18F-FDG PET/CT in Late Acquisition Identifies Sites of Active Disease in Treated Takayasu Arteritis

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Objective: Few studies have taken advantage of 18F-fluorodeoxyglucose positron emission tomography associated with computed tomography (18F-FDG PET/CT) to personalize patient evaluation and identify sites of more active disease in Takayasu arteritis (TA)-treated patients. This study aimed to evaluate the utility of 18F-FDG PET/CT in late acquisition in identifying sites of active disease in patients under full treatment for TA.

Methods: In this cross-sectional study, patients under full treatment underwent whole-body 18F-FDG PET/CT. Sites of increased 18F-FDG uptake were classified by a score of 3 on the visual scale using the liver uptake as reference. A quantitative analysis was also performed by measuring the maximum standardized uptake value (SUV) of the vascular wall of affected arteries. Disease activity using the National Institutes of Health criteria was also evaluated.

Results: Of the 20 patients, there were 18 female and 2 male patients, with a mean age of 43.6 (±11.58) years and a disease duration of 8.3 (±6.25) years. Thirteen participants (65%) were in inflammatory activity according to the criteria proposed by the National Institutes of Health. All patients received immunosuppressive agents, and one of them received immunobiological treatment. The highest SUV value was 6.2 in the aortic arch, and the lowest was 1.0 in the subclavian artery. The mean maximum SUV did not differ between clinically active and inactive patients. In the visual analysis, all participants had at least 1 vascular site with inflammatory activity, with an uptake ≥2 in relation to the liver. The aortic arch was the most frequently involved site.

Conclusions: This study showed that 18F-FDG PET/CT in late acquisition is an effective imaging method to assess TA activity even in fully treated patients.

Key Words: Takayasu arteritis, 18F-fluorodeoxyglucose positron emission tomography, disease activity

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Takayasu arteritis (TA) is a vasculitis of large vessels defined as inflammation in the aorta and its main branches.1 Evaluation of disease activity in large vessel vasculitis (LVV) is still a challenge with a direct impact on the therapeutic decision and prevention of severe ischemic complications. Patients in apparent clinical remission often show progression of the disease even in the course of treatment and with evidence of normal values of inflammatory activity markers.2,3

18F-Fluorodeoxyglucose positron emission tomography (18F-FDG PET) is based on the identification of areas of increased glucose metabolism in inflammatory cells, showing hypermetabolic areas in neoplastic, infectious, and inflammatory processes. Several studies have documented the usefulness of 18F-FDG PET in granulomatous inflammation such as sarcoidosis, granulomatosis with polyangiitis, giant cell arteritis, and TA, as well as to evaluate the extent of vascular involvement in inflammatory diseases.4,5 The use of 18F-FDG PET associated with computed tomography (CT) increases the accuracy of the anatomic localization of radiopharmaceutical accumulation on the vessel wall in rheumatic diseases.6 To date, the use of 18F-FDG PET has become a valuable complement in assessing activity in TA. The increase in FDG uptake in the arterial wall may precede clinical relapses in clinical remission patients and future structural changes detected by other imaging modalities.7,8

Glucocorticoids, immunosuppressants, and immunobiological agents inhibit the release of cytokines and inflammatory mediators, which may alter the assessment of disease activity.9 Many studies that described a correlation between vascular uptake by 18F-FDG PET/CT and disease activity include other LVV and different imaging techniques, either by visual or semiquantitative analysis.10,11 During examination, image acquisition generally occurs 60 minutes after the radiopharmaceutical injection.11 Image acquisition time differs among studies of LVV patients. Images were acquired early (40 and 60 minutes) in previous TA studies using 18F-FDG PET/CT.10,12–15 Martinez-Rodriguez et al16 showed that late imaging (180 minutes) resulted in increased FDG uptake in the arterial wall, increasing the possibility that PET/CT could be interpreted as active vasculitis. A more recent study demonstrated that image acquisition after 2 hours of tracer injection was better than conventional imaging after 1 hour to detect vascular activity inflammation in LVV, increasing PET sensitivity from 56% to 77%.17

To date, there are few reports that use late 18F-FDG PET/CT image acquisition to study LVV. The objective of this study is to evaluate the utility of 18F-FDG PET/CT in monitor sites of active disease in TA patients under full treatment, 120 minutes after the radiopharmaceutical infusion.

