Arthritis Care & Research
Vol. 70, No. 9, September 2018, pp 1406–1411
DOI 10.1002/acr.23498
© 2017, American College of Rheumatology

BRIEF REPORT

# Large-Vessel Dilatation in Giant Cell Arteritis: A Different Subset of Disease?

FRANCESCO MURATORE,[1] TANAZ A. KERMANI,[2] CYNTHIA S. CROWSON,[3] MATTHEW J. KOSTER,[3] ERIC L. MATTESON,[3] CARLO SALVARANI,[1] AND KENNETH J. WARRINGTON[3]

***Objective.*** **To compare patients with large-vessel giant cell arteritis (LV-GCA) characterized by wall thickening, stenosis, and/or occlusion of subclavian arteries to those with subclavian dilatation.**

***Methods.*** **For the purposes of the present retrospective study, 2 different subsets of LV-GCA were identified and compared from an established cohort of patients with radiographic evidence of subclavian artery vasculitis secondary to GCA: LV-GCA with wall thickening, stenosis, and/or occlusion of subclavian arteries (Group 1), and LV-GCA with dilatation of subclavian arteries without wall thickening or stenotic changes (Group 2).**

***Results.*** **The study included 109 patients in Group 1 and 11 in Group 2. Large-vessel involvement secondary to GCA was diagnosed significantly later in patients from Group 2 compared to those from Group 1 (median 15.3 versus 0.0 months; $P = 0.010$). Compared to patients from Group 1, those from Group 2 were more frequently male (17% versus 45%; $P = 0.027$), ever smokers (42% versus 73%; $P = 0.048$), and more frequently had a history of coronary artery disease (11% versus 36%; $P = 0.018$). At LV-GCA diagnosis, 10 of the 11 patients (91%) from Group 2 had aortic dilatation compared to 13 of 109 patients (12%) from Group 1 ($P < 0.001$). During the followup period, the prevalence of aortic aneurysm was significantly higher in patients from Group 2 compared with those from Group 1 (64% versus 7% at 5 years; $P < 0.001$).**

***Conclusion.*** **Two different subsets of LV-GCA were identified. Given the strong association between subclavian artery dilatation and aortic aneurysm, such patients should be evaluated and monitored carefully for aortic dilatation.**

## Introduction

Giant cell arteritis (GCA) is the most common systemic vasculitis in western countries in individuals ages <50 years. It mainly involves large and medium-sized arteries, resulting in a wide spectrum of clinical symptoms (1). The increased availability of imaging techniques is making a profound impact in the evaluation and management of patients with GCA. Systematic screening of patients with radiographic imaging has yielded a variable prevalence of large-vessel involvement, depending on the technique employed. In prospective imaging studies of patients with a new diagnosis of GCA, large-vessel disease (LV-GCA) was seen in 29–83% of patients, and the subclavian arteries were frequently involved (1). Involvement of the aortic arch branches is generally characterized by circumferential wall thickening, long segments of smooth arterial stenosis, and occlusion (2).

Dilatation of the subclavian/axillary vessels as a manifestation of GCA has been only rarely reported in the literature (2,3). The aim of this study was to compare patients with subclavian artery stenosis, wall thickening, or occlusions to the rare subset of patients who had dilatation of the subclavian arteries.

## Patients and methods

This retrospective study included patients from an established cohort with radiographic evidence of subclavian artery vasculitis attributed to LV-GCA, diagnosed at Mayo Clinic between January 1, 1999 and December 31, 2008 (1). Methods have been described in detail elsewhere (1). Briefly, patients with LV-GCA were identified using an electronic clinical notes search tool (Enterprise Data Trust portal). Due to the poor performance in patients with extracranial disease, we did not require that patients fulfill the American College of Rheumatology 1990 criteria for classification of GCA (4), with the exception that they

[1]Francesco Muratore, MD, Carlo Salvarani, MD: Azienda USL-IRCCS di Reggio Emilia, Reggio Emilia and University of Modena and Reggio Emilia, Modena, Italy; [2]Tanaz A. Kermani, MD, MS: David Geffen School of Medicine, University of California at Los Angeles; [3]Cynthia S. Crowson, MS, Matthew J. Koster, MD, Eric L. Matteson, MD, MPH, Kenneth J. Warrington, MD: Mayo Clinic, Rochester, Minnesota.

