Epidermal growth factor receptor mutation

- Colorectal Cancer with Ovarian Metastasis After Panitumumab and FOLFOX4: A Case Report
- Endocrine manifestations of lung adenocarcinoma with epidermal growth factor receptor mutation mimicking tuberculosis: A case report and literature review
- Targeting both wild-type EGFR and its drug-resistant mutants with erlotinib-aptamer conjugates
- Diagnostic accuracy of the Idylla mutation test for detecting EGFR mutations in non-small cell lung cancer: a meta-analysis
- Prevalence of osimertinib-induced cardiotoxicity in non-small cell lung cancer patients: a systematic review and meta-analysis
- Comprehensive genomic profiling by liquid biopsy in refractory metastatic colorectal cancer patients who are candidate for anti-EGFR rechallenge therapy: findings from the CAVE-2 GOIM trial
- Design, synthesis, and evaluation of 1,3,4-oxadiazole-based EGFR inhibitors
- Traditional Chinese Medicines as Anticancer Agents for Non-Small Cell Lung Cancer with EGFR Mutations: A Review

Mutations affecting EGFR expression or activity could result in cancer.

CSF and plasma cell-free DNA (cfDNA) can be retrieved for Epidermal growth factor receptor mutation testing.

Epidermal growth factor and its receptor was discovered by Stanley Cohen of Vanderbilt University. Cohen shared the 1986 Nobel Prize in Medicine with Rita Levi-Montalcini for their discovery of growth factors.

EGFR mutations, primary cancer pathology, and Recursive partitioning analysis class may be proposed as prognostic factors for intracranial progression-free survival (PFS) in non-Small-cell lung cancer (NSCLC) patients after GKRS for brain metastases in a study [1].

The receptor for epidermal growth factor (EGFR) is a prime target for cancer therapy across a broad variety of tumor types. As it is a tyrosine kinase, small molecule tyrosine kinase inhibitors (TKIs) targeting signal transduction, as well as monoclonal antibody against the EGFR, have been investigated as anti-tumor agents [2].

Development of epidermal growth factor receptor tyrosine kinase inhibitors (EGFR-TKIs): gefitinib or erlotinib, was an improvement in treatment of advanced NSCLC patients. EGFR mutations are present in 10-25% of NSCLC (mostly adenocarcinoma), and up to 55% in never-smoking women of East Asian descent. In the non-selected group of patients with BMF-NSCLC, the overall response rates after gefitinib or erlotinib treatment range from 10% to 38%, and the duration of response ranges from 9 to 13.5 months. In the case of present activating EGFR mutation, the response rate after EGRF-TKIs is greater than 50%, and in selected groups (adenocarcinoma, patients of Asian descent, never-smokers, asymptomatic BMF-NSCLC) even 70%. Gefitinib or erlotinib treatment improves survival of BMF-NSCLC patients with EGFR mutation in comparison to cases without the presence of this

mutation. There is no data on the activity of the anti-EML4-ALK agent crizotinib. Bevacizumab, recombinant humanised monoclonal antibody anti-VEGF, in the treatment of advanced non-squamous NSCLC patients is a subject of intense research. Data from a clinical trial enrolling patients with pretreated or occult BMF-NSCLC proved that the addition of bevacizumab to various chemotherapy agents or erlotinib is a safe and efficient treatment, associated with a low incidence of CSN haemorrhages. However, the efficacy and safety of bevacizumab used for therapeutic intent, regarding active brain metastases is unknown [3].

Epidermal growth factor receptor 3 in glioblastoma

see Epidermal growth factor receptor 3 in glioblastoma.

1)
Yang SH, Kim HY, Lee SI, Jin SJ. The Effect of Epidermal Growth Factor Receptor Mutation on Intracranial Progression-Free Survival of Non-Small-cell lung cancer Patients with Brain metastases Underwent Gamma Knife Radiosurgery. Brain Tumor Res Treat. 2020 Oct;8(2):103-108. doi: 10.14791/btrt.2020.8.e15. PMID: 33118342.

2)
Westphal M, Maire CL, Lamszus K. EGFR as a Target for Glioblastoma Treatment: An Unfulfilled Promise. CNS Drugs. 2017 Aug 8. doi: 10.1007/s40263-017-0456-6. [Epub ahead of print] PubMed PMID: 28791656.

3)
Cedrych I, Kruczała MA, Walasek T, Jakubowicz J, Blecharz P, Reinfuss M. Systemic treatment of non-Small-cell lung cancer brain metastases. Contemp Oncol (Pozn). 2016;20(5):352-357. doi: 10.5114/wo.2016.64593. Epub 2016 Dec 20. Review. PubMed PMID: 28373815; PubMed Central PMCID: PMC5371701.