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The Relationship between CEA Levels and Survival Rate in Lung Adenocarcinoma Patients with EGFR Mutations and Wild Type

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Abstract

Background: Carcinoembryonic Antigen (CEA), typically present at elevated levels in lung adenocarcinoma, may serve as a prognostic biomarker. However, its relationship with outcomes across Epidermal Growth Factor Receptor (EGFR) molecular subtypes remains uncertain. This study evaluated post-therapy CEA in relation to survival and compared concentrations between the variant and wild-type groups.

Method: This retrospective cohort included 50 individuals treated for lung adenocarcinoma at Ulin General Hospital, Banjarmasin, from January 2023 to December 2024. There were 31 variant-positive cases received tyrosine kinase inhibitors, whereas 19 wild-type cases received chemotherapy. CEA was classified as ≤10 or >10 ng/mL. Kaplan-Meier curves and the log-rank test assessed survival, and Cox regression examined relevant associations; $p<0.05$ indicated statistical significance.

Result: Elevated CEA (>10 ng/mL) occurred in 58% of the cohort, and 62% carried an EGFR variant. Median concentrations were 43.3 ng/mL in the variant group versus 4.3 ng/mL in the wild-type group ($p=0.012$). Six-month survival for the ≤10 and >10 ng/mL categories was 100% and 82.6%, respectively, among variant carriers ($p=0.221$), and 92.3% and 83.3% among wild-type cases ($p=0.596$). Multivariable analysis found no significant relationship between biomarker concentration and outcome in either molecular group ($p=0.338$ and $p=0.942$, respectively); age >60 years independently predicted mortality ($p=0.029$).

Conclusion: Post-therapy CEA did not correlate significantly with survival in either molecular subgroup. Nevertheless, concentrations were higher in variant-positive than in wild-type cases at Ulin Hospital, Banjarmasin, Indonesia.

Keywords: Lung adenocarcinoma; EGFR; CEA; Survival Rate; Cell lung

Abbreviations: AKT: Protein Kinase B; ALK: Anaplastic Lymphoma Kinase; BMI: Body Mass Index; BRAF: *B-Raf Proto-Oncogene, Serine/Threonine Kinase*; BSA: Body Surface Area; CEA: Carcinoembryonic Antigen; CEACAM: Carcinoembryonic Antigen-Related Cell Adhesion Molecule; ctDNA: Circulating Tumor DNA; EGFR: Epidermal Growth Factor Receptor; ERBB2: Erb-B2 Receptor Tyrosine Kinase 2; HR: Hazard Ratio; KRAS: KRAS Proto-Oncogene, GTPase; MET: MET Proto-Oncogene, Receptor Tyrosine Kinase; NSCLC: Non-Small Cell Lung Cancer; NTRK1-3: Neurotrophic Receptor Tyrosine Kinase 1-3; OR: Odds Ratio; OS: Overall Survival; PFS: Progression-Free Survival; PPV: Positive Predictive Value; RET: RET Proto-Oncogene; ROS1: ROS Proto-Oncogene 1, Receptor Tyrosine Kinase; SD: Standard Deviation; STAT3: Signal Transducer and Activator of Transcription 3; TKI: Tyrosine Kinase Inhibitor.

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INTRODUCTION

Lung cancer causes high mortality worldwide, and the incidence reaches 2.48 million new cases worldwide, ranking first with 12.4% of all cancer incidents. Lung cancer also accounts for nearly 1.81 million deaths, comprising 18.7% of global cancer mortality. Most lung cancer cases are identified as NSCLC (non-small cell lung cancer). Based on histology, adenocarcinoma constitutes the leading subtype of NSCLC. Its prognosis remains unfavorable, as the reported five-year survival rate is 12.8% [1].

Lung adenocarcinoma may be categorized according to its molecular characteristics. Genomic changes may occur as point mutations, deletions, splice variants, or translocations involving *ALK, EGFR, BRAF, NTRK1-3, RET, ROS1, MET, KRAS*, and *ERBB2*. Among Asian populations, EGFR is the most frequently altered gene, with mutations occurring more commonly among women, younger individuals, and non-smokers. These molecular abnormalities drive adenocarcinoma development and progression while also influencing therapeutic decisions [2].