Address correspondence to Francesco Muratore, MD, Department of Rheumatology, Arcispedale Santa Maria Nuova IRCCS, Viale Risorgimento, 80, 42123 Reggio Emilia, Italy. E-mail: francesco.muratore@ausl.re.it.

Submitted for publication August 27, 2017; accepted in revised form December 12, 2017.

## Significance & Innovations

- Two different subsets of large-vessel giant cell arteritis were identified: the first, more common, was characterized by wall thickening, stenosis, and/or occlusion of subclavian arteries, and the second, less common, was characterized by dilatation of subclavian arteries.
- Patients with subclavian artery dilatation have more aortic dilatation and less aortic wall thickening at diagnosis.
- Given the strong association between subclavian artery dilatation and aortic aneurysm, such patients should be evaluated and monitored carefully for aortic dilatation.

were required to be ages >50 years. Findings of subclavian artery involvement attributed to GCA were confirmed by computed tomography angiography (CTA), magnetic resonance angiography (MRA), or conventional angiography.

Two subsets of LV-GCA were identified and compared. LV-GCA with wall thickening, stenosis, and/or occlusion of subclavian arteries comprised Group 1, defined as the presence of circumferential wall thickening/wall edema with or without contrast enhancement and/or the presence of vascular stenosis/occlusion not attributed to atherosclerotic changes on MRA or CTA, or the presence of long segments of smooth arterial stenosis or smooth tapered occlusion without adjacent atherosclerotic plaques on angiography (2). Group 2 contained LV-GCA with dilatation of subclavian arteries without wall thickening or stenotic changes. Abnormal dilatation of the subclavian arteries was considered when aneurysm formation was detected or when the physiologic progressive reduction of the diameter was not observed (5). Patients with poststenotic dilatation of the subclavian arteries were included in Group 1.

Vascular imaging was performed at Mayo Clinic using a defined clinical protocol and was evaluated by an expert vascular radiologist. Patients with a report by the Mayo Clinic radiologist specifically stating that findings of the subclavian arteries were consistent with atherosclerosis (i.e., short, eccentric, focal stenosis, plaque, or calcification) or fibromuscular dysplasia (i.e., the classic string-of-beads appearance on angiography) were excluded. In the presence of subclavian focal concentric narrowing without arterial wall thickening, edema, or contrast uptake on cross-sectional vascular imaging, a diagnosis of fibromuscular dysplasia could not be excluded. In the absence of signs or symptoms of GCA, elevation of inflammatory markers, evidence of aortic involvement at imaging studies, or histologic confirmation of vasculitis, these patients were excluded. Patients with a diagnosis of Behçet's disease, Takayasu arteritis, sarcoidosis, or other connective tissue disease were excluded as well.

The available medical records of study participants were reviewed from the date of GCA diagnosis to the end of the study followup (December 31, 2010), the last visit at Mayo Clinic, or death. Only patients who were followed at Mayo Clinic for at least 6 months after GCA diagnosis were considered for the outcomes analysis. Relapse was defined as the reappearance of symptoms of GCA and/or polymyalgia rheumatica associated with a rise in erythrocyte sedimentation rate and/or C-reactive protein level. Isolated increases in inflammatory markers in the absence of other causes were considered relapses only if the treating rheumatologist increased the glucocorticoid/immunosuppressive therapy with subsequent improvement. The study was approved by the Institutional Review Board at Mayo Clinic.

**Statistical analysis.** Continuous data were described as mean ± SD or median and 25th/75th percentiles, and categorical variables as percentage. Wilcoxon's rank sum test was used to analyze continuous variables, and chi-square tests were used for categorical variables. Kaplan-Meier methods and log rank tests were used to estimate the rate of development of outcomes during followup, which is especially necessary because the length of followup differs between the 2 study cohorts. The relapse rate was calculated using person-year methods, and differences in relapse rates between the groups were computed assuming the relapse rates follow a Poisson distribution. Analyses were performed using SAS software, version 9.4.

