Diagnosing giant cell arteritis

a comprehensive practical guide for the practicing rheumatologist

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Abstract

Giant cell arteritis (GCA) is the most common large vessel vasculitis in the elderly population. In recent years, advanced imaging has changed the way GCA can be diagnosed in many locations. The GCA fast-track clinic (FTC) approach combined with ultrasound (US) examination allows prompt treatment and diagnosis with high certainty. FTCs have been shown to improve prognosis while being cost effective.

However, all diagnostic modalities are highly operator dependent, and in many locations expertise in advanced imaging may not be available.

In this paper, we review the current evidence on GCA diagnostics and propose a simple algorithm for diagnosing GCA for use by rheumatologists not working in specialist centres.

Keywords

Giant cell arteritis, fast track clinic, large vessel vasculitis

Key messages

- Giant cell arteritis is the most common large vessel vasculitis in the elderly population.
- Correct, timely diagnosis and treatment can prevent an adverse outcome and glucocorticoid-related toxicity.
- Ultrasound of cranial and supra-aortic arteries should be an integral part of any fast-track clinic approach.

Introduction

Giant cell arteritis (GCA) is a systemic vasculitis of medium and large-sized arteries that mainly affects adults older than 50 years, has a female predominance, and is relatively more common in the northern hemisphere (1, 2). Emerging evidence based on extended use of imaging modalities shows that GCA can be divided into three groups with potentially different prognoses: isolated cranial arteritis (c-GCA), isolated large vessel vasculitis (LV-GCA), and coexisting cranial and large vessel vasculitis (mixed-GCA) (3). GCA is considered a medical emergency, with complications such as blindness and stroke (4). Prompt treatment initiation is therefore recommended if GCA is suspected (5, 6). Treatment is usually based on
long-term administration of glucocorticoids (GCs), however with serious side effects even for maintenance doses lower than 5 mg Prednisolone (7-9). Targeted biologic treatments like interleukin-6 (IL-6) inhibitors may not replace GCs as a first-line treatment but can be used as GC-sparing agents and can help to sustain remission (10). However, biologics are associated with high costs and are not free from side effects (10).

European League Against Rheumatism (EULAR) recommendations and British Society of Rheumatology (BSR) guidelines outline how GCA can be diagnosed (6, 11). The use of fast-track clinics (FTCs) is emphasised by both as prompt treatment and diagnosis within 48 h has been shown to reduce vision loss and use of healthcare resources (6, 11-13).

EULAR recommends vascular ultrasound (US) and magnetic resonance imaging (MR) for diagnosing c-GCA (11). Temporal artery biopsy (TAB) is advocated if imaging modalities are not available, local imaging expertise is not ensured, or the outcome of the chosen imaging modality is uncertain despite high pretest probability. Suggested for the diagnosis of LV-GCA are US, MR, computed tomography (CT) including CT angiography (CT/CTA), and metabolic imaging with 18F-fluorodeoxyglucose positron emission tomography combined with CT (PET), while conventional US is of limited use in thoracic aortitis (11, 14).

The BSR guidelines recommended either US or TAB as primary diagnostic modalities in c-GCA. MR is advised if US and TAB are not available. The recommendations on LV-GCA do not differ from those of the EULAR (6).

EULAR and BSR do not recommend to use CT/CTA or PET as first line modality in patients with suspected c-GCA (6, 11).

Both societies based their statements on sensitivity and specificity values reported in systematic literature reviews (SLRs). However, the absence of a shared diagnostic standard in all published studies make them difficult to compare.

This paper summarises the evidence on the diagnostic process of GCA that led to BSR guidelines and EULAR recommendations as well as relevant research that emerged afterwards. For this purpose we performed a Pubmed/MEDLINE research with the search term “giant cell arteritis” between March 2017 and February 2021. Relevant publications were identified by title and abstract, results reviewed in full text.

