Diagnosing vasculitis with ultrasound: findings and pitfalls

Wolfgang A. Schmidt ⓘD and Valentin S. Schäfer ⓘD

Abstract: Rheumatologists are increasingly utilizing ultrasound for suspected giant cell arteritis (GCA) or Takayasu arteritis (TAK). This enables direct confirmation of a suspected diagnosis within the examination room without further referrals. Rheumatologists can ask additional questions and explain findings to their patients while performing ultrasound, preferably in fast-track clinics to prevent vision loss. Vascular ultrasound for suspected vasculitis was recently integrated into rheumatology training in Germany. New European Alliance of Associations for Rheumatology recommendations prioritize ultrasound as the first imaging tool for suspected GCA and recommend it as an imaging option for suspected TAK alongside magnetic resonance imaging, positron emission tomography and computed tomography. Ultrasound is integral to the new classification criteria for GCA and TAK. Diagnosis is based on consistent clinical and ultrasound findings. Inconclusive cases require histology or additional imaging tests. Robust evidence establishes high sensitivities and specificities for ultrasound. Reliability is good among experts. Ultrasound reveals a characteristic non-compressible 'halo sign' indicating intima-media thickening (IMT) and, in acute disease, artery wall oedema. Ultrasound can further identify stenoses, occlusions and aneurysms, and IMT can be measured. In suspected GCA, ultrasound should include at least the temporal and axillary arteries bilaterally. Nearly all other arteries are accessible except the descending thoracic aorta. TAK mostly involves the common carotid and subclavian arteries. Ultrasound detects subclinical GCA in over 20% of polymyalgia rheumatica (PMR) patients without GCA symptoms. Patients with silent GCA should be treated as GCA because they experience more relapses and require higher glucocorticoid doses than PMR patients without GCA. Scores based on intima-thickness (IMT) of temporal and axillary arteries aid follow-up of GCA, particularly in trials. The IMT decreases more rapidly in temporal than in axillary arteries. Ascending aorta ultrasound helps monitor patients with extracranial GCA for the development of aneurysms. Experienced sonologists can easily identify pitfalls, which will be addressed in this article.

Plain language summary

Diagnosing vasculitis with ultrasound

Rheumatologists use ultrasound to diagnose two types of blood vessel inflammation: giant cell arteritis (GCA) or Takayasu arteritis (TAK). They can do this right in their office during the examination, without sending patients elsewhere. During the ultrasound, rheumatologists can talk with patients about what they see. This is especially helpful in fast-track clinics to prevent vision loss. In Germany, doctors training to become rheumatologists learn how to use ultrasound to check for problems like these. An organization called 'European Alliance of Associations for Rheumatology (EULAR)' recommends using ultrasound as the main way to look for GCA and, if needed, for TAK. Ultrasound is also an important part of the

Correspondence to:
Wolfgang A. Schmidt
Immanuel Krankenhaus
Berlin, Medical Centre for
Rheumatology Berlin-
Buch, Lindenberger Weg
19, Berlin 13125, Germany
schmidt.wa@t-online.de

Valentin S. Schäfer
Department of
Rheumatology and
Clinical Immunology,
Clinic of Internal Medicine
III, University Hospital
Bonn, Bonn, Nordrhein-
Westfalen, Germany

new classification criteria for GCA and TAK. However, doctors do not rely on ultrasound alone. They also look what patients are feeling and do other medical tests. If ultrasound is not clear enough, doctors might need to do more tests like taking a small piece of tissue (biopsy) or using other kinds of imaging like MRI or CT scans. Ultrasound can show some characteristic signs of blood vessel inflammation, like a 'halo sign,' which tells doctors that the blood vessel walls are thicker than normal. It can also spot other problems like blockages or bulges in the blood vessels. When doctors suspect GCA, they should at least examine the arteries at the forehead and at the armpit. Most of the time, these areas are easy to see with ultrasound, but some areas might be harder to reach. Sometimes, people can have blood vessel inflammation without feeling any typical symptoms. Ultrasound can still find this silent inflammation in more than 20% of people with a condition called polymyalgia rheumatica (PMR). Even though these patients do not have typical symptoms of GCA, it is important to treat them the same way as those with GCA. Otherwise, they may have more flare-ups and need higher doses of glucocorticoids. Doctors may measure the thickness of the artery walls over time in research studies. This helps them to see if treatments are working well. The wall thickness decreases faster in arteries of the head than in larger arteries outside the head. Ultrasound of the aorta close to heart helps to find out if a widening of the aorta develops. This can be dangerous because of rupture.