## Results

From the original cohort of 120 patients with LV-GCA, 109 cases (91%) of LV-GCA with wall thickening, stenosis, and/or occlusion (Group 1) and 11 cases (9%) of LV-GCA with dilatation of subclavian arteries (Group 2) were identified. All 120 patients had imaging of the thoracic aorta and its major branches; 71 patients also had imaging of the abdominal aorta and its major branches. Baseline demographic and clinical characteristics for the 2 study groups are reported in Table 1.

Temporal artery biopsy (TAB) was performed in 73 of the 109 patients from Group 1 (67%) and in 6 of the 11 patients from Group 2 (55%) and was positive for GCA in 36 of 73 (49%) and 5 of 6 (83%), respectively ($P = 0.11$). At LV-GCA diagnosis, thoracic aorta involvement was observed in 56 patients (52%) from Group 1 and in 10 patients (91%) from Group 2 ($P = 0.013$). The type of aortic involvement in the 2 subsets of LV-GCA was significantly different; 10 of 11 patients (91%) in Group 2 had aortic dilatation (aneurysm or ectasia) compared to 13 of 109 (12%) in Group 1 ($P < 0.001$). Aortic aneurysm was also statistically different and noted in 8 of 11 patients (73%) in Group 2 compared to 6 of 109 patients (6%) in Group 1 ($P < 0.001$). Conversely, only 2 of 11 patients (18%) from Group 2 had aortic wall thickening compared to 48 of 109 patients (44%) from Group 1 ($P = 0.097$).

A total of 91 patients (84%) from Group 1 and 11 patients (100%) from Group 2 had a followup period longer than 6 months and were included in the outcomes analysis. Treatment and outcome variables for the 2

**Table 1. Baseline demographics, clinical manifestations, and laboratory findings of LV-GCA patients with wall thickening, stenosis, and/or occlusion of subclavian arteries (Group 1) and those with LV-GCA with dilatation of subclavian arteries (Group 2)***

| Variable | Group 1 (n = 109) | Group 2 (n = 11) | *P* |
|---|---|---|---|
| Age at GCA diagnosis, mean ± SD years | 68.0 ± 7.6 | 70.5 ± 6.5 | 0.26 |
| Women | 90/109 (83)† | 6/11 (55)† | 0.027† |
| Time from symptom onset to diagnosis, median (Q1, Q3) months | 3.5 (1.8, 7.2) | 3.2 (2.4, 4.0) | 0.78 |
| Time from GCA to LVI diagnosis, median (Q1, Q3) months | 0.0 (−0.2, 0.8)† | 15.3 (0.0, 64.9)† | 0.010† |
| History of PMR prior to GCA diagnosis | 30/109 (28) | 1/11 (9) | 0.18 |
| Prednisone use prior to GCA diagnosis | 19/109 (17) | 1/11 (9) | 0.48 |
| Hypertension | 50/106 (47) | 6/10 (60) | 0.44 |
| Hyperlipidemia | 42/108 (39) | 5/11 (45) | 0.67 |
| Ever smoker | 45/108 (42)† | 8/11 (73)† | 0.048† |
| Current smoker | 10/108 (9) | 0/11 (0) | 0.29 |
| Diabetes mellitus | 4/109 (4) | 0/11 (0) | 0.52 |
| Coronary artery disease | 12/109 (11)† | 4//11(36)† | 0.018† |
| Symptoms | | | |
| Any cranial symptoms | 46/109 (42) | 3/11 (27) | 0.34 |
| Permanent vision changes | 4/109 (4) | 1/11 (9) | 0.39 |
| Any vision changes | 13/109 (12) | 2/11 (18) | 0.55 |
| Constitutional symptoms | 59/109 (54) | 5/11 (45) | 0.58 |
| PMR symptoms | 24/109 (22) | 1/11 (9) | 0.31 |
| Upper-extremity claudication | 62/109 (57)† | 1/11 (9)† | 0.002† |
| Physical examination findings | | | |
| Temporal artery abnormalities | 12/79 (15) | 1/10 (10) | 0.66 |
| Vascular bruits | 42/102 (41)† | 0/10 (0)† | 0.010† |
| Abnormal radial pulse | 66/103 (64)† | 0/10 (0)† | < 0.001† |
| Upper-extremity blood pressure discrepancy | 60/103 (58)† | 0/10 (0)† | < 0.001† |
| Aortic regurgitation murmur | 4/103 (4)† | 4/10 (40)† | < 0.001† |
| Laboratory | | | |
| ESR, mean ± SD mm/hour | 67.3 ± 35.9 | 61.4 ± 51.2 | 0.57 |
| CRP, mean ± SD mg/liter | 64.4 ± 64.5 | 62.8 ± 66.0 | 0.84 |
| Hemoglobin, mean ± SD grams/dl | 13.0 ± 11.9 | 12.4 ± 2.0 | 0.43 |
| White blood count, mean ± SD $\times 10^3/\mu l$ | 8.5 ± 2.3 | 7.8 ± 2.7 | 0.11 |
| Platelets, mean ± SD $\times 10^3/\mu l$ | 407.5 ± 132.9† | 308.4 ± 156.9† | 0.033† |
| Temporal artery biopsy positive | 36/73 (49) | 5/6 (83) | 0.11 |
| American College of Rheumatology criteria | 42/109 (39) | 5/11 (45) | 0.65 |
| Prednisone started at GCA diagnosis | | | |
| Prednisone dose, mean ± SD mg | 54.3 ± 14.1 | 51.1 ± 17.6 | 0.54 |

