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Clinicopathologic parameters associated with the FDG-avidity in staging of early gastric cancer using ^{18}F-FDG PET

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Abstract

This study investigated the clinicopathologic factors associated with 2-[^{18}F]fluoro-2-deoxy-D-glucose (^{18}F-FDG) uptake of early gastric cancer (EGC) and used them to design a clinical scoring method to predict FDG-avidity of EGC.

Two hundred twenty-nine retrospectively enrolled patients underwent preoperative ^{18}F-FDG positron emission tomography/computed tomography (PET/CT). Histologic information was obtained by gastrectomy (n = 195) or endoscopic mucosal dissection (n = 34). The association between clinicopathologic factors and ^{18}F-FDG uptake by the primary tumor was determined. The results were used to develop a clinical scoring method.

^{18}F-FDG uptake was detected in 49 (17.5%) patients. According to univariate analysis, location, gross type, World Health Organization classification, Lauren classification, size, depth of invasion, and lymphatic invasion were significant variables affecting ^{18}F-FDG uptake (all $P < .05$). According to multivariate analysis, location (lower 3rd, $P = .035$), gross type (0–I, 0–IIa, $P < .001$), size (≥ 2.5 cm, $P = .026$), and depth of invasion (submucosa, $P = .007$) were significantly associated with FDG-avidity. A clinical scoring system, ranged from 0 to 4, was developed by giving one score to 4 independent variables. A cut-off value of 2.5 showed good prediction of FDG-avidity in EGCs, with a sensitivity and specificity of 65.0% and 85.2%, respectively.

^{18}F-FDG uptake by EGC depends on location, gross type, size, and depth of invasion of the primary tumor. A clinical scoring system based on clinicopathologic variables can predict the FDG-avidity of primary tumors in patients with EGC.

Abbreviations: AGC = advanced gastric cancer, EGC = early gastric cancer, ESD = endoscopic submucosal dissection, ^{18}F-FDG = 2-[^{18}F]fluoro-2-deoxy-D-glucose, PET/CT = positron emission tomography/computed tomography, SUV$_{max}$ = maximum standardized uptake value.

Keywords: ^{18}F-fluorodeoxyglucose, neoplasm staging, patient selection, positron-emission tomography, stomach neoplasms

1. Introduction

Early detection and therapeutic improvement over the past 2 decades have dramatically increased the 5-year survival rate of patients with stomach cancer (from 42.8% to 71.5%).[1] Patients with early gastric cancer (EGC) without lymph node metastasis have an excellent prognosis, evidenced by a survival rate of 98% to 100%.[2] EGC comprises about 50% to 60% of all gastric cancers in Asian countries, while the proportion of EGC is only 10% to 20% in Western countries. Such differences arise partly from the nation-wide cancer screening programs of Asian countries. Disagreement in histologic interpretation between Asian and Western countries also contributes to this discrepancy.[3–5]

For the preoperative staging of stomach cancer, 2-[^{18}F]fluoro-2-deoxy-D-glucose (^{18}F-FDG) positron emission tomography/computed tomography (PET/CT) has a high sensitivity for detecting the primary tumor in patients with advanced gastric cancer (AGC). By World Health Organization (WHO) classification, the detection rate and ^{18}F-FDG uptake is higher in tubular (intestinal) type than in poorly cohesive (diffuse) type. The low FDG uptake of signet ring cell type of poorly cohesive carcinoma is due to the reduced expression of glucose transporter-1. ^{18}F-FDG PET/CT has a high specificity for detecting lymph node metastases.[6–10]

The use of ^{18}F-FDG PET/CT for EGC staging is not generally recommended because of its low sensitivity (25.9–50.3%).[6–8] However, extra-abdominal metastasis from gastric cancer is not uncommon, and even in EGC, distant metastasis to lymph node, liver, lung, or bone can be present.[5,11][18]F-FDG PET/CT, as a whole body imaging, is useful in the detection of distant metastasis both for intestinal and diffuse types.[7,12] Patients with EGC may benefit from ^{18}F-FDG PET/CT in the detection of distant metastasis.