Additionally, taking into account availability, feasibility of reproducible satisfactory standards and costs of the diverse diagnostic modalities, we propose a simple diagnostic algorithm for use by rheumatologists practicing outside a specialized centre.
Classification criteria

For GCA, neither validated diagnostic criteria nor specific outcome measures to assess disease activity and progression exist. The American College of Rheumatology (ACR) 1990 classification criteria are aimed at homogenising study populations and not at diagnosing GCA where they perform poorly (15, 16). Since then revised criteria sets have been proposed to better reflect diagnostic practice but currently await endorsement (Supplementary Table S1, available at *Rheumatology* online) (3, 17).

Signs and symptoms

Patients with typical symptoms and signs may not be difficult to diagnose for the experienced rheumatologist (18). However, a considerable number of patients present with atypical features, not satisfying the high degree of certainty needed to treat a patient with GCs for many years (19).

A recent meta-analysis by van der Geest et al showed that typical symptoms like headache, scalp tenderness, and constitutional complaints are so common in the general population that they hardly help to distinguish between individuals with and without GCA (20). Only limb and jaw claudication, double vision, and previous polymyalgia increased the likelihood of GCA in this SLR. Typical signs like temporal artery abnormalities on palpitation/auscultation and to a lesser degree anterior ischemic optic neuropathy seemed to be more predictive (20). Available publications report substantial differences in the frequencies of symptoms, as shown in Table 1. Anorexia, malaise and stroke might indicate large vessel involvement but no symptom is exclusive for any particular subtype (21). As public awareness of GCA is low, patients may not be aware of the relevance of their symptoms.

Laboratory tests

An elevated erythrocyte sedimentation rate (ESR) may be less sensitive than C-reactive protein (CRP), but values above 60 mm/h and especially above 100 mm/h seem to increase the likelihood of GCA, while no such relationship could be demonstrated for elevated CRP above normal values or above 2.5 mg/dl (20, 22). CRP and ESR together may have a higher positive predictive value than either alone (19, 22, 23). An intense inflammatory response
measured by ESR, CRP, or IL-6 correlates inversely with the risk of ischemic complications but bears an increased risk for relapse (24, 25). Although rare, patients with proven GCA and a normal ESR and CRP before initiation of GC treatment are reported in the literature and may be as frequent as 1–4% (22, 26).

The full blood count may show anaemia and thrombocytosis in about half of GCA patients at initial presentation. While anaemia is so common in the general population that it hardly helps diagnosis, thrombocytosis may be more specific than CRP or anaemia (20). Consequently, thrombocytosis above 400 x10\(^3\)/µl increases the likelihood of GCA when present (19, 20, 27). However, because of a low sensitivity, normal values cannot predict negative GCA status.

Other inflammatory markers and genetics have been investigated for research purposes but currently play no role in clinical practice.

**Pretest probability scores**

Pretest probability scores are meant to identify patients that would benefit from further testing. In the TABUL study, patients with jaw or tongue claudication and relevant CRP or ESR levels were considered high-risk (28). The low-risk group was defined as absence of elevated ESR and CRP as well as jaw and tongue claudication. The remaining patients were categorised as medium risk.

The Giant Cell Arteritis Probability Score (GCAPS) proposed by Laskou et al was developed to help general practitioners select appropriate patients for referral to further diagnostic evaluation (29). GCAPS was shown to be useful as part of a diagnostic algorithm in an US-based FTC setting, categorising patients as having low, intermediate, and high pretest probability (Supplementary Table S2, available at *Rheumatology* online) (30).

A neural network containing clinical and laboratory features of c-GCA to predict TAB outcome was recently developed by Ing et al (31). Whether it could be of help in diagnosing c-GCA in an FTC setting is still to be shown (31).

**Temporal artery biopsy**

TAB has long been considered the gold standard in clinical practice and has also been widely used to evaluate diagnostic imaging (4, 15, 28, 32, 33). As GCA is a systemic vasculitis with
mixed-GCA in up to 80% and LV-GCA in up to 30% of patients, TAB might not be an optimal test for diagnosing GCA (34-43).