Diagnosis is confirmed by thoracic Computed Tomography (CT) scan and tissue biopsy. Contrast-enhanced CT scans are used to establish the size, location, and growth of the tumor. Tissue biopsy and cytology are useful for determining histological morphology as well as classifying tumor type and grade [3,4]. Molecular testing is also performed to understand carcinogenesis, which is necessary for targeted therapy and monitoring [5]. *EGFR* and *ALK* mutations are associated with *NSCLC* [6].

There are various treatment options for lung adenocarcinoma, encompassing radiotherapy, surgery, immunotherapy, chemotherapy, and targeted therapy. The selection of treatment counts on the clinical extent of the adenocarcinoma and the patient's clinical condition [7]. Targeted therapy is widely developed, based on the presence of specific mutations that encode proteins important for cell growth and replication. This blocks the mutation pathways and improves survival rates. Each type of mutation has a specific inhibitor, and *EGFR* mutations are inhibited by Tyrosine Kinase Inhibitors (TKI) [8,9].

The prognosis of lung adenocarcinoma is contingent on the *TNM* stage at diagnosis and is influenced by several factors. Poor prognosis is influenced by Performance Status (PS) at diagnosis, anorexia, weight loss, and the presence of extrathoracic metastases. A recent study reports a link between *EGFR* mutations and patient prognosis, in which adenocarcinoma patients have a better prognosis than those without.The occurrence of mutations in lung adenocarcinoma is associated with longer survival rates because of the use of

targeted therapy [10,11].

Tumor markers such as Carcinoembryonic Antigen (CEA) can be considered to classify patients according to their risk of recurrence and poor survival. Recent studies show growing evidence that serum CEA acts as a useful prognostic marker in NSCLC [12,13]. Higher serum CEA concentrations have been linked to enhanced response to EGFR-TKI. Increased CEA levels over the course of molecularly targeted therapy could represent a substantially more sensitive indicator of lung adenocarcinoma advancement for patients carrying *EGFR* mutations than traditional imaging methods [14]. This motivated the investigation of the relationship linking CEA levels and survival rates in lung adenocarcinoma patients carrying *EGFR* mutation and wild-type lung adenocarcinoma at Ulin Hospital, Banjarmasin, Indonesia. Consequently, the present investigation seeks to examine the relationship between CEA levels and survival rates as well as differences in post-therapy CEA levels in patients carrying *EGFR* mutation and wild-type lung adenocarcinoma at Ulin Hospital, Banjarmasin, Indonesia.

MATERIALS AND METHODS

This retrospective cohort study received ethical clearance from the Ethics Committee of the Medicine and Health Sciences Faculty, Universitas Lambung Mangkurat (No. 049/KPEK-FKIK-ULM/EC/VI/2025). Additional approval was acquired from the Ethics Committee of the Research and Development Division, Ulin Hospital, Banjarmasin, Indonesia (No. 85/VII-Reg-Riset/RSUDU/25). Secondary data were obtained from the clinical records of patients managed for lung adenocarcinoma during 2023-2024. Patients were categorized into *EGFR*-mutant and wild-type groups. All eligible records were included through total sampling. Eligibility required a diagnosis of NSCLC adenocarcinoma, completion of EGFR mutation testing, and a documented *EGFR* status. Records containing insufficient clinical information were excluded.

RESULTS

This study obtained a population of 279 subjects from medical records. After excluding 211 patients because of incomplete personal data, missing laboratory test results, or other reasons, 68 met the initial inclusion criteria. Subsequently, 18 were excluded due to incomplete therapy (n=13) or liver disorders (n=5), leaving 50 patients carrying *EGFR* mutation and wild-type lung adenocarcinoma for the final analysis.

The findings of this study are presented in Table 1. The gender predominance was 31 men (62.0%) and 19 women (38.0%). Meanwhile, the mean age was 57 years with a standard deviation and a maximum distribution of 11 years and 50-59 years (30%), respectively. Smoking status was highest among active smokers (14 subjects; 28%), nonsmokers (14 subjects; 28%), passive smokers (11 subjects; 22%), and former smokers (11 subjects; 22%). The median CEA level was 29.3 ng/mL, with a min of 1.3 ng/mL and a max of 1,500 ng/mL.

Table 1. Demographic and clinical profile of the study cohort.

