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*Research Article*

# Prognostic Significance of Pre- and Posttreatment $^{18}$F-FDG PET/CT-Derived SUV-max in Esophageal Cancer: A South Asian Perspective

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**Background:** Esophageal cancer (EC) is a leading cause of cancer-related deaths globally, with South Asia bearing a particularly high disease burden. While $^{18}$F-FDG PET/CT-derived SUV-max values are explored for their prognostic relevance, previous findings remain inconsistent, and data from South Asian populations are limited.
**Methods:** This retrospective study analyzed data from 42 biopsy-confirmed EC patients treated between June 2019 and December 2024 at a tertiary care center. All included patients had initially received standardized neoadjuvant chemoradiotherapy and underwent both pre- and posttreatment 18F-FDG PET/CT imaging. Associations between clinicopathological factors and survival status were evaluated using the independent *t*-test, Chi-square test, and Fisher's exact test. Receiver operating characteristic (ROC) analysis was performed to determine optimal SUV-max cutoffs. Survival outcomes were assessed using Kaplan–Meier estimates, log-rank test, and Cox proportional hazards regression to identify independent predictors of mortality.
**Results:** Kaplan–Meier analysis showed significantly reduced survival in patients with elevated nodal SUV-max values both pre- and posttreatment. In multivariate Cox regression, posttreatment nodal SUV-max was the strongest independent predictor of mortality (HR = 52.9, $p = 0.012$), followed by pretreatment nodal SUV-max (HR = 15.3, $p = 0.034$), while tumor SUV-max values (pre- and posttreatment) did not demonstrate independent prognostic significance.
**Conclusions:** Nodal SUV-max on PET/CT is a strong, independent predictor of survival in EC and may effectively guide personalized treatment decisions.

**Keywords:** esophageal cancer; positron emission tomography; prognosis; South Asia; standardized uptake value

**Key Findings**

- Mean pre- and posttreatment standardized uptake value (SUV) values for both tumor and lymph nodes were higher among expired patients.
- Nodal maximum SUV (SUV-max) were significantly associated with an increased risk of mortality.

- What is known and what is new?

- High pre- and posttreatment SUV-max on $^{18}$F-fluorodeoxyglucose positron emission tomography/computed tomography ($^{18}$F-FDG PET/CT) is associated with a poorer prognosis and reduced survival in esophageal cancer (EC).
- Higher pre- and posttreatment nodal SUV-max values were independently and significantly associated with increased mortality, emphasizing their prognostic potential.

- What is the implication, and what should change now?

- SUV-max nodal serves as a more reliable prognostic biomarker than tumor SUV-max for outcomes and survival in EC. Clinicians and researchers should consider incorporating nodal SUV-max into routine PET/CT evaluation and future risk stratification, particularly for patients with advance disease.

## 1. Introduction

*1.1. Background.* EC remains a significant global health challenge, ranking ninth in incidence and sixth in mortality, with over half a million deaths annually [1–3]. Due to its often late diagnosis, treatment options are frequently limited, and outcomes remain poor despite multimodal management involving surgery, chemotherapy, and radiotherapy [4–6]. Current staging guidelines recommend an initial CT scan to exclude distant metastases, followed by further staging with PET-CT, endoscopic ultrasound, or laparoscopy as clinically indicated [7, 8]. Among these, PET-CT has shown superior accuracy in detecting both locoregional disease and distant metastases, particularly in locally advanced EC. [9].

Beyond anatomical staging, 18F-FDG PET/CT provides functional insights into tumor biology by measuring glucose metabolism [10]. The SUV, particularly the SUV-max, reflects tumor metabolic activity and has been associated with tumor aggressiveness and prognosis. FDG-PET also aids restaging by identifying interval metastases and evaluating treatment response [11].

Tumor SUV-max is used to assess metabolic intensity and predict prognosis, with higher values often correlating with more aggressive disease [12]. However, nodal SUV-max is increasingly recognized as a potentially strong prognostic indicator, especially in advanced-stage EC, where it may better indicate systemic spread and metastatic potential [13]. In cases where tumor SUV-max is low or misleading due to necrosis or posttreatment changes, elevated nodal SUV-max can provide critical insights into disease severity and prognosis. Evaluating both parameters together may improve prognostic accuracy and support personalized treatment plan [14].

*1.2. Rationale and Knowledge Gap.* EC is an aggressive malignancy with early metastatic potential and poor prognosis, especially in low- and middle-income regions such as South Asia, where late-stage presentation is common [15]. PET/CT-derived SUV-max has emerged as a useful prognostic biomarker [12]. While tumor SUV-max has been extensively studied, nodal SUV-max may offer superior prognostic value in advanced disease, such as EC [13]. Despite its potential, the prognostic value of SUV-max remains unexplored in South Asian population, where late diagnosis is common, and current evidence is largely extrapolated from other regions. This study aims to evaluate prognostic significance of pre- and posttreatment $^{18}$F-FDG PET/CT-derived SUV-max in EC. Findings may enhance risk stratification and support more tailored treatment strategies in this high-risk group.

*1.3. Objective.* The objective of this study is to evaluate the prognostic value of pre- and posttreatment $^{18}$F-FDG PET/CT-derived SUV-max in predicting treatment response and survival outcomes in EC patients from a South Asian population.

This manuscript is written following the STROBE checklist.

## 2. Methods

*2.1. Study Design and Setting.* All patients provided informed consent. The study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board of Dow University of Health Sciences (IRB-2780/DUHS/1148). A retrospective design was employed, and medical records of EC patients treated at the Department of Upper GI Surgery, Surgery Unit-I, Dr. Ruth K.M. Pfau Civil Hospital, were retrieved for the period between June 2019 and December 2024.

