Cardiac and aortic involvement in patients with polymyalgia rheumatica: a study with echocardiography and FDG-PET/CT

Sirs,

Polymyalgia rheumatica (PMR) is an inflammatory syndrome, which affects people over 50 years of age, characterised by pain and stiffness in the shoulder and pelvic girdles (1). PMR can occur as an isolated condition or concomitantly with giant cell arteritis (GCA) (2).

18F-fluorodeoxyglucose (FDG) uptake in large arteries is present in about 30% of patients with PMR, the most specific site of inflammation being the thoracic aorta (3, 4).

To investigate the relationship between inflammatory involvement of the ascending aorta and its possible dilatation, we examined PMR patients with transthoracic echocardiography and positron emission tomography with FDG (PET) fused with computed tomography (CT).

Transthoracic echocardiography may play an important role as first diagnostic approach because not expensive, non-invasive and widely diffused.

The study was performed on 24 patients with PMR, diagnosed according to the 2012 criteria proposed by the American College of Rheumatology (ACR) and the European League against Rheumatism (EULAR) (5), of whom 4 had also GCA. Patients with PMR were compared with 24 controls matched for age, sex and cardiovascular risk factors (CRFs). Seventeen out of 24 patients were assuming prednisone, at doses between 2.5 and 25 mg/day; nine out of 24 were assuming methotrexate, between 7.5 and 15 mg/week.

Transthoracic echocardiography examination was performed by the same investigator and all measurements were performed in accordance with the recommendations of the American Society of Echocardiography (6). Twenty-two out of 24 patients underwent FDG-PET/CT.

Ten out of the 24 patients had an increased uptake of FDG-PET in the thoracic aorta, indicating large-vessel vasculitis.

Echocardiographic results are summarised in Table I. No significant differences were observed between patients and controls in terms of aortic diameters. Aortic regurgitation was significantly more frequent in patients than in controls (15/24 *vs*. 7/24, p=0.04).

In addition, also the mean regurgitation grade of the aortic valve was significantly higher in patients than in controls (1±0.9 *vs*. 0.3±0.4, p=0.005).

Echocardiographic data of patients with PMR and vasculitis seen at PET/CT did not differ statistically from those of patients with PMR without vasculitis (Table I), although the frequency and severity of aortic regurgitation tended to be higher in patients with aortitis (1.4 in patients with a positive PET *vs*. 0.7 in patient with a negative PET, on a semi-quantitative scale from 0=absence of regurgitation to 3=severe regurgitation). Within the PMR group, no correlation was found between echocardiographic changes and demographic data, PMR variables, and type or duration of treatment.

Table I. Echocardiographic data of the studied population.

	PMR (n=24)	Controls (n=24)	*p*-value
AAO (diameter, mm)	19.5 ± 3.2	19.8 ± 1.7	ns
AsAO (diameter, mm)	33.9 ± 3.4	33.8 ± 2.3	ns
AsAO ectasia (n)	5	1	ns
AOArch (diameter, mm)	24 (19 – 33)	24 (20 – 26)	ns
AOArch ectasia (n)	2	0	ns
LA (mm)	35.4 ± 5.3	39.3 ± 3.7	0.005
Aortic stenosis	0	0	ns
Aortic regurgitation (n)	15	7	0.04
Mean aortic regurgitation grade	1 ± 0.9	0.3 ± 0.4	0.005
Mitral stenosis	0	0	ns
Mitral regurgitation (n)	22	22	ns
Mean mitral regurgitation grade	1 (0 – 3)	1 (0 – 3)	ns
PAPS (mean, mmHg)	29±4.8	27.8±3.1	ns
PAPS >35 mmHg (n)	2	0	ns
LVEDD (mm)	46 (41 – 59)	49 (44 – 56)	0.014
LVESS (mm)	27.7 ± 6	27.8 ± 5.5	ns
IVST (mm)	11 ± 2	12.1 ± 1.5	0.036
PWT (mm)	10 (7 – 15)	10 (8 – 13)	ns
LVEF (%)	59.7 ± 2.8	58.5 ± 3.1	ns

AAO: abdominal aorta; AsAO: ascending aorta; AOArch: Aortic Arch; LA: left atrium; PAPS: pulmonary artery systolic pressure; LVEDD: left ventricular end-diastolic dimension; LVESD: left ventricular end-systolic dimension; IVST: interventricular septum thickness; PWT: posterior wall thickness; LVEF: left ventricular ejection fraction.

To our knowledge, this study is the first to address echocardiographic involvement in patients with PMR, correlating it with the presence of large-vessel vasculitis. In this case-control study, we found no evidence of an increase in aortic diameters in patients with PMR. Our main finding was that patients with PMR showed more frequently aortic valve regurgitation than controls. Furthermore, aortic regurgitation in patients with PMR was more severe with a higher mean regurgitation grade. The causes of aortic regurgitation may be essentially two: a pathological alteration of the valve leaflets or an annuloaortic ectasia. We feel that in our patients aortic regurgitation could be due to an initial inflammatory annular dilatation, because no significant deterioration of the valve leaflets was observed. Although no significant correlation was found between echocardiographic changes and the presence of aortitis seen with PET/CT, the frequency and severity of aortic regurgitation tended to be higher in patients with aortitis.

In conclusion, our results suggest that PMR patients may show early signs of aortic dilatation at the annular level. Since PMR is a common disease in patients over 50 years of age, and because this age group is at high risk of cardiovascular events, the role of echocardiographic screening should be investigated in this population. Echocardiography is presently not recommended by the recent European guidance for the management of cardiovascular risk in inflammatory joint disorders (7), but its role in predicting the occurrence of aneurysms in PMR patients should be tested.

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