Participant characteristic	Category/statistic	Overall cohort (n=50)
Sex, n (%)	Male	31 (62.0)
	Female	19 (38.0)
Age, years	Mean ± SD	57 ± 11
Age distribution, n (%)	40-49	13 (26.0)
	50-59	15 (30.0)
	60-69	14 (28.0)
	70-79	7 (14.0)
	≥ 80	1 (2.0)
History of cancer, n (%)	Present	1 (2.0)
	Absent	49 (98.0)
Smoking status, n (%)	Nonsmoker	14 (28.0)
	Active smoker	14 (28.0)
	Passive smoker	11 (22.0)
	Former smoker	11 (22.0)
CEA, ng/mL	Median (min-max)	29.3 (1.3-1500.0)
CEA category, n (%)	>10 ng/mL	29 (58.0)
	≤ 10 ng/mL	21 (42.0)
EGFR status, n (%)	Mutation	31 (62.0)
	Wild type	19 (38.0)
BMI, kg/m²	Mean ± SD	21.0 ± 3.4
BSA, m²	Mean ± SD	1.52 ± 0.17
BMI classification, n (%)	Normal	24 (48.0)
	Underweight	13 (26.0)
	Overweight	7 (14.0)
	Obese	6 (12.0)

This study found that 29 patients (58%) exhibited CEA levels >10 ng/mL, while 21 (42%) exhibited CEA levels <10 ng/mL. Generally, 31 patients (62%) had EGFR mutations, compared to 19 with *EGFR* wild type (38%). The types of therapy received by lung adenocarcinoma patients were TKI and chemotherapy administered to 31 and 19 subjects, representing 62% and 38%, respectively.

The mean BMI (body mass index) was 21.0 kg/m2, accompanied by a standard deviation of 3.4 kg/m2. The study found that 24 patients (48%) had a normal BMI, compared with 13 (26%) and 7 (14%) who were underweight and overweight, respectively. The overall body surface area was 1.52 m2 with a standard deviation of 0.17 m2 (Figure 1).

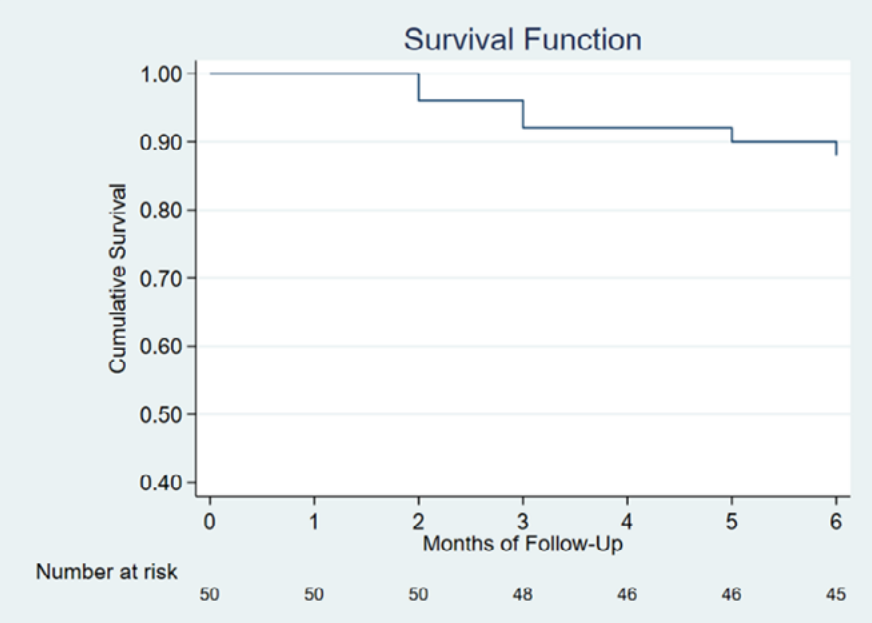


Figure 1. 6-month survival rate of lung adenocarcinoma patients following 6 months of therapy.

A comprehensive analysis of 50 lung adenocarcinoma patients who were observed for 6 months after therapy revealed a 6-month survival rate of 88%, with a mean of 5.7 months. Age (p=0.029) was statistically associated with the 6-month survival rate, with higher mean survival identified in the <60-year group, as shown in Table 2.

Patients aged ≤60 years had an estimated six-month survival probability of 96.6% and an *RMST* of 5.9 months (95% CI, 5.7-6.0). In comparison, patients aged>60 years had a survival probability of 76.2% and an RMST of 5.4 months (95% CI, 4.9-6.0). Survival distributions differed significantly between the age groups (log-rank p=0.029), with more favorable survival observed in the younger group (Table 2 and Figure 2).

