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Correlation of molecular response as measured by 18-FDG PET with outcome after chemo-radiation in patients with esophageal carcinoma

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Abstract

Purpose—Determine if 18-FDG PET-CT scans predict pathologic complete response, disease-free and overall survival in patients with esophageal carcinoma undergoing definitive or pre-operative chemoradiation.

Material & Methods—Patients with esophageal carcinoma presenting for definitive or pre-operative treatment undergoing pre- and post-treatment 18-FDG PET-CT scans were retrospectively reviewed. Histology, T-stage, nodal status, radiation dose, days from end of radiation to PET scan and surgery were the variables investigated to determine a relationship to baseline SUV of the primary tumor at the time of diagnosis. We also attempted to determine if a relationship existed between % decrease SUV and pathologic complete response, overall and disease-free survival.

Results—Eighty-one patients, 14 female and 67 male, underwent 18-FDG PET-CT scanning prior to treatment and 63 had post-treatment scans. T-stage and tumor location predicted in univariate but not multivariate analysis for initial SUV. Sixty-six percent of patients with a post-chemoradiation

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SUV <2.5 had tumor seen in the surgical specimen and 64% of patients had positive lymph nodes at surgery not imaged on the post-chemoradiation PET scan. A trend existed for post-treatment SUV and days from radiation to surgery to predict for pathologic complete response, p=0.09 and p=0.08, respectively. Post-treatment SUV predicted for disease-free survival in the definitive chemoradiation group, p=0.01.

Conclusion—A correlation existed between depth of tumor invasion and baseline SUV level. Post-treatment SUV predicted for disease-free survival in the definite chemoradiation group. Caution should be exercised in utilizing post-treatment PET scans to determine the necessity of surgical resection.

Keywords

Molecular imaging; esophageal cancer; chemoradiation

Introduction

Molecular imaging is playing an increasing role in the management of patients with esophageal carcinoma. However, the use of PET scanning in assessing treatment response is not completely defined. Downey et al. reported 15% of patients with esophageal cancer with undetected sites of metastatic disease after standard staging studies were found to have metastatic disease with PET scans.[1] Re-staging PET scans after induction chemoradiation, however, failed to predict disease progression. Two-year disease-free survival and overall survival was marginally improved in patients who underwent a R_0 esophagectomy who had a >60% decrease in Standardized Uptake Value (SUV) after induction chemoradiation. Weber et al. reported on the predictive response of PET scans in 40 patients with adenocarcinoma of the esophagus receiving induction chemotherapy prior to esophagectomy.[2] Patients without a metabolic response in the PET scan 14 days after induction chemotherapy exhibited a shorter time to progression/recurrence and a shorter overall survival compared to patients with a metabolic response.

The optimal treatment for patients with locally advanced esophageal cancer is not known with certainty. Pre-operative chemoradiation prior to surgery has been compared to chemoradiation alone in two recent randomized trials.[3, 4] Both trials concluded that overall survival was equivalent between the surgery and non-surgical arms although local control was improved in the surgical arm. Identification of patients at high risk for local failure who then could undergo immediate esophagectomy may spare some patients surgery.

This retrospective study had two goals, first, to determine if 18-FDG PET-CT scans can help predict pathologic complete response, disease-free and overall survival in patients with carcinoma of the esophagus undergoing pre-operative chemoradiation prior to planed esophagectomy. We hypothesize patients with a greater % decrease in SUV and lower post-treatment SUV will have a greater chance to experience a pathologic complete response. The second goal was to determine if 18-FDG PET-CT scans can predict overall and disease-free survival in patients receiving definitive chemoradiation. Because accurate pathologic response cannot be determine by biopsy alone, correlation of PET response with pathologic complete response was not attempted in this group. We hypothesize patients having lower pre and post-treatment SUV as well as a greater % decrease SUV will have longer disease-free and overall survival.