A recent meta-analysis found a pooled sensitivity for TAB of 77.3% for the diagnosis of GCA, comparable to MR and US, but with high between-study heterogeneity and lower sensitivity in newer studies (44). Specificity is 90–95%, but considerable heterogeneity in performance and inter-rater reliability reflects the relevance of GCA-dedicated and experienced surgeons and raters as publications with the highest predictive values for TAB would often present data from highly experienced centres with vigorous TAB protocols (28, 44-46). Consequently, more than 7% of biopsy specimens may be inconclusive or do not contain arterial tissue at all (28, 47). Compared to US, TAB is associated with higher resource use and patient burden (28). Complications after TAB are rare (0.5%) but include hematomas, scalp necrosis, infections, and nerve damage (28, 48-51).

Varying lengths of TAB specimens have been proposed in the literature (27, 32, 46, 51, 52). Muratore et al proposed recently that TABs with post-fixation lengths of at least 5 mm may be sufficient to diagnose GCA (52). The fresh biopsy should be at least 10% longer to allow for shrinking. The same study advised evaluating at least three further sections at deeper levels to not miss inflammatory changes if the initial result is negative (52).

Cases with inflammation in adventitial tissue or vasae vasorum that may not be visible on US have been described (53). Whether these changes are specific for GCA has not been determined (54, 55). US-guided TAB did not demonstrate increased diagnostic accuracy (56). This might be a consequence of the usually symmetrical involvement of the temporal arteries (46).

Maleszewski et al demonstrated that even if sensitivity decreases during GC treatment, up to 44% of GCA patients may still have a positive TAB result 1 year after beginning GS therapy (57). TAB may therefore be an appropriate option if US or MR fails to show distinct pathology in cases with moderate to high pretest probability of having GCA (28, 57, 58).

**Ultrasound**

In c-GCA, the halo sign, described by Schmidt et al and defined by the Outcome Measures in Rheumatology initiative (OMERACT), is the best-studied diagnostic US feature (Figure 1) (59-70). It represents an increased intima-media thickness (IMT) as a consequence of oedema
and the accumulation of inflammatory cells and myofibroblasts in the vessel wall (71, 72). Sensitivity for the halo sign is 77% tested against TAB and clinical diagnosis in the SLR informing EULAR recommendations (14). Although the halo sign is not pathognomonic, specificity may reach 96% (14, 61, 73-75). More recent studies from expert centres consistently reported an even higher diagnostic accuracy, as well as high inter-and intra-operator reliability (30, 60, 64, 67, 69, 70, 76). Yet, comparable results may not be reproducible for rheumatologists outside of specialized centres.

The heterogeneity between studies with regard to sensitivity highlights the importance of training, experience, and the appropriate time and equipment for US (14, 77). Chrysidis et al recently proposed and successfully tested a possible curriculum for an US training programme, however all participants had extensive experience on musculoskeletal US (78). Less extensive training interventions have also been reported on, albeit with ambiguous results (28, 79).

Halo scores, which weigh the severity and dissemination of US findings with or without clinical features, have been developed and may help to diagnose GCA or even predict outcome (80, 81). However, no scoring system can currently replace an experienced sonographer's expertise, as the overall performance of these scores does not exceed fair to moderate discrimination (80, 81).

The halo sign's reproducibility is good enough for structured reporting and machine learning algorithms, as a recent study demonstrated (82). Grayscale and Doppler settings depend on the vendor, the type of US machine, and the transducer frequency used and are the subject of ongoing development (83-85).

With modern equipment, it is often possible to measure IMT directly (Figure 1), though it is not included in the EULAR, BSR, or OMERACT recommendations (11, 60, 71). IMT cut-off values for a multitude of arteries have been published as shown in Table 2 (42, 65, 71, 86-89). IMT thickening is, however, not specific for GCA, because it correlates with atherosclerosis and is occasionally seen with other types of vasculitis (90).

The compression sign demonstrates the incompressibility of the inflamed vessel wall (70). It can help to verify the halo-sign and distinguish it from situations where too low colour gain settings mimic vessel wall thickening (pseudo-halo). The compression sign is included in OMERACT’s definitions but not in EULAR or BSR documents (60). It is easy and quick to incorporate into the US examination, has a steep learning curve, high diagnostic accuracy, and
excellent inter- and intra-observer reliability (60, 70, 91). The finding of stenosis and occlusion do not improve sensitivity or specificity but may be clinically relevant, especially in follow-up settings (60).