METHODS

Study Design and Settings

This is a cross-sectional study conducted at the Rheumatology Unit of the State University of Campinas, São Paulo, Brazil.

Ethics and Informed Consent

This study was performed in accordance with the Declaration of Helsinki and was approved by the ethics committee of the Faculty of Medical Sciences of the University of Campinas—Unicamp (CAAE-51793915.5.0000.5404). All subjects provided a written informed consent before study.
Subjects

Between July 2017 and January 2018, 25 consecutive patients with a diagnosis of TA were selected for the study. Inclusion criteria were the fulfillment of the American College of Rheumatology classification criteria for TA, aged older than 18 years, and regular follow-up. The exclusion criteria included history of iodine allergy, renal insufficiency, heart failure, asthma, pregnancy, and sickle cell anemia.

Assessment Measurements for Disease Activity

All patients had their clinical history registered and underwent detailed physical examination and laboratory tests on the day of the 18F-FDG PET/CT examination. Disease activity was assessed according to the criteria proposed by the National Institutes of Health (NIH), which consists of a worsening of 2 or more of the following criteria: (1) systemic complaints with no other identified cause (fever, arthralgia, myalgia, weight loss, and asthenia); (2) increased erythrocyte sedimentation rate (ESR); (3) ischemia (claudication, heart murmur, decreased or absent murmur, blood pressure asymmetry, pain in vessel pathways); and (4) new arteriographic changes.19

Disease duration was defined as less than 3 years, between 3 and 6 years, and more than 6 years. The ESR and C-reactive protein (CRP) tests were used to assess disease activity, with normal values below 20 mm/h for ESR and 0.3 mg/dL for CRP.

All patients were on immunosuppressive and/or immunobiological treatment. Patients considered in disease remission stage were treated with prednisone at doses below 20 mg daily.

Classification of obesity followed the recommendations of the World Health Organization,20 and dyslipidemia followed the recommendations of the V Brazilian Guidelines on Dyslipidemias and Prevention of Atherosclerosis.21

Examination Protocol

Capillary glycemia was measured after an 8-hour fast. Fluorodeoxyglucose 4.44 GBq/kg (0.12 mCi/kg) was administered 120 minutes before examination in a Biograph Truepoint PET/CT model mCT (Siemens USA). All images were acquired after a 35-mA topogram from the head to the root of the thighs, followed by a whole-body CT with kV and mA modulated by the topogram, and finally a PET with a duration of 90 seconds per bed position every 20 cm. The acquisitions of the images were performed at 120 minutes to increase the sensitivity of the method.

Interpretation of Findings

The examinations were interpreted by 2 experienced nuclear physicians without knowing the patients' identities and clinical histories. Divergent cases were discussed in a consensus meeting.

The anatomic locations selected for analysis included the aortic arch, brachiocephalic trunk, right and left subclavian arteries, right and left carotid arteries, ascending aorta, descending aorta, abdominal aorta, and renal arteries. 18F-FDG uptake was measured according to visual and quantitative analysis.

Quantification was performed using standardized uptake value (SUV). The area of greatest vascular uptake of 18F-FDG was considered for calculating maximum SUV (SUVmax).

Visual analysis was performed following the technique proposed by Meller et al.22 The vascular uptake was compared with liver uptake: degree 0 (no uptake), degree 1 (lower uptake than liver), degree 2 (same uptake as liver), and degree 3 (greater uptake than liver). Equivocal cases (when deciding between grades 1 and 2 or between 2 and 3) were defined using SUV quantification. In these cases, liver mean SUV (SUVmean) measurements were taken from normal-appearing right lobe of the liver. Grade 2 was considered when lesion uptake was equal to liver SUVmean ± 2 standard deviations.