Introduction

Rheumatologists routinely diagnose and manage vasculitides. The Chapel Hill nomenclature categorizes primary vasculitides based on vessel size. It distinguishes vasculitides affecting large, medium, small and variable vessels.[1] This review focuses on large-vessel vasculitis (LVV), specifically giant cell arteritis (GCA) and Takayasu arteritis (TAK), where ultrasound effectively depicts distinct pathology. Other large-vessel-affecting vasculitides exist. Active vasculitis poses a risk factor for venous thrombosis.[2] Thrombosis is caused directly by vasculitis in Behçet's syndrome, where increased vein wall diameters are noted even in veins without thrombosis.[3]

Musculoskeletal ultrasound has become an important diagnostic tool for rheumatologists, playing a significant role in rheumatology training across many countries.[4] With widespread ultrasound equipment and expertise in rheumatology practice, it is increasingly employed for examining various organs in conditions such as Sjögren's syndrome affecting salivary glands,[5] interstitial lung disease,[6] systemic sclerosis affecting the skin,[7] and connective tissue diseases through echocardiography.[8]

Temporal artery colour Doppler ultrasound in GCA was initially described in 1995,[9–11] and many subsequent studies have solidified its role. Ultrasound is now integral to the 2022 American College of Rheumatology (ACR)/European Alliance of Associations for Rheumatology (EULAR) classification criteria for GCA[12] and TAK.[13]

The 2018 EULAR recommendations on imaging in LVV were the first to recommend imaging as equivalent to histology in diagnosis of GCA.[14] The 2023 update strengthened the role of ultrasound, recommending it as the primary imaging modality to investigate for suspected GCA.[15] Various other European guidelines including those from EULAR on management of LVV,[16] the European Headache Federation,[17] the British Society for Rheumatology,[18] the Swedish Society of Rheumatology,[19] the Norwegian Society of Rheumatology[20] and German societies[21] recommend ultrasound for confirming GCA.

Probably the first country to include ultrasound for suspected vasculitis in the curriculum for rheumatology training was Germany in 2023.[22] This review aims to provide an overview of current literature and practical aspects of ultrasound scanning in clinical practice.

Giant cell arteritis and Takayasu arteritis

Both diseases share similarities, with CD4+ T cells and macrophages forming granulomatous lesions in the walls of large arteries, particularly in the media. However, the composition of the wall-infiltrating immune cell compartment differs between TAK and GCA. The ratio of CD4/CD8 T cells is high in GCA and low in TAK. TAK aortitis exhibits a higher proportion of natural killer cells, and the adventitia is typically more expanded in TAK than in GCA, suggesting distinct inflammatory effector pathways.[23]

Some clinical features also differ between GCA and TAK:

- Age: GCA predominantly affects individuals around 70 years, with almost all patients over 50. TAK has a mean onset age of approximately 25 years, affecting mostly individuals under 40.
- Artery involvement: While extracranial artery involvement may be similar, temporal, facial and occipital arteries are never involved in TAK. Axillary vasculitis is more common in GCA, whereas the abdominal aorta, mesenteric and renal arteries are more frequently affected in TAK. The subclavian and carotid arteries and the aorta are the most affected arteries in TAK.[24,25]
- Prognosis: The disease course is longer, and stenoses of extracranial arteries are more prevalent in TAK.
- Treatment: Glucocorticoids are affective in both diseases, but responses to other treatments vary. For instance, tumor necrose factor (TNF) inhibitors are effective in TAK, but not in GCA.[26,27]

In the author's GCA fast-track clinic in Berlin-Buch, out of 367 GCA patients with newly diagnosed GCA in a 4-year period, 48% had cranial GCA, 21% of them had extracranial GCA and 32% of them had mixed GCA involving both cranial and extracranial arteries.[28] Extracranial vasculitis, mostly of the aorta, subclavian and axillary arteries, correlates with younger age and female sex, with less common symptoms like headache, jaw claudication and vision loss.[29,30] Patients with extracranial GCA tend to require glucocorticoids for a longer duration with a higher cumulative dose.[31]