EULAR and BSR recommend examining temporal and axillary arteries in suspected GCA (6, 11). However, the examination of additional cranial and extracranial arteries can improve the sensitivity of US and also show prognostic value: Facial artery involvement may predict jaw claudication and vision loss, while arteritis of the vertebral artery is associated with posterior stroke (38-42, 92-96).

Concentric vessel wall thickening is a characteristic feature of LV-GCA and can be demonstrated by direct IMT measurement (38, 41, 89). The slope or slide sign may also be critical for LV-GCA diagnosis (Figure 1) (97, 98). The axillary arteries are frequently affected in LV-GCA and are easy to assess by US (11, 35, 36, 39, 42, 71, 83). Nielsen et al showed an absolute increase in sensitivity of 20% if axillary arteries were included in the US procedure while specificity was 100% for LV-GCA US in the same study that compared US against PET in the setting of clinically diagnosed GCA (36). Large vessel US delivers highly reproducible results and may detect wall changes in supra-aortic vessels in larger numbers of GCA patients than 1.5 T MR (99).

US should be performed as early as possible related to the initiation of GC therapy. GC treatment may decrease the sensitivity by 50% within 4 days, while specificity seems relatively unaffected (28, 59, 64, 99-104). The time to regression of the halo-sign under GC treatment is variable and potentially longer in large vessels and long standing GCA (39, 59, 62, 64, 65, 99, 103, 105).

US’s role in follow-up is under investigation. EULAR recommends imaging in follow-up on an individual basis (5, 71). However, an increase in the size of the halo and more affected areas during treatment may indicate relapse (71, 104).

Machine learning and very high-resolution vascular US with frequencies of 40–55 MHz that have the potential to visualize changes in active disease may further increase the value of US in the future (82, 106).

**Magnetic resonance imaging (MR) and magnetic resonance angiography (MRA)**
The meta-analysis leading to EULAR recommendations showed a sensitivity of 73% and a specificity of 88% for MR in detecting c-GCA compared to clinical diagnosis (14). However, in all MR studies included in this review, patients were already treated with GCs. As sensitivity may decrease to 56% after more than 4 days under such treatment, it may be even higher under optimal circumstances, while specificity seems unaffected by GC treatment (14, 99, 101, 107, 108). The inter-observer agreement is highly dependent on expertise but seems to be at least moderate (99, 109).

Technical requirements are high to reliably delineate GCA lesions (11, 94, 110, 111). However, a recently published smaller study showed that even 1.5 Tesla (T) MR had a lower sensitivity than US for depicting vessel wall pathology in supra-aortic LV-GCA (99).

MR is a useful tool for delineating GCA lesions in the whole body (107). It enables visualisation of the entire extent of the disease without radiation, even though such extended procedures would be time-consuming. Intra- and extracranial arteries can be assessed within one examination that combines MRA with optimised pre- and post-gadolinium-acquired sequences for each area (112, 113).

MRA is an essential component of a MR examination undertaken to confirm a diagnosis of GCA (114). Regarding the assessment of large vessels, contrast-enhanced MRA, enables detection of vessel stenosis or occlusion (113). Exemplary MR findings in GCA are shown in Figure 2, typical MR techniques are listed in Supplementary Table S3, available at Rheumatology online.

MR allows for a high standardisation of data acquisition and thereby for structured reporting and machine learning, but requires uniform protocols to do so.

To which extent MR may be useful for monitoring and evaluating the response to ongoing treatment remains unclear (115).

18F-fludeoxyglucose positron emission tomography with low dose CT (PET)

Metabolic imaging is usually combined with low-dose CT to more precisely correlate positive findings to anatomical structures (6, 11). According to EULAR and BSR documents, PET is primarily used in patients with suspected LV-GCA (6, 11). In the EULAR recommendations, the use of PET is emphasised in cases where malignancy or infection may be relevant differential diagnoses (11). Previously, the proximity of the cranial arteries to the high
metabolic activity of the brain was thought to limit the possibilities of PET in diagnosing c-GCA (116, 117). However, recent studies using modern machines demonstrate that PET is a reasonable technique to assess cranial arteries (118-120). These studies show a sensitivity between 75% and 83% and a specificity between 75% and 100% for c-GCA, albeit measured against different standards (118-120).