Disease activity was considered when the degree was ≥2 in one of the analyzed arterial locations.

Statistical Analysis

The data were processed in the SAS System for Windows (Statistical Analysis System, version 9.4; SAS Institute Inc, 2002–2012, Cary, NC).

To describe the profile of the sample under study, the frequency of categorical variables were described in absolute (n) and percentage (%) frequency values, and for quantitative variables, descriptive measures (mean, standard deviation, minimum, median, and maximum) were obtained.

Fisher test was used to assess the association of PET/CT with the use of immunosuppressants, the duration of the disease, CRP level >0.3, and disease activity according to the criteria proposed by the NIH. In the quantitative assessment of the SUV; the values were grouped using the mean of each vascular site as the cutoff point.

The level of significance adopted for the study was 5%.

RESULTS

Twenty-five patients were initially selected for the study, 4 of whom were excluded due to renal failure and one due to acute myocardial infarction. This study included 20 patients, and 18 (90%) were women. The mean age at the time of examination was 43.6 years (±11.58), and the mean age at diagnosis was 36.6 years (±9.69). Most patients had more than 6 years of illness (45%), 6 patients between 3 and 6 years (30%), and 5 patients less than 3 years (25%). There was a predominance of patients who were overweight (40%) and with obesity (35%). Low-density lipoprotein cholesterol levels above 70 mg/dL were present in 18 participants (90%). All patients involved in the study were currently using immunosuppressive agents (methotrexate or leflunomide), and only one (5%) under immunobiological treatment at the time of the 18F-FDG PET/CT. Sixteen patients (80%) were taking prednisone, 10 of them (62.5%) with a dose above 20 mg/d. Erythrocyte sedimentation rate values were above 20 mm/h in 14 patients (70%), and CRP values were above 0.3 mg/dL in 13 patients (65%).

| Variable | Total (N = 20) |
|----------|---------------|
| Age, mean (range), y | 43.6 (23–71) |
| Age at diagnosis, mean (± SD), y | 36.6 (19–55) |
| Sex, n (% female) | 18 (90) |
| Race, n (%) | |
| White | 16 (80) |
| Asian | 1 (5) |
| African descendants | 3 (15) |
| Obesity, n (%) | 7 (35) |
| Overweight, n (%) | 8 (40) |
| Dyslipidemia, n (%) | 18 (90) |
| Immunosuppression, n (%) | 16 (80) |
| Immunobiological, n (%) | 1 (5) |
| Prednisone >20 mg/d, n (%) | 10 (62.5) |
| ESR >20 mm/h, n (%) | 14 (70) |
| CPR >0.3 mg/dL, n (%) | 13 (65) |
| Active disease (NIH criteria), n (%) | 13 (65) |
According to the NIH criteria, 13 participants (65%) experienced inflammatory activity, and there was no significant association between disease activity and CRP ($p = 0.1736$). The baseline characteristics of the patients are presented in Table 1.

In visual analysis, 19 participants (95%) presented at least 1 vascular site with active inflammation. In decreasing order of frequency, active disease (visual grade 2 or 3) was found in the following sites: aortic arch, descending aorta, ascending aorta, carotid arteries, abdominal aorta, subclavian arteries, and brachiocephalic trunk. The visual grade $\geq 2$ was found in 12 (92%) of the 13 patients with clinical disease activity, 11 with uptake grade 3 uptake. Between patients without clinical activity of the disease, all presented vascular activity on PET/CT images, 4 with grade 3 uptake.

In quantitative analysis, considering the values at each vascular site, the highest $SUV_{max}$ were 6.2, 5.0, and 4.5 in the aortic arch, ascending aorta, and descending aorta, respectively. Seven (35%) of the 20 patients had maximum $SUV_{max}$ equal to or above liver SUVs, independent of the evaluation of the disease activity according to the criteria suggested by the NIH. The mean values of $SUV_{max}$ founded in the evaluated arteries are described in Table 2.