Table 2. Six-month survival estimates and bivariate Cox regression results according to demographic and clinical characteristics.

Characteristic	n	Incidence rate, %	6-month survival probability, % (95% CI)	6-month RMST, months (95% CI)	Log-rank p value	Bivariate HR (95% CI)	Cox regression p value
All	50	2.1 (0.9 - 4.7)	88.0 (75.2 - 94.4)	5.7 (5.4 - 6.0)			
Gender							
Male	31	2.9 (1.2 - 6.9)	83.9 (65.5 - 93.0)	5.6 (5.3 - 6.0)	0.271	3.11 (0.36 - 26.64)	0.3
Female	19	0.9 (0.1 - 6.5)	94.7 (68.1 - 99.2)	5.8 (5.4 - 6.2)		1 (ref)	
Age Category							
≤ 60 years	29	0.6 (0.1 - 4.2)	96.6 (78.0 - 99.5)	5.9 (5.7 - 6.0)	0.029*	1 (ref)	
> 60 years	21	4.4 (1.8 - 10.5)	76.2 (51.9 - 89.3)	5.4 (4.9 - 6.0)		7.51 (0.88 - 63.99)	0.067
Smoking Status							
Nonsmoker/ Former smoker	25	1.4 (0.3 - 5.5)	92.0 (71.6 - 97.9)	5.8 (5.6 - 6.1)	0.445	1 (ref)	
Active smoker	14	3.9 (1.3 - 12.1)	78.6 (47.3 - 92.5)	5.5 (4.8 - 6.2)		2.83 (0.47 - 16.95)	0.254
Passive smoker	11	1.6 (0.3 - 11.5)	90.9 (50.8 - 92.5)	5.6 (4.9 - 6.3)		1.18 (0.11 - 13.08)	0.889
CEA Category							
≤10 ng/mL	21	0.8 (0.1 - 5.8)	95.2 (70.7 - 99.3)	5.9 (5.6 - 6.1)	0.186	1 (ref)	

>10 ng/mL	29	3.1 (3.1 -7.4)	82.8 (63.4 - 92.4)	5.6 (5.2 - 6.0)		3.81 (0.45 - 32.37)	0.222
EGFR							
Mutation	31	2.3 (0.9 - 6.1)	87.1 (69.2 - 94.9)	5.6 (5.2 - 6.0)	0.773	1.28 (0.25 - 6.70)	0.775
Wild Type	19	1.8 (0.5 - 7.2)	89.5 (64.1 - 97.3)	5.8 (5.5 - 6.2)		1 (ref)	
BMI Class							
Underweight	13	2.7 (0.7 - 10.8)	84.6 (51.2 - 95.9)	5.7 (5.1 - 6.3)	0.834	1.23 (0.21 - 7.38)	0.529
Normal	24	2.2 (0.7 - 6.8)	86.2 (67.3 - 94.6)	5.6 (5.2 - 6.0)		1 (ref)	
Overweight/ Obese	13	1.3 (0.2 - 9.5)	92.3 (56.6 - 98.9)	5.8 (5.3 - 6.2)		0.60 (0.06 - 5.77)	0.612
Note: Six-month survival was derived from Kaplan-Meier analysis with Greenwood 95% CIs; comparisons used the log-rank test. Mean survival denotes the 6-month RMST. HRs were obtained from bivariate Cox regression. CI, confidence interval; HR, hazard ratio; ref, reference; RMST, restricted mean survival time. *p<0.05.							

