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PET Scans as a Predictive Marker of Survival in Advanced Colorectal Cancer

Minsig Choi, M.D.[1], **Sri lakshmi S. Kollepara, M.D.**[2], **Lance K. Heilbrun, Ph.D.**[3], **Daryn Smith, M.S.**[3], **Anthony F. Shields, M.D., Ph.D.**[3], and **Philip A. Philip, MD, Ph.D., FRCP**[3]

[1]Department of Medicine, Stony Brook University, Stony Brook, NY

[2]Department of Internal Medicine, DMC Sinai Grace Hospital, Detroit, MI

[3]Karmanos Cancer Institute, Wayne State University School of Medicine, Detroit, MI

Abstract

Background—The clinical utility of FDG-PET scan in predicting the outcome of patients with metastatic colorectal cancer (mCRC) has not been well studied. We hypothesized that standardized uptake value (SUV) in post treatment FDG-PET predicts outcomes among patients with mCRC.

Methods—We retrospectively reviewed mCRC patients who had FDG - PET before their treatment and measured their SUV on follow-up imaging at the Karmanos Cancer Institute. Primary end points of time to progression (TTP) and overall survival (OS) were compared in two groups: follow up (post-treatment) SUV of 0 versus > 0.

Results—The study population consisted of 44 patients (median age of 58.1 years). Forty (91%) of the patients were treated first line, 34 (77%) received an oxaliplatin based regimen and 7 (16%) received irinotecan based regimen. Thirty-four (77%) patients received concurrent bevacizumab. Median pre-treatment SUV was 9.2 (range 1.7 – 46.3), while median post-treatment SUV (in N=41) was 4.0 (range 0 – 14). The median % change in SUV was −68.5 % (range −9.2% to −100%). The median time interval between scans was 2.6 months. There was no statistically significant difference noted between metabolic responders and non-responders in regards to TTP and OS. However, patients with post-treatment SUV of 0 had significantly longer OS than those with post-treatment SUV of > 0 (median 42 vs 25.2 months, respectively), and slightly longer TTP (median 8.2 vs 6.9 months, respectively).

Conclusion—Systemic therapy significantly decreased SUV on follow-up PET scans in advanced CRC patients Absence of FDG uptake on follow up PET scan was associated with markedly longer OS and slightly longer TTP.

Corresponding Author: Minsig Choi, M.D., Associate Professor of Medicine, Stony Brook University, HSC 15-053C, Stony Brook, New York 11794-8191, Phone 631-444-3578, Fax 631-638-0915, Minsig.choi@stonybrookmedicine.edu.

Keywords

Prognostic marker; PET scan; metastatic colorectal cancer

Introduction

Colorectal cancer (CRC) is one of the leading causes of cancer related death across the world. It is the second most common cause of cancer related mortality in United States, accounting for nearly fifty thousand deaths every year.[1] With the availability of newer chemotherapeutic and targeted agents, overall survival (OS) of advanced colorectal cancer has improved from 12 months to 24–29 months in recent clinical trials. [2–4] Current practice utilizes structural imaging like computer tomography (CT) every 2–3 months to monitor treatment response. Although few clinicians use PET-CT for monitoring treatment response in advanced CRC, it is not recommended since PET can transiently give false negative results following chemotherapy administration. [5,6]

Functional imaging with fluorodeoxyglucose positron emission tomography (FDG-PET) visualizes the distribution of glucose uptake and higher accumulation of metabolite in tumor cells.[7] Imaging of changes in glucose metabolism, as assessed by FDG-PET can provide timely response assessment as there are rapid changes in cellular metabolism before the structural changes. FDG-PET is already used in staging prior to surgical resection of recurrent and metastatic CRC (mCRC), localizing the origin of disease recurrence in CRC patients with rising carcinoembryonic antigen level (CEA) and in the assessment of residual tumor after treatment.[8,9] However, the utility of FDG-PET in prediction and evaluation of response to treatment mCRC is not clearly understood.

