

The Effect of PET/CT SUV max Value on Anti-EGFR Treatment Response in Lung Adenocarcinoma

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ABSTRACT

In this study, we aimed to evaluate the effect of pre-treatment positron emission tomography-computed tomography (PET/CT) maximum standardized uptake value (SUV max) on the response to tyrosine kinase inhibitor (TKI) treatment and survival in patients with metastatic lung adenocarcinoma. We retrospectively evaluated data from 77 patients diagnosed with advanced-stage lung adenocarcinoma, who had activating EGFR mutations and received first-line anti-EGFR TKI treatment between 2011 and 2022. We determined the effect of the PET/CT SUV max value on the anti-EGFR TKI treatment response by identifying a cut-off with ROC analysis. Median age was 63 years (range 30-86), 46 patients were female (59.7%). Median follow-up time was 24 months (range 3-122), median overall survival (OS) was 25 months, median progression-free survival (PFS) was 14 months, and the median SUV max was 9 (range 2-35). Ideal cut-off value of pretreatment SUV max that predicted 1-year PFS was 9 in the ROC analysis [AUC: 0.649 (0.524-0.774) / $p=0.025$] with a sensitivity of 65% and specificity of 65%. While the >1-year PFS rate was 65% in patients with pretreatment SUV max ≤ 9 , it was 35% in patients with SUV max > 9 ($p=0.012$). Patients with a pretreatment PET/CT SUV max median ≤ 9 had significantly longer median PFS (18 months vs. 11 months) ($p=0.038$) and median OS (30 months vs. 20 months) ($p=0.023$) compared to those with SUV max median > 9 . An increase in baseline PET/CT SUV max value before treatment negatively predicts response to anti-EGFR treatments, PFS, and OS.

Keywords: Lung cancer, Adenocarcinoma, EGFR TKI, PET/CT, SUV max

INTRODUCTION

Lung cancer remains the leading cause of cancer-related mortality worldwide, accounting for 1.8 million deaths in 2020¹. However, survival rates have improved owing to advances in screening techniques and the development of targeted therapies and immunotherapies.²

Lung cancer treatment is tailored according to the histological subtype, molecular profile, disease stage, and the patient's overall health status. In patients with advanced-stage non-small cell lung cancer (NSCLC), EGFR exon 19 deletions and exon 21 L858R mutations are predictive of re-

sponse to EGFR tyrosine kinase inhibitors (TKIs). EGFR mutations are more commonly observed in women, non-smokers, and individuals with adenocarcinoma histology. In advanced-stage lung adenocarcinoma, the prevalence of EGFR mutations is approximately 10-15% in Western populations and 22-62% in Asian populations.³ EGFR TKIs have been shown to improve survival outcomes in patients with advanced-stage lung adenocarcinoma harboring activating EGFR mutations when compared to platinum-based chemotherapy.⁴⁻⁶

According to previous studies, while first-generation (erlotinib, gefitinib) and second-generation

(erlotinib, gefitinib) and second-generation (afatinib) TKIs are considered standard first-line treatments for EGFR-mutant advanced lung cancer, more recent data indicate superior efficacy with third-generation TKIs (osimertinib). Nevertheless, resistance to TKIs eventually develops, with a median progression-free survival (PFS) of approximately 9-14 months.⁷⁻⁹ Proposed mechanisms of resistance include the emergence of the T790M mutation in exon 20, c-MET amplification, and activation of alternative signaling pathways.¹⁰

FDG PET/CT is an imaging modality based on the elevated glucose metabolism characteristic of tumor cells and is widely used in the diagnosis, staging, and assessment of treatment response in lung cancer. The maximum standardized uptake value (SUV max), a commonly used indicator of glucose metabolic activity,¹¹ has been associated with shorter PFS and overall survival (OS) in many studies involving NSCLC patients.^{12,13}

Although the prognostic value of PET/CT-derived SUV max has been studied in NSCLC, its independent role in predicting survival outcomes and resistance to therapy in EGFR-mutant lung adenocarcinoma remains unclear. This study aims to evaluate whether pretreatment SUV max can serve as a prognostic biomarker for survival and treatment resistance in patients undergoing first-line EGFR TKI therapy.

MATERIALS AND METHODS

Patients

We retrospectively reviewed the medical records of patients diagnosed with advanced non-small cell lung cancer (NSCLC) who were followed and treated in the medical oncology clinic. Patients were included if they had a histologic subtype of adenocarcinoma, carried activating EGFR mutations (exon 19 deletion or exon 21 L858R mutation), received first-line EGFR tyrosine kinase inhibitor (TKI) therapy for metastatic disease, and underwent staging PET/CT within one month prior to treatment initiation.

Patients were excluded if they lacked a pretreatment PET/CT scan, did not harbor activating EGFR mutations, had an unknown EGFR mutation status, were not in the metastatic stage, or received anti-EGFR TKI treatment in later lines of therapy.

