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Standardised FDG uptake: A prognostic factor for inoperable non-small cell lung cancer

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Abstract

The aim of this study was to investigate the relationship between standardised uptake value (SUV) obtained from [^{18}F]fluorodeoxyglucose positron emission tomography (FDG PET) and treatment response/survival of inoperable non-small cell lung cancer (NSCLC) patients treated with high dose radiotherapy. Fifty-one patients were included recording stage, performance, weight loss, tumour volume, histology, lymph node involvement, SUV, and delivered radiation dose. The maximum SUV (SUV_{max}) within the primary tumour was a sensitive and specific factor for predicting treatment response. Apart from SUV_{max}, stage and performance were also independent predictive factors for treatment response. In a multivariate disease-specific survival (DSS) analysis, SUV_{max} ($P = 0.01$), performance status ($P = 0.008$) and stage ($P = 0.04$) were prognostic factors. For overall survival (OS), SUV_{max} ($P = 0.001$) and performance ($P = 0.06$) were important prognostic factors. SUV_{max} was an important prognostic factor for survival of inoperable NSCLC patients and a predictive factor for treatment response. Although the number of patients was small, the treatment was not homogeneous and the use of FDG SUV may have had constraints, we still conclude that the FDG SUV is potentially a good indicator for selecting patients for different treatment strategies.

Keywords: Prognosis; Radiotherapy; NSCLC; FDG PET; SUV

1. Introduction

TNM stage, performance status and weight loss are important prognostic factors used to stratify non-small cell lung cancer (NSCLC) patients for the most optimal treatment regimen [1]. However, these factors do not always provide a satisfactory explanation for differences in outcome between patients. Molecular markers and other features, such as tumour doubling time, are also closely related to prognosis [2]. These factors are, however, not always available in cases of inoperable NSCLC patients (e.g., no pathology). A non-invasive prognostic classifier may improve selection of inoperable NSCLC patients for individually adapted therapy (dose escalation, chemoradiation) and to potentially improve their poor prognosis.

Neoplastic cells demonstrate upregulation of glucose metabolism in order to obtain energy needed for proliferation [3]. Consequently, uptake of glucose or glucose analogues like deoxy-glucose is increased. Labelling deoxy-glucose with the positron emitting radionuclide ^{18}F to form [^{18}F]fluorodeoxyglucose (FDG) renders these cells detectable using positron emitting tomography (PET) as FDG is not a substrate for hexokinase and is therefore trapped inside the cells. The standardised

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uptake value (SUV) is a semi-quantitative method for assessing glucose metabolism, which is often used in clinical studies. Higher SUVs were observed in NSCLC with higher proliferation rates [4–6]. Moreover, it was demonstrated that SUV was a significant prognostic factor in the survival analysis of NSCLC patients [7–12].

The purpose of the present study was to investigate whether SUV could also be used as a classifier for predicting which patients will have favorable treatment response and to assess the significance of SUV as a prognostic factor in survival analysis of inoperable NSCLC patients treated with high dose radiotherapy.

2. Patients and methods

2.1. Patients

Patients included in this study were diagnosed and treated at the Department of Radiation Oncology in The Netherlands Cancer Institute – Antoni van Leeuwenhoek Hospital. The FDG scan was performed at the Department of Nuclear Medicine and PET research of the VU University Medical Centre, Amsterdam. PET imaging was requested for the evaluation of mediastinal lymph node involvement and as a screening method for the presence of clinically unsuspected distant metastasis in patients diagnosed with NSCLC. For patients with small progressive peripheral lesions on computed tomography (where histology or cytology was not feasible) increased FDG uptake was used as a criterion to confirm diagnosis.

From January 1999 and November 2001, data from 60 consecutive patients meeting the eligibility criteria were evaluated in the present analysis. Patient criteria included having a histologically proven NSCLC or a progressive lesion on computed tomography with increased FDG uptake on the PET image. Both transmission and emission scan were required to analyse the SUV retrospectively. Nine of the 60 patients were suspected to have distant metastases from the PET images and were excluded from the study as curative therapy was not feasible. Follow up of patients was closed on 31 July 2004.