*2.2. Sample Size, Inclusion Criteria, and Exclusion Criteria.* Biopsy-confirmed EC patients with complete medical records, including both pre- and posttreatment $^{18}$F-FDG-PET/CT scans, along with documented details of the treatment received during the defined study period (June 2019 to December 2024), were included.

Exclusion criteria involved patients with incomplete records. All retrieved information underwent quality control checks to ensure data accuracy. A total of 42 patients met the inclusion criteria and were included in the final analysis.

*2.3. Data Collection and Analysis.* Patient data were systematically retrieved from institutional proformas/hospital records, encompassing all details, from the time of admission to the last documented follow-up. Collected information included clinicopathological characteristics, details of the concurrent neoadjuvant therapy regimen administered, type of surgery (if performed), and operative parameters such as operative time (in minutes) and estimated blood loss (in milliliters), which were used to assess

the quality of resection and the immediate impact of surgical intervention on prognosis.

All included patients had undergone imaging, pathological, and biopsy assessments at a single laboratory facility, ensuring consistency and standardized evaluation. Data retrieval was supported by a structured quality control process, which involved a thorough review of medical records, pathology reports, and surgical notes. Survival outcomes were assessed through weekly follow-up clinical records at the unit. For patients who missed scheduled visits, follow-up data were obtained via telephonic contact, and survival status was recorded.

Data analysis was conducted using SPSS Version 24. Descriptive statistics, including frequencies, percentages, means, and standard deviations, were reported. Receiver operating characteristic (ROC) curve analysis was performed to calculate the area under the curve (AUC) and to determine the optimal cutoff values for SUV of tumor and lymph nodes in predicting overall survival (OS) and progression-free survival (PFS). Bivariate associations between clinicopathological characteristics and patient survival status (alive or expired) were examined using the independent *t*-test, Chi-square test, or Fisher's exact test. The OS was measured from the date of cancer diagnosis to the last date of follow-up. PFS was calculated from the date of cancer diagnosis to the last date of the disease reoccurrence, progression, or last follow-up, whichever occurred first. Survival curves were plotted using the Kaplan–Meier estimates, and differences were compared with the log-rank test. A Cox proportional hazards regression was employed for both univariate and multivariate analyses. Hazard ratios (HRs) with corresponding 95% confidence intervals (CIs) were reported. A $p$ value $\leq 0.05$ was considered statistically significant.

## 3. Results

A total of 42 patients were included in the study, with a mean age of $45.5 \pm 12.0$ years (range: 22–73 years). Among them, 22 (52.4%) were female and 20 (47.6%) were male. Histologically, 36 (85.7%) patients were diagnosed with squamous cell carcinoma, while 6 (14.3%) had adenocarcinoma. Tumor grading showed that 6 (14.3%) patients had well-differentiated tumors, 32 (76.2%) had moderately differentiated tumors, and 4 (9.5%) had poorly differentiated tumors. The lower esophagus was the most commonly involved site, observed in 22 (52.4%) patients, followed by the middle thoracic region in 13 (31%) and the upper thoracic region in 6 (14.3%) (Table 1).

All patients received a standardized concurrent neoadjuvant therapy regimen, regardless of whether they proceeded to definitive surgical resection. This regimen consisted of four cycles of chemotherapy, including carboplatin, paclitaxel, cisplatin, and fluorouracil, in combination with 25–28 sessions of radiotherapy, delivering a total dose of 45 Gy. Of the 42 patients, 35 (83.3%) underwent surgical resection, where 25 (59.5%) had laparoscopic McKeown esophagectomy, 4 (9.5%) had laparoscopic Ivor Lewis esophagectomy, while 6 (14.3%) had their surgery abandoned, and 7 (16.7%) did not undergo any surgery. The median operative time was 320 min (range: 260–380 min), and the median estimated intraoperative blood loss was 100 mL (range: 90–200 mL). These treatment and surgical parameters established a baseline for further evaluating outcomes.

During the follow-up period, 12 patients (28.6%) died. The median duration of follow-up was 1.3 years, ranging from 0.3 to 4.5 years. The OS rate was 71.4% (30 patients), with a mean OS of 3.2 years (Figure 1). On subgroup analysis, patients who died were more likely to have higher radiological N stage ($p = 0.037$), pathological T stage ($p = 0.012$), and pathological N stage ($p = 0.016$). Furthermore, patients with pathological tumor length $< 5$ cm were more likely to survive, indicating a significantly better prognosis ($p = 0.041$). Vascular involvement was also more prevalent among those who died ($p = 0.033$), suggesting an association with more aggressive disease behavior (Table 1).

Further analysis revealed that mean pre- and post-treatment SUV values for both tumor and lymph nodes were higher among expired patients. Notably, posttreatment SUV node values showed a statistically significant difference (mean difference: 2.3; $p$ value = 0.035), as detailed in Table 1. This observation prompted an evaluation of metabolic response to therapy.

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To further refine prognostic stratification, ROC analysis was conducted. The optimal cutoff values were determined to be 7.5 for pretreatment SUV tumor, 2.4 for pretreatment SUV node, 1.7 for posttreatment SUV tumor, and 3.4 for posttreatment SUV node (Table 2). The Kaplan–Meier survival analysis based on these thresholds revealed that patients with higher pretreatment SUV node values had significantly poorer OS (83.9% vs. 36.4%; log-rank $p = 0.002$). Similarly, patients with elevated posttreatment SUV node values (78.9% vs. 0%; log-rank $p < 0.001$) and high posttreatment SUV tumor values (81.5% vs. 53.3%; log-rank $p = 0.033$) demonstrated significantly reduced survival. In contrast, the pretreatment SUV tumor did not show a statistically significant difference with OS ($p = 0.051$) (Figure 2).