New ACR/EULAR classification criteria distinguish between vasculitides, but have low diagnostic accuracy for diagnosing GCA or TAK. They have not been developed and should not be used as diagnostic criteria due to low specificity. A score of ≥6 is needed for **classifying GCA**, with temporal ultrasound and histology contributing the highest number of points (5 points). Ultrasound contributes two additional points for axillary involvement (Table 1).[12] Two studies tested these classification criteria in their cohorts of suspected and newly diagnosed GCA patients. In one study, the sensitivity was 94% and the specificity was 72% for the clinical diagnosis of GCA.[32] In the other study, the sensitivity was 98%, and the specificity was 58% for the clinical diagnosis of GCA.[33]

For **TAK classification**, a score of ≥5 is required. Up to 3 points are provided when imaging including ultrasound exhibits vasculitis in up to three arteries. An additional point is given for symmetric vascular involvement; and additional 3 points relate to the involvement of the aorta together with the renal or mesenteric arteries (Table 2).[13] Again, the classification criteria were validated in an external cohort and performed well regarding sensitivity (96%) but poor with respect to specificity (64%).[34]

Diagnosing GCA and TAK relies on **typical clinical findings confirmed by histology or imaging**, with conclusive results. Otherwise, a second diagnostic test is recommended.[15] Randomized controlled trials for GCA adhere to these principles, requiring typical clinical symptoms such as headache or polymyalgia rheumatica (PMR) and confirmation through histology or imaging, including ultrasound, magnetic resonance imaging (MRI), positron emission tomography (PET) or computed tomography (CT).[35–39]

Technical requirements and settings for temporal artery ultrasound

Table 3 lists the technical requirements and settings for temporal artery ultrasound. Facial arteries require similar settings. For the occipital

Table 1. ACR/EULAR classification criteria for giant cell arteritis.[12]

Criteria (≥6 points needed for the classification of GCA)	Points
Consider prior to application	
Diagnosis of medium- or large-vessel vasculitis has been made	
Alternate diagnoses mimicking vasculitis haven been excluded	
Absolute requirement	
Age ≥50 years	
Clinical criteria	
Morning stiffness in shoulders/neck	+2
Sudden visual loss	+3
Jaw or tongue claudication	+2
New temporal headache	+2
Scalp tenderness	+2
Abnormal examination of temporal artery	+2
Diagnostic tests	
Maximum ESR ≥50 mm/h or maximum CRP ≥10 mg/l	+3
Positive TAB or temporal artery ultrasound (halo sign)	+5
Bilateral axillary artery involvement (any imaging)	+2
FDG-PET activity throughout the aorta	+2

ACR, American College of Rheumatology; CRP, C-reactve protein; ESR, erythrocyte sedimentaton rate; EULAR, European Alliance of Associations for Rheumatology; FDG, 18-F fluorodesoxiglucose; GCA, giant cell arteritis; PET, positron emission tomography; TAB, temporal artery biopsy.

arteries, image depth needs to be slightly increased, Doppler frequency should be low and pulse repetition frequency (PRF) should be decreased to 1–2 kHz for achieving sufficient Doppler sensitivity.[15]

Small foot print or hockey stick probes with frequencies exceeding 20 MHz are increasingly prevalent in rheumatology practice, offering excellent resolution, particularly for visualizing temporal arteries. These probes allow measuring intima-media thickening (IMT) even in normal temporal arteries.

Probes with frequencies ranging from 50 to 70 MHz are also available, providing an axial resolution of about 0.04 mm. This high resolution allows for the distinction of thickened intima, media and adventitia in GCA, exhibiting excellent correlation with histological measurements.[40] Occasionally, such pathology can also be displayed using 20–30 MHz footprint probes in temporal and axillary arteries.[28,41] However, very high frequency probes are expensive and cannot be used for musculoskeletal ultrasound because of low penetration.

Ultrasound findings

A normal artery exhibits a distinctive anechoic (black) lumen representing fluid (blood). Ultrasound renders anatomical interfaces as hyperechoic (bright) lines. The interface between the arterial lumen and the intima appears as a thin hyperechoic line. Another hyperechoic line represents the interface between media and adventitia. The region between these lines is hypoechoic (dark) representing the intima media complex (IMC; Figure 1). Ultrasound cannot discern the thin intima in normal arteries. The interface between adventitia and the surrounding tissue is usually invisible. The normal IMC is defined as a 'homogenous, hypoechoic, or anechoic echostructure delineated by two parallel hyperechoic margins ("double line pattern"), which is surrounded by mid-echoic to hyperechoic tissue'.[42]