Recorded characteristics were age, gender, performance status, histology, tumour volume, weight loss, treatment response, and tumour and lymph node stage. Weight loss was scored into three groups defined as 0–5%, 5–10% or >10% loss of the original weight during 3 months prior to therapy. Treatment response was scored 3 months after treatment and defined according to the Response Evaluation Criteria in Solid Tumours (RECIST) criteria [13]. The performance status of the patient was defined according to the World Health Organization (WHO) criteria.

The mean age of the 51 patients (34 men and 17 women) was 69 years. Fifteen patients had WHO performance status 0, 33 performance status 1, and only 3 patients performance status 2, respectively. Stage I disease was diagnosed in 21 patients and 11 patients had stage II disease. These patients were medically inoperable or refused surgery. Nineteen patients had stage III disease and were technically inoperable. Concerning the lymph nodes, 23 patients had N0 disease, 10 patients N1 and 18 patients proved to have N2–3 disease based on CT and PET (Table 1).

Tumour volume was computed as the volume of the primary tumour delineated by the radiation oncologist on the pre-radiotherapy CT scan. The mean tumour volume was 80 cm^3 (range: 4.2–455.6 cm^3). The smallest and largest tumour had a maximum diameter of 1.5 and 12.3 cm, respectively. Pathology was not available for 11 patients with small peripheral tumour, which was progressive on CT and positive on PET. Seventeen patients had squamous cell carcinoma, 13 patients ade-

Table 1
Patient characteristics

Characteristic	Number of patients
	Mean (range)
Total	51
Age	69 (32–88)
Gender	
Male	34
Female	17
WHO performance status	
0	15
1	33
2	3
Stage	
I	21
II	11
III	19
Lymph node	
N0	23
N1	10
N2–3	18
Tumour volume (cm^3)	80.0 (4.2–455.6)
Histology	
Squamous cell	17
Adenocarcinoma	13
Large cell	10
Unknown	11
SUV_{max}[a]	17.0 (3.4–40.8)
Chemotherapy	
Sequential	6
Concurrent	4
Radiotherapy dose (Gy)	77.0 (60.0–94.5)

[a] SUV_{max} is the maximum standardised uptake value within the primary tumour.

nocarcinoma and 10 patients large cell carcinoma (Table 1).

Thirty-four patients were included in a Phase I/II dose escalation study [14] and were treated with doses between 60.8 and 94.5 Gy (2.25 Gy per fraction, fixed overall treatment time of six weeks). Six of these dose escalation patients had stage IIIA or IIIB NSCLC and received induction chemotherapy (gemcitabin with either carboplatin or cisplatin). Four patients received 66 Gy (2.75 Gy per fraction) with concurrent daily intravenous cisplatin (6 mg/m^2). Two of those patients had stage IIIB NSCLC and 2 patients had stage IIB NSCLC. Three patients received 70 Gy in 2 Gy fractions. Ten patients were irradiated with a total dose of 67.5 Gy in 2.25 Gy per fraction. The mean dose was 77 Gy (range 60–94.5 Gy) (Table 1). PET positive lymph nodes were included in the gross tumour volume together with the primary tumour. Therefore, these nodes were irradiated to the same dose as the primary tumour.

2.2. FDG-PET

PET imaging was performed with a median of 19 days (minimum 4, maximum 66 days) before start of treatment. Scans were performed on a dedicated ECAT EXACT HR+ PET scanner (Siemens/CTI, Knoxville, TN, USA). This scanner has an axial field of view of 15 cm, divided into 63 contiguous planes.

All patients fasted for at least 6 h before scanning. FDG was injected in the arm contra lateral to the tumour and prior to injection a blood sample was taken from this arm for the serum glucose measurement.

Approximately 60 min after injection of FDG (5.5 MBq × body weight, with a minimum of 340 MBq, and a maximum of 550 MBq), an emission scan (2D mode), and a 10–15 min transmission scan were acquired.

The FDG scan was corrected for dead time, decay, scatter, random coincidences and photon attenuation. Scans were reconstructed using ordered subset expectation maximisation (OSEM) with 2 iterations and 16 subsets, followed by post-smoothing of the reconstructed image using a Hanning 0.5 filter. An image matrix size of 256 × 256 was used.