The European Organization for Research and Treatment of Cancer (EORTC) has defined criteria of cancer treatment response using FDG-PET based on lesion specific region of interest that are followed on subsequent scans. Complete metabolic response (CMR) was complete resolution of ^{18}F-FDG uptakes within all lesions making them indistinguishable from surrounding tissue. Partial metabolic response (PMR) was a reduction in SUV of at least 25% after more than 1 treatment cycle. Progressive metabolic disease (PMD) was an increase of at least 25% in SUV or a new^{18}F-FDG–avid lesion. Stable metabolic disease (SMD) was a response between PMR and PMD. [10] PET response criteria in solid tumors (PERCIST) operates with fixed region of interest using single most metabolically active tumor in the patient's PET scan using SUV normalized to lean body mass termed SUL. The four categories are similar to EORTC criteria. However, PMR is defined as a reduction of at least 30% in SUL peak and PMD was an increase of at least 30% in SUL peak or a new ^{18}F-FDG–avid lesion.[11] We hypothesized that post-treatment SUV level may be associated with overall survival (OS) and time to progression (TTP) in mCRC patients.

Methods

This is a retrospective study performed at Karmanos Cancer Institute (KCI) and was approved by the Wayne State University Institutional Review Board. From January 2005 to June 2012 44 patients who were treated at KCI with diagnosis of mCRC and had a FDG -

PET before the treatment were included as the study population. Of these 44 patients, 41 had two or more serial FDG-PET scans. Their response to treatment was assessed on the follow-up (post-treatment) FDG-PET scans. In all patients the treatment decisions were made by the multidisciplinary team involved in their care.

Data were collected from the electronic medical records of the study group. This included demographics, type of chemotherapy, SUV on both initial and follow up scans, objective response to treatment, as well as the progression status were verified by two independent investigators. To calculate the response criteria, SUVmax from initial and subsequent PET was used. PET response was classified into, complete responders (CR), metabolic responders (MR), and non responders (NR) using the PERCIST criteria. All images were done approximately 1 hour post injection of the tracer and standardized protocols were followed for all mCRC patients. For simplicity, when patient had complete normalization of FDG activity in the tumor that is indistinguishable from background blood-pool level; SUV was assigned 0. The Social Security Administration death index (SSADI) and electronic medical records were used to determine survival status of the patients.

Statistical Methods

Baseline demographic and clinical variables were summarized with descriptive statistics. TTP was measured from the date of SUV measurement until the first documented CT evidence of disease progression. Patients without progression were censored as of the date of their most recent CT indicating that they were still progression-free. Patients who died from other causes (unrelated to their mCRC) were censored for TTP as of their date of death. OS was measured from the date of SUV measurement until the date of death from any cause. Patients still alive as of the most recent date on which that was confirmed were censored as of the date of that confirmation. The absence of a death date in the SSADI was not accepted as a date of censoring for OS.

For all 44 patients having an initial (pre-treatment) FDG-PET, their censored TTP and censored OS distributions were measured from the date of initial scan, and estimated using the Kaplan-Meier (K-M) method. The 41 patients having at least one follow-up (post-treatment) scan were divided into two groups: SUV = 0 and SUV > 0, based on their first follow-up (post-treatment) SUV. Using a landmark approach their TTP and OS distributions were measured from the date of the follow-up scan, and estimated using the K-M method. [5]

Due to the small sample sizes, TTP and OS statistics (e.g., median, 12 month rate, etc.) were estimated more conservatively using linear interpolation among successive event times on the K-M curves. [12] Censored TTP or censored OS curves were compared by category of follow-up SUV using the log-rank test. The Cox proportional hazards (PH) model was used to adjust for prior bevacizumab therapy (yes/no) as a covariable. The PH assumption was checked for all dichotomized predictor variables via inspection of log(-log)[survival time] plots, and smoothed hazard functions estimated with the Epanechnikov kernel smoother in SAS 9.3.

Results

Table 1 summarizes the patient characteristics, chemotherapy received, and SUV on the initial and follow up PET scans. Forty (91%) of the patients were treated first line, 34 (77%) received an oxaliplatin based regimen, and 7 (16%) received irinotecan based regimens. Majority of patients (77%) received concurrent bevacizumab. Median pre-treatment SUVmax (N=44) was 9.2 (range 1.7 – 46.3), while median post-treatment SUVmax (N=41) was 4.0 (range 0 – 14.0). The median % change in SUV was −68.5 (range −9.2% to −100%). Median interval between scans was 2.6 months and seven patients had no tumor uptake on post treatment scans. Five patients (11%) had complete response, 26 patients (59%) had partial response and 13 patients (30%) had stable disease as defined by Response Evaluation Criteria in Solid Tumors (RECIST criteria).[13]

As shown in Table 2, among the 41 patients having both pre- and post-treatment (follow-up) scan, those with follow-up SUV= 0 (n-12) had median TTP of 8.2 months, and median OS of 42 months. In comparison, the patients with follow-up SUV > 0 (n=29) had worse outcomes with a median TTP of 6.9 months and a median OS of 25.2 months. Figures 1a and 1b graphically display the OS and TTP curves in the full study population (N=44).