In LVV, the artery wall is thickened due to infiltration with lymphocytes and macrophages leading to myointimal hyperplasia. In acute disease, artery wall oedema contributes to the hypoechoic appearance on ultrasound. Ultrasound shows hypoechoic, non-compressible material causing a thickening of the IMC. It is uncompressible while the artery lumen can be compressed. Colour Doppler facilitates the detection of smaller arteries like the temporal arteries, with colour coding correlating with blood flow. Transverse scans show a dark ring around the artery lumen, termed the '**halo sign**' (Figures 2 and 3).[9] Longitudinal views display the thickened hypoechoic walls on both sides of the lumen. The halo sign is defined as 'homogenous, hypoechoic wall thickening, well delineated towards the luminal side, visible both in longitudinal and transverse planes, most commonly concentric in transverse scans'.[42] In the temporal arteries, 'the thickened arterial wall remains visible upon compression; the hypoechogenic vasculitic vessel wall thickening contrasts with the mid-echogenic to hyperechogenic surrounding tissue (compression sign)'.[42]

Vasculitic wall thickening may lead to **stenosis**, characterized by increased peak systolic velocity, intra- and poststenotic turbulences, and persistent diastolic flow in colour Doppler ultrasound (Figures 3 and 4). In temporal arteries, the maximum systolic flow velocity determined within the stenosis by pulsed wave-Doppler ultrasound is ≥2 times higher than the flow velocity proximal or distal to the stenosis.[10,42] Describing stenoses aided in identifying more inflamed temporal arteries with low resolution probes.[10] Modern equipment usually reveals a halo sign at the stenosis level in active disease. In extracranial arteries, stenoses help assess the severity of pathology.

Occlusions are defined by 'absence of colour Doppler signals in a visible artery filled with hypoechoic material, even with low PRF and high colour gain'.[42] They occur in 25–30% of patients with GCA, primarily in the temporal, axillary and vertebral arteries.[25] In TAK, occlusions are more common, particularly in subclavian and carotid arteries.[24]

How extensively should ultrasound be performed?

In suspected GCA, at least the common superficial **temporal arteries** with the frontal and parietal branches bilaterally and both **axillary arteries** should be examined.[15] Including the axillary arteries alongside the temporal arteries increases ultrasound sensitivity from 70% to 89%, maintaining a specificity of >90%,[43] a consensus supported by various studies.[29,41,44–48]

We examine the axillary arteries from the axillary side if the patient can abduct the arm. This scan accesses the middle and the distal segments. The axillary artery is also visible from anterior at the cranio-lateral aspects of the subscapularis fossa. As the axillary artery lies deeper when examined anteriorly, a lower ultrasound frequency is necessary leading to lower resolution. However, the anterior scan provides access also to the proximal segments of the axillary artery, but it does not increase sensitivities for GCA or extracranial in GCA in established GCA.[49]

Protocols may additionally include the extracranial **carotid arteries**, **vertebral arteries** and **subclavian arteries**. Although subclavian arteries do not contribute to increased sensitivity,[50] the addition of vertebral arteries to the protocol enhances detection of extracranial vasculitis.[49]

Table 2. ACR/EULAR classification criteria for Takayasu arteritis.[13]

Criteria (≥5 points needed for the classification of TAK)	Points
Consider prior to application	
Diagnosis of medium- or large-vessel vasculitis has been made	
Alternate diagnoses mimicking vasculitis haven't been excluded	
Absolute requirements	
Age ≤60 years	
Evidence of vasculitis on imaging (ultrasound, MRI, CT, PET)	
Clinical criteria	
Female sex	+1
Angina or ischaemic cardiac pain	+2
Arm or leg claudication	+2
Vascular bruit	+2
Reduced pulse in upper extremity	+2
Tenderness, absence or reduction of pulse of carotid artery	+2
Systolic blood pressure difference in arms ≥20 mmHg	+1
Imaging criteria	
One arterial territory affected	+1
Two arterial territories affected	+2
Three or more arterial territories affected	+3
Symmetric involvement of paired arteries	+1
Abdominal aorta with renal or mesenteric arteries involved	+3

ACR, American College of Rheumatology; CT, computed tomography; EULAR, European Alliance of Associations for Rheumatology; MRI, magnetic resonance imaging; PET, positron emission tomography; TAK, Takayasu arteritis.

According to the author's experience, patients with cranial GCA and exclusive vertebral involvement more frequently exhibit the cranial phenotype of GCA.