The overall PFS rate was 66.7% (28 patients), with a mean PFS of 3.0 years (Figure 3).

The Kaplan–Meier PFS analysis revealed that patients with higher pretreatment nodal SUV values had significantly poorer PFS (67.0% vs. 21.2%; log-rank $p < 0.001$), and a similar trend was observed for those with elevated pretreatment tumor SUV values (89.5% vs. 31.5%; log-rank $p = 0.013$). Likewise, patients with high posttreatment nodal SUV values (60.4% vs. 0%; log-rank $p = 0.031$) and high posttreatment tumor SUV values (65.1% vs. 32.4%, log-rank $p = 0.031$) demonstrated significantly reduced PFS, as shown in Figure 4.

Univariate Cox regression analysis further supported all previous associations as described, showing that elevated

Table 1: Characteristics of esophageal cancer patients ($n = 42$).

| Characteristics | Total $n$ (%) | Alive ($n = 30$) | Expired ($n = 12$) | $p$ value* |
|---|---|---|---|---|
| *Age, years* | | | | |
| Mean ± SD | 45.5 ± 12.0 | 44.6 ± 13.0 | 47.8 ± 9.3 | 0.445 |
| Minimum–maximum | 22–73 | | | |
| *Gender* | | | | |
| Male | 20 (47.6) | 15 (50.0) | 5 (41.7) | 0.625 |
| Female | 22 (52.4) | 15 (50.0) | 7 (58.3) | |
| *Tumor site* | | | | |
| Cervical, < 20 cm from incisors | 1 (2.4) | 0 | 1 (8.3) | 0.237 |
| Upper thoracic, 20–25 cm from incisors | 6 (14.3) | 5 (16.7) | 1 (8.3) | |
| Mid thoracic, 25–30 cm from incisors | 13 (31.0) | 11 (36.7) | 2 (16.7) | |
| Lower esophagus | 22 (52.4) | 14 (46.7) | 8 (66.7) | |
| *Histopathology* | | | | |
| Adenocarcinoma | 6 (14.3) | 5 (16.7) | 1 (8.3) | 0.655 |
| Squamous cell carcinoma | 36 (85.7) | 25 (83.3) | 11 (91.7) | |
| *Tumor grade* | | | | |
| Well differentiated | 6 (14.3) | 4 (13.3) | 2 (16.7) | 0.529 |
| Moderately differentiated | 32 (76.2) | 24 (80.0) | 8 (66.7) | |
| Poorly differentiated | 4 (9.5) | 2 (6.7) | 2 (16.7) | |
| *Tumor length radiological* | | | | |
| Less than 5 cm | 13 (31.0) | 11 (36.7) | 2 (16.7) | 0.081 |
| 5–10 cm | 23 (54.8) | 17 (56.7) | 6 (50.0) | |
| More than 10 cm | 6 (14.3) | 2 (6.7) | 4 (33.3) | |
| *T stage radiological* | | | | |
| T2 | 5 (11.9) | 4 (13.3) | 1 (8.3) | 0.999 |
| T3 | 28 (66.7) | 20 (66.7) | 8 (66.7) | |
| T4 | 9 (21.4) | 6 (20.0) | 3 (25.0) | |
| *N stage radiological* | | | | |
| N0 | 5 (11.9) | 4 (13.3) | 1 (8.3) | 0.037* |
| N1 | 32 (76.2) | 25 (83.3) | 7 (58.3) | |
| N2 | 5 (11.9) | 1 (3.3) | 4 (33.3) | |
| *M stage radiological* | | | | |
| M0 | 41 (97.6) | 30 (100.0) | 11 (91.7) | 0.286 |
| M1 | 1 (2.4) | 0 | 1 (8.3) | |
| *Vascular involvement* | | | | |
| No vascular involvement | 9 (21.4) | 7 (23.3) | 2 (16.7) | 0.033* |
| Abutting aorta less than 90 degrees | 30 (71.4) | 23 (76.7) | 7 (58.3) | |
| Encasing aorta/abutting aorta more than 90 degrees | 3 (7.1) | 0 | 3 (25.0) | |
| *Clinical stage* | | | | |
| II | 8 (19.0) | 8 (26.7) | 0 | 0.089 |
| III | 28 (66.7) | 19 (63.3) | 9 (75.0) | |
| IV | 6 (14.3) | 3 (10.0) | 3 (25.0) | |
| *Pretreatment SUV tumor* | | | | |
| Mean ± SD | 8.9 ± 4.9 | 8.3 ± 5.3 | 10.5 ± 3.7 | 0.187 |
| Minimum–maximum | 1.5–20.2 | | | |
| *Pretreatment SUV node* | | | | |
| Mean ± SD | 1.3 ± 2.5 | 0.7 ± 1.8 | 3.0 ± 3.2 | 0.035* |
| Minimum–maximum | 0–9.1 | | | |
| *Treatment* | | | | |
| Neoadjuvant | 5 (11.9) | 2 (6.7) | 3 (25.0) | 0.270 |
| Neoadjuvant followed by surgery | 36 (85.7) | 27 (90.0) | 9 (75.0) | |
| Surgery followed by adjuvant therapy | 1 (2.4) | 1 (3.3) | 0 | |
| *Posttreatment SUV tumor* | | | | |
| Mean ± SD | 1.6 ± 2.3 | 1.2 ± 2.2 | 2.5 ± 2.5 | 0.110 |
| Minimum–maximum | 0–7.2 | | | |
| *Posttreatment SUV node* | | | | |
| Mean ± SD | 0.6 ± 1.8 | 0.1 ± 0.7 | 1.9 ± 3.0 | 0.074 |
| Minimum–maximum | 0–8 | | | |

