Relationship between fluorodeoxyglucose uptake in the large vessels and late aortic diameter in giant cell arteritis


Objective. GCA carries an increased risk of developing thoracic aortic aneurysms. Previous work with fluorodeoxyglucose (FDG)-PET has shown that the aorta is frequently involved in this type of vasculitis. We wanted to investigate whether there is a correlation between the extent of vascular FDG uptake during the acute phase of GCA and the aortic diameter at late follow-up.

Methods. All patients with biopsy-proven GCA who ever underwent an FDG-PET scan in our centre were asked to undergo a CT scan of the aorta. The diameter of the aorta was measured at six different levels (ascending aorta, aortic arch, descending aorta, abdominal suprarenal, juxtagenital and infrarenal aorta) and the volumes of the thoracic and of the abdominal aorta were calculated.

Results. Forty-six patients agreed to participate (32 females, 14 males). A mean of 46.7 ± 29.9 months elapsed between diagnosis and CT scan. All aortic dimensions were significantly smaller in women than in men, except for the diameter of the ascending aorta. Patients who had an increased FDG uptake in the aorta at diagnosis of GCA, had a significantly larger diameter of the ascending aorta (P = 0.006), and descending aorta (P = 0.044) and a significantly larger volume of the thoracic aorta (P = 0.029). In multivariate analysis, FDG uptake at the thoracic aorta was associated with late volume of the thoracic aorta (P = 0.039).

Conclusion. GCA-patients with increased FDG uptake in the aorta may be more prone to develop thoracic aortic dilatation than GCA patients without this sign of aortic involvement.

Keywords: Giant cell arteritis, Temporal arteritis, Vasculitis, Large-vessel vasculitis, Aorta, Positron Emission tomography, Fluorodeoxyglucose, Aortic dilatation, Vascular inflammation, Aortic aneurysm.

Introduction

GCA is the most frequent vasculitis of elderly persons. The age-adjusted annual incidence of GCA is 188–220 cases per million in the United States and the United Kingdom [1, 2] and 290 per million in Norway [3]. Although GCA patients have similar survival curves than their age-controlled peers, the two most feared complications of GCA are blindness and aortic rupture.

GCA carries a 17.3-fold increased risk of developing thoracic aortic aneurysms and a 2.4-fold increased risk of developing abdominal aortic aneurysms [4].

In a study of 210 biopsy-proven GCA patients, 20 (9.5%) developed aortic aneurysmal disease, 16 times at the thoracic level and 6 times at the abdominal level. The incidence of aortic aneurysm and/or dissection was 18.9/1000 person-years at risk [5].

In a study of 1064 patients with aortic aneurysms, aortitis not associated with prior surgery, infection or atherosclerosis was found in 4.4% of surgical specimens, predominantly from women and almost completely restricted to the thoracic level (96%). In 26% (12 of 47), aortitis occurred in patients with a prior history of a systemic illness for which aortitis was a known complication, of whom four had had GCA. In 16 of 36 patients with aortitis not related to prior systemic disease, giant cells were seen in the surgical specimens. Post-operatively, recurrent aneurysms were not identified among 11 corticoid-treated patients, while new aneurysms were found in 6 of 25 not so treated [6].

Studies with FDG-PET in GCA have shown that the aorta, both abdominal and thoracic, is involved in about 50% of the patients. In elderly patients with atypical symptoms such as fever, weight loss or malaise and biochemical signs of inflammation, the finding of increased FDG uptake in the larger thoracic vessels may directly lead to a diagnosis of GCA [7–9].

In the present study, we wanted to investigate if FDG-PET scintigraphy can predict which GCA patients are prone to aortic dilatation. We wondered if there was a correlation between the intensity of vascular FDG uptake during the acute phase of GCA and the aortic diameter at late follow-up.

Patients and methods

Patients

All patients with biopsy-proven GCA who ever underwent an FDG-PET scan in our centre were contacted, first by letter (as was their general practitioner), then by phone to explain the present study and to ask for their participation, i.e. to undergo a CT scan of the aorta. Several of these patients had been previously included in other studies of our group on FDG-PET scintigraphy in GCA [7–9]. All patients who accepted signed informed consent. Patients who had no transportation available were transported to the hospital and back home by our study nurses. The study was approved by the local ethics committee of our university hospital.