2.3. Standardised uptake value (SUV)

FDG uptake was quantified using SUV. In this study, SUV_{max} was defined as the maximum tumour concentration of FDG divided by the injected dose and corrected for the body weight of the patient: [SUV = maximum activity concentration/(injected dose/body weight)]. For the determination of the SUV_{max}, the maximum FDG-uptake was searched within the region of the primary tumour on the PET image. This region was manually drawn. The mean SUV (SUV_{mean}) was defined as the mean concentration of FDG divided by the injected dose and corrected for the body weight of the patient. The mean FDG-uptake was determined by a threshold method, whereby the mean of all pixel values above 50% of the maximum value was calculated. Assuming a normal fasting plasma glucose concentration [Glc] of 100 mg/dl (i.e. 5.55 mmol/L), both SUVs were corrected for glucose by multiplying the SUV with the measured glucose concentration divided by this normal value [15].

2.4. Statistical analysis

For the disease-specific survival (DSS), an event was defined if lung cancer was the cause of death. One patient with a partial response developed metastasis 5 months after treatment and received palliative chemotherapy, which was complicated by renal insufficiency. For this patient, renal failure was the cause of death, but it was still defined as an event in the DSS analysis.

Death of any cause was defined as an event in the overall survival (OS) analysis. Survival time was defined as the time interval between the date of treatment and an event. These events were censored in the DSS analysis.

Correlations between two variables were calculated with the Pearson correlation coefficient. Logistic regression analysis was performed to assess which factors were significant explanatory variables to predict treatment response. The sensitivity and specificity of the SUV to predict therapy response was evaluated using the receiver operating characteristic (ROC) curve. Survival probabilities were estimated using the Kaplan–Meier method. Significance of the difference between groups with respect to the studied parameters was assessed using the log-rank test. To assess the joint effects and interactions of the significant variables in the univariate analysis, multivariate analysis was carried out with the Cox proportional hazards model. A significance level of 0.05 was used for covariate entry. To avoid "over-interpretation" of the SUV analysis, the median SUV was used for univariate analysis and SUV was incorporated as a continuous variable in the multivariate analysis.

3. Results

3.1. Treatment response and follow up

Thirty-three percent ($n = 17$) of the patients experienced a complete response (CR). These patients had a median survival of 38 months. Fifty percent ($n = 25$) of the patients had a partial response (PR) with a median survival of 14 months. Stable disease (SD) was achieved in 8% ($n = 4$, 10 months median survival) of the patients and 10% ($n = 5$) of the patients suffered from progressive disease (PD, 9 months median survival) (Table 2).

Table 2
Response and follow-up data of all patients

Response	Number of patients	No evidence of disease	Local progression of disease	Metastasis	Death due to disease	Death due to other cause	Median survival (months)
Complete response							38
	15	10				5	
	1		1				
	1			1	1		
Partial response							14
	9					4	
	7		7		6	1	
	5			5	5		
	4		4	4	3	1	
Stable disease							11
	2			1	1	1	
	2		2	2	1		
Progressive disease							9
	5				4		
Total	51	10	14	13	21	12	Overall median 17

The overall median follow-up was 17 months (range: 3–57 months). Of the 17 patients with CR, one developed local progression 6 months after treatment and one patient developed distant metastasis at 24 months follow up. Sixteen of the 25 patients with PR developed disease progression and/or metastases, diagnosed nine months (median) after treatment. Ten months (median) after treatment, 3 of the 4 patients with SD developed metastases and 5 patients did not respond to therapy (PD). For 21 patients, lung cancer was the cause of death after a median survival time of 12 months. Twelve patients died from other causes than lung cancer. Six patients died of vascular diseases, 3 patients of respiratory diseases and 3 patients from other causes. These 12 patients had a median survival of 14 months. At the end of the study the 19 surviving patients had a median survival time of 24 months.

3.2. Standardised uptake value

The median SUV_{max} was 15 (mean = 17, SD = 9) and the minimum and maximum SUV_{max} values were 3 and 41, respectively. No statistical correlation was observed between tumour volume and SUV_{max} ($r = 0.17$, $P = 0.2$), and between lymphnode status and SUV_{max} ($r = 0.15$, $P = 0.3$). The mean SUV_{max} for squamous cell carcinoma was 15. For adenocarcinoma and large cell carcinoma, SUV_{max} was 16 and 20, respectively. The mean SUV_{max} was 17 for the 11 patients with unknown histopathology. Stage I and II patients had a mean SUV_{max} of 14. Stage III patients had a mean SUV_{max} of 16. The median SUV_{mean} was 9 (mean = 11, SD = 6) and the minimum and maximum SUV_{mean} values were 2 and 25, respectively.