* Values are the number of patients who were positive/number of patients for whom data were available (%) unless indicated otherwise. LV-GCA = large-vessel giant cell arteritis; Q1, Q3 = quintile 1, quintile 3; LVI = large-vessel involvement; PMR = polymyalgia rheumatica; ESR = erythrocyte sedimentation rate; CRP = C-reactive protein.
† Statistically significant.

study groups are reported in Table 2. During the followup period, the relapse rate was higher in patients from Group 1 compared to those from Group 2, and a greater proportion of patients in Group 1 were treated with additional immunosuppressive agents. There was no difference in the cumulative glucocorticoid dose at 1 year, nor in the median time to discontinue glucocorticoid therapy between the 2 groups. The prognosis of LV-GCA was excellent in both study groups. No patients developed major upper-extremity ischemic complications or subclavian dissection/rupture during followup.

The prevalence of aortic aneurysm during followup was significantly higher in patients from Group 2 compared with those from Group 1 (Table 2). None of the 109 patients from Group 1 developed subclavian dilatation during the followup period. Ten patients from Group 2 (91%) and 2 patients from Group 1 (2%) underwent surgical aortic aneurysm repair. Active giant cell aortitis was found in 10 of 12 patients (83%; 8 from group 2, and 2 from group 1); severe cystic medial degeneration was found in the remaining 2 patients, in 1 with evidence of laminar necrosis. At the time of aortic surgery, none of the 12 patients were using glucocorticoid therapy. Six of the 12 patients (50%) had no symptoms and/or sign of active GCA and had normal inflammatory markers; 3 of the 12 patients (25%) had isolated elevation of inflammatory markers without symptoms and/or sign of active GCA. The remaining 3 patients (25%) had systemic symptoms (in 1 with associated polymyalgic symptoms) and elevation of inflammatory markers. There were no significant differences in the prevalence of the other outcomes evaluated during followup (angina or myocardial infarction, transient ischemic attack or stroke, and lower limb arterial disease; data not shown).