Both the EULAR and the Society of Nuclear Medicine and Molecular Imaging together with the European Association of Nuclear Medicine have published recommendations on how PET images should be obtained and interpreted (11, 121). These recommendations mirror commonly used acquisition standards in other indications, while evidence is emerging that a longer time between 18F-FDG injection and imaging (delayed imaging) could benefit diagnostic accuracy (122). Typical findings are shown in Figure 3.

PET’s sensitivity for LV-GCA is reported to be 67%–77% in the SLR informing EULAR guidelines, but only two studies met the systemic requirements, and in both studies, patients were already treated with GCs (11, 14, 123, 124). Recent studies show a combined sensitivity for PET of over 80% (117, 120, 125).

Specificity for PET is as high as 88% compared with clinical diagnosis, yet vascular remodelling and atherosclerosis can mimic vasculitis (11, 109, 126). A pattern recognition approach may help, since GCA differs from other types of vasculitis and atherosclerosis with regard to location, and in the combination with CT, plaque calcification can be differentiated as well (127, 128). 18-F-FDG blood activity variations may mask artery wall uptake.

However, this challenge can be overcome by new PET scanning systems (dynamic PET) in which non-metabolised tracer is not visualised (129). The interrater-reliability appears to be very high (109).

PET sensitivity diminishes rapidly within 3 days during GC treatment and offers only a short window for image acquisition (37, 121, 130). PET regularly shows vasculitic activity during clinical remission (131-133). Although this may reflect residual changes, it has been shown that even in the absence of ESR or CRP elevation, vascular uptake on PET in GCA patients correlates with escalation of treatment (134). The same study demonstrated that a higher level of arterial PET tracer uptake in patients in clinical remission was associated with relapse.

**Computed tomography with or without venous phase contrast (CT) and angiography (CTA)**
Due to lack of evidence for visualizing temporal arteritis in GCA, CT and CTA have no role in a FTC or whenever c-GCA is suspected. In LV-GCA, CTA combined with venous phase contrast acquisitions allows assessment of vascular changes such as stenosis, occlusions and aneurysms, but also arterial wall enhancement similar to that provided by MR. Inflammation-induced mural thickening and perivascular contrast enhancement can be depicted with higher spatial resolution and shorter acquisition time compared with MR. CT and CTA are usually more accessible than MR outside referral institutions and involve considerably lower costs. The SLR informing EULAR recommendations could only identify one relevant study that satisfied the criteria of the SLR, highlighting a sensitivity of 73% and a specificity of 78% for CTA in LV-GCA compared to clinical diagnosis after 6 months (14, 124).

Aortitis in CTA is often defined as a concentric wall thickening >2 mm with or without contrast enhancement of the vessel wall in zones without adjacent atheroma (Supplementary Figure S1, available at Rheumatology online) (135). Of the few dedicated studies assessing CT/CTA diagnostic accuracy in GCA, two revealed aortitis in 46%–65% of newly diagnosed GCA patients (135, 136). Another study showed that enhancement decreased during GC treatment after 1 year, while partial wall thickening remained in two-thirds of patients (137). Haemorrhage, stenosis, infarction, and aneurysmatic lesions can be seen as secondary GCA complications (18, 138, 139). Consequently, because the risk for aortic aneurysm is increased in GCA, CT/CTA may be of interest as a follow-up tool.

Although not recommended by either EULAR or BSR for the diagnosis of GCA, venous phase contrast-enhanced CT is often used to investigate fever of unknown origin or constitutional symptoms in clinical practice, with random arterial wall changes triggering the diagnostic possibility of large vessel vasculitis (18, 138). Mural thickening and especially contrast enhancement in the venous phase may represent active vasculitis, although no universal criteria exist and vigilance for possible vasculitis is needed (135, 137, 138). No data exist on the diagnostic accuracy of venous phase-contrast CT.