Although ^{18}F-FDG uptake is tissue-specific and associated with tumor clinicopathologic factors, the nature of this relationship in EGC has yet to be determined.[10,13,14] Thus, in the present study, we retrospectively investigated the clinicopathologic factors of EGCs associated with FDG-avidity in the primary tumor during preoperative PET/CT. These factors were then used to develop a clinical scoring system to predict the FDG-avidity of EGC.

2. Materials and methods

2.1. Patients

The study patients were recruited retrospectively through a review of medical records acquired between June 2004 and July 2014. The 229 consecutive patients identified had undergone preoperative ^{18}F-FDG PET/CT for a staging work-up after endoscopic diagnosis of EGC. Any patient with both EGC and AGC was excluded from the study to avoid a false-positive result. All patients underwent either gastrectomy with lymph node dissection (n = 195) or endoscopic submucosal dissection (ESD, n = 34).

Histopathologic information, including tumor size, location, gross type, WHO classification, Lauren classification, depth of invasion, lymphatic/venous/perineural invasion of the primary tumor, and lymph node staging, was collected retrospectively from each patient based on endoscopic or histologic reports. Lymph node staging was determined for those who underwent gastrectomy, based on the 8th edition of the staging manual of the American Joint Committee on Cancer.[15] The gross EGC type was determined according to the Macroscopic Classification of Early Gastric Cancer published by the Japanese Gastric Cancer Association.[16]

All procedures were performed in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) and the ethical standards of our Institutional Review Board on Human Experimentation (approval no. AJIRB-MED-MDB-15). The requirement for obtaining informed consent from the patients was waived by the institutional review board.

2.2. ^{18}F-FDG PET/CT acquisition

All patients fasted for at least 6 hours prior to the PET/CT examination. The blood glucose concentration was measured to ensure a level of <150 mg/dL. After intravenous injection of 370 MBq ^{18}F-FDG, all patients were instructed to rest comfortably for 60 minutes. Immediately before PET, each patient drank a 500-mL bottle of water to flush out physiologic secretions within the gastric lumen.

Emission PET data were acquired from the skull base to the upper thigh using a PET/CT scanner (Discovery ST or Discovery STE, GE Healthcare, Milwaukee, WI). CT was performed without the administration of contrast material and using the following parameters: tube rotation rate 1 second/revolution, 120 kV, 60 mA, 7.5 mm/rotation, acquisition time 60.9 seconds, scan length 867 mm. PET data were reconstructed using non-

contrast CT by iterative reconstruction (ordered-subsets expectation maximization with 2 iterations and 30 subsets, field of view = 600 mm, slice thickness=3.27 mm).

2.3. Image analysis

Two nuclear medicine physicians analyzed the PET/CT images, reaching a diagnosis based on consensus. Histologic information obtained before the PET/CT acquisition was blinded to the interpreters. Primary tumors with a higher ^{18}F-FDG uptake than the rest of the gastric wall were considered FDG-avid. ^{18}F-FDG confined to the gastric lumen was considered to be a physiologic secretion. For the semi-quantitative analysis of FDG-avid gastric tumors, volumes of interest were placed on attenuation-corrected transaxial PET images. The maximum standardized uptake value (SUV_{max}) was then calculated from the injected dose and the patient's body weight.

For nine patients with multiple EGCs, only the largest tumor was considered for analysis. In patients with 2 or more coexisting gross types of tumor, the type with the furthest spread was considered the representative gross type of that tumor.