Figure 1c (N=41) shows that when compared to the SUV > 0 group, patients in the SUV= 0 group had significantly longer OS ($p = 0.031$). That result persisted ($p = 0.0443$) even after Cox model adjustment for prior bevacizumab therapy (yes/no). At one and two years of follow–up, the SUV = 0 group had 90% and 83% OS rates, compared to the 79% and 51% in the group with follow-up SUV> 0. Patients with follow-up SUV = 0 had slightly longer TTP than did the patients with follow-up SUV > 0 ($p = 0.0628$). That result persisted ($p = 0.0663$) even after Cox model adjustment for prior bevacizumab therapy (yes/no).

A similar analysis was conducted for metabolic responders (MR) and non-responders (NR) with decrease in SUVmax of ≥ 30%. There were 34 MR patients and 7 NR patients. The median TTP estimates for MR and NR were 7.4 and 8.4 months, respectively. The median OS estimates for MR and NR were 30.4 and 32.5 months, respectively. Neither TTP or OS differed significantly between MR and NR patients ($p > 0.70$ for each comparison). Those results were virtually unchanged ($p > 0.74$ for each comparison) after Cox model adjustment for prior bevacizumab therapy (yes/no).

Discussion

The utility of FDG-PET in monitoring therapeutic response in Non-Hodgkin's lymphoma (NHL) [14] and esophageal cancer is well established.[15,16] However, the clinical utility in predicting the outcome of advanced CRC patients is limited to small case series.[17–19] In a study of twenty CRC patients with liver metastasis, Findlay et al demonstrated that a 15% decrease in tumor: liver FDG uptake ratio on the follow up scan of mCRC patients, predicted the objective response as measured by CT.[18] Dimitrakopoulou et al demonstrated that the median SUV on the pre-treatment PETcould predict progressive and stable disease more accurately than the partial response group in a study of 28 mCRC patients who were treated with FOLFOX chemotherapy. [17] De Bruyne et al studied 19 mCRC patients with

potentially resectable liver lesions to evaluate the role of FDG-PET in evaluating response to chemotherapy and bevacizumab as well as in predicting progression free survival (PFS). They have demonstrated that the median baseline SUVmax was higher in non-responders than in responders,(7.20 vs 3.77, respectively) and that higher follow- up SUVmax was associated with worse PFS. [19]

The challenges with interpreting the results of the small studies conducted in mCRC have been lack of clear definition of PET response and use of different parameters to define clinical response. In our cohort of 41 patients with both pre and post treatment scans, only complete metabolic responders (SUV = 0 post treatment) had improved OS compared to non-responders (42 months vs. 25.2 months). However, we could not validate the PERCIST criteria using an SUV reduction cut off of 30% in the long term outcome of advanced CRC. There was no statistically significant difference noted between PERCIST metabolic responders and non-responders in regards to TTP and OS.

Our study outcome is very similar to a randomized trial of 51 mCRC patients who received irinotecan based chemotherapy in 1st line advanced CRC. [20] That study demonstrated that PET response was not statistically significantly associated with TTP or OS using the EORTC PET response criteria. For MR and NR patients the median TTP was 10.8 and 8.8 months respectively (p=0.7). The median OS for PET responders was 24.9 months compared to 21.5 months in NR (p=0.1). Hence two cohorts of advanced CRC patients treated in the first line setting have shown the limitation of the currently available criteria used in PERCIST and EORTC for long term outcome in advanced CRC. Interestingly another study done by de Geus-oei et. al in 50 heterogeneous CRC patients showed that PET response (SUV decreased of ≥20%) was highly predictive for patient outcome including OS and PFS .[21] In that study, 26 patients received chemotherapy in first line, 16 patients in second line, 7 patients in 3rd line and one in fourth line.. The study demonstrated that both quantitative Patlak analysis and simplified SUV changes at 2 months after treatment could delineate PET responders and non-responders with corresponding long term outcome.[21]

Although our study had a heterogenous group of mCRC patients like the study conducted by de Geus et. al. 91% of the patients received first line chemotherapy. In this setting, 5 patients had complete response, 26 patients with partial response and 13 patients had stable disease using conventional RECIST criteria. In 1st line mCRC patients where effective polychemotherapy is given, using PET to delineate progressive and non-progressive patients will be more difficult. Many clinical trials using doublet and triplet chemotherapy have shown that clinical benefit in this setting is about 90%. [3,22] The clinical value of PET might be limited in first line treatment of mCRC where most of the patients benefit from chemotherapy using oxaliplatin and irinotecan based regimens.