Common carotid arteries are easily accessible with ultrasound. In TAK, exclusive vasculitis of the carotid arteries is considered specific. However, in suspected GCA patients, arteriosclerosis in carotid arteries is common, potentially leading to confusion with vasculitis, particularly

Table 3. Technical requirements and settings for temporal artery ultrasound.[15]

Feature	Explanation
Ultrasound machine	Modern high-quality equipment
Probe	Linear, ≥15 MHz, preferably ≥18 MHz, preferably footprint probe
Image depth	10–20 mm
Focus	At artery level, usually about 5 mm below skin surface
Gain (brightness)	Artery walls should not appear anechoic
Doppler mode	Colour Doppler preferred over other modalities, Grey scale (B-mode) may be sufficient with the use of high-resolution probes
Colour gain	Avoid over- or underfilling of the artery lumen
Colour box	Angle in longitudinal scans to avoid perpendicularity between sound waves and artery
Doppler frequencies	About half of B-mode frequency depending on equipment
PRF	2–7 kHz depending on equipment

PRF, pulse repetition frequency.

when soft plaques are present. Exclusive GCA involvement of carotid arteries is uncommon, and vasculitis should be detected in additional arteries. Plaques are particularly common at the carotid bifurcation. The proximal internal and external carotid arteries are rarely involved in GCA and TAK, but in the age group of GCA patients, arteriosclerotic stenoses are common.[28] Stenoses in common carotid arteries occur rarely in GCA but more frequently in TAK.

Other arteries will be listed in the 'standard scans' section.

Does limiting the ultrasound examination to temporal and axillary arteries overlook patients with GCA or GCA patients with extracranial involvement? A study with 72 consecutive, newly diagnosed GCA patients found that while PET-CT detected aortitis in 33%, ultrasound of temporal and axillary arteries only missed two patients, one with isolated aortitis, actually not classified as GCA,[1] and one with aortitis and vasculitis of the iliac arteries.[51]

Standard scans

Prior to the ultrasound examination, **structured history and focused clinical examination** are essential. History taking may continue during the ultrasound examination. The sequence of the ultrasound examination depends on clinical findings, particularly the palpation of temporal arteries. The probe may be placed directly on a thickened, rigid temporal artery segment. Alternatively, in a female patient aged 60 and exclusively with constitutional symptoms, the examination may commence with the axillary arteries, as axillary vasculitis is more likely in such cases. For a young patient suspected of TAK, the examination may initiate with the carotid or subclavian arteries.
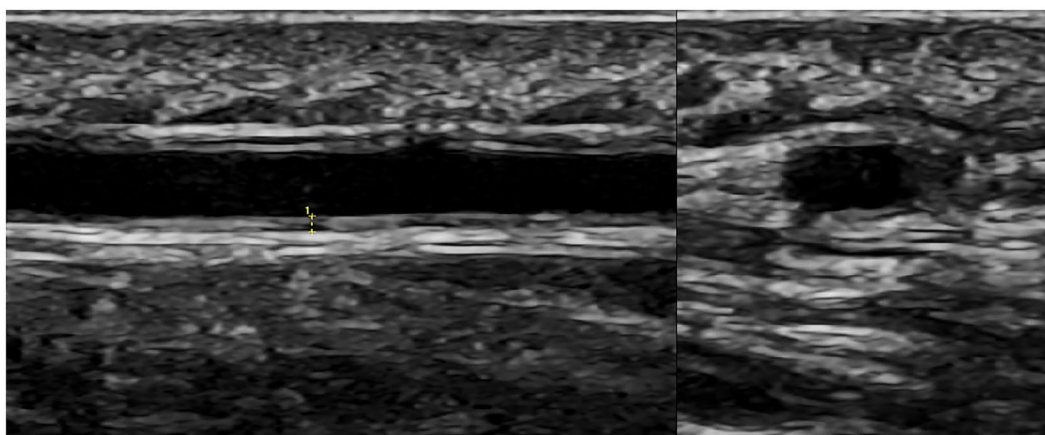

