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Editorial

Biomarker Testing for Treating Non-Small Cell Lung Cancer

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In patients with non-small cell lung cancer (NSCLC), mutations in anaplastic lymphoma kinase (*ALK*), epidermal growth factor receptor (*EGFR*), and V-Ki-ras2 Kirsten rat sarcoma viral oncogene homolog (*KRAS*) are mutually exclusive. One mutation in lieu of another can influence response to targeted therapy. Testing for these mutations and tailoring therapy is generally accepted as standard practice. Tyrosine kinase inhibitors (TKIs) demonstrates biological and clinical activity in only a relatively limited subset of lung cancers. The predominant mechanism by which NSCLC escapes from detection and elimination is by the immune system is by exploiting one such inhibitory pathway via the expression of programmed death ligand 1 (PD-L1, B7-H1). Patients with somatic mutations within the EGFR-TK domain, especially exon 19 deletion, exon 21 L858R, and exon 18 G719X demonstrate the highest response rates to these TKIs. In contrary, the exon 20 T790M mutation is associated with acquired resistance to TKI therapy. Generally, activating *EGFR* mutation are frequently identified in patients with adenocarcinoma of lung. Approximately, 50% of Asians and 10% of non-Asians are associated with activating *EGFR* mutations. Patients with adenocarcinoma of lung have poor responses to EGFR-TKIs (afatinib, erlotinib, and gefitinib). The activity of the *EGFR* monoclonal antibody (cetuximab) seems to be independent of *EGFR* mutation status. Unfortunately, how the presence of an acquired *EGFR* mutation, such as T790M should influence the therapeutic decisions is unclear.

Approximately, 25% of patients with adenocarcinoma of lung are associated with *KRAS* mutation. There are less common among those of Asian descent, but are common in smokers. Patients with *KRAS* mutations seem to be resistant to EGFR-TKIs, whereas they influence the treatment selection remains unclear. Approximately, 2 - 7% of patients with NSCLC adenocarcinomas demonstrate fusion between *ALK* and echinoderm microtubule-associated protein-like 4 (*EML4*). This and other *ALK* rearrangements are more common in those with adenocarcinomas of lung and nonsmokers or light smokers. Patients with *ALK* rearrangements are benefit from treatment with an *ALK* inhibitor (brigatinib, ceritinib and crizotinib). Approximately, 1% of patients with NSCLC demonstrates ROS-1 gene alteration.

In conclusion, targeted therapy is already a reality for many patients with NSCLC. It is not farfetched to expect the current consensus guidelines being obsolete due to constant generating of overwhelming amount of information into the molecular derangements associated with the development of lung cancer. Recently, the United States Food and Drug Administration (US FDA) approves nivolumab and pembrolizumab in treating NSCLC. It appears that both anti-PD1 antibodies and the therapeutic anti-PD-L1 antibodies that is in development will have comparable efficacy and toxicity with serious autoimmune toxicities in 5 - 10% of patients and response in approximately 15 - 20% of unselected NSCLC patients.

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