However, an important observation that metabolic complete responders have long term clinical benefit in mCRC would indicate that there is prognostic value in PET scans. Kalff et. al found a similar outcome in locally advanced rectal cancer using FDG-PET after neoadjuvant chemoradiotherapy and a similar experience has been noted in esophageal cancer as well. [23] However, normalization of FDG uptake does not translate to pathologic complete response. Our data suggest that median TTP in metabolic complete responders is

8.2 months, only slightly better than non-responders 6.9 months. Tan et. al. have shown in pathologic specimens that 85% of metabolic complete response tumor specimens contained viable cancer cells. [24] So treatment should not be changed or interrupted based solely on PET scan results and routine monitoring for mCRC is not recommended using PET scans.

A major finding of our study is that post-treatment SUV of 0 in 17% of patients was a strong predictor of the long-term outcome. Patients with SUV of 0 may be managed differently including earlier discontinuation of therapy to avoid cumulative toxicities and reduce cost. PET/CT might also have a role in the management of colon cancer patients with oligometastatic disease who were potentially curable where a rapid assessment of therapeutic response would be useful and could potentially impact decision-making in this high-risk subset of patients.

The key limitation in our cohorts is that the group of mCRC is a heterogeneous group and that 2nd FDG-PET was performed in various time points. Recent development in cancer response monitoring have shown that early assessment of FDG_PET even 2 weeks after chemotherapy corresponded nicely to anatomic response and correlated with long term outcome. [25,26] However many of the imaging studies conducted in the past have similar limitation and limited number of patients using various PET parameters. Larger studies with specific time points for 2nd PET scan should be conducted to validate the 25% and 30% cut-off in SUV as suggested by EORTC and PERCIST.

Conclusion

Systemic therapy decreased SUV on follow-up PET scans in all of the advanced CRC patients in our study. Complete absence of FDG uptake on follow up PET scan was associated with markedly longer OS and was suggestive of longer TTP in patients with mCRC. Overall, using FDG-PET as treatment monitoring in standard clinical practice has limited value at this time. Larger studies are required to validate the clinical role of PET imaging in treatment decisions for mCRC patients including the risk stratification of patients in clinical trials.

Acknowledgments

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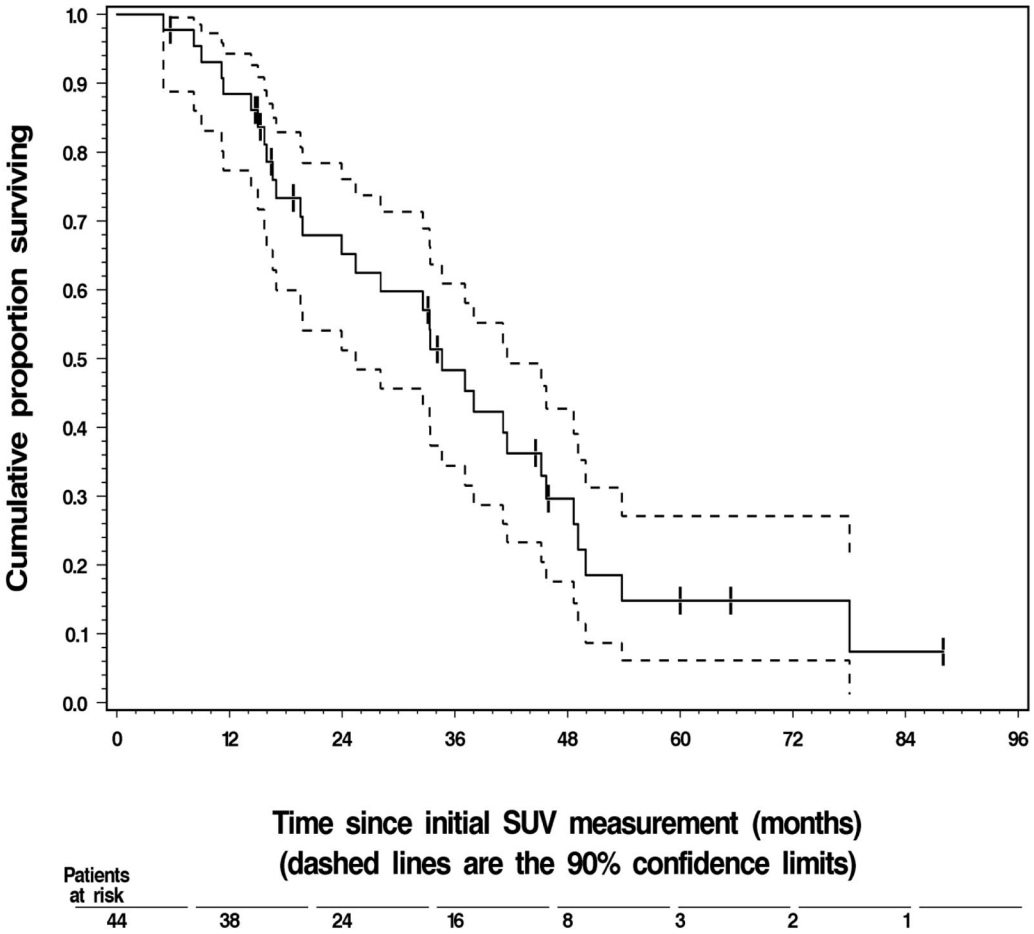
References