Figure 1. Normal temporal artery frontal branch in longitudinal (left) and transverse planes examined with a 6–24 MHz hockey stick probe. The callipers in the longitudinal view indicate a normal IMT of 0.28 mm. IMT, intima-media thickening.
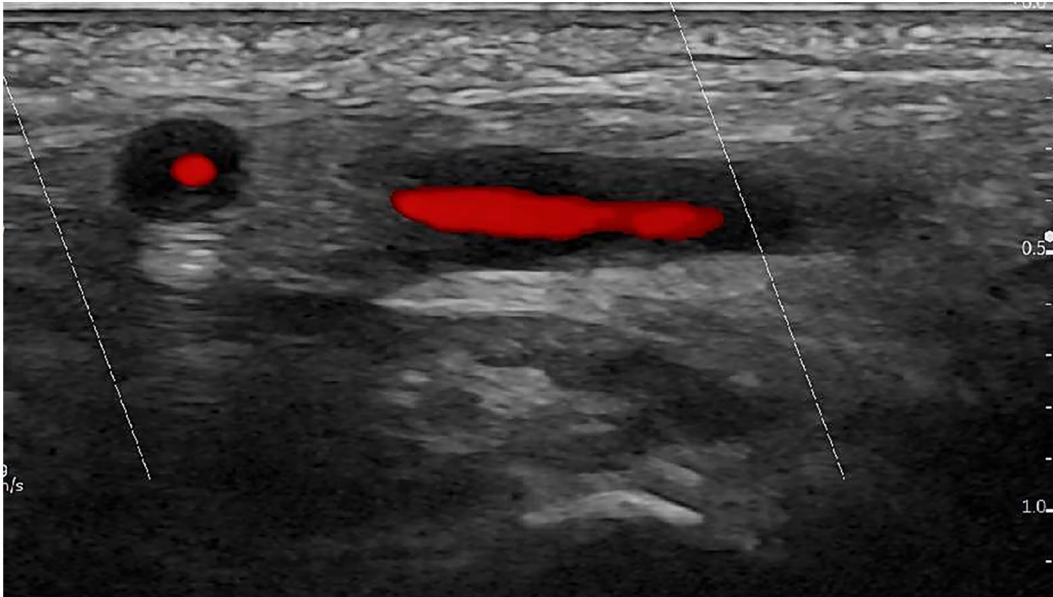
Figure 2. Halo sign in the parietal and frontal branch of a temporal artery near the bifurcation in GCA, using the same probe as in Figure 1.
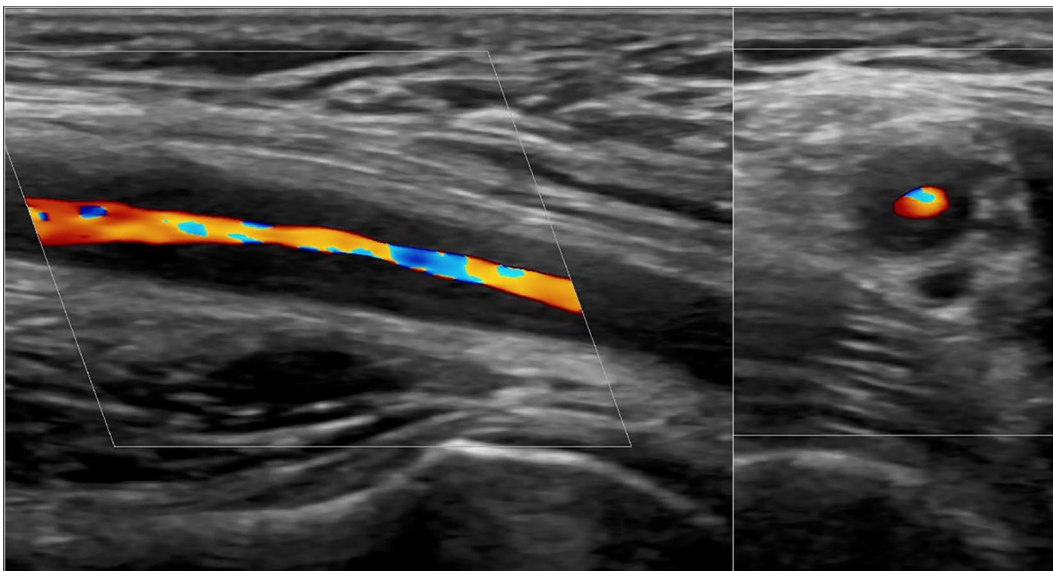GCA, giant cell arteritis.

Figure 3. Halo sign in the axillary artery in extracranial GCA in longitudinal (left) and transverse planes (right) with a 2–9 MHz linear probe. Mixed colour signals suggest turbulent flow (aliasing) with increased velocities due to stenosis. The hyperechoic line at the bottom represents the humeral head and neck, with the right side being proximal in the longitudinal image.
GCA, giant cell arteritis.

