PET scan with [11C]erlotinib may provide noninvasive method to identify TKI-responsive lung tumors

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A non-invasive PET imaging technique may identify lung cancers that respond best to tyrosine kinase inhibitors (TKIs), allowing doctors to better select patients for personalized therapy, according to research presented at the 14th World Conference on Lung Cancer in Amsterdam, hosted by the International Association for the Study of Lung Cancer (IASLC).

"As more and more therapeutic agents are becoming available for non-small cell lung cancer therapy, selecting the best drug for each individual patient becomes increasingly challenging," said principal investigator Dr. Idris Bahce, of VU University Medical Center in Amsterdam, the Netherlands. "Predictive markers may offer guidance in personalizing therapy."

One marker that predicts tumor response to TKIs is the activating mutation of the epidermal growth factor receptor (EGFR) gene of the tumor cells, Dr. Bahce said. But it's not easy to obtain adequate tumor tissue from the patient for DNA analysis to determine whether the mutation exists.

In the study, non-small cell lung cancer (NSCLC) patients underwent PET (positron emission tomography) scans using radiolabeled erlotinib, a TKI.

"We found that patients who had an activating EGFR mutation also had an increased tracer uptake and were more sensitive to treatment with erlotinib as compared to those who did not have this mutation," Dr. Bahce said. "This is an important finding, as it indicates that this new imaging PET technique may be a non-invasive predictive marker that identifies NSCLC patients who benefit from treatment with TKIs."

Ten NSCLC patients, five with wild-type EGFR and five with activating EGFR mutations -- determined by DNA sequencing on tumor tissue -- were included in the study. Each was scanned twice using a procedure that included a low-dose computed tomography (CT) scan, a 10 minute [15O]water dynamic PET scan and a one-hour [11C]erlotinib dynamic PET scan.

Tumor uptake of [11C]erlotinib was significantly higher in the mutated group (median uptake (VT) = 1.70;
range 1.33-2.30) than in the wild-type group (median uptake (VT) = 1.18; range, 0.75-1.34; p = 0.03). This difference was not due to differences in tumor perfusion. Tracer [11C]erlotinib uptake correlated with tumor response to subsequent erlotinib treatment, as only high-uptake tumors responded to treatment.

Provided by International Association for the Study of Lung Cancer