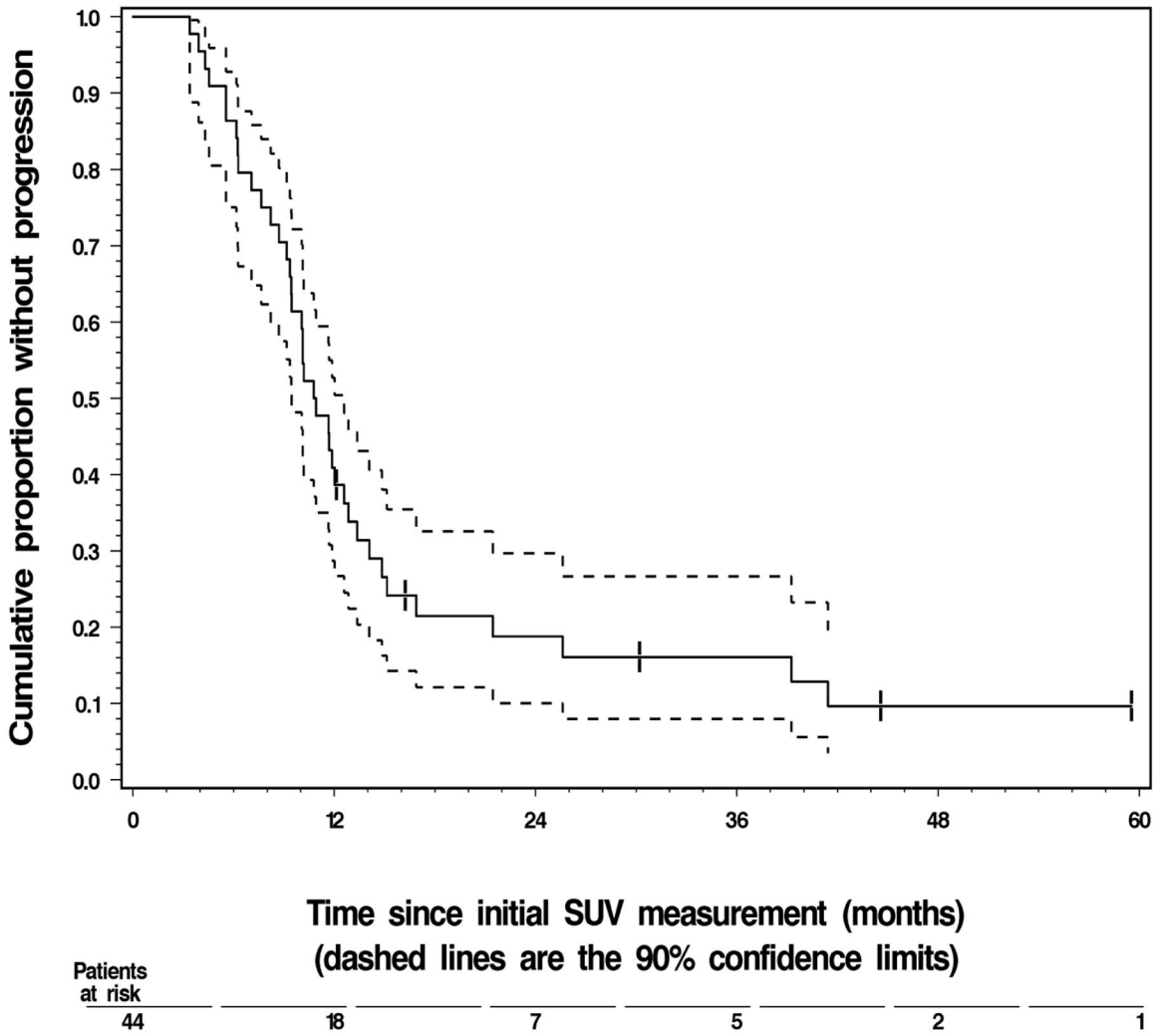
1. Siegel R, Desantis C, Jemal A. Colorectal cancer statistics, 2014. CA Cancer J Clin. 2014; 64:104–17. [PubMed: 24639052]
2. Venook A, Niedzwieki D, Lenz H, et al. CALGB/SWOG 80405: Phase III trial of irinotecan/5-FU/leucovorin (FOLFIRI) or oxaliplatin/5-FU/leucovorin (mFOLFOX6) with bevacizumab (BV) or cetuximab (CET) for patients (pts) with KRAS wild-type (wt) untreated metastatic adenocarcinoma of the colon or rectum (MCRC). J Clin Oncol. 2014; 32(5s suppl):abstr LBA3.
3. Saltz LB, Clarke S, Diaz-Rubio E, et al. Bevacizumab in combination with oxaliplatin-based chemotherapy as first-line therapy in metastatic colorectal cancer: a randomized phase III study. J Clin Oncol. 2008; 26:2013–9. [PubMed: 18421054]