FDG-PET scintigraphy

Original FDG-PET scintographies, performed between 1996 and 2006, were reread by two independent nuclear medicine specialists, who were unaware of CT findings. Aortic FDG uptake was reported as negative (score 0 or 1) or positive (score 2 and 3), as was done in former publications [8] (Fig. 1).

CT scans of the aorta

A CT scan of the entire aorta was made, without contrast injection, with multiplanar reconstructions (transverse, coronal and sagittal). The diameter of the aorta was measured at six different levels (ascending aorta, aortic arch, descending thoracic...
aorta, abdominal suprarenal, juxtarenal and infrarenal aorta) and the volumes of the thoracic aorta and of the abdominal aorta were calculated (Fig. 2a and b). The diameter of the ascending thoracic aorta was usually measured in a transverse and sagittal plane, distal to the aortic root/sinus of Valsalva. The aortic arch was usually measured in a transversal and sagittal plane. The descending aorta was usually measured in a transversal and sagittal/coronal plane, most often at the first part, distal to the aortic arch, unless a larger diameter was found at its course down to the transition zone at the level of the diaphragm. The suprarenal part of the abdominal aorta was usually measured transversely; another plane was chosen in case of tortuosity. The diameter was usually measured at the level of the origin of the superior mesenteric artery. The juxtarenal abdominal aorta was usually measured transversely, except in case of tortuosity, where a sagittal/coronal plane was chosen to obtain correct measurement perpendicular to the aorta, at the level of the origin of the (main) renal arteries. For the diameter of the abdominal aorta (also usually measured transversely, except in case of tortuosity), the largest diameter of the aorta between the origin of the renal arteries and the aortic bifurcation was given.

To obtain the volume of the thoracic and abdominal aorta, the thoracic and abdominal aorta were segmented semi-automatically using the auto-snap tool of Voxar 3D (Barco, Kortrijk, Belgium). Next, the volumes of both parts were calculated automatically. All measurements were done by radiologists who were unaware of the FDG-PET findings.

Statistics

Data are given as mean ± s.d. The Pearson chi-square test and the Kruskal–Wallis H-test or the Mann–Whitney U-test were used to compare categorical and continuous variables, respectively. Spearman test was used to calculate bivariate correlations. For multivariate analysis, linear regression analysis was performed. Thoracic and abdominal aorta volumes were chosen as dependent variables; as independent variables, age, gender and body length were included, together with any individual parameter that would show a significant correlation with aortic diameters in univariate analysis.

All statistical testing was performed using two-tailed tests, with significance at P < 0.05. SPSS software, version 12.0, was used for statistical analyses (SPSS, Chicago, IL, USA).

Results

Population characteristics

Seventy-nine patients with biopsy-proven GCA underwent an FDG-PET scan in our centre between 1996 and June 2006. At the start of the study, 8 patients were lost to follow-up and 12 patients had died. Causes of death were myocardial infarction and calciphylaxis in two patients each, stroke, bowel ischaemia, lung carcinoma and terminal chronic obstructive lung disease in one patient each. In four other patients, no exact cause of death could be determined, although none died suddenly, as from aortic dissection.

The 59 remaining patients were contacted. Thirteen patients refused to participate. Forty-six patients (14 men, 32 women) accepted and signed informed consent. They underwent FDG-PET scintigraphy between March 1997 and June 2006 and underwent CT scan of the aorta between January and July 2006. The number of patients who agreed to participate is given in Fig. 3a according to the year of diagnosis and the number of those who could not be included is given in Fig. 3b.

At diagnosis, the age of the 46 included patients was 72.6 ± 6.4 yrs (range 49–85 yrs). The age of the 32 women at diagnosis was 73.3 ± 6.9 yrs (range 49–85 yrs); the age of the 14 men at diagnosis was 70.9 ± 4.8 yrs (P = 0.12).

The age of the 46 patients at the time of CT scan was 76.6 ± 6.3 yrs (range 55–89 yrs), the age of the 32 women at the time of CT scan was 77.1 ± 6.8 yrs (range 55–89 yrs), while the age of the 14 men was 75.4 ± 4.9 yrs (range 65–82 yrs), (P = 0.22). A mean of 46.7 ± 29.9 months expired between FDG-PET scan and CT scan (range 1–110 months).