3.3. Univariate response analysis

The ability of SUV_{max} to predict initial therapy response is depicted by the receiver operating curve (ROC) curve shown in Fig. 1. Using the median SUV_{max} of 15 yielded a sensitivity of 77% and specificity 84% in predicting CR. The area under the curve of 82% (95% confidence interval (CI), 69–95%) is indicative of the level of accuracy (Fig. 2). Stage ($P = 0.02$), performance status ($P = 0.04$) and positive lymph nodes ($P = 0.04$) were significant factors correlated with CR. In contrast, chemotherapy, dose, tumour volume, weight loss and histology were not significant.

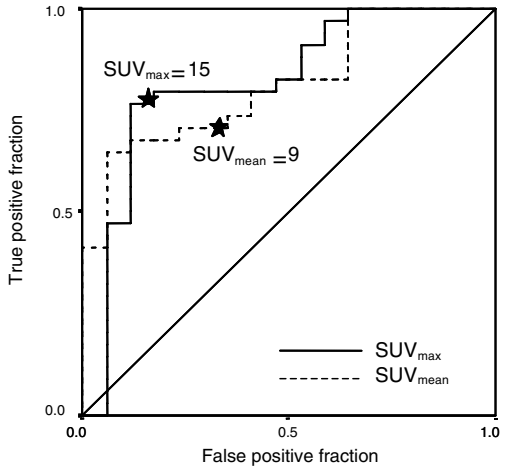

Fig. 1. Receiver operating characteristic curves using standardised uptake value (SUV) to predict complete response. The maximum SUV (SUV_{max}) and the mean SUV (SUV_{mean}) were tested.

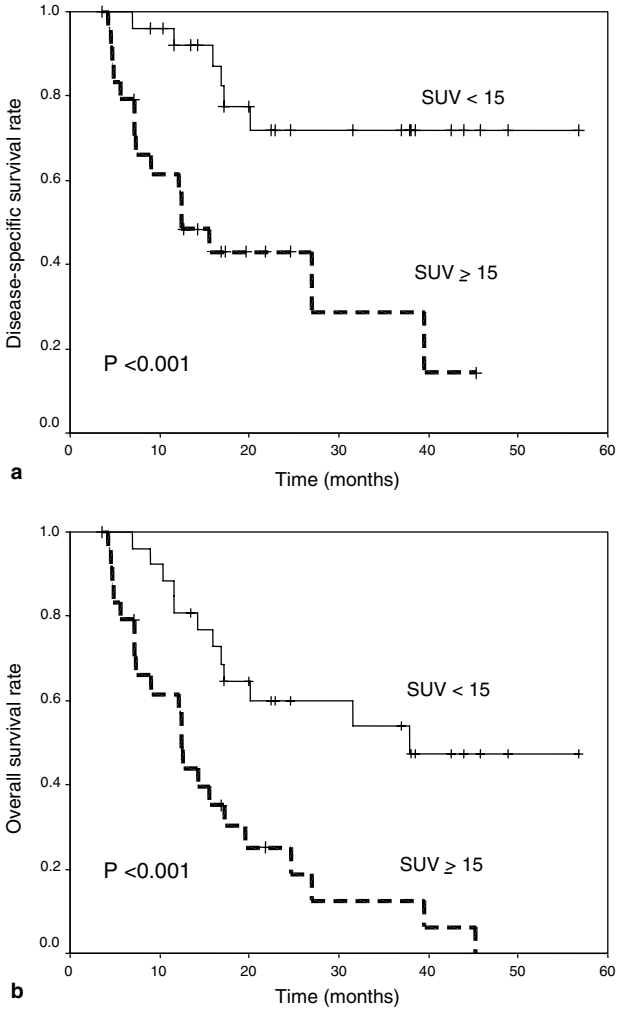

Fig. 2. Kaplan–Meier survival curves for the disease-specific survival and the overall survival and the P values of the log-rank test.