2.4. Statistical analysis

All continuous values are presented as mean ± standard deviation. The Chi-squared test or Fisher exact test was used for the univariate analysis of ^{18}F-FDG uptake with respect to the clinicopathologic variables. For variables with values of 0 in the cells of a 2×2 table, the median unbiased estimate was adopted as the odds ratio. The Mann–Whitney test was used for group comparisons with respect to a particular variable. Only those variables that were significant on univariate analysis were included in the binomial logistic regression analysis. A clinical scoring system was developed in accordance with previous reports.[17,18] Based on the multivariate analysis, one point was assigned to each independent factor. The optimal cut-off values for the clinical scores were determined by receiver operating characteristic curve analysis. All statistical analyses were performed using SPSS software (version 22.0; SPSS, Inc, Chicago, IL). A P-value <.05 was considered to indicate statistical significance.

3. Results

3.1. Patient characteristics

The mean interval from the PET/CT acquisition to treatment was 10 days (range, 0–60). The most common gastric location of the tumors was the lower 3rd of the stomach, with types 0-IIb and 0-IIc as the major gross types (77.8%, Table 1). The most common histologic type according to the WHO classification was tubular adenocarcinoma, followed by signet ring cell carcinoma. The average tumor size was 2.8 cm (median 2.5 cm). Among the 229 patients, 49 patients had FDG-avid gastric tumors. The majority of the patients had N0 disease; none had EGC with N3 lymph node metastasis. Neither PET/CT nor cytologic (or histologic) examination (omentum in 7, peritoneal fluid in 6, and ovary in 1 patient) found distant metastasis.

3.2. Univariate analysis of ^{18}F-FDG uptake with respect to clinicopathologic factors in patients with EGC

According to univariate analyses, tumor location, gross type, WHO classification, Lauren classification, size, depth of invasion,

Table 1

Patient characteristics.

Variable	Value
Number of patients	229
Age, yrs	60 ± 12 (31–78)*
Sex, M:F	163:66
Interval from PET/CT to treatment, d	10 ± 8 (0–60)*
Location, n (%)	
Upper	27 (11.8)
Middle	80 (34.9)
Lower	122 (53.3)
Gross type, n (%)	
0–I	12 (5.2)
0–IIa	27 (11.8)
0–IIb	78 (34.1)
0–IIc	100 (43.7)
0–III	12 (5.2)
WHO classification, n (%)	
Papillary adenocarcinoma	2 (0.9)
Tubular adenocarcinoma	168 (73.4)
Mucinous adenocarcinoma	2 (0.9)
Signet-ring cell carcinoma	53 (23.1)
Carcinoma with lymphoid stroma	5 (2.2)
Size of primary tumor, cm	2.8 ± 2.5 (0.1–9.0)*
Treatment, n (%)	
Endoscopic submucosal dissection	34 (14.8)
Gastrectomy with lymph node dissection	195 (85.2)
FDG-avidity, n (%)	49 (17.5)
SUV_{max} of FDG-avid gastric cancers	5.2 ± 2.4 (2.2–12.5)*
Lymph node staging for patients with gastrectomy, n (%)	
N0	160 (81.9)
N1	17 (7.4)
N2	18 (7.9)
N3a	0 (0.0)
N3b	0 (0.0)

FDG = 2-[18F]fluoro-2-deoxy-D-glucose, PET/CT = positron emission tomography/computed tomography, SUV_{max} = maximum standardized uptake value.
* Mean ± standard deviation (range)

and lymphatic invasion were significantly associated with 18F-FDG uptake (Table 2). EGCs characterized by larger tumor sizes (≥2.5 cm, FDG-avidity 29.2%), submucosal invasion (26.7%), or lymphatic invasion (36.7%) had higher rates of FDG-avidity than did EGCs characterized by smaller tumor sizes (<2.5 cm, FDG-avidity 10.0%), mucosal invasion (7.3%), or no lymphatic invasion (12.2%). Tumors located in the lower 3rd of the stomach (FDG-avidity 23.8%) were more likely to show 18F-FDG uptake than were those in the middle 3rd (10.0%, P = .014). Tumors of gross type 0-I had a higher rate of FDG-avidity (23.8%) than did those of other gross types, while gross type 0-IIa tumors had a higher FDG-avidity rate (37.0%) than did type 0-IIb (9.0%) or type 0-IIc tumors (10.1%). Among all WHO classifications, signet ring cell carcinomas had the lowest FDG-avidity (5.7%). Tumors with a diffuse type (6.1%), as determined by the Lauren classification, had lower FDG-avidity than did those with a mixed (61.5%), indeterminate (37.5%), or intestinal type (21.4%). Representative examples of FDG-avid EGC and FDG-negative EGC tumors are shown in Figure 1.