There was no significant difference between the means or medians of the SUV values at each vascular site evaluated among the patients with and without disease activity according to NIH criteria. The mean $SUV_{max}$ did not differ between clinical active and inactive results, with values of 3.53 and 3.21, respectively.

There was no correlation between disease duration and mean $SUV_{max}$ values at each vascular site.

In both visual and quantitative analysis, prednisone doses and immunosuppressive therapy had no influence on radiopharmaceutical uptake among patients considered active and inactive at each vascular site.

Only in abdominal aorta, a significant association was found between CRP greater than 0.3 mg/dL and $^{18}$F-FDG uptake greater than the mean of all abdominal aorta SUV measurements ($p = 0.008$).

### TABLE 2. Values of SUVs at Each Vascular Site Evaluated

| Location | Mean | Median | Standard Deviation | Minimum | Maximum | $p$ value |
|----------|------|--------|--------------------|---------|---------|-----------|
| AoA      |      |        |                    |         |         |           |
| Total group (n = 20) | 3.0   | 2.8    | 1.0                | 1.5     | 6.2     |           |
| Active NIH (n = 13)  | 3.1   | 2.7    | 1.1                | 2.0     | 6.2     |           |
| Inactive NIH (n = 7) | 2.7   | 2.8    | 0.9                | 1.5     | 4.3     | 0.6060    |
| AbA      |      |        |                    |         |         |           |
| Total group (n = 20) | 2.9   | 2.8    | 0.6                | 1.8     | 4.2     |           |
| Active NIH (n = 13)  | 3.1   | 3.1    | 0.7                | 1.8     | 4.2     |           |
| Inactive NIH (n = 7) | 2.6   | 2.6    | 0.3                | 2.2     | 3.1     | 0.0677    |
| DA       |      |        |                    |         |         |           |
| Total group (n = 20) | 2.9   | 2.8    | 0.8                | 1.8     | 4.5     |           |
| Active NIH (n = 13)  | 2.8   | 2.8    | 0.6                | 1.9     | 3.8     |           |
| Inactive NIH (n = 7) | 3.0   | 2.8    | 1.1                | 1.8     | 4.5     | 0.8736    |
| AsA      |      |        |                    |         |         |           |
| Total group (n = 20) | 2.8   | 2.7    | 0.8                | 1.7     | 5.0     |           |
| Active NIH (n = 13)  | 3.0   | 2.9    | 0.9                | 1.7     | 5.0     |           |
| Inactive NIH (n = 7) | 2.5   | 2.5    | 0.3                | 2.0     | 3.1     | 0.2332    |
| BB       |      |        |                    |         |         |           |
| Total group (n = 20) | 2.3   | 2.1    | 0.6                | 1.4     | 3.3     |           |
| Active NIH (n = 13)  | 2.1   | 2.0    | 0.4                | 1.4     | 3.1     |           |
| Inactive NIH (n = 7) | 2.5   | 2.8    | 0.7                | 1.7     | 3.3     | 0.3179    |
| LC       |      |        |                    |         |         |           |
| Total group | 2.3   | 2.5    | 0.6                | 1.2     | 3.2     |           |
| Active NIH   | 2.4   | 2.5    | 0.5                | 1.6     | 3.2     |           |
| Inactive NIH | 2.1   | 2.4    | 0.6                | 1.2     | 2.8     | 0.2321    |
| RS       |      |        |                    |         |         |           |
| Total group | 2.1   | 2.2    | 0.5                | 1.2     | 3.2     |           |
| Active NIH   | 2.1   | 2.1    | 0.5                | 1.4     | 3.2     |           |
| Inactive NIH | 2.0   | 2.2    | 0.7                | 1.2     | 2.9     | 1.0000    |
| RC       |      |        |                    |         |         |           |
| Total group | 2.1   | 2.0    | 0.5                | 1.1     | 3.4     |           |
| Active NIH   | 2.1   | 2.0    | 0.5                | 1.4     | 3.4     |           |
| Inactive NIH | 1.9   | 1.9    | 0.6                | 1.1     | 2.7     | 0.5508    |
| LS       |      |        |                    |         |         |           |
| Total group | 1.9   | 2.0    | 0.5                | 1.0     | 2.9     |           |
| Active NIH   | 1.8   | 1.8    | 0.5                | 1.0     | 2.9     |           |
| Inactive NIH | 2.1   | 2.2    | 0.4                | 1.5     | 2.5     | 0.1733    |