FDG PET/CT

All patients underwent baseline PET/CT imaging before initiating treatment. PET/CT scans were performed after a fasting period of at least 6 hours, with blood glucose levels confirmed to be below 150 mg/dL. The maximum standardized uptake value (SUV max) was defined as the highest SUV observed across all cross-sectional planes (sagittal, coronal, or transaxial).

SUV max values were determined using a GE Discovery PET/CT scanner. A standardized region of interest (ROI) with a 1.0 cm diameter was manually placed at the site of maximum FDG uptake in the primary lung lesion. The same imaging protocol was applied consistently across all patients. Measurements were performed by an experienced nuclear medicine specialist, with multiple readings averaged to enhance reliability. The PET/CT device underwent regular calibration and quality control according to international standards.

EGFR Mutation Assessment

DNA extraction was performed on paraffin-embedded tissue samples using the DNA Sample Preparation Kit (BIO-RAD CFX96/AmoyDx EGFR 29 Mutation Detection Kit). Mutation analysis was conducted using reverse transcription-polymerase chain reaction (RT-PCR). The AmoyDx™ EGFR 29 Mutation Detection Kit is a CE-approved PCR-based assay capable of detecting 29 common somatic mutations within exons 18-21 of the EGFR gene, with a 1% false-negative rate and 99% accuracy.

Validation procedures followed the manufacturer's protocol and included both internal quality controls (positive controls with known mutations, wild-type DNA, and water as a negative control) and external quality control measures. Each test run incorporated these controls, and approximately 10% of the sample subgroup was retested to assess both intra-assay and inter-assay repeatability and ensure the reliability of results.

Ethical Approval: The study was approved by the Clinical Research Ethics Committee of Marmara University, Faculty of Medicine (March, 10, 2023, No: 276).

Table 1. Follow-up and patient characteristics

		min-max	median	mean	±	sd./n-%
Age		30-86	63	61.8	±	1.4
	≤ 65			43		55.8%
	> 65			34		44.2%
Follow-up Duration (Months)		3-122	24	32.97	±	3.1
ECOG PS	0-1			67		87
	2			10		13
Status	Died			54		70.0%
	Alive			23		30.0%
Progression-free Survival		3-110	14	21.1	±	2.67
Gender	Female			46		59.7%
	Male			31		40.3%
LDH	Normal			28		45.2%
	High			34		54.8%
Initial Stage	I-III			13		16.9%
	IV			64		83.1%
Mutation Type	Exon 19 deletion			54		70.1%
	Exon 21 mutation			23		29.3%
Smoking	Yes			16		20.8%
	No			61		79.2%
Weight Loss	Present			7		9.1%
	Absent			70		90.9%
Brain Met	Present			28		36.4%
	Absent			49		63.6%
FDG PET SUV max	≤ 9			40		51.9%
	> 9			37		48.1%
TKI	Erlotinib			66		85.7
	Afatinib			7		9.1%
	Gefitinib			4		5.2%
Progression-free Survival (PFS) (Months)	≤ 1 Year PFS			34		44.2%
	> 1 Year PFS			43		55.8%

Statistical Analysis

Overall survival (OS) and progression-free survival (PFS) were calculated using the Kaplan-Meier method, with the start date defined as the first day of EGFR TKI therapy. Univariate analyses were used to evaluate the association between prognostic variables and survival outcomes, and multivariate analyses were performed to assess the independent impact of variables found significant in univariate analysis.

Because SUV max values were not normally distributed, nonparametric tests were used for inter-group comparisons (PFS ≤ 1 year vs. >1 year). Receiver operating characteristic (ROC) analysis was conducted to determine the optimal SUV max cut-off value for predicting resistance to EGFR TKI therapy, defined as PFS less than 1 year.

Based on the ROC analysis, an SUV max threshold of 9 was identified, with an area under the curve

(AUC) of 0.649 (95% CI: 0.524-0.774). This value provided optimal sensitivity (65%) and specificity (65%) for predicting 1-year PFS. ECOG performance status (PS) and lactate dehydrogenase (LDH) levels were included in multivariate analysis due to their known prognostic value in NSCLC and their statistical significance in univariate testing ($p < 0.05$).

RESULTS

A total of 77 patients with advanced-stage lung adenocarcinoma who received first-line EGFR TKI therapy were retrospectively evaluated. The median follow-up duration was 24 months. During this period, 79% (61/77) of patients experienced disease progression, and 70% (54/77) had died. The cohort was composed of 60% (46/77) females, with a median age of 63 years (range: 30-86 years).