3.3. Multivariate analysis of SUV_{max} with respect to clinicopathologic factors in patients with EGC

According to multivariate analyses of the clinicopathologic variables determined to be significant in the univariate analysis,

tumor location, gross type, size and depth of invasion were independent factors correlated with 18F-FDG uptake in EGC (Table 3). A clinical scoring system was developed using these 4 independent variables. The WHO classification, Lauren classification, and lymphatic invasion of the tumor did not significantly correlate with 18F-FDG uptake. Thus, these variables were not used for clinical scoring system.

3.4. Clinical scores for FDG-avidity

For each independent variable, one point was assigned to the 18F-FDG uptake status of EGC: lower 3rd for location, types 0-I and 0-IIa for gross type, ≥2.5 cm for tumor size, and submucosal invasion for depth of invasion, resulting in clinical scores ranging from 0 to 4 for each patient with EGC. The mean score was significantly higher in patients with FDG-avid than in those with non-FDG-avid tumors (2.7 ± 1.1 vs 1.4 ± 0.9, P < .001). The primary tumors of patients with a clinical score of 4 had a high rate of 18F-FDG uptake (84.6%) (Table 4). The percentages of FDG-avidity in patients with clinical score 3, 2, and 1 were 36.1%, 15.6%, and 4.9%, respectively. Only 2 of 35 patients with clinical score 0 showed 18F-FDG uptake. This clinical scoring system performed well (area under the receiver operating characteristic curve, 0.801, P < .001) in predicting the FDG-avidity of EGC. Based on a cut-off value of 2.5, the clinical score had moderate sensitivity (65.0%) and high specificity (85.2%).

4. Discussion

The aim of our investigation was to evaluate a large number of patients with EGC to identify those likely to benefit from 18F-FDG PET/CT staging. Our clinical scoring system, developed using significant clinicopathologic predictors of FDG-avidity in EGC, showed modest sensitivity and high specificity in identifying these patients. To the best of our knowledge, this is the 1st study to evaluate this type of clinical scoring system in patients with EGC. Importantly, unlike previous reports that have analyzed both AGC and EGC together, only patients with EGC were enrolled in large numbers.

Previous reports demonstrated a relationship between tumor size and the detection rate of primary gastric cancers by 18F-FDG PET.[7,19–21] The tumors analyzed in those studies were divided into 2 or 3 categories, with larger tumors detected more readily than smaller tumors.[7,19,21] In addition, tumor size was shown to correlate not only with detectability but also with the SUV of the primary gastric cancer.[20,22,23] For example, Han et al showed that gastric tumor size was positively related to the SUV_{max} (3.0 ± 0.4 for tumors <1 cm, 3.9 ± 2.1 for those 1–3 cm, and 5.7 ± 3.2 for those <3 cm). Other studies, however, did not find an effect of the primary tumor size on tumor detectability by 18F-FDG PET/CT.[10,24,25]

Our study also showed a higher rate of FDG-avidity for tumors in the lower 3rd than those in the upper or middle 3rd of the stomach. According to Stahl et al, AGCs in the proximal 3rd of the stomach are detected more frequently by 18F-FDG PET are than those in the distal 3rd (74% vs 41%) and are more likely to be of the intestinal growth type (65% vs 41%).[26] The Lauren classification has been consistently related to 18F-FDG uptake in gastric tumors, with a higher rate of FDG-avidity and a higher SUV for intestinal type tumors than for tumors of the non-intestinal type.[7,19,22–24,26] Our patients with intestinal type EGCs also had a higher rate of 18F-FDG uptake than did those

Table 2

Univariate analysis between 2-[^{18}F]fluoro-2-deoxy-D-glucose uptake and clinicopathologic variables.