Material & Methods

Beginning in June 2002, patients diagnosed with adenocarcinoma or squamous cell carcinoma of the esophagus underwent PET/CT, in addition to standard staging studies including

esophageal ultrasound (EUS) and computed tomography (CT), prior to undergoing combined modality chemotherapy and radiation therapy at Fox Chase Cancer Center. The PET/CT scans were usually obtained on the same day as the CT simulation prior to the initiation of any cytotoxic therapy and were repeated 4–6 weeks after completion of treatment, prior to planned esophagectomy in patients undergoing pre-operative chemoradiation. Our PET/CT procedures have been published previously.[5] A maximum Standardized Uptake Value (SUV) was obtained from the tumor by a nuclear medicine physician (A.M.) without knowledge of the clinical outcome.

All patients underwent CT simulation with the PET scan images fused with the CT simulation images to determine the Gross Tumor Volume (GTV) and Planning Tumor Volume (PTV). Patients were initially treated with anterior/posterior (AP/PA) and posterior/anterior (PA) fields with 6 or 10 MV photon beams. An AP and two posterior oblique fields were incorporated into the treatment to limit the spinal cord to no more than 4500 cGy. Customized blocks were used to protect normal tissue. The usual field borders were 5 cm superior, 3 cm inferior and 2.5–3 cm lateral to the GTV as outlined by CT and PET scans. Chemotherapy regimen was at the discretion of the treating medical oncologist.

The clinical data were obtained retrospectively from chart review. Survival analysis was performed from the start of treatment. Patients dying of non-cancer related causes after completion of therapy without evidence of cancer were censored at the time of death. Patients with locally advanced disease receiving palliative radiation dying during treatment were coded as having local persistence of disease.

The Wilcoxon test was used to determine association of selected variables to initial tumor SUV.[6] The Spearman correlation was calculated to determine if a relationship existed between endoscopic length and SUV.[6] A generalized linear model was used with backward selection to find significant independent predictors for % decrease in SUV.[7] Logistic regression with backward selection was used to find significant predictors of no evidence of tumor in surgical specimen.[7] Finally, a Cox proportional hazards model with backward selection was used to investigate if an association existed between selected variables and disease-free survival.[8]

This research was reviewed and approved by the Institutional Review Board (IRB) of Fox Chase Cancer Center. All patient identifiers were removed prior to the analysis of the data.

Results

Eighty-one patients with esophageal cancer, 14 female and 67 male, underwent staging18-FDG PET-CT scans between June 2002 and July 2006 prior to initiation of definitive treatment. Endoscopic ultrasound was performed on 66 patients allowing T-stage determination with 5 T2, 56 T3 and 5 T4 tumors. The mean pre-treatment SUV was significantly lower in the T2 tumors, 3.6, compared to T3/T4 tumors, 10, p=0.005. There was no statistical difference in pre-treatment SUV when analyzed by stage or histology. The pre-treatment SUV of adenocarcinomas was 9.2 compared to 11.2 for squamous cell carcinomas, p=0.14. Tumor location did, however, influence pre-treatment SUV. Pre-treatment SUV for cancers in the cervical esophagus was 10.2, middle esophagus 11.0, lower esophagus 11.2, and GE junction 7.2, p=0.005. Factors predicting pre-treatment SUV in univariate analysis were T stage and tumor location but there were no significant independent predictors in multivariable analysis.

Pre-Operative Chemoradiation Group

Forty-four patients underwent esophagectomy, 3 female and 41 male. Forty-two patients had adenocarcinoma and 2 patients had squamous cell carcinoma. The primary site included 3 middle esophageal, 20 lower esophageal and 21 gastroesophageal primaries. Patients were

staged according to the AJCC 6th edition.[9] EUS was performed in 41 patients resulting in 2 T2, 36 T3, and 3 T4 tumors. Four patients were stage IIA, 2 stage IIB, 27 stage III, and 9 stage IVA.