4. Kopetz S, Chang GJ, Overman MJ, et al. Improved survival in metastatic colorectal cancer is associated with adoption of hepatic resection and improved chemotherapy. J Clin Oncol. 2009; 27:3677–83. [PubMed: 19470929]
5. Anderson JR, Cain KC, Gelber RD. Analysis of survival by tumor response. J Clin Oncol. 1983; 1:710–9. [PubMed: 6668489]
6. Delbeke D, Martin WH. FDG PET and PET/CT for colorectal cancer. Methods Mol Biol. 2011; 727:77–103. [PubMed: 21331930]
7. Pauwels EK, McCready VR, Stoot JH, et al. The mechanism of accumulation of tumour-localising radiopharmaceuticals. Eur J Nucl Med. 1998; 25:277–305. [PubMed: 9580862]
8. Flamen P, Hoekstra OS, Homans F, et al. Unexplained rising carcinoembryonic antigen (CEA) in the postoperative surveillance of colorectal cancer: the utility of positron emission tomography (PET). Eur J Cancer. 2001; 37:862–9. [PubMed: 11313174]
9. de Geus-Oei LF, Wiering B, Krabbe PF, et al. FDG-PET for prediction of survival of patients with metastatic colorectal carcinoma. Ann Oncol. 2006; 17:1650–5. [PubMed: 16936185]
10. Young H, Baum R, Cremerius U, et al. Measurement of clinical and subclinical tumour response using [18F]-fluorodeoxyglucose and positron emission tomography: review and 1999 EORTC recommendations. European Organization for Research and Treatment of Cancer (EORTC) PET Study Group. Eur J Cancer. 1999; 35:1773–82. [PubMed: 10673991]
11. Wahl RL, Jacene H, Kasamon Y, et al. From RECIST to PERCIST: Evolving Considerations for PET response criteria in solid tumors. J Nucl Med. 2009; 50 (Suppl 1):122S–50S. [PubMed: 19403881]
12. Lee, ET.; Wang, JW.; Wiley, John, et al. Statistical methods for survival data analysis, Wiley series in probability and statistics. 3. New York: J. Wiley; 2003.
13. Therasse P, Arbuck SG, Eisenhauer EA, et al. New guidelines to evaluate the response to treatment in solid tumors. European Organization for Research and Treatment of Cancer, National Cancer Institute of the United States, National Cancer Institute of Canada. J Natl Cancer Inst. 2000; 92:205–16. [PubMed: 10655437]
14. Schoder H, Noy A, Gonen M, et al. Intensity of 18fluorodeoxyglucose uptake in positron emission tomography distinguishes between indolent and aggressive non-Hodgkin's lymphoma. J Clin Oncol. 2005; 23:4643–51. [PubMed: 15837966]
15. Flamen P, Lerut A, Van Cutsem E, et al. Utility of positron emission tomography for the staging of patients with potentially operable esophageal carcinoma. J Clin Oncol. 2000; 18:3202–10. [PubMed: 10986052]
16. Kato H, Miyazaki T, Nakajima M, et al. The incremental effect of positron emission tomography on diagnostic accuracy in the initial staging of esophageal carcinoma. Cancer. 2005; 103:148–56. [PubMed: 15558794]
17. Dimitrakopoulou-Strauss A, Strauss LG, Rudi J. PET-FDG as predictor of therapy response in patients with colorectal carcinoma. Q J Nucl Med. 2003; 47:8–13. [PubMed: 12714949]
18. Findlay M, Young H, Cunningham D, et al. Noninvasive monitoring of tumor metabolism using fluorodeoxyglucose and positron emission tomography in colorectal cancer liver metastases: correlation with tumor response to fluorouracil. J Clin Oncol. 1996; 14:700–8. [PubMed: 8622014]
19. De Bruyne S, Van Damme N, Smeets P, et al. Value of DCE-MRI and FDG-PET/CT in the prediction of response to preoperative chemotherapy with bevacizumab for colorectal liver metastases. Br J Cancer. 2012; 106:1926–33. [PubMed: 22596235]
20. Bystrom P, Berglund A, Garske U, et al. Early prediction of response to first-line chemotherapy by sequential [18F]-2-fluoro-2-deoxy-D-glucose positron emission tomography in patients with advanced colorectal cancer. Ann Oncol. 2009; 20:1057–61. [PubMed: 19164458]
21. de Geus-Oei LF, van Laarhoven HW, Visser EP, et al. Chemotherapy response evaluation with FDG-PET in patients with colorectal cancer. Ann Oncol. 2008; 19:348–52. [PubMed: 17962202]
22. Van Cutsem E, Kohne CH, Hitre E, et al. Cetuximab and chemotherapy as initial treatment for metastatic colorectal cancer. N Engl J Med. 2009; 360:1408–17. [PubMed: 19339720]
23. Kalff V, Duong C, Drummond EG, et al. Findings on 18F-FDG PET scans after neoadjuvant chemoradiation provides prognostic stratification in patients with locally advanced rectal

