EDITORIAL

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Realities of KRAS-mutated non-small cell lung cancer

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The most common histologic subtype of non-small cell lung cancer is adenocarcinoma. Adenocarcinoma has targetable gene alterations such as epidermal growth factor receptor (EGFR) mutations and anaplastic lymphoma kinase and ROS1 translocations, and targeting of these molecules has dramatically improved therapeutic results over the past decade. KRAS mutations, which occur more often in smokers and Western populations, are also found in adenocarcinomas. The development of therapeutic agents targeting KRAS mutations has been attempted, but no effective ones have been available before now [1]. In contrast to EGFR mutations, of which deletion 19 and L858R account for the majority, there is a wide variety of KRAS mutations. In addition, the KRAS pathway is involved in cross-talk with several signal pathways, rendering it difficult to target this pathway due to easy bypassing of signal blocking. Therefore, it has been very difficult to develop an effective therapeutic agent for adenocarcinomas harboring KRAS mutations. Because of this, the results of this study are clinically meaningful, despite the retrospective nature [2]. The findings suggest that the sensitivities of pemetrexed- and gemcitabine-based chemotherapies may differ depending on the specific KRAS mutation. The authors concluded that since existing cytotoxic chemotherapies are the only primary treatment options for patients with KRAS mutations, for whom other effective targeted therapies are lacking, the most sensitive cytotoxic agent should be selected based on the KRAS mutation status. However, prospective studies are needed to gather reliable evidence. Finally, one of the hurdles with treatment of cancers with KRAS mutations is that genetic testing of KRAS is not reimbursed by the government in Korea.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

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