Table 1: Continued.

| Characteristics | Total $n$ (%) | Alive ($n = 30$) | Expired ($n = 12$) | $p$ value* |
|---|---|---|---|---|
| *Surgical procedure* | | | | |
| Not done | 7 (16.7) | 5 (16.7) | 2 (16.7) | 0.011* |
| Lap McKeown esophagectomy | 25 (59.5) | 21 (70.0) | 4 (33.3) | |
| Abandoned esophagectomy | 6 (14.3) | 1 (3.3) | 5 (41.7) | |
| Lap Ivor Lewis esophagectomy | 4 (9.5) | 3 (10.0) | 1 (8.3) | |
| *T stage pathological* | | | | |
| T0 | 20 (47.6) | 18 (60.0) | 2 (16.7) | 0.012* |
| T1 | 5 (11.9) | 3 (10.0) | 2 (16.7) | |
| T2 | 3 (7.1) | 3 (10.0) | 0 | |
| T3 | 1 (2.4) | 0 | 1 (8.3) | |
| Not identified | 13 (31.0) | 6 (20.0) | 7 (58.3) | |
| *N stage pathological* | | | | |
| N0 | 20 (47.6) | 18 (60.0) | 2 (16.7) | 0.016* |
| N1 | 7 (16.7) | 5 (16.7) | 2 (16.7) | |
| N2 | 1 (2.4) | 1 (3.3) | 0 | |
| N3 | 1 (2.4) | 0 | 1 (8.3) | |
| Not identified | 13 (31.0) | 6 (20.0) | 7 (58.3) | |
| *Tumor length pathological* | | | | |
| Less than 5 cm | 28 (66.7) | 23 (76.7) | 5 (41.7) | 0.041* |
| 5–10 cm | 1 (2.4) | 1 (3.3) | 0 | |
| Not identified | 13 (31.0) | 6 (20.0) | 7 (58.3) | |

*Note:* Data are reported as $n$ (%).
*$p$ value was calculated by independent $t$ test/Chi-square test/Fisher's exact test.

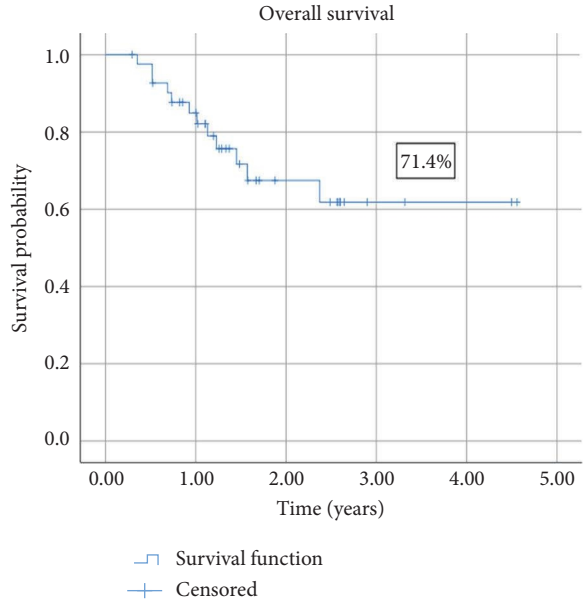


Figure 1: Overall survival of esophageal cancer patients.

pretreatment SUV node (HR = 5.2, $p = 0.005$), posttreatment SUV tumor (HR = 3.2, $p = 0.045$), and posttreatment SUV node (HR = 11.7, $p < 0.001$) were each significantly associated with increased mortality risk. Variables with a $p$ value < 0.25 in a univariate analysis were then included in multivariate models to account for potential confounding. Model 1 included pretreatment SUV parameters, while Model 2 focused on posttreatment parameters. Multivariate Cox regression validated that higher pretreatment SUV node (HR = 15.3, $p = 0.034$) and higher posttreatment SUV node (HR = 52.9, $p = 0.012$) were independently and significantly associated with an increased risk of mortality (Table 3), confirming their role as critical indicators of survival in EC.

## 4. Discussion

This study investigated the prognostic relevance of pre- and posttreatment SUV-max values derived from $^{18}$F-FDG PET/CT in patients with EC. Notably, higher pretreatment (≥ 2.4) and posttreatment (≥ 3.4) nodal SUV-max values were independently and significantly associated with increased mortality, emphasizing their prognostic potential. These findings reinforce the clinical value of PET/CT imaging in the management of EC, particularly in stratifying patients based on metabolic tumor burden.

However, the existing literature presents a varied representation. Some studies support the prognostic value of PET-derived parameters, while others report limited utility [16–18]. This inconsistency is not unique to EC. In other malignancies, such as head and neck cancers, FDG PET metrics have shown a limited prognostic value [19], whereas in cholangiocarcinoma, lung adenocarcinoma, and endometrial cancer, they have been found to significantly correlate with survival outcomes [20–22]. These interstudy disparities likely stem from multiple confounding variables that influence SUV-max values.