carcinoma subsequently treated by radical surgery. J Nucl Med. 2006; 47:14–22. [PubMed: 16391182]

24. Tan MC, Linehan DC, Hawkins WG, et al. Chemotherapy-induced normalization of FDG uptake by colorectal liver metastases does not usually indicate complete pathologic response. J Gastrointest Surg. 2007; 11:1112–9. [PubMed: 17623263]
25. Maisonobe JA, Garcia CA, Necib H, et al. Comparison of PET metabolic indices for the early assessment of tumour response in metastatic colorectal cancer patients treated by polychemotherapy. Eur J Nucl Med Mol Imaging. 2013; 40:166–74. [PubMed: 23151911]
26. Buvat I, Necib H, Garcia C, et al. Lesion-based detection of early chemosensitivity using serial static FDG PET/CT in metastatic colorectal cancer. Eur J Nucl Med Mol Imaging. 2012; 39:1628–34. [PubMed: 22710958]





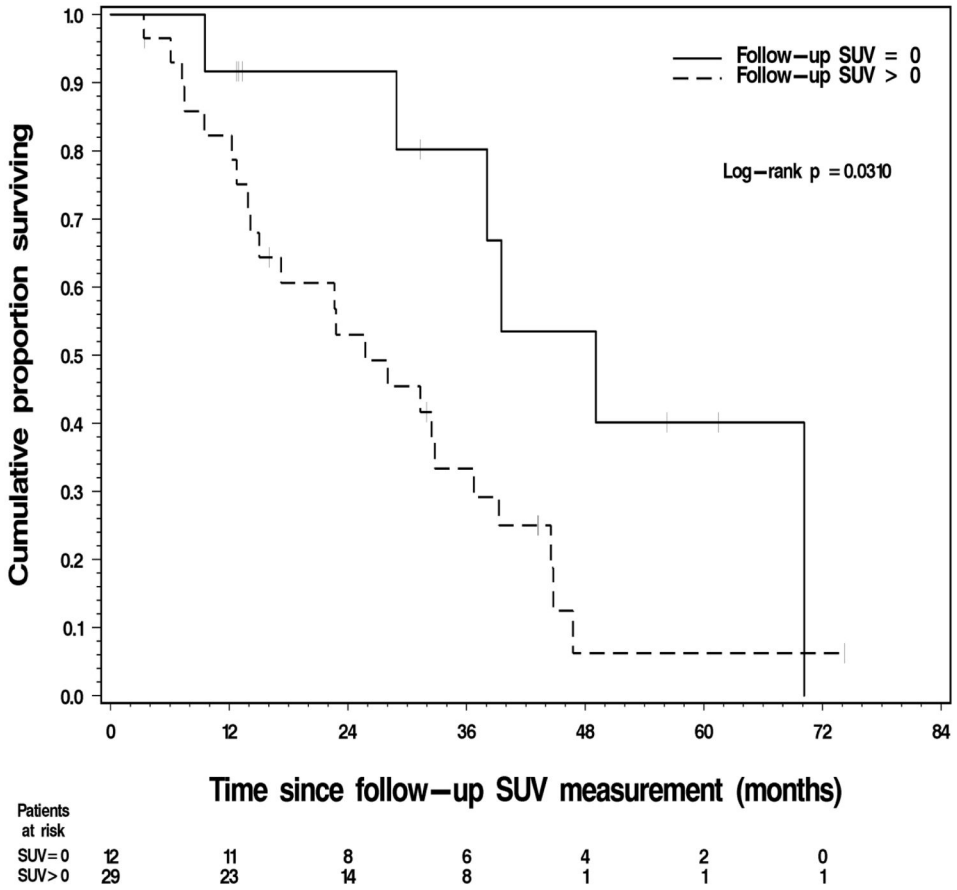


Figure 1.

Figure 1a. Kaplan-Meier graph of overall survival (OS) for all 44 patients with an initial (pre-treatment) scab. Dashed lines identify the pointwise 90% confidence limits for successive rates of survival.

Figure 1b. Kaplan-Meier graph of time to progression (TTP) for all 44 patients with an initial (pre-treatment) scan Dashed lines identify the pointwise 90% confidence limits for successive rates of freedom from progression.

Figure 1c. Kaplan-Meier graph of overall survival (OS) for all 41 patients with scans pre- and post-treatment, by level of follow-up SUV. Dashed lines identify the pointwise 90% confidence limits for successive rates of freedom from progression.

Table 1

Baseline demographics, chemotherapy, and SUV measurements