**Conclusion**

GCA is a relatively common disease, yet symptoms can be unspecific and complications in the form of permanent vision loss or stroke can be dramatic. However, GCA is still primarily a clinical diagnosis and precise history taking and knowledge about how different forms of GCA might present are paramount.
Additionally, pre-test probability scores or neural networks can provide valuable help to decide whom to refer for further testing. The GCAPS is an example of an easy-to-use pretest score that performs well in an US-based FTC setting. The FTC approach has been shown to improve the prognosis of GCA and can reduce unnecessary GC exposition. Consequently, this approach should be the standard in assessing and treating GCA patients whenever available. In Figure 4, we propose an algorithm for the diagnosis of GCA for use by rheumatologists practicing outside specialized centres.

No diagnostic modality can with certainty diagnose or exclude GCA. All modalities are highly dependent on the individual skills of the performing physician. TAB, though reported to show high specificity, has definite drawbacks regarding invasiveness and lower sensitivity than modern imaging modalities. A longer procession-time compared with imaging exposes patients to GCs without final diagnosis. If the result is negative, GC treatment might consequently complicate subsequent imaging (140). However, TAB may have a role whenever imaging is inconclusive, after prolonged GC treatment at initial assessment, or if imaging expertise is not available.

US fits the role as the first diagnostic modality in the FTC setting best. It is a worthwhile step in every diagnostic algorithm for GCA, especially in the hands of the diagnosing rheumatologist, given sufficient training and showed good results (78, 79). These studies indicate that it is feasible to build up sufficient competency also outside of highly specialized centres even though diagnostic accuracy will certainly be lower than publications from specialist centres showed. MR may have the same role as US in FTCs. It is currently restricted to specialist centres, but sensitivity to GC is reported to be high and specificity is high. PET has the restriction of radiation exposure to patient and can be used to confirm disease activity. However, radiation exposure to patient and cost is the major restriction for widespread use of PET. MR technology and availability are evolving and newer promising applications such as diffusion-weighted sequences have not been widely investigated (141).
If used as single modality without TAB or US, CT/CTA does not have a role in a FTC setting due to its limited ability to visualise temporal arteritis. Together with US and especially with TAB it might increase the diagnostic performance and better depict the extend of the disease. CT/CTA may be of use in the follow-up of patients with LV-GCA to screen for complications such as aortic aneurysms or stenosis. A critical approach to local expertise and possibly the consecutive use of diagnostic modalities might help to achieve a high diagnostic yield outside of highly specialized centres, too.

Technical evolution will presumably change the practice of diagnosing GCA in the future due to the emergence of new imaging technologies, biomarkers and better neural networks for predicting diagnosis or assisting imaging interpretation. However, implementation of most of them will increase costs and will also require building up local expertise. An FTC approach including clinical judgement, basic inflammatory markers and US, optimally based on a standardized training curriculum, may therefore remain the backbone of diagnostic algorithms in the coming years.

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Author Contributions

APD and PMA planned the scope of the article. PMA prepared the manuscript with input from all co-authors.

Conflict of interest statement

The authors have declared no conflicts of interest.

Data availability statement

No new data were generated or analysed in support of this research.