The median radiation dose was 4,500 cGy range: (720–5,040 cGy). All patients received chemotherapy: 33 cisplatin and 5-Fluorouracil (5-FU) (5-FU 1000mg/m^2/day continuous infusion for 96–120 hours and Cisplatin 75 mg/m^2 i.v. day 1 with this cycle repeated weeks 1 and 5). Eleven patients were also treated on an in-house phase II trial consisting of paclitaxel, cisplatin, and 5-FU Induction cisplatin 75mg/m^2 i.v. and paclitaxel 175mg/m^2 i.v. was given weeks 1 and 3 followed by cisplatin 25mg/m^2 i.v., paclitaxel 50mg/m^2 i.v., and 5-FU 200 mg/m^2/day continuous infusion 7 days a week during the 5-week course of radiation. A preliminary report of toxicity from this regimen has been previously reported.[10] The median follow-up after surgery was 14.1 (range: .67–49.4). The median survival of patients undergoing surgery was 16.7 (range: 3.5–50.5) months.

All patients in this group underwent attempted Ivor-Lewis esophagectomy (39 R0, 4 R1 and 1 R2 resection), 38 of which had pre and post chemoradiation 18-FDG PET-CT scans. The median % decrease in SUV from the initial to post-chemoradiation PET scan was 57% (range +15 to −86%). Radiation dose, T-stage, pre-treatment nodal status and days from the end of radiation to the second PET scan were not found to independently predict for % SUV decrease. The pathologic complete response was 25% in the patients undergoing esophagectomy. Radiation dose, T-stage, nodal status,, initial SUV, % SUV decrease, and endoscopic length were not found to be predictive of complete pathologic response. Multi-variate analysis found a trend for post-treatment SUV and days from the end of radiation to surgery to be predictive of pathologic complete response in the primary, p=0.09 and p=0.08 respectively. There was no significant difference between the mean % SUV decrease in patients with complete pathologic response, 50% (Std. error .289), and patients without a complete pathologic response, 53% (Std. error .133), p=0.90. Sixty-six percent of patients with a post-chemoradiation SUV <2.5 had persistent disease found on the pathologic specimen at the time of surgery. Sixty-four percent of patients with positive lymphadenopathy at the time of surgery had no evidence of lymphadenopathy seen on the post-chemoradiation PET scan. Mean initial SUV, mean post-treatment SUV, or mean % SUV decrease did not predict for site of failure..

Definitive Chemoradiation Group

Thirty-seven patients, 11 female and 26 male, did not undergo surgery because of advanced disease, medical contraindications to surgery or patient refusal. Twenty-four had adenocarcinoma and 13 squamous cell carcinoma. This anatomic distribution is as follows: 1 cervical esophagus, 12 middle, 12 lower and 12 gastroesophageal. There were 3 T2, 20 T3, and 2 T4 tumors. Accurate staging information was available on 27 patients resulting in the stage distribution of 6 IIA, 1 IIB, 13 III, 4 IVA and 3 IVB. Endoscopic ultrasound was not performed in patients referred from outside hospitals if it was determined that the treatment would not be changed regardless of the outcome of the endoscopic ultrasound. The median radiation dose in this group was 5,040 (range: 720–6,208) cGy. Thirty-two patients received the following chemotherapy; 20 cisplatin and 5-FU, (5-FU 1000mg/m^2/day continuous infusion for 96–120 hours and Cisplatin 75 mg/m^2 i.v. day 1 with this cycle repeated weeks 1 and 5), 8 5-FU (225 mg/m^2 continuous infusion daily during radiation), 1 5-FU (225 mg/m^2 continuous infusion daily during radiation) and paclitaxel (50 mg/m^2 intravenously weekly), 1 paclitaxel (50 mg/m^2 intravenously weekly during radiation), 1 paclitaxel, cisplatin, and 5-FU, (same chemotherapy schedule as pre-operative group) and 1 capecitabine (825 mg/m^2 orally b.i.d).