Importantly, SUV-max is influenced by several variables. Factors such as serum glucose levels, FDG uptake timing, tumor-intrinsic characteristics, and the heterogeneity of neoadjuvant chemoradiotherapy protocols across studies can all impact SUV measurements [23]. Additionally, the timing of the posttreatment PET/CT scan plays a critical role, as therapy-induced inflammation and necrosis may

Table 2: Optimal cutoff values for overall survival prediction.

| Inflammatory markers | AUC (95% CI) | Cutoff values | Sensitivity (%) | Specificity (%) |
|---|---|---|---|---|
| Pretreatment SUV tumor | 0.65 (0.48–0.82) | 7.5 | 83.3 | 60.0 |
| Pretreatment SUV node | 0.72 (0.53–0.91) | 2.4 | 58.3 | 86.7 |
| Posttreatment SUV tumor | 0.65 (0.46–0.84) | 1.7 | 58.3 | 73.3 |
| Posttreatment SUV node | 0.64 (0.44–0.84) | 3.4 | 33.3 | 100.0 |

*Note:* AUC = area under the curve.
Abbreviation: CI = confidence interval.

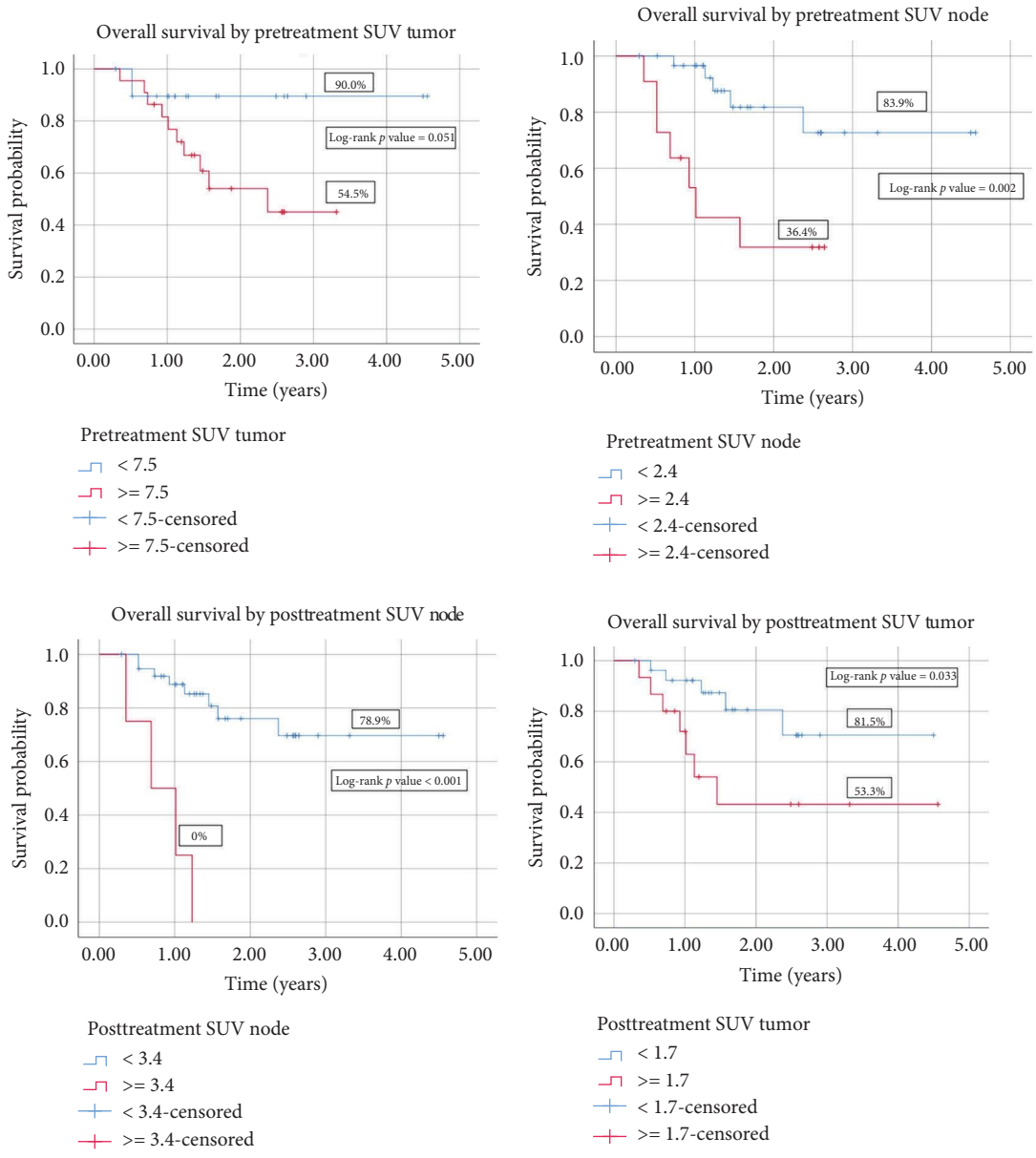


Figure 2: Overall survival by pre- and posttreatment SUV tumor size and node.

result in nonspecific FDG uptake, potentially mimicking residual tumor activity or masking viable disease [24]. Therefore, careful consideration and control of these factors are essential when interpreting PET/CT findings in clinical practice. In our cohort, all patients received a standardized neoadjuvant regimen comprising four cycles of chemotherapy (carboplatin, paclitaxel, cisplatin, and fluorouracil) along with 25–28 sessions of radiotherapy, followed by a posttreatment PET/CT conducted 8 weeks after completion of chemoradiotherapy.