AoA, aortic arch; AbA, abdominal aorta; DA, descending aorta; AsA, ascending aorta; BB, brachiocephalic branch; LC, left carotid; LS, left subclavian; RC, right carotid; RS, right subclavian.
Table 3 shows the characteristics, laboratory results, and visual scale results of 18F-FDG PET/CT of each patient. Figure shows the 18F-FDG PET/CT images of a patient with clinical active disease.

**DISCUSSION**

Nuclear imaging has gained importance as a noninvasive method for diagnosis and monitoring of inflammatory diseases. In TA, even with the improvement of imaging techniques, assessing disease activity is still a challenge. This study showed that 18F-FDG PET/CT in late acquisition is an effective imaging method to assess TA activity even in full treatment patients.

The activity criterion proposed by Kerr et al was most widely used in clinical practice, although clinical characteristics do not correlate with inflammatory evidence in approximately 50% of cases. In this study, CRP was correlated with 18F-FDG uptake only in abdominal aorta in quantitative analysis. At other sites, ESR and CRP levels were not related to intensity of uptake in 18F-FDG PET/CT, neither in visual nor in quantitative analysis.

Corticosteroids, immunosuppressants, and immunobiological agents are known to reduce FDG uptake. Nevertheless, in the present study, all patients were under immunosuppression and most presented activity at the image examination, both by visual and quantitative analyses, regardless of the presence of clinical activity according to the criteria proposed by Kerr et al. Furthermore, even those patients who used more than 20 mg/d of prednisone had disease activity in PET evaluation. When the visual analysis was considered, 95% of patients presented upgrade 2 or 3 in at least 1 anatomic location, also independently of clinical activity (Table 4). Recent evidence demonstrates that immunobiological therapy (infliximab and tocilizumab) results in fewer relapses than the disease-modifying antirheumatic drugs (DMARDS). Gudbrandsson et al reports the appearance of new arterial lesions in 40% of patients treated with DMARDS, whereas this occurs in only 10% in those patients treated with immunobiological therapy. These findings also support our results that even in patients under immunosuppression with DMARDS, 18F-FDG PET/CT can identify inflammatory activity on the vessel wall, probably due to smoldering inflammatory vessel activity and might be a predictor of clinical recurrence.

Many previously published studies acquired images 60 minutes after 18F-FDG infusion. In a comparing image acquisition 60 and 180 minutes after 18F-FDG infusion in patients with LVV, the authors reported more accurate results at 180 minutes, with better quality images. The authors suggest that later acquisitions are more accurate to assess inflammation on the vascular wall using 18F-FDG PET. A recent study has shown that the time taken to acquire images significantly influences the interpretation of disease activity in PET scans performed on patients with TA and giant cell arteritis. The later evaluation, up to 120 minutes, results in increased radiopharmaceutical uptake in the vascular wall, increasing the likelihood of