The median SUV_{mean} had a sensitivity of 71% and a specificity of 65% to predict CR, and the area under the curve was 77 % (95% CI, 63–91%) (Fig. 1).

3.4. Multivariate response analysis

To assess whether SUV was an independent predictive factor for treatment response, logistic regression analysis was used. Because the predictive value of SUV_{max} was better than the predictive value of SUV_{mean}, we included only SUV_{max} in the multivariate analysis. SUV_{max} was incorporated as a continuous parameter. As stage was strongly correlated with the lymph node status ($r^2 = 0.86$, $P < 0.001$) only SUV_{max}, stage and performance status (and not the lymph node status) were included in this model.

Stage ($P = 0.02$), performance status ($P = 0.01$) and SUV_{max} ($P = 0.05$) were independently associated with complete response (Table 3). The odds ratio (OR) of SUV_{max} was 1.13, which number indicates the relative increase in odds of having no complete response when the SUV_{max} value increased by one-unit. The ORs of stage and performance status were 3.29 and 6.92, respectively (Table 3).

Table 3
Logistic regression analysis to predict treatment response

	Odds ratio	95% CI[a]	P
SUV_{max}[b]	1.13	1.01–1.27	0.05
Stage, I–II *vs.* III	3.29	1.23–8.77	0.02
Performance (WHO), 0 *vs.* 1 *vs.* 2	6.92	1.53–31.32	0.01

[a] 95% Confidence interval of the odds ratio.
[b] SUV_{max} is the maximum standardised uptake value within the primary tumour; SUV_{max} is incorporated as a continuous variable.

3.5. Univariate survival analysis

Kaplan–Meier plots for the disease-specific survival (DSS) and overall survival (OS) illustrate large survival differences between patients with $SUV_{max} < 15$ and $SUV_{max} \geqslant 15$ in favour of the low SUV_{max} group (Fig. 2). Survival differences were statistically significant for both the DSS ($P < 0.001$) and OS ($P < 0.001$) (Table 4). The survival benefit for patients of the low SUV_{mean} group ($SUV_{mean} < 9$) was less significant than the survival benefit observed for the low SUV_{max} group (DSS, $P = 0.04$ and OS, $P = 0.02$). For DSS and OS, median survival times were 39 and 17 months, respectively. The 2-year survival rates for DSS and OS were 57% and 43%, respectively. Stage ($P = 0.04$), performance status ($P = 0.01$), lymphnode status ($P = 0.04$), treatment response ($P = 0.02$), chemotherapy ($P = 0.01$) and tumour volume ($P = 0.03$) were significantly associated with DSS. A trend for a better DSS was observed in patients irradiated with doses of 70 Gy or higher compared with patients irradiated with doses lower than 70 Gy ($P = 0.06$).

Significant factors for OS were performance status ($P = 0.04$), chemotherapy ($P = 0.01$) and treatment response ($P = 0.01$) (Table 4). Weight loss and histology were not significant factors.

3.6. Multivariate survival analysis

For the multivariate survival analysis the significant variables in the log-rank test ($P < 0.05$) were examined in the Cox proportional hazards model to evaluate their interaction and joint effect on DSS and OS. The SUV_{max} was included in this analysis because SUV_{max} was more significant in the log-rank test than the SUV_{mean}. The SUV_{max} was incorporated as continuous parameter. We excluded the treatment response as a parameter in the multivariate analysis, since this parameter is not known prior to therapy. Chemotherapy, tumour volume and lymph node status were not included, because of the strong correlation of these factors with stage.