High baseline SUV-max values have also been associated with more advanced tumor stages (cT3/4) and aggressive

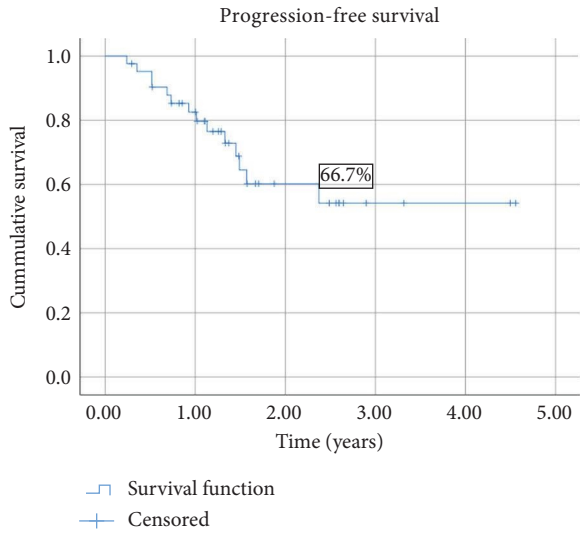


Figure 3: Progression-free survival of esophageal cancer patients.

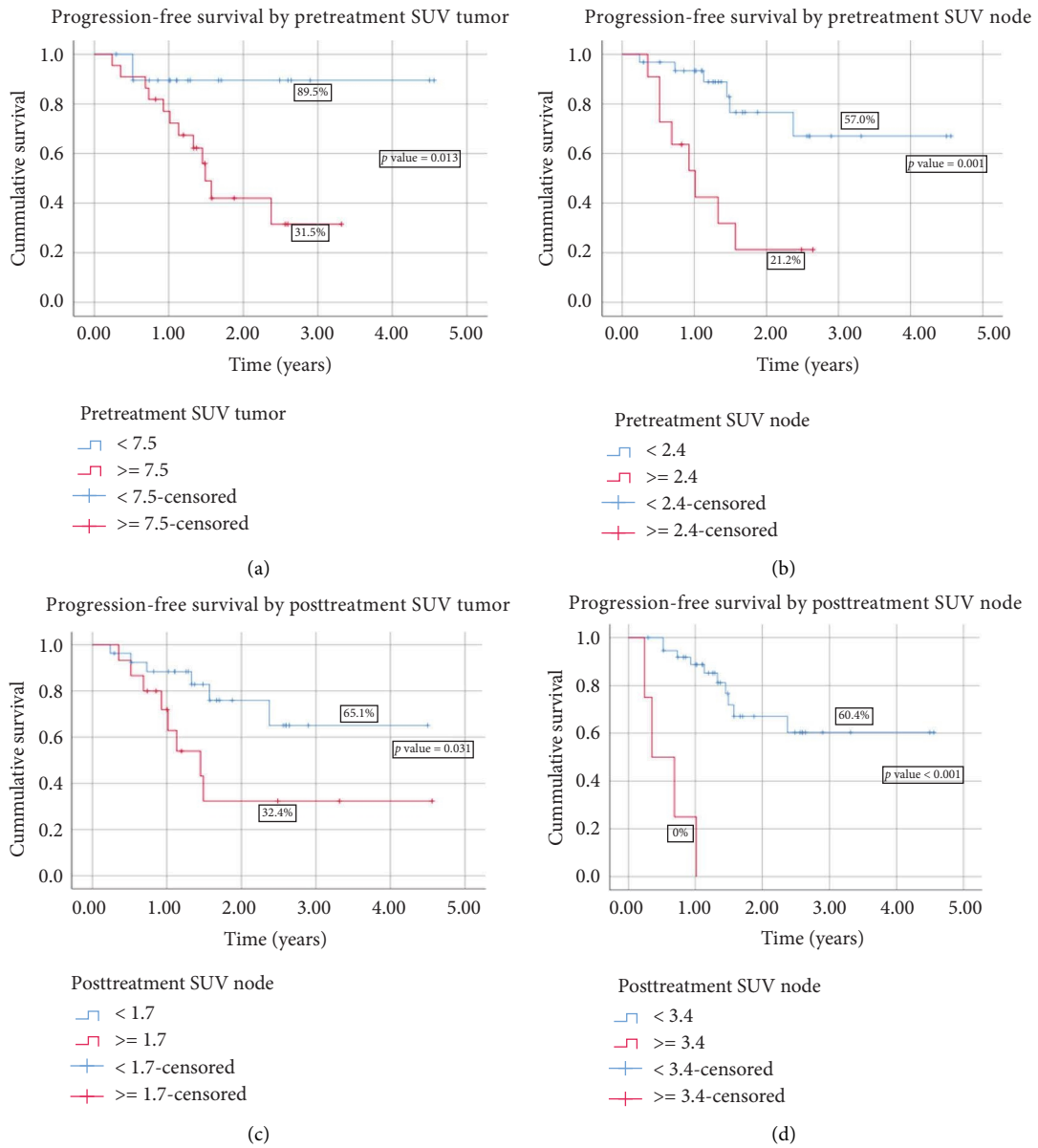


Figure 4: Progression-free survival by pre- and posttreatment SUV tumor and node.