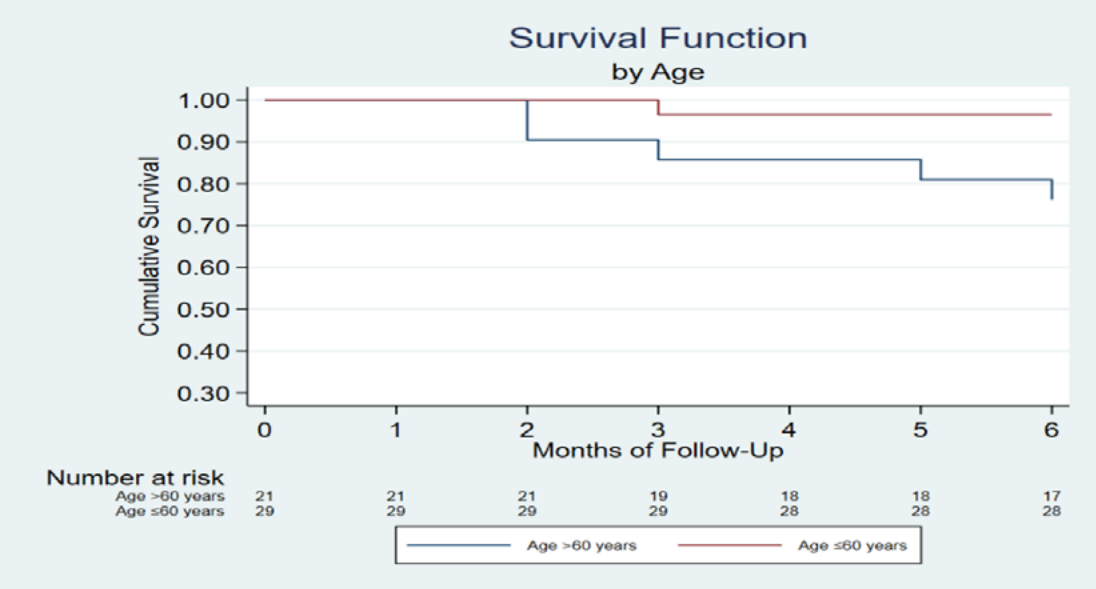


Figure 2. 6-month survival rate of lung adenocarcinoma paatients following 6 months of therapy based on age.

At six months, the estimated survival probabilities were 87.1% in the *EGFR*-mutant group and 89.5% in the *EGFR* wild-type group. The corresponding RMST values were respectively 5.6 and 5.8 months. No significant difference in survival was identified across both groups (log-rank p=0.773; Table 2). Analysis of potential predictors for death in lung adenocarcinoma patients undergoing six months of therapy was conducted using a Cox proportional hazard regression model. This approach allows for a bivariate assessment of each variable's influence on time to death (without adjustment for other factors). The Kaplan-Meier graph shows a difference, but not statistically significant (Figure 3).

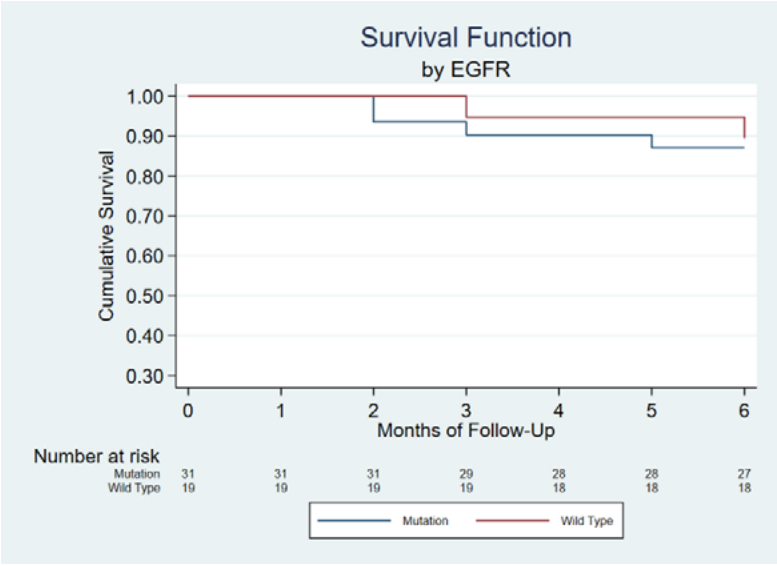


Figure 3. Kaplan-Meier curves for six-month survival stratified by EGFR mutation status.

DISCUSSION

The findings demonstrated that the male gender was more prevalent (62%), with a mean age of 57 years. This is in line with Haryati, et al., where the majority of primary lung cancer patients were male (76.1%) with a mean age of 57 years and adenocarcinoma as the most common histological type (61.96%) [15]. Similar findings were also documented by Kusumawardhani, et al. [16] among lung adenocarcinoma patients at Ulin Regional Hospital from 2020-2022, with the predominant group being male (64.1%) and aged <60 years (65.6%). The consistency of male patient dominance was also seen in an international study by Franceschini JP, et al. [17] The largest proportion of NSCLC lung cancer patients in this study were male (62.4%) and demonstrated a higher mean age of 64 ± 11 years. Ermayanti, et al. [18] also reported male dominance reaching 77.8% in lung cancer patients at M Djamil Hospital, Padang, Indonesia. Aini, et al. [19] showed that 62.5% of lung cancer cases were male smokers at Dr. Soetomo Hospital, Surabaya, Indonesia.