Table 4
Univariate analysis of the disease-specific survival and overall survival

Factor	Number of patients	Disease-specific survival			Overall survival		
		Median survival (months)	2-Year survival (%)	Log-rank (*P*)	Median survival (months)	2-Year survival (%)	Log-rank (*P*)
Total group	51	39	57		17	43	
SUV_{max}							
<15	26	NR	72	<0.001	38	60	<0.001
⩾15	25	12	43		12	27	
Treatment response							
Complete	17	NR	92	0.02	38	50	0.01
Partial	25	12	40		14	26	
Stable/progressive disease	9	12	41		10	33	
Stage							
I, II	32	NR	68	0.04	25	50	0.3
III	19	16	41		16	32	
Performance (WHO)							
0	15	NR	81	0.01	38	61	0.04
1	33	20	51		16	39	
2	3	12	0		12	0	
N stage							
N0	23	NR	72	0.04	25	54	0.2
N+	28	16	44		16	34	
Chemotherapy							
No	41	NR	64	0.01	20	50	0.01
Yes	10	12	26		12	13	
Dose (Gy)							
<70	17	17	42	0.06	16	37	0.4
⩾70	34	NR	65		20	47	
Tumour volume (cm^3)							
⩽18	17	NR	69	0.03	12	56	0.3
(19, 74)	17	NR	67		17	44	
⩾75	17	12	36		31	34	
Weight loss (%)							
<5	41	17	60	0.6	17	42	0.8
(5–10)	10	40	48		17	48	
>10	0						
Histology							
Squamous	17	NR	50	0.5	14	32	0.8
Adenocarcinoma	13	27	47		20	39	
Large cell	10	20	60		17	50	
Unknown	11	NR	79		31	64	

NR, not reached.

For DSS, all significant factors in the univariate analysis (SUV_{max}, performance status and stage) remained significant in the Cox proportional hazard model (SUV_{max}, $P = 0.01$; performance status, $P = 0.008$; stage, $P = 0.04$, Table 5). The hazard ratio (HR) of SUV_{max} was 1.06, which indicated that a one-unit increase of SUV_{max} correspond to a 6% increase of hazard of lung cancer related death. The HRs for stage and performance status were 1.6 and 3.86, respectively.

For OS, SUV_{max} ($P = 0.001$) and performance status ($P = 0.06$) remained significant factors in the Cox proportional hazard model (Table 6). Equally to the DSS, a one-unit increase of SUV_{max} corresponded with a 6% increase of hazard of death due to any cause.

Table 5
Cox proportional hazards model for the disease-specific survival

	Hazard ratio	95% CI[a]	*P*
Performance (WHO), 0 *vs.* 1 *vs.* 2	3.86	1.43–10.43	0.008
Stage I, II *vs.* III	1.60	1.02–2.51	0.04
SUV_{max}[b]	1.06	1.01–1.10	0.01

[a] 95% Confidence interval of the hazard ratio.
[b] SUV_{max} is the maximum standardised uptake value within the primary tumour; SUV_{max} is incorporated as a continuous variable.

Table 6
Cox proportional hazards model for the overall survival

	Hazard ratio	95% CI[a]	*P*
Performance (WHO), 0 *vs.* 1 *vs.* 2	1.93	1.41–3.86	0.06
SUV_{max}[b]	1.06	1.02–1.10	0.001

[a] 95% Confidence interval of the hazard ratio.
[b] SUV_{max} is the maximum standardised uptake value within the primary tumour; SUV_{max} is incorporated as a continuous variable.

4. Discussion

In the present study, FDG SUV_{max} was predictive for treatment response and the median SUV_{max} was a good variable to predict complete response in inoperable NSCLC patients. The multivariate survival analysis showed that SUV_{max} was an explanatory prognostic factor for both disease-specific (DSS) and overall (OS) survival. We could not perform a disease free survival analysis because only 2 patients with complete response ($n = 17$) developed metastases or progression of disease.

Differences in patients selection makes it difficult to compare our results with previous studies. Most studies included mainly surgically treated NSCLC patients and observed better survival rates than in the present study [8–10]. Ahuja and colleagues [11] found poorer survival rates however, 20% of those patients had stage IV disease and were treated palliatively. Only Sasaki [7] evaluated the prognostic value of SUV_{max} for radiotherapy patients. They found that a cut off value of 5 (median SUV_{max} was 8) provided the most significant survival difference between patients above and below this cut off value. Their radiotherapy patients had a high 2-year OS of 71 %, which was not different from the 2-year OS of surgically treated patients. The importance of SUV for inoperable NSCLC patients was confirmed in our analysis, where SUV_{max} was statistically associated with both treatment response and survival. The lower survival rate in the present study may be explained by both higher SUV_{max} values and higher incidence of medical inoperability observed in our patients. Downey [10] also incorporated SUV_{max} as a continuous variable in the Cox proportional hazards model, and observed a 7% increase in hazard of death after a one-unit increase in SUV_{max}. This is in agreement with the present data. It is important to note that Downey and colleagues observed SUV_{max} values in the same range as in the present study.