Twenty-four patients were being treated for arterial hypertension while 12 were not. Ten patients got treatment for diabetes
mellitus while 36 did not. Mean sedimentation rate at diagnosis was 82 ± 30 mm/1 h (median 82 mm/h), mean CRP level at diagnosis was 82 ± 64 mg/l (median 62 mg/l).

By January 2007, 25 out of 46 patients had had at least one relapse of GCA, 17/46 did not yet relapse and 4/46 were still on steroid treatment. Mean total methylprednisolone intake until the time of CT scan was 5524 ± 2538 mg.

Twenty-two patients (48%) had a moderate to extensive FDG uptake at the thoracic aorta at diagnosis of GCA (FDG-PET scored 2 or 3 and reported positive); 24 patients (52%) had no or minimal FDG uptake at the thoracic aorta (FDG-PET scored 0 or 1 and reported negative). Ten patients (22%) had a moderate to extensive FDG uptake at the abdominal aorta at diagnosis, 35 patients (76%) had no or minimal FDG uptake at the abdominal aorta; in 1 patient (2%), the abdominal aorta was not visualized entirely up to the bifurcation.

Univariate analysis of possible risk factors for aortic dilatation

All aortic dimensions, except the diameter of the ascending aorta, were significantly larger in men than in women, as is shown in Table 1. There was no significant correlation between the age of the patients at CT scan and any aortic dimension, except for a negative correlation with the volume of the abdominal aorta ($P = 0.033$, Table 2).

The only aortic dimension that was very significantly correlated with body length was the volume of the abdominal aorta (positive correlation, $P = 0.004$), while there was a borderline significant positive correlation between body length and diameter of the aortic arch ($P = 0.049$, Table 2).
There was no relation between the number of months elapsed between diagnosis and CT scan on the one hand and any aortic dimension on the other hand, except for the diameter of the ascending aorta (positive correlation, \( P = 0.006 \), Table 2).

There was no relationship between the presence of arterial hypertension (Table 1), the presence of diabetes mellitus (Table 1) or the cholesterol level (Table 2) and any aortic dimension.

There was no relationship between the height of the sedimentation rate or the CRP level at diagnosis of GCA and any aortic dimension at follow-up CT scan (Table 2).

The aortic dimensions did not differ between patients who relapsed and those who did not, except that there was a negative correlation between relapse and the diameter of the ascending aorta (\( P = 0.024 \)). Patients who had undergone a relapse had a significantly smaller diameter than those who did not relapse (Table 1). There was no relation between the total steroid dose taken and any aortic dimension.

The relationship of the initial aortic FDG uptake with the corresponding aortic dimensions is given in Table 3. \( P \)-values <0.05 were obtained for the diameter of the ascending aorta (\( P = 0.025 \)), the diameter of the descending aorta (\( P = 0.044 \)) and the volume of the thoracic aorta (\( P = 0.029 \)). These were all positive correlations. At the level of the aortic arch and the entire abdominal aorta, no significant relationship was found with the initial FDG uptake at the corresponding level. Eleven patients out of 46 had a diameter of the ascending aorta exceeding 40 mm; nine of these had a positive FDG-PET scan at diagnosis (positive correlation, \( P = 0.0097 \)).

We could not find any relationship between FDG uptake at 3 months and any thoracic aortic dimension (data not shown). The same was true for residual FDG uptake at 6 months of treatment, except that there was a relation with the diameter of the descending thoracic aorta (\( P = 0.017 \)).

### Multivariate analysis of risk factors for aortic dilatation

The only independent variable that correlated with the volume of the thoracic aorta, was thoracic aortic FDG uptake at the time of diagnosis (\( P = 0.039 \)). Gender (\( P = 0.252 \), age at CT scan (\( P = 0.569 \)), body length (\( P = 0.470 \)) or the time elapsed since diagnosis (\( P = 0.572 \)) did not correlate independently with the volume of the thoracic aorta. For the abdominal aorta, no independent risk factor could be identified (gender: \( P = 0.195 \); age at CT scan: \( P = 0.228 \); body length: \( P = 0.379 \); months elapsed since diagnosis: \( P = 0.889 \); abdominal aortic FDG uptake: \( P = 0.646 \)).

