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Concurrent sarcomatoid carcinoma transformation and EGFR T790M mutation occurrence after gefitinib

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Background: EGFR tyrosine kinase inhibitor (TKI) shows dramatic response but eventually develops resistance in EGFR-mutant lung cancer. Several resistance mechanisms include EGFR T790M mutation, activation of alternative pathways, small cell lung cancer transformation and epithelial-to-mesenchymal transition. Among them, the T790M mutation is the most common resistance mechanism, which accounts for about 60% of the patients. However, the resistanttumors harboring T790M mutation show a favorable prognosis and more indolent progression than those without the mutation. We reported a case of EGFR-mutant lung cancer which acquired a rare and aggressive transformation to sarcomatoid carcinoma as well asthe common and indolent T790M mutation after response to EGFR-TKI. **Case Report:** A 50-year-old woman was diagnosed with stage IV lung adenocarcinoma with brain metastasis. She underwent craniotomy and tumor removal. EGFR 19 deletion mutation was found in the brain tumor tissue. Thus, she was treated with 9 cycles of pemetrexed plus cisplatin with intercalated gefitinib in a clinical trial. She showed huge tumor shrinkage and then maintained gefitinib alone. After 3rd cycle of gefitinib maintenance, chest CTindicated disease progression including markedly increased subcarinal lymph node (LN). She underwent biopsy of mediastinal LNs. The histology of LNs was proven as sarcomatoid carcinoma. The mutation test found EGFR T790 mutation. She received radiation therapy to subcarinal LN due to bronchus narrowing. After 10 days, she was presented with nausea, vomiting, and diplopia. Brain MRI and CSF cytology suggested leptomeningeal metastasis. After 3 months, she died of a rapid disease progression despite of supportive care. **Conclusions:** The biological behavior of each drug resistance mechanism is quite different in EGFR-mutant lung cancer. In this case, the histology transformation and genetic alteration was simultaneously occurred. However, sarcomatoid carcinoma transformation more significantly contributed to her rapid deterioration. Thus, knowing resistance mechanism is important to provide appropriate clinical care and predict accurate prognosis of these patients.

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Pattern of metastasis related to clinicopathological features in curatively resected HNSCC

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Background: Epithelial mesenchymal transition (EMT) means that the epithelial cells lose their properties of adhesion and polarization and gain properties of invasion and migration and has an important role in invasion and metastasis of tumor cells. EZH2 have an effect on EMT through regulating E-cadherin expression. However, the exact role of EZH2 and its clinical significance in head and neck squamous cell carcinoma (HNSCC) are not yet known. We tried to examine EZH2 expression with clinic-pathological characteristics to clarify the role of the prognosis in HNSCC. **Methods:** We obtained formalin-fixed paraffin-embedded tissues of 118 patients who received curative surgery for HNSCC. All patients received R0 resection. Six blocks of TMA were made for immunohistochemical study. Paraffin blocks of the tissue microarray weresectioned at a 4um thickness. The nuclear staining of EZH2 was evaluated semi-quantitatively on the basis of staining intensity and distribution using the immunoreactive scores. **Results:** Total 29 recurrences were observed. Among them, 17 patients showed local recurrence only, and 12 patients showed distant metastasis with or without local recurrence. We analyzed distinct patterns of metastasis according to protein expression profile. A significant association between high expression of EZH2 which is a kind of mesenchymal marker and the presence of distant progression was demonstrated ($p = .026$). In patients with node-negative HNSCC, total 15 patients had recurrences. Among them, 7 patients had distant metastasis and all of these 7 patients exhibited EZH2 atthe time of initial diagnosis. However, we did not find its correlation into overall survival. **Conclusions:** EZH-2, one of EMT-associated protein expressed HNSCC was associated with more distant metastasis. It could be a useful marker for determination ofadditional treatment (e.g. adjuvant chemotherapy and/or radiotherapy) in currently resected HNSCC patients in the future.