Factors	FDG (−)	FDG (+)	P-value	OR	95% CI
Age, yrs			.155	1.650	0.824–3.303
≤61	99 (86.1%)	16 (13.9%)			
>61	90 (78.9%)	24 (21.1%)			
Sex			.572	0.809	0.389–1.686
Female	53 (80.3%)	13 (19.7%)			
Male	136 (83.4%)	27 (16.6%)			
Location			.027		
Upper	24 (88.9%)	3 (11.1%)			
Middle	72 (90.0%)	8 (10.0%)			
Lower	93 (76.2%)	29 (23.8%)			
Gross type			<.001		
0–I	2 (16.7%)	10 (83.3%)			
0–IIa	17 (63.0%)	10 (37.0%)			
0–IIb	71 (91.0%)	7 (9.0%)			
0–IIc	90 (90.0%)	10 (10.0%)			
0–III	9 (75.5%)	3 (25.0%)			
WHO classification			.002*		
Papillary adenocarcinoma	0 (0.0%)	2 (100.0%)			
Tubular adenocarcinoma	133 (79.8%)	34 (20.2%)			
Mucinous adenocarcinoma	1 (50.0%)	1 (50.0%)			
Signet-ring cell carcinoma	50 (94.3%)	3 (5.7%)			
Carcinoma with lymphoid stroma	4 (80.0%)	1 (20.0%)			
Lauren classification			.016		
Diffuse	62 (93.9%)	4 (6.1%)			
Mixed	5 (38.5%)	8 (61.5%)			
Indeterminate	5 (62.5%)	3 (37.5%)			
Intestinal	88 (78.6%)	24 (21.4%)			
Tumor size, cm			<.001	3.714	1.814–7.605
<2.5	126 (90.0%)	14 (10.0%)			
≥2.5	88 (70.8%)	26 (29.2%)			
Depth of invasion			<.001	3.831	1.729–8.488
pT1a	101 (92.7%)	8 (7.3%)			
pT1b	88 (73.3%)	32 (26.7%)			
Lymphatic invasion			<.001	4.170	2.005–8.673
Absence	158 (87.8%)	22 (12.2%)			
Presence	31 (63.3%)	18 (36.7%)			
Venous invasion			.211†	3.263	0.527–20.198
Absence	186 (83.0%)	38 (17.0%)			
Presence	3 (60.0%)	2 (40.0%)			
Perineural invasion			.759‡	0.690	0–3.900
Absence	184 (82.1%)	40 (17.9%)			
Presence	5 (100.0%)	0 (0.0%)			
Lymph node staging			.161		
N0	131 (81.9%)	29 (18.1%)			
N1–3b	25 (68.6%)	10 (31.4%)			

CI = confidence interval, FDG = 2-[^{18}F]fluoro-2-deoxy-D-glucose, OR = odds ratio.

WHO classification was subdivided according to the differentiation in performing analysis.

* Fisher exact test.
† n = 199.
‡ Median unbiased estimate.

with diffuse type EGCs (21.4% vs 6.1%, P = .016). Furthermore, tumors in the lower 3rd of the stomach were more frequently of the intestinal type than were those in the upper or middle 3rd (56.6% vs 45.3%, P = .037). Therefore, the higher detectability of tumors in the lower 3rd of the stomach may be attributed to the higher incidence of intestinal type tumors at this location.