### Table 1. Relationship between gender, relapse status, the presence or absence of arterial hypertension and diabetes mellitus on the one hand and the different aortic dimensions on the other hand

<table>
<thead>
<tr>
<th>Aortic dimensions (mean ± s.d.)</th>
<th>Gender</th>
<th>Relapse of GCA</th>
<th>Arterial hypertension</th>
<th>Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male patients</td>
<td>Female patients</td>
<td>Yes (n = 25)</td>
<td>No (n = 17)</td>
</tr>
<tr>
<td>Diameter ascending aorta (mm)</td>
<td>38.7 ± 3.8</td>
<td>38.6 ± 6.0</td>
<td>0.474</td>
<td>37.1 ± 3.4</td>
</tr>
<tr>
<td>Diameter aortic arch (mm)</td>
<td>32.7 ± 3.1</td>
<td>29.8 ± 3.5</td>
<td>0.002</td>
<td>29.9 ± 3.6</td>
</tr>
<tr>
<td>Diameter descending thoracic aorta (mm)</td>
<td>34.1 ± 3.4</td>
<td>31.1 ± 5.1</td>
<td>0.009</td>
<td>31.2 ± 4.0</td>
</tr>
<tr>
<td>Diameter suprarenal abdominal aorta (mm)</td>
<td>25.7 ± 2.0</td>
<td>23.2 ± 3.1</td>
<td>0.004</td>
<td>23.7 ± 3.1</td>
</tr>
<tr>
<td>Diameter infrarenal abdominal aorta (mm)</td>
<td>36.6 ± 2.0</td>
<td>21.2 ± 2.3</td>
<td>0.001</td>
<td>22.2 ± 2.7</td>
</tr>
<tr>
<td>Diameter infrarenal abdominal aorta (mm)</td>
<td>22.2 ± 2.4</td>
<td>19.4 ± 2.8</td>
<td>0.002</td>
<td>20.4 ± 3.1</td>
</tr>
<tr>
<td>Volume thoracic aorta (cm³)</td>
<td>303 ± 144</td>
<td>264 ± 77</td>
<td>0.005</td>
<td>262 ± 58</td>
</tr>
<tr>
<td>Volume abdominal aorta (cm³)</td>
<td>77 ± 14</td>
<td>56 ± 15</td>
<td>&lt;0.005</td>
<td>67 ± 19</td>
</tr>
</tbody>
</table>

### Table 2. Relation between age, body length, number of months between diagnosis and CT scan, sedimentation rate, CRP level and cholesterol level on the one hand and the different aortic dimensions on the other hand

<table>
<thead>
<tr>
<th>Aortic dimensions (mean ± s.d.)</th>
<th>Age at CT scan</th>
<th>Body length</th>
<th>Number of months between diagnosis and CT scan</th>
<th>Sedimentation rate at diagnosis</th>
<th>CRP at diagnosis</th>
<th>Cholesterol level</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correlation coefficient</td>
<td>P-value</td>
<td>Correlation coefficient</td>
<td>P-value</td>
<td>Correlation coefficient</td>
<td>P-value</td>
</tr>
<tr>
<td>Diameter of the ascending aorta (mm)</td>
<td>−0.121</td>
<td>0.424</td>
<td>−0.253</td>
<td>0.177</td>
<td>0.400</td>
<td>0.006</td>
</tr>
<tr>
<td>Diameter of the aortic arch (mm)</td>
<td>−0.124</td>
<td>0.410</td>
<td>0.363</td>
<td>0.049</td>
<td>0.128</td>
<td>0.396</td>
</tr>
<tr>
<td>Diameter of the descending thoracic aorta (mm)</td>
<td>−0.225</td>
<td>0.133</td>
<td>0.232</td>
<td>0.217</td>
<td>0.225</td>
<td>0.133</td>
</tr>
<tr>
<td>Diameter of the suprarenal abdominal aorta (mm)</td>
<td>−0.149</td>
<td>0.324</td>
<td>0.148</td>
<td>0.435</td>
<td>0.210</td>
<td>0.160</td>
</tr>
<tr>
<td>Diameter of the infrarenal abdominal aorta (mm)</td>
<td>−0.127</td>
<td>0.400</td>
<td>0.184</td>
<td>0.330</td>
<td>0.189</td>
<td>0.208</td>
</tr>
<tr>
<td>Diameter of the infrarenal abdominal aorta (mm)</td>
<td>−0.259</td>
<td>0.082</td>
<td>0.319</td>
<td>0.086</td>
<td>0.074</td>
<td>0.626</td>
</tr>
<tr>
<td>Volume of the thoracic aorta (cm³)</td>
<td>−0.111</td>
<td>0.462</td>
<td>0.354</td>
<td>0.055</td>
<td>0.281</td>
<td>0.059</td>
</tr>
<tr>
<td>Volume of the abdominal aorta (cm³)</td>
<td>−0.319</td>
<td>0.033</td>
<td>0.523</td>
<td>0.004</td>
<td>0.070</td>
<td>0.646</td>
</tr>
</tbody>
</table>
FDG uptake and late aortic diameter in GCA