Twenty-five patients underwent a post-treatment PET scan with a median post-treatment SUV of 2.9 (range: 0–16.3) with the median %SUV decrease −65% (range: +254 to −91). As was seen in the pre-operative group, mean initial SUV, mean post-treatment SUV, or mean % SUV decrease did not predict for site of failure.. The median overall survival was 5.2 (range: .5–31.4) months with disease-free survival of 4 (range: 0–31.4) months. Univariate analysis revealed post-treatment SUV to significantly predict disease specific survival with one unit increase in post-treatment SUV increasing disease specific mortality by 30% (p=0.01). No variables, however, were significant in multivariate analysis.

Discussion

18-FDG PET-CT evaluation allows for an evaluation of cancer metabolic activity providing physicians with another modality to aid in the staging and evaluation of treatment response in patients with esophageal cancer.[11–15] Kato et al. evaluated 18-FDG uptake in 32 patients with squamous cell carcinoma of the esophagus undergoing radical esophagectomy.[14] A significant association between FDG uptake and depth of tumor invasion and lymph node metastasis was noted corresponding to our results of higher SUV for patients with T3 tumors compared to T2 tumors as staged by EUS.

A number of studies have also evaluated whether the response to treatment, either chemotherapy alone or chemoradiation, as measured by 18-FDG PET-CT uptake is prognostic of pathologic response.[1, 2, 12, 16, 17] Table 1 shows studies published to date comparing 18-FDG PET-CT response to pathologic response. Weber et al. had the lowest mean % SUV reduction but this may be a function of fewer days between the end of treatment and the second PET scan as well as patients not receiving radiation.[2] Cutoff values ranging from 35%–40% have been used to evaluate metabolic response predicting clinical response.[18, 19] Higher complete or subtotal tumor regression was reported in 44% of patients with a metabolic response compared to only 5% without a metabolic response. Patients without a metabolic response also exhibited a shorter time to progression or recurrence and shorter overall survival. Song et al. reported a 66% pathologic complete response in a group of 32 patients undergoing neoadjuvant chemoradiotherapy.[20] They too found a correlation of metabolic to pathologic response only in cancers with initially high metabolic primary tumor, i.e. ≥ 4.0 SUV. Swisher et al. also reported that post-chemoradiation PET scan did identify pathologic responders but failed to identify microscopic tumor in 18% of patients and had a false positive rate of 71%.[8] We did not find a difference in initial SUV or %SUV decrease between patients having or not having a complete pathologic response after chemoradiation. We did find, however, a trend for pathologic complete response and lower post-treatment SUV. Other reports have found a difference in SUV response to treatment but combined complete with partial responders. Combining complete responders with almost complete pathologic response was not performed in this analysis because we tried to determine if 18-FDG PET-CT response could be used as a surrogate marker to determine which patients could be spared an esophagectomy if they possessed a complete pathologic response.

The optimal time to obtain the post-treatment PET scan is not known with certainty. Increased post-chemoradiation inflammation may contribute to higher than expected SUV's. Song et al. performed the post-treatment PET scan at a mean of 5.1 weeks after treatment but still had a 41% incidence of esophagitis.[20] Post-treatment inflammation could falsely elevate the post-treatment SUV if the scans are obtained too soon after treatment. Weber et al., with the shortest time between treatment and post-treatment PET scans, had only a 31% mean %SUV reduction after treatment even though they had the highest initial mean pre-treatment SUV. Levine et al had one of the longest intervals between the end of treatment and the post-treatment scan and they had one of the largest mean %SUV reduction. The timing of the post-treatment scans should be standardized to eliminate this source of variability. Having the post-treatment scan

obtained the day before surgery may be the most optimal time for the post-treatment scan. We are currently evaluating adding a qualitative assessment to the quantitative assessment of post-treatment PET scans in this group of patients.

The baseline SUV is comparable in all studies as is the %SUV decrease after treatment. Our study, however, did not find a statistical difference in disease-free or overall survival based upon PET scan response. We did find on univariate analysis post-treatment SUV to predict for disease-specific mortality in patients not undergoing surgery but this difference did not continue on multivariate analysis.