Another finding of our study was that the depth of tumor invasion (submucosa) was an independent factor predicting high FDG PET/CT detectability. Although there are no published reports of a relationship between submucosal invasion of EGC and ^{18}F-FDG uptake, AGCs were previously shown to be more easily visible than EGCs on ^{18}F-FDG PET/CT.[7,19–21,23–25] The pooled data from multiple studies revealed that the FDG-avidity of AGCs is much higher than that of EGCs, which, together with our data from patients with EGC, implies that the depth of tumor invasion is an important factor for FDG-avidity in gastric cancer.[19]

This study is also the 1st to show that the endoscopic type of EGC is related to ^{18}F-FDG uptake. Thus, types 0-I (83.3%) and 0-IIa (37.0%) had higher FDG-avidity than did other gross types (10.5%), which suggests that tumors expanding toward the gastric lumen take up more ^{18}F-FDG than do those along the
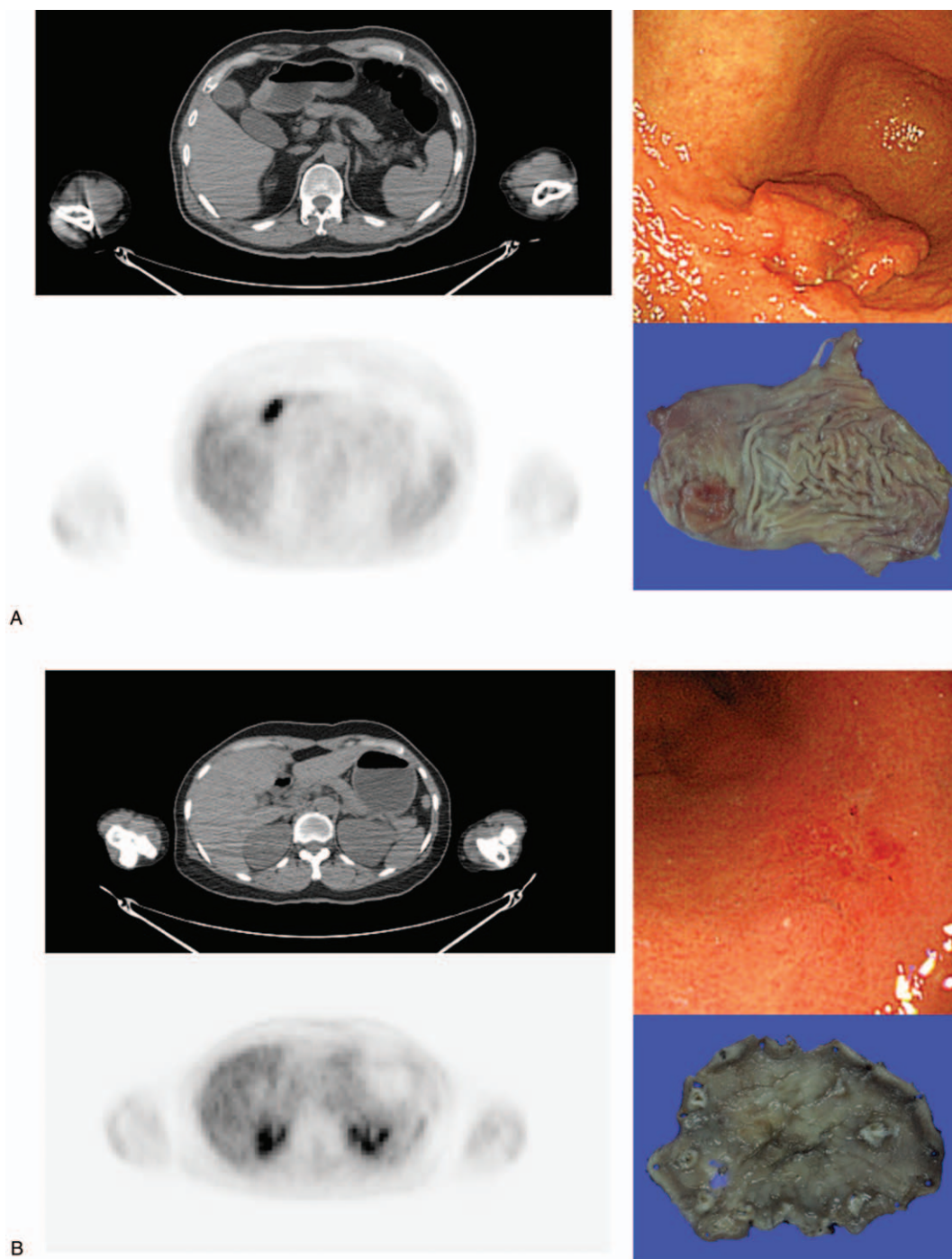
Figure 1. Representative cases of ^{18}F-FDG PET imaging in EGC. (A) A 57-year-old male patient with tubular adenocarcinoma at lower 3rd of stomach (3.5 × 3 cm, gross type 0–I, submucosal invasion, lymphatic invasion, mixed by Lauren classification) showing ^{18}F-FDG uptake in antrum (SUV$_{max}$ = 9.5). (B) A 46-year-old female patient with signet ring cell carcinoma at middle 3rd of stomach (1.1 × 1 cm, gross type 0–IIb, mucosal invasion, diffuse by Lauren classification) showing no significant ^{18}F-FDG uptake. FDG = 2-[^{18}F]fluoro-2-deoxy-D-glucose, EGC = early gastric cancer, SUV$_{max}$ = maximum standardized uptake value.