**Table 2. Comparison of treatment and outcome variables in LV-GCA patients with wall thickening, stenosis, and/or occlusion of subclavian arteries (Group 1) and those with LV-GCA with dilatation of subclavian arteries (Group 2)***

| Outcome | Group 1 (n = 91) | Group 2 (n = 11) | *P*† |
|---|---|---|---|
| Duration of followup, median (IQR) years | 3.3 (2.1–6.4)‡ | 5.5 (4.5–6.6)‡ | 0.031‡ |
| Relapses, no. | 198 | 17 | – |
| Relapse rate per 10 person-years | 5.2 (4.5–6.0)‡ | 2.7 (1.6–4.3)‡ | < 0.001‡ |
| Cumulative corticosteroid dose at 1 year, mean ± SD gm | 11.6 ± 5.5 | 10.0 ± 8.9 | 0.39 |
| Additional immunosuppressive therapy, no.§ | 50 | 3 | – |
| Within 1 year of GCA diagnosis | 40 (28–49)‡ | 20 (0–41)‡ | 0.049‡ |
| Within 2 years | 49 (37–58)‡ | 20 (0–41)‡ | – |
| Within 5 years | 61 (47–72)‡ | 20 (0–41)‡ | – |
| Rate of aortic aneurysm development, no.§ | 5 | 9 | – |
| Within 1 year after GCA diagnosis | 2 (0–5)‡ | 54 (13–76)‡ | < 0.001‡ |
| Within 2 years | 4 (0–8)‡ | 54 (13–76)‡ | – |
| Within 5 years | 7 (1–12)‡ | 64 (20–83)‡ | – |

* Values are the rate (95% confidence interval) unless indicated otherwise. LV-GCA = large-vessel giant cell arteritis; IQR = interquartile range (25th and 75th percentiles).
† Differences between groups were tested using rank sum tests for duration of followup and cumulative corticosteroid dose, Poisson methods for relapse rate, and log rank test for all others. For medications used by only 1 or 0 patients in a group, log rank *P* values were not available.
‡ Statistically significant.
§ Rates determined using Kaplan-Meier estimate.

## Discussion

The present study is a novel analysis aimed at comparing 2 different subsets of LV-GCA patients involving the subclavian arteries according to lesion type: wall thickening, stenosis, and/or occlusion versus dilatation. Patients with subclavian artery dilatation are more frequently male and smokers (ever), more frequently have a history of coronary artery disease, and have fewer clinical features of vascular insufficiency. Aortic dilatation was more frequent at diagnosis, and a higher prevalence of aortic aneurysm was observed in these patients during followup.

Dilatation of the aortic arch branches as a manifestation of GCA has been rarely reported. In the present study, we found subclavian artery dilatation in 11 of 120 patients (9%) with LV-GCA, all with histologic confirmation of GCA (6 with evidence of giant cell aortitis, 3 with TAB positive for GCA, and 2 with both giant cell aortitis and TAB positive for GCA). In a retrospective study of 65 patients with LV-GCA diagnosed by conventional angiography, Stanson (2) reported 3 patients (5%) with dilated arterial segments, almost aneurysmal in size, a frequency slightly lower than that seen in the present study. Similarly, aneurysm of the subclavian arteries was reported by conventional angiography in 1 of 32 GCA patients (3%) enrolled in a longitudinal multicenter observational cohort of patients with established LV-GCA (3). To date, only 1 study has prospectively evaluated the prevalence, characteristics, and topography of large-vessel involvement using CTA in 40 newly diagnosed, biopsy-proven, untreated GCA patients. In this study, dilatation of the aortic branches was not seen, while thickening was found in 23 of 40 patients with newly diagnosed GCA (57.5%) (5). Differences in the study design may explain the discrepant results observed (prospective study of unselected, newly diagnosed, consecutive, biopsy-proven GCA patients versus retrospective studies of cohorts of patients with established radiographic evidence LV-GCA).

In the current study, large-vessel involvement secondary to GCA was diagnosed significantly later in patients from Group 2 compared to those from Group 1, likely due to the absence of signs and/or symptoms of upper-extremity vascular insufficiency in patients with subclavian dilatation. Subclavian dilatation is likely a late complication of vascular inflammation, and delayed diagnosis may have contributed to these findings. In this regard, only 1 study prospectively evaluated the outcome of CTA findings of large-vessel inflammation (wall thickening) in 35 newly diagnosed biopsy-proven GCA patients after a median of 13.5 months of glucocorticoid therapy (6). Wall thickening of the aortic branches was still present in 12 of the 19 patients (63%) who had this finding at diagnosis, while development of aortic branch dilatation was not reported.