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Table 3. Association Between Disease Activity at Each Vascular Site Evaluated and Time of Disease, in Visual Analysis Compared With Liver Uptake

|        | <3 y, n = 5 (%) | 3–6 y, n = 6 (%) | >6 y, n = 9 (%) | Total, n = 20 (%) | p value |
|--------|----------------|-----------------|----------------|------------------|---------|
| AsA    |                |                 |                |                  | 1.000   |
| Active disease | 4 (80)          | 5 (56)          | 7 (78)         | 16 (80)          |         |
| Inactive disease | 1 (20)          | 1 (44)          | 2 (22)         | 4 (20)           |         |
| AoA    |                |                 |                |                  | 0.047   |
| Active disease | 4 (80)          | 3 (50)          | 7 (78)         | 14 (70)          |         |
| Inactive disease | 1 (20)          | 3 (50)          | 2 (22)         | 6 (30)           |         |
| DA     |                |                 |                |                  | 0.625   |
| Active disease | 2 (40)          | 3 (50)          | 6 (67)         | 11 (55)          |         |
| Inactive disease | 3 (60)          | 3 (50)          | 3 (33)         | 9 (45)           |         |
| AbA    |                |                 |                |                  | 0.310   |
| Active disease | 1 (20)          | 0               | 3 (33)         | 4 (20)           |         |
| Inactive disease | 4 (80)          | 6 (100)         | 6 (67)         | 16 (80)          |         |
| BB     |                |                 |                |                  | 1.000   |
| Active disease | 3 (60)          | 3 (50)          | 5 (56)         | 11 (55)          |         |
| Inactive disease | 2 (40)          | 3 (50)          | 4 (44)         | 9 (45)           |         |
| RC     |                |                 |                |                  | 0.728   |
| Active disease | 3 (60)          | 0               | 2 (22)         | 5 (25)           |         |
| Inactive disease | 2 (40)          | 6 (100)         | 7 (78)         | 15 (75)          |         |
| LC     |                |                 |                |                  | 0.319   |
| Active disease | 3 (60)          | 1 (44)          | 5 (56)         | 9 (45)           |         |
| Inactive disease | 2 (40)          | 5 (56)          | 4 (44)         | 11 (55)          |         |
| RS     |                |                 |                |                  |         |
| Active disease | 0               | 0               | 2 (22)         | 2 (10)           |         |
| Inactive disease | 5 (100)         | 6 (100)         | 7 (78)         | 18 (90)          |         |
| LS     |                |                 |                |                  | 0.415   |
| Active disease | 0               | 1 (44)          | 3 (33)         | 4 (20)           |         |
| Inactive disease | 5 (100)         | 5 (56)          | 6 (67)         | 16 (80)          |         |

AsA indicates ascending aorta; AoA, aortic arch; DA, descending aorta; AbA, abdominal aorta; BB, brachiocephalic branch; RC, right carotid; LC, left carotid; RS, right subclavian; LS, left subclavian.
Late FDG PET/CT images reduce the possibility of interference by the presence of circulating FDG. Studies with early image acquisition suggest that $^{18}$F-FDG PET/CT is a useful tool to diagnose and follow-up LVV patients. Lee et al. found a 75% correlation in clinical activity using $^{18}$F-FDG PET according to the NIH criteria, and Teruza et al. evidenced a sensitivity of 92.6% and specificity of 91.7% of the PET to evaluate vascular activity in TA. Our study did not find any association of clinical activity with the presence of activity by $^{18}$F-FDG PET/CT. Our findings corroborate those described by Arnaud et al., who demonstrated a low correlation between inflammatory evidence on PET images and clinical activity. More recently, Zhang et al. found a positive correlation between clinical disease activity and SUV values, which was not found in our study. We found similar values of mean SUV$_{\text{max}}$ among clinically active and inactive patients with values of 2.86 and 2.85, respectively. In agreement with Soriano et al. and Janes et al., our study showed the highest values of SUV$_{\text{max}}$ in the aorta thoracic segments of the aorta. Although it is unclear whether increased uptake of $^{18}$F-FDG in the vascular wall is directly correlated with current inflammatory activity or is a consequence of vascular remodeling or atherosclerosis, facing these results, we believe that our findings are related to disease activity. According to the Cardiovascular Committee of the European Association of Nuclear Medicine (EANM), for the assessment of atherosclerosis by PET, it is recommended to acquire images 2 hours after the infusion of $^{18}$F-FDG. This time interval is important due to the lower concentration of the circulating radiopharmaceutical and, consequently, greater accumulation in the arterial wall. The objective of our study was to evaluate the utility of $^{18}$F-FDG PET/CT in monitoring sites of active disease in TA patients under full treatment 120 minutes after the radiopharmaceutical infusion. Takayasu arteritis is strongly associated with atherosclerosis and arterial calcification, even in younger patients, especially where there is inflammation. Differentiating arterial vascular lesions of atherosclerotic or vasculitic causes using PET is challenging. The anatomical location, distribution, and intensity of radiopharmaceutical uptake are important factors to be considered in interpretation and differentiation. The vessels preferentially involved in atherosclerosis are the abdominal aorta, popliteal arteries, descending thoracic aorta, and carotids. In vasculitis, the thoracic vessels, common carotid, subclavian, and axillary arteries are usually the most involved site. Regarding the distribution of radiopharmaceuticals, when presenting a linear aspect, they are more suggestive of vasculitis. Finally, marked uptake (grade 2 or 3) is more consistent with arterial vascular inflammation than atherosclerosis. Our study evaluated 20 patients with marked arterial activity in the ascending aorta and aortic arch, both at visual