concavity. Specific ^{18}F-FDG uptake by tumors of a particular gross type can be explained by their relationship with the depth of invasion. Submucosal invasion of type 0-I or 0-IIa tumors was 84.3%, compared with 43.3% for types 0-IIb, 0-IIc, and 0-III tumors ($P < .001$).

The ^{18}F-FDG PET/CT has low sensitivity for signet ring cell gastric carcinoma. The FDG-based detectability of this tumor ranges from 14.3% to 70.6%, which is significantly lower than

that of non-signet ring cell carcinoma (52.9–100%) and is consistent with its significantly lower SUV$_{max}$.[10,19,21,24] In our univariate analysis, non-signet ring cell carcinomas were more FDG-avid than were signet ring cell carcinomas, but this difference was not confirmed in the multivariate analysis.

Lymph node metastasis is not a feature of primary tumors, and related data were not obtainable from our patients with ESD. Nonetheless, a close relationship between lymph node status and

Table 3

Multivariate analysis between 2-[¹⁸F]fluoro-2-deoxy-ᴅ-glucose uptake and clinicopathologic variables.

Factor	P-value	Exp (B)	95% CI
Location	.035	1.941	1.048–3.593
Gross type	<.001	0.444	0.294–0.672
WHO classification	.078	0.803	0.629–1.025
Tumor size	.026	2.527	1.117–5.716
Depth of invasion	.007	3.481	1.398–8.669

CI = confidence interval, FDG = 2-[¹⁸F]fluoro-2-deoxy-ᴅ-glucose, WHO = World Health Organization.

the FDG-avidity of primary tumors has been reported.[7,25] In a study of 27 patients with EGC, Mukai et al determined that 7 patients had FDG-positive tumors, including 2 patients with lymph node metastasis. In a series of 20 patients with FDG-negative EGC, no patient had lymph node metastasis.[7] We found that among the 195 patients with surgically resected EGCs, 39 had FDG-positive tumors, of whom 10 (25.6%) also had lymph node metastasis, compared with 25 (16.0%) of 156 FDG-negative EGCs. Accurate assessment of lymph node status is crucial in treatment planning for EGC, because ESD is indicated when the likelihood of lymph node metastasis is extremely low.[27] Together with the results of the present study, predicting FDG-avidity of EGC by PET/CT would be of benefit to clinicians in determining the most appropriate treatment in patients with EGC.