This male predominance and advanced age are closely related to long-term exposure to risk factors, particularly smoking, pollution, and biological changes due to aging [20]. Lung cancer primarily happens in older individuals, and the incidence increases with age or biological aging, as reported in the literature [21]. The incidence of lung cancer in older adults reflects exposure to risk factors in previous years, and there are differences in the pattern of the most affected age groups in developed and developing countries. Previous research has associated biological-aging markers, including telomere length and DNA methylation patterns, with susceptibility to lung cancer [22].

EGFR mutations were identified in 62% of the present cohort. This proportion is consistent with the relatively high mutation prevalence reported across the Asia-Pacific region (20%-76%), compared with Europe (6-41%) and the United States (3%-42%) [23]. In Indonesia, Sampurno, et al. [24] detected *EGFR* mutations in 34 of 76 NSCLC patients (44.7%) at Saiful Anwar Hospital, Malang. Aini, et al. [19] also reported frequent EGFR mutation detection in adenocarcinoma and adenosquamous carcinoma. Consistent with the high mutation frequency, 68% of patients in the present study received TKI-based targeted therapy. TKIs represent a principal treatment option for EGFR-mutant NSCLC and have been associated with longer survival. Geographic differences in mutation prevalence may reflect both genetic and environmental influences [25]. Lung adenocarcinoma is also frequently observed among women who have never smoked [26].

EGFR constitutes a transmembrane receptor tyrosine kinase undergoing ligand-induced activation to regulate cellular differentiation, proliferation, and survival [27]. Its overexpression or mutation has been described in several malignancies and contributes to tumor growth and progression [28]. *EGFR* mutations affect 15%-20% of lung adenocarcinomas as well as are particularly linked to Asian ethnicity and never-smoking status [29]. Their reported prevalence in East Asian populations is approximately 38.4% [30]. At Ulin Hospital, a 2017 study identified *EGFR* mutations in 13 of 38 patients with cytologically diagnosed lung adenocarcinoma (34.2%) [29]. A subsequent study in 2019 found that never-smokers represented the largest subgroup of patients with adenocarcinoma, accounting for 64.7% [30].

Hayati, et al. [15] detected *EGFR* mutations in 14 of 18 patients (77.8%) using plasma ctDNA analysis and histopathological assessment of tumor specimens. Riensya, et al. [31] reported an EGFR mutation prevalence of 39.2% in a cohort of 102 patients with lung adenocarcinoma. EGFR mutation status was substantially linked to former smoking (OR, 4.3; 95% CI, 1.24-15.57; p=0.022), male sex (OR, 3.409; 95% CI, 1.406-8.268; p=0.007), and M1a metastatic status (OR, 0.203; 95% CI, 0.05-0.821; p=0.025).

A meta-analysis of 456 articles encompassing 30,466 patients with EGFR mutations revealed that exon 19 alterations were reported in 343 studies and accounted for 16.7% of cases. Exon 21 alterations were reported in 330 studies, representing 12.3% of cases [32]. Sun, et al. [33] also identified a significant connection between EGFR mutations and the predominantly micropapillary subtype of lung cancer (p=0.003).

Tobacco exposure contributes to lung carcinogenesis through DNA-adduct formation and structural injury to the bronchial epithelium. Among former smokers, DNA-adduct levels were inversely associated with age at smoking initiation. Individuals who began smoking earlier demonstrated greater DNA damage than those who started at an older age. This accumulated genomic injury may contribute to EGFR alterations [34].

At six months, the estimated survival probabilities were 87.1% in the EGFR-mutant group and 89.5% in the EGFR wild-type group. The corresponding RMST values were respectively 5.6 and 5.8 months. No significant difference in survival was identified across both groups (log-rank p=0.773; Table 2). The six-month survival probability was 95.2% among patients with CEA ≤10 ng/mL and 82.8% among those with CEA >10 ng/mL. The corresponding RMST values were 5.9 and 5.6 months. However, survival distributions did not differ significantly between the CEA categories (log-rank p=0.186). Within the EGFR-mutant subgroup, estimated six-month survival was 100% among patients with CEA ≤10 ng/mL and 82.6% among those with CEA>10 ng/mL (log-rank p=0.221). In the EGFR wild-type subgroup, the corresponding estimates were 92.3% and 83.3% (log-rank p=0.596). Multivariable analysis showed no substantial connection between post-treatment CEA levels and survival in either subgroup.

