KRAS and EGFR mutations coexisting in lung adenocarcinoma

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Lung adenocarcinoma represents about 42% and 28% of NSCLC in women and men. Adenocarcinomas incidence is still rising being the most frequent type of NSCLC diagnosed in USA. Both *EGFR* and *KRAS* gene mutations can contribute to the development of NSCLC, namely adenocarcinomas. *EGFR* and *KRAS* mutations are considered by some authors as mutually exclusive explained by the fact that KRAS-MAPK pathway is one of the downstream signalling pathways of EGFR. Lung cancers with *KRAS* mutations are resistant to EGFR tyrosine kinase inhibitors.

Sections of the adenocarcinoma of the lung, formalin-fixed paraffin-embedded tissues (FFPE), were selected to analyze mutations in *EGFR* exons 19 and 21 and *KRAS* - codons 12 and 13 by DNA extraction for polymerase chain reaction (PCR). Exon 19 was studied by fragment analysis and exon 21, codons 12 and 13 were studied by direct sequencing. The analysis of FISH results was done by Cappuzzo's score to *EGFR* gene. Determination of EGFR protein expression was done by immunohistochemistry (IHC) (Zymed Laboratories).

The authors present a rare case with synchronous *EGFR* and *KRAS* mutations. The patient is a 77 years old, male with a central 3cm mixed adenocarcinoma. The tumor showed EGFR protein overexpression identified by IHC and chromosome 7 high polyssomy by FISH.

The authors call attention to the fact that although *EGFR* and *KRAS* mutations are almost always mutually exclusive in some cases they may coexist in the same neoplasia.

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