In the present study, the 4 significant variables identified in the multivariate analysis were used as indicators in a clinical scoring system, using a cut-off value of 2.5 (based on a score of 0–4). This scoring system was developed to select patients with EGC who will benefit from ¹⁸F-FDG PET/CT. Kaneko et al also developed a pretreatment PET-based scoring system for gastric cancer, but their weighted scores were obtained by transforming the odds ratios of the 4 significant variables to a logarithmic scale.[19] In their system, large (>3.0 cm), glucose transporter 1-positive tumors resulted in high scores, even in patients with EGC. Despite differences in the patient population, the measured variables, and the scoring method, the accuracy of detecting FDG-avid gastric

cancers (sensitivity/specificity = 85%/71%) was similar to that obtained by our own method.

There were several limitations to our study. First, it was retrospective in its design and involved patients from a single center, such that selection bias was unavoidable. Second, in general, ¹⁸F-FDG PET has a low rate of detection of EGC, which may explain why there were only 49 true-positive cases (17.5%). Nonetheless, the population analyzed in this study was larger than those in previous ¹⁸F-FDG PET-based investigations of EGC. Finally, using ¹⁸F-FDG PET, tumors with a small volume can be underestimated by partial volume averaging effects, which may affect the positivity rate of ¹⁸F-FDG PET. Therefore, the results and the scoring system of this investigation can be generally applied after being validated further in other population.

In conclusion, among the many clinicopathologic factors evaluated in this study, the location, gross type, size, and depth of invasion of the primary tumor were independently related to ¹⁸F-FDG uptake in EGC. A clinical scoring system based on these variables was used to predict the FDG-avidity of EGC, with modest sensitivity and high specificity. Because ¹⁸F-FDG PET/CT is not routinely performed for EGC staging, our clinical scoring system may help clinicians identify those patients with EGC likely to benefit from this imaging modality, for instance, predicting the risk of lymph node metastasis.

Author contributions

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Table 4

Clinical scores and 2-[¹⁸F]fluoro-2-deoxy-ᴅ-glucose avidity.

score	Clinicopathologic variables				No. of patients	FDG-avidity, %
	Location	Gross type	Size	DOI		
4	Lower	0–I, 0–IIa	≥2.5	Submucosa	13	84.6
3	Lower	0–IIb, 0–IIc, 0–III	≥2.5	Submucosa	23	34.8
3	Lower	0–I, 0–IIa	≥2.5	Mucosa	6	33.3
3	Upper, mid	0–I, 0–IIa	≥2.5	Submucosa	6	33.3
3	Lower	0–I, 0–IIa	<2.5	Submucosa	1	100.0
2	Lower	0–IIb, 0–IIc, 0–III	<2.5	Submucosa	19	21.1
2	Lower	0–IIb, 0–IIc, 0–III	≥2.5	Mucosa	17	5.9
2	Upper, mid	0–IIb, 0–IIc, 0–III	≥2.5	Submucosa	16	12.5
2	Upper, mid	0–I, 0–IIa	<2.5	Submucosa	8	25.0
2	Upper, mid	0–I, 0–IIa	≥2.5	Mucosa	2	50.0
2	Lower	0–I, 0–IIa	<2.5	Mucosa	2	0.0
1	Lower	0–IIb, 0–IIc, 0–III	<2.5	Mucosa	41	4.9
1	Upper, mid	0–IIb, 0–IIc, 0–III	<2.5	Submucosa	29	3.4
1	Upper, mid	0–IIb, 0–IIc, 0–III	≥2.5	Mucosa	10	0.0
1	Upper, mid	0–I, 0–IIa	<2.5	Mucosa	1	100.0
0	Upper, mid	0–IIb, 0–IIc, 0–III	<2.5	Mucosa	35	5.7

DOI = depth of invasion, FDG = 2-[¹⁸F]fluoro-2-deoxy-ᴅ-glucose.

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