Difference in PET scanning techniques is another potential problem in comparing studies that attempt to evaluate the prognostic value of SUV. Differences in injected FDG-dose, scanning time (i.e. time after injection), reconstruction algorithms, filters, scanner characteristics, sinogram noise and quantification methods might lead to (structural) inter-institutional SUV differences [16].

Even the calculation of a SUV may differ between SUV studies. SUV_{mean} calculated with a threshold method was shown to have slightly better reproducibility than the SUV_{max}. However, the SUV_{mean} might include pixel values of non-tumour tissue [17]. We observed that the SUV_{max} was better predictive for treatment response than the SUV_{mean}, which might be due to the fact that the maximum SUV represents the most metabolic active (i.e. most aggressive) part of the tumour better.

Another factor that influences the SUV is the level of plasma glucose of the patient during PET scanning. Two studies observed a reduced variability and improved reproducibility of SUV after glucose correction [18,19]. In our study, we observed a similar predictive value for both glucose corrected SUV and uncorrected SUV and for survival analysis also, glucose correction did not influence the results (data not shown).

Notwithstanding the mentioned constraints of SUV, it is a clinically feasible and often used quantification parameter of PET images. Hoekstra compared different SUV calculation methods with the non-linear regression method that is used as a golden standard to quantify FDG uptake (but which is not clinically feasible) [20]. To verify the best possible quantification method with the appropriate cut off values for NSCLC, similar studies are required with prospective clinical patients data. In addition, PET-scanning techniques and quantification methods should be more uniform, before guidelines for general use of a particular SUV can be implemented [21].

Currently, the application of FDG PET in the treatment of NSCLC patients has become increasingly important. First, because it can detect otherwise unknown metastases and distinguish benign from malignant lymphnodes to improve staging, and thereby potentially also the choice of treatment for NSCLC patients [22,23]. Second, FDG PET is used in inoperable NSCLC patients to obtain information that is important in delineating gross tumour volume [24]. Lastly, studies have shown that PET is a sensitive method for evaluating tumour response [25], even more accurate than response monitoring by CT [26]. Apart from these diagnostic, treatment and response monitoring purposes, the prognostic value of SUV adds another dimension to PET imaging of inoperable NSCLC patients. Prognostic information obtained from a tumour biopsy is not always available in inoperable NSCLC patients (in this study pathology was not avaliable in 11 patients). Pathology, tumour doubling time, glucose transporter proteins (Glut 1 and Glut 3) and proliferation markers (Ki-67) are prognostic factors obtained from biopsy material [1,27,28] and SUV was associated with them. Squamous cell carcinoma had a higher SUV than adenocarcinoma [10,12] and strong correlations were observed between SUV and tumour doubling time, glucose transporters (Glut1 and Glut 3) and the proliferation marker Ki-67 [4,6,29].

SUV could have an important impact for inoperable NSCLC patients as their prognosis is poor. Although the poor prognosis is also due to the co-morbidity in inoperable NSCLC patients, better selection for individually adapted treatment strategies will undoubtedly improve outcome. SUV has shown to accurately predict treatment response and may contribute to select patients for the appropriate treatment strategies. Quantification of PET images, in combination with conventional prognostic factors, might indicate which patients are appropriate candidates for more aggressive treatment strategies (chemoradiation, dose escalation) or should better be treated in a palliative setting.

To conclude, this study has shown that FDG SUV_{max} was an important complementary prognostic factor for survival in 51 irradiated inoperable NSCLC patients. Moreover, this SUV provided good prediction of response to radiotherapy. In current diagnostic and treatment settings of (inoperable) NSCLC patients, the use of FDG PET imaging is increasing. The FDG SUV_{max} obtained from these images might help to determine the most appropriate treatment strategy, and consequently improve treatment efficiency. Due to the limited number of patients in this study, a prospective clinical study with a larger group of patients is necessary to determine the optimal cut off values.

Conflict of interest statement

None declared.

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