Table 3: Cox regression analysis for the risk factors associated with mortality among patients.

| Characteristics | Model 0 HR (95% CI) | *p* value | Model 1 HR (95% CI) | *p* value | Model 2 HR (95% CI) | *p* value |
|---|---|---|---|---|---|---|
| Age, years | 1.02 (0.97,1.07) | 0.399 | — | | — | |
| *Gender* | | | | | | |
| Male | Ref | | | | | |
| Female | 1.18 (0.37,3.73) | 0.773 | — | | — | |
| *Tumor site* | | | | | | |
| Cervical, < 20 cm from incisors | Ref | | | | | |
| Upper thoracic, 20–25 cm from incisors | 0.21 (0.01,3.40) | 0.272 | — | | — | |
| Mid thoracic, 25–30 cm from incisors | 0.16 (0.01, 1.84) | 0.144 | | | | |
| Lower esophagus | 0.41 (0.05, 3.41) | 0.414 | | | | |
| Adenocarcinoma | Ref | | | | | |
| Squamous cell carcinoma | 1.25 (0.15, 9.85) | 0.832 | — | | — | |
| *Tumor grade* | | | | | | |
| Well differentiated | Ref | | | | | |
| Moderately differentiated | 0.64 (0.13, 3.15) | 0.591 | — | | — | |
| Poorly differentiated | 1.61 (0.22, 11.61) | 0.637 | | | | |
| *Tumor length radiological* | | | | | | |
| Less than 5 cm | Ref | | Ref | | Ref | |
| 5–10 cm | 1.28 (0.25, 6.47) | 0.759 | 0.78 (0.06, 8.96) | 0.844 | 2.26 (0.25, 19.86) | 0.460 |
| More than 10 cm | 4.20 (0.76, 23.03) | 0.098 | 0.06 (0.01, 2.59) | 0.148 | 0.03 (0.01, 1.55) | 0.082 |
| *T stage radiological* | | | | | | |
| T2 | Ref | | | | | |
| T3 | 2.58 (0.31, 21.10) | 0.375 | — | | — | |
| T4 | 2.24 (0.23, 21.69) | 0.484 | | | | |
| *N stage radiological* | | | | | | |
| N0 | Ref | | Ref | | Ref | |
| N1 | 1.76 (0.21, 14.51) | 0.599 | 0.36 (0.02, 5.48) | 0.463 | 1.21 (0.09, 15.93) | 0.883 |
| N2 | 6.49 (0.71, 59.30) | 0.097 | 2.58 (0.13, 48.52) | 0.525 | 0.87 (0.03, 24.66) | 0.939 |
| *Vascular involvement* | | | | | | |
| No vascular involvement | Ref | | Ref | | Ref | |
| Abutting aorta less than 90 degrees | 1.61 (0.33, 7.86) | 0.552 | 1.39 (0.08, 22.56) | 0.814 | 1.06 (0.07, 14.14) | 0.965 |
| Encasing aorta/abutting aorta more than 90 degrees | 5.50 (0.91, 33.05) | 0.062 | 10.42 (0.25, 432.03) | 0.217 | 49.14 (0.41, 5854.54) | 0.110 |
| *Pretreatment SUV tumor* | | | | | | |
| < 7.5 | Ref | | Ref | | | |
| ≥ 7.5 | 4.04 (0.88, 18.49) | 0.071 | 11.33 (0.82, 155.13) | 0.069 | — | |
| *Pretreatment SUV node* | | | | | | |
| < 2.4 | Ref | | Ref | | | |
| ≥ 2.4 | 5.28 (1.66, 16.78) | 0.005 | 15.34 (1.23, 191.15) | 0.034 | — | |
| *Posttreatment SUV tumor* | | | | | | |
| < 1.7 | Ref | | | | Ref | |
| ≥ 1.7 | 3.27 (1.02, 10.39) | 0.045 | — | | 3.57 (0.67, 18.94) | 0.135 |

Table 3: Continued.

| Characteristics | Model 0 HR (95% CI) | *p* value | Model 1 HR (95% CI) | *p* value | Model 2 HR (95% CI) | *p* value |
|---|---|---|---|---|---|---|
| *Posttreatment SUV node* | | | | | | |
| < 3.4 | Ref | | | | Ref | |
| ≥ 3.4 | 11.71 (3.09, 44.28) | < 0.001 | — | | 52.99 (2.39, 1174.28) | 0.012 |
| *Surgical procedure* | | | | | | |
| Not done | Ref | | Ref | | Ref | |
| Lap McKeown esophagectomy | 0.23 (0.03, 1.43) | 0.115 | 0.17 (0.01, 2.50) | 0.201 | 0.45 (0.02, 8.79) | 0.600 |
| Abandoned esophagectomy | 2.38 (0.45, 12.47) | 0.303 | 0.49 (0.05, 4.32) | 0.525 | 12.70 (0.50, 318.74) | 0.122 |
| Lap Ivor Lewis esophagectomy | 1.10 (0.09, 12.49) | 0.938 | 6.60 (0.32, 136.34) | 0.222 | 7.82 (0.26, 233.99) | 0.235 |

*Note:* Model 0 = unadjusted model; Model 1 = adjusted for pre-SUV parameters; Model 2 = adjusted for post-SUV parameters; multivariate models were adjusted for those variables whose *p* value > 0.25 in a univariate model.

Abbreviations: CI = confidence interval; HR = hazards ratio.