Median Age at Diagnosis in years (range)	58.1 (35–84.7)
Gender	
Male	23 (52.3)
Female	21 (47.7)
Race	
Caucasian	26 (59.1)
Afro American	6 (13.6)
Asian	3 (6.8)
Other	9 (20.5)
Prior Treatment	
None	16 (36.4)
Chemo Rx/Surgery	10 (22.7)
Surgery	18 (40.9)
Treatment received	
FOLFOX	27 (61.4)
CAPOX	7 (15.9)
FOLFIRI	5 (11.4)
CAP/IRIN	2 (4.6)
Capecitabine	2 (4.6)
OTHER	1 (2.3)
Line of treatment	
First line	40 (90.9)
Second line	2 (4.6)
Third line	2 (4.6)
Monoclonal Antibody Used	
None	5 (11.4)
bevacizumab	34 (77.3)
cetuximab	5 (11.4)
Best CT Response	
Complete Response	5 (11.4)
Partial Response	26 (59.1)
Stable Disease	13 (29.6)
Median Initial SUVmax (range)	9.2 (1.7–46.3)
Median Follow up SUVmax(range)	4.0(0.0–14.0)
Median % Change in SUVmax(range)	68.5(9.2–100)
Median Inter-Scan time interval in months, (range)	2.6 (1.3–13.7)

Table 2

Summary statistics of TTP and OS by level of follow-up SUV

Follow-up SUV value	Statistic	N	Events	Point estimate	90 % CI	
					Lower limit	Upper Limit
Time to Progression						
SUV = 0		12	8			
	Median			8.2 mo.	4.5 mo.	30.1 mo.
	12 month rate			39 %	13 %	65 %
	24 month rate			27 %	1 %	53 %
SUV > 0		29	28			
	Median			6.9 mo.	4.4 mo.	9.3 mo.
	12 month rate			19 %	6 %	31 %
	24 month rate			10 %	1 %	20 %
Overall Survival						
SUV = 0		12	6			
	Median			42.0 mo.	21.8 mo.	63.1 mo.
	12 month rate			90 %	69 %	100 %
	24 month rate			83 %	62 %	100 %
	36 month rate			70 %	43 %	97 %
SUV > 0		29	23			
	Median			25.2 mo.	14.4 mo.	32.2 mo.
	12 month rate			79 %	66 %	92 %
	24 month rate			51 %	36 %	67 %
	36 month rate			30 %	15 %	45 %

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