Age was not a risk factor for aortic dilatation in our study population, but then most of our patients were elderly people (as one can expect in GCA patients) with rather narrow s.d. for age (6.4 yrs), while the age-related increase in aortic diameters is only 1 mm per decade [11]. In the general population, age, gender and body surface area are major determinants of thoracic aortic dimensions [12]. In our study, body length correlated strongly with the volume of the abdominal aorta, depicting the relation between total body length and length of the abdominal aorta.

There was no relation between the classic risk factors for atherosclerosis (hypertension, diabetes mellitus and cholesterol level) and any aortic dimension. This is in line with the results of the second phase of the Stroke Prevention: Assessment of Risk in a Community (SPARC) Study performed in Olmsted County, in which atherosclerosis played only a minor role in thoracic aortic dilatation, in contrast to age, male gender and body surface area or to the dominant role of atherosclerosis in abdominal aortic dilatation [12].

As is also found by other groups studying aortic diameters in GCA patients [13], the sedimentation rate and the CRP level did not correlate with future aortic enlargement.

For aortic dilatation, the intensity of the initial inflammation at the thoracic aorta seems to be important, but not the duration of the initial aortic inflammation, since there was no relation with residual aortic FDG uptake after 3 months of steroid treatment. The diameter of the descending aorta seemed to correlate with residual FDG uptake at 6 months (which was found in 5/12 patients) but the low number of repeat PET scans at that time may render interpretation of data more difficult. The finding that people who had a relapse of GCA, were not more prone to aortic dilatation, and even had a smaller diameter of the ascending aorta, cannot be explained by a protective effect of long-standing steroid therapy, since there was no relation between total steroid dose and aortic dimension.

In multivariate analysis, we used those parameters that showed any significance in univariate analysis. To our surprise, male gender, which was a strong risk factor in univariate analysis, did not prove to be an independent risk factor in multivariate analysis, probably due to its relation with body length. Strong FDG uptake at the thoracic aorta at diagnosis was the only independent risk factor identified for future aortic dilatation at the thoracic level. At the abdominal level, no independent risk factors could be identified.

In conclusion, our results indicate that GCA indeed is a risk factor for thoracic aortic dilatation, especially at the level of the ascending aorta. All GCA patients should be carefully followed up for early detection of this complication, and especially those with strong thoracic aortic FDG uptake. These findings also indicate that FDG-PET scintigraphy not only has a place in the diagnosis of atypical cases of GCA, but also might have a prognostic value for aortic dilatation.

There are, however, some limitations to our study. First, many patients from the ‘earliest years’ (1996–98) could not be included in the study, because they had died, refused to participate or were lost to follow-up. Second, we do not have baseline—or even better, sequential—CT scans from our patients, and therefore we do not know exactly when and how fast the aorta is growing in size. We hope that these limitations can be overcome in a comprehensive prospective study on this topic.

**Rheumatology key messages**

- GCA is a risk factor for thoracic aortic dilatation, especially at the ascending aorta.
- Those GCA patients with strong aortic FDG uptake at PET-scintigraphy may be especially prone to this complication.

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**Discussion**

In recent years, it has become more and more evident that GCA can involve arteries far beyond the temporal ones. Where we once thought that extra-temporal involvement was rather an exception than the rule, especially studies with FDG-PET scintigraphy have learned that the aorta, for instance, is involved in about half of the patients, although at a subclinical level. Some patients, however, become symptomatic, with aortic dilatation and even rupture as a fatal consequence. Until now, one could not predict which patients were especially prone to aortic dilatation. What was known was that the ascending aorta was the main site of aortic rupture in GCA [6], probably due to its high elastin content.