For examining the **temporal arteries**, the patient is supine. Placing the probe longitudinally and anteriorly to the cranial half of the ear, the examination begins with the left site to facilitate patient communication with the sonologist, and explain findings on the monitor. If the artery is not immediately visible, the probe is shifted anteriorly and posteriorly. Once the artery is visible,
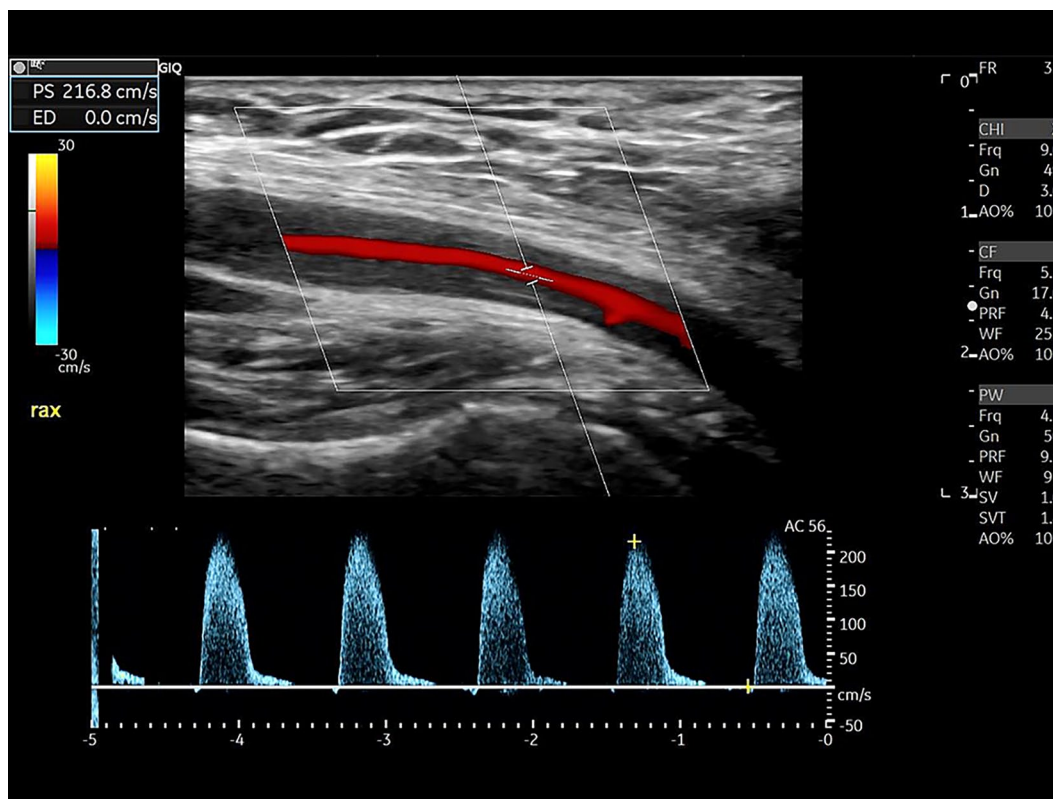
Figure 4. Pulsed-wave Doppler curves in the same artery as in Figure 4, typical for stenosis. Peak systolic flow velocity is elevated (217 cm/s), the spectrum is broader, and the systolic flow velocity increases more slowly while remaining high for a longer duration. The colour signal exhibits no aliasing because the PRF is adjusted for high velocity (9.1 kHz).
PRF, pulse repetition frequency.

the probe is shifted proximally and distally. Subsequently, the probe is rotated for a transverse scan and shifted from the proximal aspects of the artery to its bifurcation. The parietal and frontal branch are then examined in transverse and longitudinal scans with compression and decompression. A normal artery is well compressible. The examination will then be continued on the right side.

The **axillary arteries** localize between the first rib and the distal margin of the teres major muscle. The clavicle is easily visible and close to the junction of the subclavian and axillary artery. The teres major muscle is not visible with ultrasound from the axillary aspect. Its distal margin is located about 5 cm distal to the humeral head, with variability depending on arm position. The anatomy of the axillary artery is also variable.[52] Circumflex humeri arteries are close to the humeral head. Vasculitis may occur up to 5–10 cm distal to the humeral head, but usually not distal to the bifurcation of the profunda brachii artery.