### TABLE 4. TA Patients Who Underwent an $^{18}$F-FDG PET CT Examination—Visual Analysis

| Sex | Age, y | Stage of Disease | ESR | CRP | Visual Scale$^a$ |
|-----|--------|------------------|-----|-----|-----------------|
| M   | 38     | Active           | 61  | 0.95| 3               |
| F   | 36     | Active           | 57  | 1.62| 1               |
| F   | 51     | Active           | 49  | 1.18| 3               |
| F   | 46     | Inactive         | 46  | 1.5 | 3               |
| F   | 32     | Active           | 43  | 0.56| 3               |
| F   | 51     | Active           | 39  | 1.07| 3               |
| F   | 32     | Active           | 38  | 1.49| 3               |
| F   | 46     | Active           | 36  | 0.56| 3               |
| F   | 23     | Active           | 30  | 0.23| 3               |
| F   | 34     | Active           | 26  | 0.59| 3               |
| F   | 48     | Active           | 24  | 0.53| 2               |
| F   | 71     | Inactive         | 23  | 1.19| 3               |
| F   | 50     | Active           | 21  | 3.04| 3               |
| F   | 40     | Inactive         | 15  | 0.13| 3               |
| F   | 60     | Inactive         | 14  | 0.03| 3               |
| F   | 30     | Active           | 10  | 0.86| 3               |
| F   | 54     | Inactive         | 9   | 0.31| 2               |
| F   | 33     | Inactive         | 7   | 0.2 | 2               |
| F   | 47     | Inactive         | 6   | 1.22| 2               |
| F   | 50     | Active           | 3   | 0.02| 3               |

$^a$ Grade of vascular uptake compared with liver uptake.
and quantitative analysis. In addition, the present study did not find association between disease duration and vascular activity by PET, also suggesting the presence of disease activity rather than atherosclerosis. These results lead us to believe that the findings are due to disease activity and not just atherosclerosis. There was also no correlation between time of disease and SUVs. The frequency of disease activity according to NIH criteria and mean SUV are shown in Table 3.

PET/CT is an image test with high sensitivity for diagnosis and follow-up of patients with LVV and has the advantage of detecting metabolic alteration and evaluating the extension of the inflammatory process in the same test. A crucial point observed in this study is that vascular structures with higher FDG uptake vary significantly among patients, suggesting that the response to treatment may be heterogeneous and that $^{18}$F-FDG PET/CT allows the individualization of locations at higher risk of ischemic complications.

The limitations of the study include the small number of patients studied in a single center and not including control patients. The absence of a control group did not allow us to determine a cut-off value for the SUV to define disease activity.

Our study demonstrates that $^{18}$F-FDG PET/CT is an effective imaging method to assess arterial wall inflammation, 120 minutes of radiopharmaceutical infusion, even in patients under treatment. This finding may be of great importance to detect subclinical activity and possible later ischemic complications.

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