Supplementary Table S1. The vascular imaging modalities used to detect LVI. Number of individuals with involvement of each vessel segment, by imaging modality.

Vessels involved		Imaging modalities						
		CTA	MRA	DSA	US	PET	CT	Conventional radiology
Aorta	Ascendens	8			8	1	5	
	Descendens	2				1	2	2
	Abdominal	2		1	1	1	7	1
Tributaries	Carotid	2	1		5			
	Subclavian	1				1	1	
	Innominate	1						
	Coeliac trunc	1	1				1	
	Superior mesenteric	1	1					
	Renal		1		1			
	Iliac	2	2	2			1	
	Femoral		4	3				
	Lower leg arteries	1	4	1				
Other vessels	Intracranial	1	1					

CTA: computed tomography angiography; MRA: magnetic resonance angiography; DSA: digital subtraction angiography; US: ultrasound, comprising carotid duplex, ultrasonography of abdomen and echocardiography; PET: ^{18}fluoro-2-deoxy-d-glucose positron emission tomography–computed tomography; CT: computed tomography.

Supplementary Table S2. Vascular imaging modalities used in i patients with GCA, by evidence of large vessel involvement.

	LVI (n=52*)	No LVI (n=38*)
CT angiography	11	9
CT other	14	14
MR angiography	9	1
PET-CT	1	0
Echocardiography	8	5
Ultrasonography	10	25
Conventional radiography	3	3

CT: computed tomography; MR magnetic resonance tomography; PET-CT: ^{18}fluoro-2-deoxy-d-glucose positron emission tomography-computed tomography.
*Several patients in both groups had more than one imaging modality.

Supplementary Table S3. Predictors of LVI- subgroup analysis among those with relevant imaging, n=90; unadjusted.

		HR	95 % CI
Age at disease onset (years)	<70	1.30	0.63 - 2.71
	70 - 80	1.17	0.55 - 2.49
	>80	1.00 = ref	
Age (year) (per SD)		0.96	0.73 - 1.28
Female sex		0.82	0.44 - 1.55
PMR at onset		1.55	0.80 - 2.99
Pre-existing PMR		0.91	0.37 - 2.26
Visual symptoms at onset		1.34	0.74 - 2.41
ESR (mm/h) (per SD)		1.05	0.81 - 1.36
CRP (mg/L), (per SD)		0.85	0.61 - 1.19
CRP (per quartile), lowest ≤54 (mg/L),		1.00=ref	
Quartile 2 54.1-94		0.57	0.24 - 1.34
Quartile 3 94.1-141.25		0.66	0.31 - 1.42
Quartile 4 >141.25		0.71	0.30 - 1.67
Platelets ($\times 10^9$/L) per SD		1.04	0.78 - 1.39
Platelets (per quartile), lowest ≤319 ($\times 10^9$/L),		1.00=ref	
Quartile 2 319.1- 406		1.08	0.40 - 2.92
Quartile 3 406.1- 496		1.52	0.58 - 4.00
Quartile 4 >496		1.21	0.46 - 3.20
Giant cells		**0.49**	**0.27 - 0.87**
Granuloma		1.07	0.52 - 2.21
Disrupted IEL		0.64	0.35 - 1.15
Fibrosis		0.94	0.47 - 1.89
Luminal stenosis		1.78	0.83 - 3.80

LVI: large vessel involvement; SD: standard deviation; PMR: polymyalgia rheumatica; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; IEL: internal elastic lamina.

Supplementary Table S4. Summary of previous studies of the occurrence of giant cells in temporal artery biopsies of patients with giant cell arteritis, and their clinical correlation.

Author, Number of patients	Year	Features evaluated	Features associated with giant cells
Bevan *et al*. (1)	1968	Cranial, visual and constitutional symptoms and inflammatory parameters.	None
Huston *et al*. (2)	1978	Comparing the number of clinical signs and symptoms/ patient, erythrocyte sedimentation rate, duration of treatment with >7.5mg prednisolone/d (months), total duration of corticosteroid treatment (months)	None
Mambo *et al*. (3)	1979	Cranial, visual and constitutional symptoms,	None
Schmidt *et al*. (4)	1994	Jaw claudication, anterior ischemic optic neuropathy, central retinal artery occlusion.	None
Kaiser *et al*. (5)	1998	Cranial, visual and constitutional symptoms, PMR, inflammatory parameters, stroke/ transient ischemic attack.	Significant difference in the occurrence of giant cells in the group with intimal hyperplasia compared to those with normal-minimal intimal hyperplasia.
ter Borg EJ *et al*. (6)	2007	Age, gender, cranial and visual symptoms, PMR, inflammatory parameters.	None
Armstrong *et al*. (7)	2008	Age, gender, cranial, visual and constitutional symptoms, PMR, inflammatory parameters, muscle/joint, relapse frequency.	Trend towards increased occurrence of blindness and PMR compared to those without giant cells.
Chatelain *et al*. (8)	2009	Permanent visual loss	Quantity of giant cells was associated with permanent visual loss.
Breuer *et al*. (9)	2013	Neuro-ophthalmic ischemic manifestations	None
Hernándes-Rodrígues *et al*. (10)	2016	Cranial and constitutional symptoms, irreversible and reversible ischemic complications, PMR, carotidynia, inflammatory parameters, muscle/joint, relapses.	None
Ting *et al*. (11)	2016	Cranial, visual and constitutional symptoms, PMR, inflammatory parameters, relapses.	The presence of giant cells tended to be more frequent in patients with jaw claudication, permanent visual loss and high inflammatory markers.
Muratore *et al*. (12)	2016	Cranial, visual, constitutional symptoms, PMR, inflammatory parameters, stroke/ transient ischemic attack.	The presence of giant cells was a predictor of cranial ischemic events (CIE) (excluded visual loss) and inversely correlated to marked systemic response *i.e*. fever>38C°.

Cranial symptoms: scalp tenderness, jaw claudication, headache, trismus, earache, tender and/or abnormal temporal artery biopsy; visual: one or more of anterior ischaemic optic neuropathy, central retinal artery occlusion, partial or permanent visual loss, diplopia, amaurosis fugax, blurred vision; constitutional symptoms: fever, sweating, loss of appetite, weight loss, malaise, fatigue; inflammatory parameters: one or more of ESR, CRP, WBC, Plt.
PMR: polymyalgia rheumatica.

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