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<table>
<thead>
<tr>
<th>Percentage (%) in GCA patients</th>
<th>Symptom</th>
</tr>
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<tbody>
<tr>
<td>65 – 90%</td>
<td>Headache</td>
</tr>
<tr>
<td>30 – 60%</td>
<td>Abnormal temporal artery on examination</td>
</tr>
<tr>
<td>30 – 60%</td>
<td>Constitutional symptoms</td>
</tr>
<tr>
<td>30 – 60%</td>
<td>Polymyalgia rheumatica</td>
</tr>
<tr>
<td>30 – 50%</td>
<td>Jaw or tongue claudication</td>
</tr>
<tr>
<td>30 – 50%</td>
<td>Scalp tenderness</td>
</tr>
<tr>
<td>30 – 40%</td>
<td>Acute visual defects</td>
</tr>
<tr>
<td>20 – 40%</td>
<td>Arthralgia, myalgia, muscle weakness or (much rarer) arthritis</td>
</tr>
<tr>
<td>5 – 20%</td>
<td>Respiratory symptoms such as dry cough, sore throat, or choking sensation</td>
</tr>
<tr>
<td>3 – 10%</td>
<td>Neurological symptoms like stroke, confusion, affective symptoms, myelopathy, or mononeuritis</td>
</tr>
<tr>
<td>No valid data</td>
<td>Otolaryngologic manifestations such as hearing loss, tinnitus, vertigo, dizziness, or abnormal vestibular testing</td>
</tr>
<tr>
<td>No valid data</td>
<td>Unspecific transient oedema of the neck/face</td>
</tr>
<tr>
<td>No valid data</td>
<td>Organ-specific symptoms such as abdominal or chest pain, localised swelling, or dyspnoea.</td>
</tr>
</tbody>
</table>

Table 1: Frequency and symptoms associated with giant cell arteritis (20, 50, 89).
<table>
<thead>
<tr>
<th>Examined artery</th>
<th>IMT threshold (in mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common temporal</td>
<td>0.42</td>
</tr>
<tr>
<td>Frontal temporal</td>
<td>0.34</td>
</tr>
<tr>
<td>Parietal temporal</td>
<td>0.29</td>
</tr>
<tr>
<td>Facial artery</td>
<td>0.37</td>
</tr>
<tr>
<td>Axillary artery</td>
<td>1.0</td>
</tr>
<tr>
<td>Subclavian artery</td>
<td>1.0</td>
</tr>
<tr>
<td>Occipital artery</td>
<td>0.4</td>
</tr>
<tr>
<td>Vertebral artery</td>
<td>0.7</td>
</tr>
<tr>
<td>Common carotid</td>
<td>1.0</td>
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</table>

Table 2: Proposed threshold intima-media thickness values in US examination (86, 89). IMT: intima-media thickness.
Figure 1: Ultrasound findings in GCA. A and B show the halo sign in the temporal artery as defined by OMERACT as homogeneous, hypoechoic wall thickening, well delineated towards the luminal side, visible both in longitudinal and transverse planes, most commonly concentric in transverse scans (60). C and D show the halo sign in the facial artery. Axillary arteritis is shown in E, demonstrating typical features of large vessel GCA in the form of long stretches of intima-media thickness thickening with low echogenicity, even endothelial surface, and the slope sign. Vertebral artery involvement is shown in F. Atherosclerosis would usually more uneven in distribution and often show a high echogenicity and/or calcification of the intima-media thickness.
Figure 2: Magnetic resonance imaging showing typical changes in a patient with giant cell arteritis. A. Gadolinium contrast-enhanced high-resolution T1-weighted spin-echo sequences with fat suppression can depict temporal artery involvement (arrow). B. Contrast-enhanced magnetic resonance angiography showing luminal stenosis in bilateral subclavian and axillary arteries (arrows). C. Transversal T1-weighted, contrast enhanced sequence with fat suppression showing aortitis of the descending thoracic aorta (arrow).
Figure 3: Typical examples of large vessel FDG uptake in patients with giant cell arteritis. The arterial FDG uptake is increased and the uptake pattern is homogeneous and segmental. In the large vessels, there is a predilection for aorta and/or the supra-aortic branches, and the FDG uptake intensity is higher than the uptake in the liver. This is in contrast to atherosclerotic uptake, which is often less intense, more speckled, and typically is more pronounced in the aorta and the infra-aortic arteries. In the cranial vessels, uptake intensity is higher than in surrounding tissue. A. GCA patient with involvement of aorta, subclavian, and axillary arteries. B. GCA patient with temporal arteritis. C. GCA patient with vertebral and aortic involvement. EULAR recommends a visual grading approach where FDG uptake is compared to liver uptake (11, 121, 142, 143). Semiquantitative methods have a role in research studies (121).
Figure 4: Suggested diagnostic algorithm for diagnosing GCA for the clinical rheumatologist not working in a specialist centre. GC: glucocorticoid; FTC: fast-track clinic.