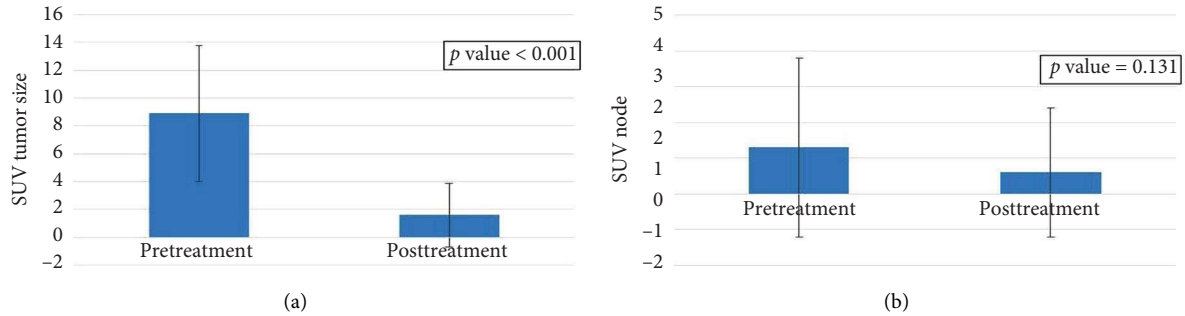


Figure 5: Pre- and posttreatment SUV tumor and node comparison.

pathological features, including lymph node metastasis and deeper invasion [25]. These observations align with the profile of our patient cohort, which includes fairly advanced-stage cases with higher initial/pretreatment SUV values (Table 1). Interestingly, tumors with higher initial SUV-max values often show a better metabolic response to preoperative treatment [16], and patients who achieve such responses tend to have significantly better survival than nonresponders [26, 27]. In our cohort, a substantial metabolic response was observed, with a prominent reduction in both primary tumor and nodal SUV-max values posttreatment (Figure 5). This correlated with a favorable OS rate of 71.4% (Figure 1), underscoring the predictive value of SUV-max not only in baseline risk stratification but also in evaluating treatment efficacy.

A meta-analysis by Port et al. [28], encompassing 47 studies and 5504 patients, confirmed the prognostic value of pretreatment FDG-PET metrics in EC. Similarly, Hamai et al. [29] demonstrated that $^{18}$F-FDG PET predicts both treatment response and survival. SUV-max has also been linked to recurrence risk [30, 31]. Lai et al. [30] reported poorer overall (62% vs. 78%) and recurrence-free survival in patients with preoperative SUV-max > 5. In our cohort, the mean pretreatment tumor SUV-max among deceased patients was 10.5, placing most above this threshold. However, significance was not achieved, likely due to the overall aggressive disease profile of our cohort (Table 1).

Notably, 12 deaths occurred within our cohort, all involving patients with advanced disease (Clinical Stages III and IV) and higher mean pre- and posttreatment SUV-max values compared to surviving patients (Table 1). Surgical resection was performed in 29 patients (69%), while in the remaining cases, surgery was either abandoned or not pursued due to evolving complications, comorbidities, or concerns regarding treatment tolerance. Importantly, those who underwent surgery demonstrated a lower risk of mortality compared to patients who did not, as shown in Table 1, indicating that surgery itself was not a confounding factor for mortality in our analysis. These decisions were guided by a multidisciplinary evaluation during a tumor board to ensure individualized patient-centered care [31].

In this study, nodal SUV-max demonstrated a stronger and more consistent prognostic association with OS than tumor SUV-max (Figure 2), likely reflecting its ability to capture systemic disease burden and biologically aggressive behavior. Notably, this is the first study from South Asia to stratify and evaluate nodal SUV-max in this context, contributing novel insights into its clinical relevance.

Despite its strengths, the study has several limitations. Its retrospective design and relatively small sample size may restrict the generalizability of the findings. In particular, the high HR (HR = 52.9) observed for posttreatment nodal SUV-max in Cox regression was derived from a limited subgroup ($n = 12$) and should therefore be interpreted with caution due to potential overestimation and reduced model stability. Additionally, although efforts were made to standardize imaging protocols, interpatient physiological variability may have influenced SUV measurements.

Nevertheless, these findings underscore the prognostic utility of SUV-max in EC, particularly in South Asia, and highlight its role in clinical risk stratification. Future prospective studies with larger, well-defined cohorts are warranted to validate these results and further define the role of metabolic imaging in guiding treatment decisions.

## 5. Conclusions

SUV-max on PET/CT is a strong, independent predictor of survival in EC and may effectively guide personalized treatment decisions.

## Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

## Ethics Statement

The study was conducted according to the guidelines of the Declaration of Helsinki, approved by the Institutional Ethical Review Board of DUHS (Approval #IRB-2780/DUHS/1148).

## Disclosure

The authors have completed the STROBE reporting checklist.

The authors are accountable for all aspects of the work (if applied, including full data access, integrity of the data, and the accuracy of the data analysis) in ensuring that questions related to the accuracy or integrity of any part of the work are

appropriately investigated and resolved. All authors have read and agreed to the published version of the manuscript.

## Conflicts of Interest

The authors declare no conflicts of interest.

## Author Contributions

Sajida Qureshi: methodology; Saba Mughal and Waqas Ahmad Abbasi: formal analysis; Saba Mughal: data curation; Mehreen Ansari, Raheel Ahmed, Mubashir Iqbal, Hira Abdul Jalil, Hanieya Saiyed, and Waqas Ahmad Abbasi: writing–original draft preparation; Waqas Ahmad Abbasi, Sajida Qureshi, Saba Mughal, Mehreen Ansari, Raheel Ahmed, Mubashir Iqbal, Hira Abdul Jalil, Hanieya Saiyed, and Muhammad Saeed Quraishy: writing–review and editing; Sajida Qureshi and Muhammad Saeed Quraishy: supervision.

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