These findings are consistent with Facchinetti, et al. [35], who reported that declining CEA levels showed a high PPV for disease control but were not associated with PFS or OS among patients with EGFR mutations. In contrast, Yang, et al. [36] and Kuo, et al. [37] associated decreasing CEA levels with improved OS and PFS in this population. The absence of a survival association in the wild-type subgroup also agrees with Kuo, et al. [37] and Zheng, et al. [38] Sánchez-Reyes, et al. [39] reported that CEA normalization following systemic treatment might indicate more favorable clinical outcomes. Differences across studies may partly reflect baseline CEA concentrations; the median CEA level in the wild-type group of the present cohort was 4.3 ng/mL. The observed between-group pattern in post-treatment CEA is also consistent with evidence linking EGFR mutations to increased CEA expression [40]. Kuo, et al. [37] and Zheng, et al. [38] similarly reported a connection between enhanced CEA and EGFR mutation detection, with different CEA dynamics across EGFR subgroups.

Previous studies have also examined the prognostic role of CEA in *EGFR*-mutant lung adenocarcinoma. Kuo, et al. [37] revealed that a baseline CEA concentration >10 ng/mL was linked to poorer OS and PFS among patients with stage IIIB/IV *EGFR*-mutant NSCLC. These results show that high post-therapy CEA tends to indicate poorer survival in the *EGFR* mutation subgroup.

Zhao, et al. [41] reported the following treatment outcome and survival findings according to baseline serum CEA levels (Table 3).

Table 3. Treatment response and time-to-event outcomes according to CEA concentration.

Clinical endpoint	Entire cohort	CEA ≤10 *vs*. >10 ng/mL	Association measure
Treatment response, %	54.8	69.2 *vs*. 43.4 (p = 0.001)	OR, 0.322 (p=0.001)
Median PFS, months	6.6	7.8 *vs*. 5.3 (p = 0.029)	HR, 1.45 (p=0.025)
Median OS, months	14.8	18.8 *vs*. 11.8 (p < 0.001)	HR, 2.133 (p<0.001)

Note: CEA results are presented for the ≤10 and >10 ng/mL groups, respectively; effect estimates compare the>10 with the ≤ 10 ng/mL group. HR: Hazard Ratio; OR: Odds Ratio; OS: Overall Survival; PFS: Progression-Free Survival

Elevated serum CEA concentrations have been connected with a greater frequency of *EGFR* mutations, suggesting a positive relationship between CEA expression and *EGFR*-mutant disease [40]. CEA is an established tumor marker that may be elevated in lung adenocarcinoma, particularly among women and never-smokers. The CEACAM (CEA-Related Cell Adhesion Molecule) family falls within the immunoglobulin superfamily and contains membrane-associated adhesion proteins. CEACAM1 has been reported to possess tumor-suppressive functions, although its expression may also promote angiogenesis in lung adenocarcinoma. In contrast, CEACAM5 and CEACAM6 have been implicated in tumor-promoting processes. CEACAM signaling may impair cellular differentiation and apoptosis, thereby supporting tumor progression. *EGFR* activation also engages downstream effectors, including AKT and STAT3, which regulate tumor-cell survival and apoptosis. Activation of this pathway may increase CEA expression; consequently, serum CEA has been investigated as a marker of EGFR-mutant disease [24].

Several limitations should be recognized in this study. First, the retrospective design depended on information documented in medical records, with data availability determined by the completeness of existing records, introducing the potential for selection bias. Second, the six-month observation period was insufficient to evaluate differences in survival outcomes across patients with EGFR mutations and the wild-type genotype.

Conclusion

As a general conclusion, there was no statistically meaningful correlation between post-therapy CEA levels and survival rates in patients with lung adenocarcinoma in the *EGFR* mutation or wild-type groups. Nevertheless, CEA levels were markedly elevated in patients carrying *EGFR* mutations relative to the *EGFR* wild-type at Ulin Hospital in Banjarmasin, Indonesia.

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CONFLICT OF INTEREST

The authors report no competing interests concerning this study.

FUNDING DISCLOSURE

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