The diameter of the ascending aorta in our population with GCA was as wide in women as in men. In the general population, this diameter is significantly larger in men than in women, and it increases with age in both the sexes, as do all aortic diameters. The diameter of the ascending aorta was reported to be 29.0 ± 3.4 mm in 24 females and 32.0 ± 4.2 mm in 46 males, ranging from 17 to 89 yrs, using helical CT [10]. Aroenberg et al. [11] showed that aortic diameters increase about 1 mm per decade during adulthood, using the same technique. In 373 patients, aged ≥45 yrs and from Olmsted County (MN, USA), the diameter of the ascending aorta measured with transoesophageal echocardiography increased from 29.9 ± 2.8 mm in women aged 50–59 yrs, over 31.6 ± 2.8 mm in women aged 60–69 yrs to 33.2 ± 3.5 mm in women aged 70–79 yrs. In men, the diameter of the ascending aorta increased from 33.3 ± 3.1 mm at 50–59 yrs to over 34.3 ± 3.4 mm at 60–69 yrs and to 36.9 ± 3.6 mm at 70–79 yrs [12]. The diameters of the ascending aorta obtained in our female GCA patients are much larger (38.6 ± 6.0 mm) than those obtained in age-matched women (33.2 ± 3.5 mm), and the same applies for our male patients, although to a lower extent (38.7 ± 3.8 mm compared with 36.9 ± 3.6 mm, in 70- to 79-yr-old men). It seems that GCA causes the ascending aorta to dilate, especially in female patients.

In univariate analysis, there was a significant positive correlation between the size of the ascending aorta and the number of months that elapsed between the diagnosis of GCA and the time of CT scanning. The longer the patients lived after initial diagnosis of GCA, the more they were at risk for developing ascending aorta dilatation, and hence the closer they should be followed up.

We mainly hypothesized that the intensity of FDG uptake at diagnosis of GCA might be a risk factor for later aortic dilatation. The results of the present study are in line with this hypothesis, and thoracic aortic FDG uptake was in fact the only parameter that stood as an independent risk factor in multivariate analysis. In univariate analysis, the correlation between initial FDG uptake and aortic diameters was especially strong for the ascending aorta, the predilection site for aortic rupture in GCA. The vast majority of patients with diameters exceeding 40 mm had thoracic aortic FDG uptake at diagnosis.

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**Table 3. Aortic dimensions at the different levels, according to the presence or absence of aortic FDG uptake**

<table>
<thead>
<tr>
<th>Aortic dimensions (mean ± s.d.)</th>
<th>FDG-uptake negative</th>
<th>FDG-uptake positive</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diameter of the ascending aorta (mm)</td>
<td>37.0 ± 2.8</td>
<td>40.4 ± 6.9</td>
<td>0.025</td>
</tr>
<tr>
<td>Diameter of the aortic arch (mm)</td>
<td>30.1 ± 3.6</td>
<td>31.2 ± 3.6</td>
<td>0.281</td>
</tr>
<tr>
<td>Diameter of the descending aorta (mm)</td>
<td>30.6 ± 4.0</td>
<td>33.5 ± 5.3</td>
<td>0.044</td>
</tr>
<tr>
<td>Volume of the thoracic aorta (cm³)</td>
<td>253 ± 51</td>
<td>301 ± 61</td>
<td>0.029</td>
</tr>
<tr>
<td>Diameter of the suprarenal abdominal aorta (mm)</td>
<td>23.5 ± 3.0</td>
<td>25.9 ± 3.3</td>
<td>0.094</td>
</tr>
<tr>
<td>Diameter of the juxtarenal abdominal aorta (mm)</td>
<td>21.7 ± 2.5</td>
<td>22.8 ± 2.1</td>
<td>0.192</td>
</tr>
<tr>
<td>Diameter of the infrarenal abdominal aorta (mm)</td>
<td>20.0 ± 2.7</td>
<td>21.1 ± 3.6</td>
<td>0.629</td>
</tr>
<tr>
<td>Volume of the abdominal aorta (cm³)</td>
<td>63 ± 19</td>
<td>64 ± 14</td>
<td>0.925</td>
</tr>
</tbody>
</table>
Acknowledgements

The authors want to thank Mrs Helga Ceunen and Mrs Marina Lejeune, study nurses, for their invaluable help in the organization of the trial and the gathering of data.

Disclosure statement: The authors have declared no conflicts of interest.

References