Patients with subclavian dilatation had similar exposure to glucocorticoids prior to GCA diagnosis and during the treatment course, compared to those with stenotic lesions, and therefore, differences in treatment are unlikely to explain the study findings. None of the 11 patients from Group 2 had subclavian artery thickening, and none of the 109 patients from Group 1 developed subclavian dilatation during the followup period. These data suggest that 2 distinct subsets of LV-GCA may be present.

One of the most intriguing findings is that aortic involvement differed significantly between the 2 subsets of LV-GCA. Compared to those with subclavian thickening, patients with subclavian artery dilatation had significantly more aortic dilatation and less aortic wall thickening at LV-GCA diagnosis. Furthermore, the prevalence of aortic aneurysm during the followup period was significantly higher in patients with subclavian dilatation.

Patients with GCA have a 6.6–17.3-fold increased risk of developing thoracic aortic aneurysm compared with the general population (7,8). Aortic aneurysm is a delayed complication of GCA (22.2% and 33.3% of patients with GCA after a median disease duration of 5.4 and 10.3 years, respectively) (9). Patients with GCA who develop aortic aneurysm are at high risk of aortic dissection and rupture and have an increased mortality compared with the general population (10,11). The incidence of aortic aneurysm/dissection increases 5 years after GCA diagnosis and continues to increase thereafter (11). Consistent predictors of aortic aneurysm/dissection in GCA are lacking (10,12). Furthermore, there is no association between aneurysm size and rate of growth and risk of dissection/rupture in patients with GCA (13). A long-term screening program for aortic aneurysm by imaging modalities is therefore mandatory in all GCA patients (10).

More recently, prospective studies have evaluated the prevalence of aortic involvement in unselected, newly diagnosed patients with GCA (5,14). Aortic wall thickening was seen in 45–65% and aortic dilatation in 15–23% of GCA patients. Only rarely were aortic wall thickening and aortic dilatation found in the same patients or in the same segment of the aorta (5,14,15).

Taken together, these findings suggest that large-vessel thickening is a common and early manifestation of GCA, while large-vessel dilatation is a less frequent manifestation that could occur both in the early and late course of the disease. Mechanisms underlying the response of the artery to injury are not fully understood, and different pathophysiologic mechanisms may account for the variable response to large-vessel inflammation. Immunologic responses appears to regulate the extent of intimal hyperplasia, resulting in wall thickening and luminal stenosis (16). In some patients, however, vascular injury may lead to arterial dilatation in the absence of intimal thickening. Large-vessel dilatation could reasonably be a consequence of previous or persistent vessel-wall inflammation (active aortitis was found in 83% of our patients who underwent aortic surgery), but a direct link between inflammation and subsequent dilatation has never been established.

In the present study, patients with subclavian artery dilatation were more frequently male, were ever smokers, and more frequently had a history of coronary artery disease. These cardiovascular risk factors have been previously associated with aortic dilatation in GCA (10,12). One hypothesis is that patients with cardiovascular risk factors may have early damage of the elastic fibers and muscular layer, or inefficient vascular repair or remodeling after injury that contribute to progressive arterial dilatation (6). Sex-associated factors may also play a significant role (5).

The major limitation of our study is its retrospective design, which relies on documentation in the medical records. The treatment of patients and the addition of adjunctive immunosuppressive therapy was not standardized, but was at the discretion of the treating physician. Finally, the number of patients with subclavian artery dilation identified was small, and the exact delineation of the differences in these 2 subsets of LV-GCA may require larger multicenter cohorts. However, the present study is to our knowledge the first of its nature and has a number of strengths, including the large cohort size and long duration of followup.

In conclusion, our findings indicate that there may be 2 different subsets of LV-GCA involving the subclavian arteries, one characterized by occlusive disease, and the second characterized by arterial dilatation. Patients with subclavian artery dilatation had more aortic dilatation at diagnosis and a higher prevalence of aortic aneurysm during followup. In view of this observation, such patients should be evaluated and monitored carefully for aortic complications.