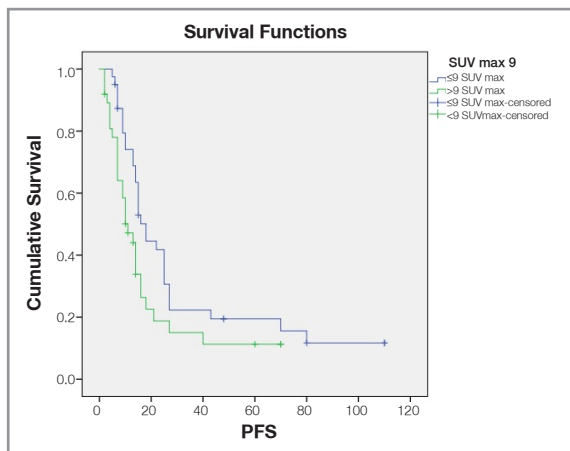


Figure 1. Progression free survival according to pretreatment PET CT SUV max

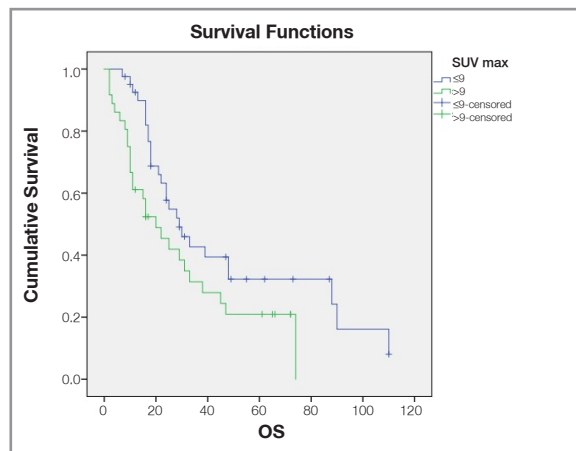


Figure 2. Overall survival according to pretreatment PET CT SUV max

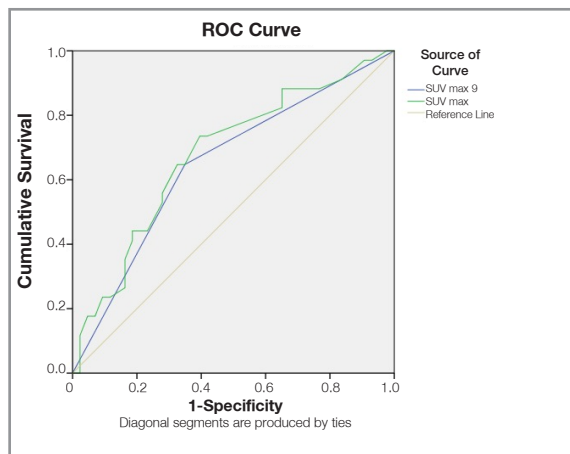


Figure 3. ROC curve ideal cut-off value predicting 1-year PFS

At the time of diagnosis, 83% of patients presented with metastatic disease, and 21% had a history of smoking. Exon 19 deletion was detected in 70% of cases, and 36% had brain metastases. The majority of patients (86%, 66/77) received erlotinib as their EGFR TKI (Table 1).

The median overall survival (OS) was 25 months (range: 3-122 months). Both univariate and multivariate analyses demonstrated that poor ECOG performance status and higher pretreatment SUV max values were significantly associated with reduced OS. The median progression-free survival (PFS) was 14 months (range: 3-110 months). Univariate analysis revealed that poor ECOG performance status, elevated LDH levels, and high pre-

treatment SUV max were all negatively associated with PFS. Multivariate analysis confirmed that high LDH was an independent predictor of shorter PFS (Table 2).

Among the 77 patients analyzed, the median pre-treatment SUV max was 9. Patients with SUV max ≤ 9 had significantly longer PFS (18 vs. 11 months, $p=0.038$) (Figure 1) and OS (30 vs. 20 months, $p=0.023$) (Figure 2) compared to those with SUV max > 9 . The ideal cut-off value of pre-treatment SUV max that predicted 1-year PFS was 9 in the ROC analysis, [AUC: 0.649 (0.524-0.774) / $p=0.025$] with a sensitivity of 65% and specificity of 65% (Figure 3). In multivariate analysis, high SUV max was an independent predictor of shorter OS (HR: 1.87, 95% CI: 1.07-3.26, $p=0.027$) and shorter PFS (HR: 1.53, 95% CI: 1.00-2.79, $p=0.049$).

