

Genomic testing and treatment landscape in patients with advanced Non-Small Cell Lung cancer (aNSCLC) using real-world data from community oncology practices.

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BACKGROUND

While aNSCLC is a leading cause of US cancer deaths, targeted therapies and immune checkpoint inhibitors (ICPi) have emerged as important new treatment options for these patients and NCCN guidelines recommend testing of eight genes in aNSCLC patients at diagnosis. Targetable alterations (TA) in four genes, EGFR, ALK, ROS1, and BRAF, are associated with FDA-approved therapies. The labels for ICPis indicate that patients with TAs in EGFR and ALK are not candidates for first line treatment with ICPi.

METHODS

The Integra Connect database, which includes electronic medical record (EMR) and claims data on approximately 600,000 cancer patients, was queried across five community oncology practices (289 oncologists) to identify aNSCLC patients (stage 3B or 4) treated since January 2017. Manual review of charts was done to abstract tumor type/stage, drug regimens, and evidence of somatic genetic testing. A Wilcoxon rank sum test was used to test difference in time to results (TTR) for blood- vs tissue-based tests.

RESULTS

A total of 1,203 aNSCLC patients were identified. Testing rates varied from 54% for EGFR to 22% for all 4 genes (table 1). 163 patients had a TA in EGFR, ALK, ROS1 or BRAF, and 55% of these pts did not receive targeted therapy. 84 pts with TA's in EGFR or ALK had no evidence of progression on targeted therapy, yet 31 (37%) received an ICPi; 24% had the TA test result prior to ICPi use and 13% received the TA result after starting ICPi. Median TTR for blood-based somatic tests was shorter than tissue-based tests (4 vs. 14 days, p-value = 3.5-e07).

CONCLUSIONS

Our analysis in the community oncology setting for aNSCLC patients finds evidence of underutilization of genomic testing, underutilization of targeted therapies, and ICPi use outside of label. Further research is needed to identify strategies to increase testing in aNSCLC patients to provide physicians with the information needed to make optimal treatment decisions.

TABLE 1

Gene	Patients tested % (n=1,203)	Patients with TA in EGFR, ALK, ROS1, BRAF (n=163)		Patients with TA in EGFR, ALK without TKI progression (n=84)		
		On targeted therapy	No targeted therapy	No ICPi	TA prior to ICPi	TA after ICPi
All 4 Genes (T4)	22%	45%	55%	63%	24%	13%
EGFR	54%					
ALK	51%					
ROS1	43%					
BRAF	29%					