## AUTHOR CONTRIBUTIONS

All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version to be submitted for publication. Dr. Muratore had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

**Study conception and design.** Muratore, Kermani, Crowson, Koster, Matteson, Salvarani, Warrington.

**Acquisition of data.** Muratore.

**Analysis and interpretation of data.** Muratore, Kermani, Crowson, Koster, Matteson, Salvarani, Warrington.

## REFERENCES

1. Muratore F, Kermani TA, Crowson CS, Green AB, Salvarani C, Matteson EL, et al. Large-vessel giant cell arteritis: a cohort study. Rheumatology (Oxford) 2015;54:463–70.
2. Stanson AW. Imaging findings in extracranial (giant cell) temporal arteritis. Clin Exp Rheumatol 2000;18 Suppl 20:S43–8.
3. Assie C, Janvresse A, Plissonnier D, Levesque H, Marie I. Long-term follow-up of upper and lower extremity vasculitis related to giant cell arteritis: a series of 36 patients. Medicine (Baltimore) 2011;90:40–51.
4. Hunder GG, Bloch DA, Michel BA, Stevens MB, Arend WP, Calabrese LH, et al. The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. Arthritis Rheum 1990;33:1122–8.
5. Prieto-González S, Arguis P, García-Martínez A, Espígol-Frigolé G, Tavera-Bahillo I, Butjosa M, et al. Large vessel involvement in biopsy-proven giant cell arteritis: prospective study in 40 newly diagnosed patients using CT angiography. Ann Rheum Dis 2012;71:1170–6.
6. Prieto-González S, García-Martínez A, Tavera-Bahillo I, Hernández-Rodríguez J, Gutiérrez-Chacoff J, Alba MA, et al. Effect of glucocorticoid treatment on computed tomography angiography detected large-vessel inflammation in giant-cell arteritis: a prospective, longitudinal study. Medicine (Baltimore) 2015;94:e486.
7. Evans JM, O'Fallon WM, Hunder GG. Increased incidence of aortic aneurysm and dissection in giant cell (temporal) arteritis: a population-based study. Ann Intern Med 1995;122:502–7.
8. Robson JC, Kiran A, Maskell J, Hutchings A, Arden N, Dasgupta B, et al. The relative risk of aortic aneurysm in patients with giant cell arteritis compared with the general population of the UK. Ann Rheum Dis 2015;74:129–35.
9. García-Martínez A, Arguis P, Prieto-González S, Espígol-Frigolé G, Alba MA, Butjosa M, et al. Prospective long term follow-up of a cohort of patients with giant cell arteritis screened for aortic structural damage (aneurysm or dilatation). Ann Rheum Dis 2014;73:1826–32.
10. Bongartz T, Matteson EL. Large-vessel involvement in giant cell arteritis. Curr Opin Rheumatol 2006;18:10–7.
11. Kermani TA, Warrington KJ, Crowson CS, Ytterberg SR, Hunder GG, Gabriel SE, et al. Large-vessel involvement in

giant cell arteritis: a population-based cohort study of the incidence-trends and prognosis. Ann Rheum Dis 2013;72: 1989–94.
12. Mackie SL, Hensor EM, Morgan AW, Pease CT. Should I send my patient with previous giant cell arteritis for imaging of the thoracic aorta? A systematic literature review and meta-analysis. Ann Rheum Dis 2014;73:143–8.
13. Kermani TA, Warrington KJ, Crowson CS, Hunder GG, Ytterberg SR, Gabriel SE, et al. Predictors of dissection in aortic aneurysms from giant cell arteritis. J Clin Rheumatol 2016;22:184–7.
14. Agard C, Barrier JH, Dupas B, Ponge T, Mahr A, Fradet G, et al. Aortic involvement in recent-onset giant cell (temporal) arteritis: a case–control prospective study using helical aortic computed tomodensitometric scan. Arthritis Rheum 2008;59:670–6.
15. Nuenninghoff DM, Warrington KJ, Matteson EL. Concomitant giant cell aortitis, thoracic aortic aneurysm, and aortic arch syndrome: occurrence in a patient and significance. Arthritis Rheum 2003;49:858–61.
16. Weyand CM, Goronzy JJ. Medium- and large-vessel vasculitis. N Engl J Med 2003;349:160–9.