DISCUSSION

First-line EGFR TKIs are the standard of care for patients with stage IV NSCLC harboring activating EGFR mutations.¹⁴⁻¹⁶ The association between high SUV max and poor prognosis may be attributed to elevated tumor metabolic activity and underlying intratumoral heterogeneity –both factors known to contribute to resistance against EGFR TKIs. Tumor metabolic activity can be assessed via SUV, and prior meta-analyses in NSCLC have demonstrated that high SUV is correlated with poor prognosis.¹⁷⁻¹⁸

Table 2. Cox-regression model of overall survival (OS) and progression free survival (PFS) in EGFR mutant advanced stage lung adenocarcinoma

	OS			Multivariate Analysis			PFS			Multivariate Analysis		
	Univariate Analysis			95% CI			95% CI			95% CI		
	HR	Lower	Upper	P	HR	Lower	Upper	P	HR	Lower	Upper	P
Age (≤ 65 / > 65)	0.78	0.45	1.34	0.377					1.37	0.82	2.29	0.230
Gender (male/female)	1.03	0.78	1.35	0.810					1.02	0.78	1.32	0.895
Smoking (-/+)	0.80	0.40	1.58	0.525					0.79	0.42	1.49	0.486
ECOG (0-1/2)	2.21	1.03	4.73	0.041				0.030	1.53	2.53	13.14	0.000
LDH High (-/+)	1.65	0.87	3.13	0.125					1.94	1.02	3.32	0.042
Weight Loss (-/+)	0.74	0.46	1.17	0.199					0.89	0.56	1.41	0.620
Mutation Type (exon 19 / exon 21)	1.10	0.82	1.49	0.526					1.19	0.89	1.60	0.237
FDG PET SUV max (≤ 9 / > 9)	1.87	1.07	3.26	0.027				0.01	1.53	1.00	2.79	0.049
Initial Stage (I-III/IV)	0.96	0.45	2.05	0.914					1.02	0.52	2.02	0.950
Cox-Regression												

Intratumoral heterogeneity refers to the presence of distinct genotypic and phenotypic subpopulations within the same tumor.¹⁹ In oncology, this heterogeneity is understood as a result of tumor evolution through the coexistence of cellular clones with diverse characteristics.²⁰ In such heterogeneous tumors, some subclones may be inherently resistant or may develop adaptive resistance to therapy. Several studies have demonstrated a strong link between tumor heterogeneity and adverse clinical outcomes.²¹⁻²³ In EGFR-mutant NSCLC, intratumoral heterogeneity may partly explain the variable response rates observed with TKI therapy.²⁴

PET/CT imaging, widely used for tumor staging and treatment response assessment, also provides an indirect measure of tumor heterogeneity through metabolic markers such as SUV max. Patients with high SUV max and substantial tumor burden may exhibit greater genomic instability, possibly due to underlying inter- and intratumoral heterogeneity. Such tumors are presumed to have shorter PFS despite higher initial response rates to EGFR TKIs. A study by Park et al. (2017) found that EGFR-mutant NSCLC patients with higher pretreatment textural heterogeneity on PET/CT experienced shorter PFS and were more likely to develop resistance to TKI therapy.²⁵

Previous studies investigating the prognostic significance of PET/CT-derived SUV max in EGFR-mutant NSCLC treated with gefitinib or erlotinib have reported that a lower SUV max in the primary tumor is associated with better survival outcomes.^{26,27} In a 2015 study, Keam et al. analyzed the impact of baseline PET/CT parameters—total lesion glycolysis (TLG) and SUV max—on TKI resistance and survival. They concluded that high TLG predicted both shorter PFS and OS, while high SUV max was associated with reduced PFS.²⁸ Similarly, Aguloglu et al. (2022) reported that pretreatment metabolic tumor volume predicted PFS independently but did not correlate with OS in a cohort of 68 EGFR-mutant patients.²⁹

In our study, patients with a pretreatment SUV max > 9 had significantly worse survival outcomes. These findings align with prior research.^{25,28,29} However, unlike those studies, we demonstrated that SUV max independently predicts both PFS and OS. This discrepancy may be due to variations in study population characteristics (e.g., ethnic

background, mutation frequency, smoking status), methodological differences (e.g., PET protocols, ROI settings, SUV cut-off definitions), sample size, and follow-up duration. Collectively, our results support the hypothesis that tumors with high metabolic activity may exhibit a more aggressive phenotype and increased genomic instability.

Clinically, identifying EGFR-mutant NSCLC patients with high pretreatment SUV max—and potentially other PET-based parameters such as TLG or MTV—may help stratify patients at higher risk of early progression and inferior survival. In such high-risk individuals, combination therapies (e.g., TKI plus chemotherapy) may be more appropriate than TKI monotherapy. Furthermore, intensified monitoring may be warranted to detect early progression.

Conclusion

This study demonstrates that pretreatment SUV max is an independent predictor of both overall survival and progression-free survival in patients with EGFR-mutant non-small cell lung cancer receiving first-line EGFR TKI therapy. These findings suggest that SUV max may serve as a useful prognostic biomarker and could be incorporated into clinical risk stratification models to identify patients at increased risk of early treatment resistance.

Further prospective studies are warranted to validate these results and to explore the utility of PET/CT-derived metabolic parameters in guiding personalized treatment strategies for this patient population.

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