The majority of the published literature to date has focused on the ability of 18-FDG PET scans to predict for pathologic response or survival in patients undergoing pre-operative chemoradiation. This is the first study to report on the use of PET scans to monitor response and outcome in patients not undergoing surgery. Correlating post-treatment PET scans with pathologic response in this group would be difficult given the potential for false negative biopsy results due to sampling error in the process of endoscopic biopsies. We have found a relationship between post-treatment SUV and disease-free survival after definitive chemoradiation. But how the information gained from this test will be integrated into the follow-up regimens of patients not undergoing surgery will need to be evaluated further. Whether patients having persistently elevated post-treatment SUV would benefit from additional therapy should be the focus of future clinical trials.

The study of other radioisotopes to investigate factors predicting tumor response and outcome are warranted. 18F-fluoro-3′-deoxy-3′L-fluorothymidine (18F-FLT) was evaluated in 10 patients with biopsy proven esophagus or gastroesophageal cancers and was found to have more false-negative findings and fewer false-positive findings compared to 18F-FDG.[21] 11C-Choline-PET was compared to 2-deoxy-2-[(18)F]fluoro-D-glucose (FDG)-PET in 38 patients with malignancies, 2 of whom had esophageal cancer.[22] 11C-Choline PET was reported to be similar to FDG-PET in differentiation between malignant and benign lesions in various tumors. 11C-acetate could be evaluated in slowly-growing esophageal tumors that have very little initial FDG uptake.[23]

18-FDG PET-CT scans, in this heterogeneous population, were not able to predict for overall or disease-free survival in patients undergoing chemoradiation prior to planned esophagectomy. A trend did exist for post-treatment SUV to predict for pathologic complete response in the primary tumor in patients undergoing chemoradiation. Thus, utilizing PET scans to decide whether esophagectomy is warranted is inappropriate at the current time. Post-treatment SUV was predictive of disease-free survival in patients undergoing definitive chemoradiaiton but how this finding should be integrated into the current management of patients with esophageal cancer should be the focus of future clinical trials. Larger prospective, multicenter studies will be necessary to confirm these results and clarify the role of 18-FDG PET-CT in the assessment of response to chemoradiation in patients with esophageal carcinoma.

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Table 1

Studies evaluating 18-FDG response in patients with esophageal cancer

Study	Pts.	M/F	Hist.	Chemo.	Mean RT Dose	Mean Pre-tx. SUV	Mean Days to Post-tx PET Scan	Mean % SUV Reduction	Patients Undergoing Surgery	Primary Pathologic CR
Weber[2]	40	37/3	Adeno.	Cisplat. 5-Fu	None	17.9	14	31%	22 (R0)	9%
Flamen[12]	36	28/8	Both	Cisplat. 5-Fu	40 Gy	NR	35	NR	30 (23R0)	20%
Wieder[16]	38	27/4	Squam.	5-Fu	40 Gy	9.3	3–4 weeks	70% Hist. Rsp. 51% Hist. Nrsp.	33	24%
Downey[1]	39	34/5	Both	Taxol Cisplat.	50.4 Gy	NR	NR	59%	17	24%
Brucher[17]	27	23/4	Squam.	5-Fu	30 Gy	8.3	3 weeks	72% Hist. Rsp. 42% Hist. Nrsp.	24	12.5%
Swisher[8]	83	74/9	Both	Multiple Regimens	50.4 Gy	NR	NR	NR	83	31%
Levine[18]	64	53/11	Both	Cisplat. 5-FU	50.4 Gy	9.9	2.5 months	52.8%	44	27%
Konski (current study surgery only)	44	41/3	Both	Multiple Regimens	45 Gy	9.2	26.5	50% pCR 56% No pCR	43 (28 R0 or R1 resection)	25%

Pts.-Patients in study; M/F- Male/Female; Chemo.- Chemotherapy; Pre-tx.- Pre-treatment; NR- Not reported; Adeno.- Adenocarcinoma; Squam.-Squamous cell; Hist Rsp.- Histologic responder; Hist. Nrsp.- Histologic non-responder; CR- Complete response; pCR- Pathologic complete response