For practical reasons, this segment should be considered as axillary artery, even though a small area may localize distal to the distal margin of the teres major muscle. Figure 5 illustrates a panoramic view of a normal middle and distal axillary artery including the anterior circumflex humeri artery and the profunda brachii artery.

Examination of **axillary arteries** is best performed with the patient supine at the axillary recess of the shoulder. The patient abducts and retroflects the arm, with the hand lying next to the head. The examination begins longitudinally at the axillary recess of the shoulder, showing the humeral head and neck. Mostly, the probe needs to be shifted slightly medially for detecting the axillary artery. Once located at the humeral head level, the probe is shifted 10 cm distally and 5–10 cm proximally. The probe needs to be heel-toed for achieving a perpendicular alignment between probe and artery to enhance visibility of the artery wall. The probe is then shifted transversely from the proximal to the distal aspect of
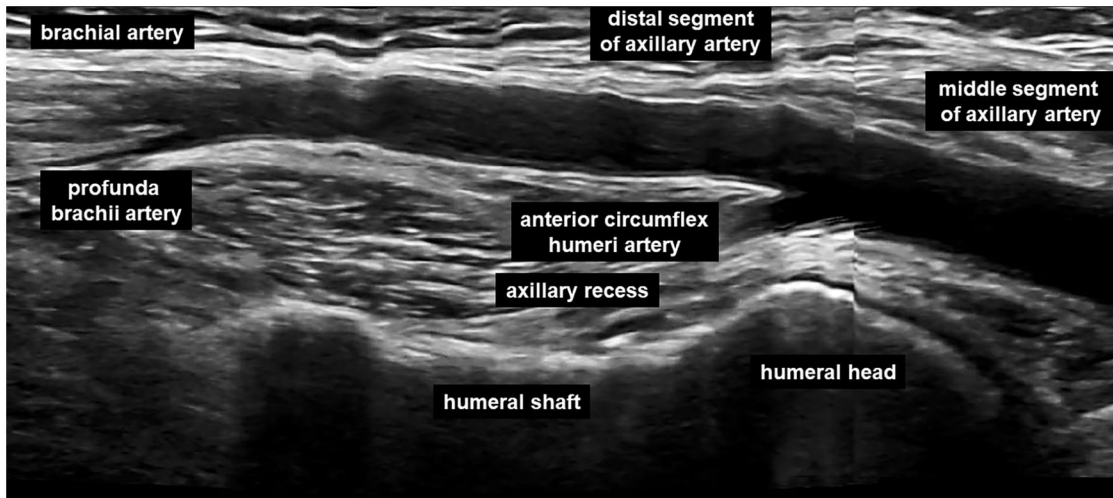
Figure 5. Panoramic ultrasound image displaying normal axillary artery anatomy seen from the axillary recess. The undulating walls reflect pulsations while generating the panoramic image.

the artery. The proximal segment of the axillary artery is visible only with an anterior oblique scan medially to the humeral head.

Examination of other arteries is optional. The patient should be supine except for the popliteal arteries which are examined in the prone position.

The **subclavian arteries** follow a wide arc under the clavicle, visible from above or sometimes below. The probe needs to be tilted for following the course of the artery.

The **common carotid arteries** are easily accessible with transverse and longitudinal scans lateral to the thyroid. The examination should include the common carotid artery and the visible aspects of the proximal internal and external carotid arteries. The proximal aspects of the common carotid arteries become visible with a caudal tilt in a transverse scan.

Vertebral arteries are accessed by shifting or tilting the probe laterally and posteriorly from the longitudinal scan of the common carotid arteries. They are partially hidden by the vertebral transverse processes. Mainly the first two of four segments are well visible. The third segment can be accessed more cranially. Lower Doppler frequency and lower PRF of 1–2 kHz are required due deeper location and slower flow compared to carotid arteries.

The **aortic arch** can be visualized in most individuals with ultrasound, along with the proximal left subclavian, proximal left common carotid and brachiocephalic arteries. A convex probe is placed cranially to the sternum and tilted caudally to the maximum. For low frequency linear probes, a trapezoid view option is recommended.

The first 6–8 cm of the **ascending aorta** are well visible in the longitudinal parasternal view of the heart with sector scanners designed for echocardiography. A convex probe may be used if a sector scanner is unavailable.

The **abdominal aorta** and its branches, such as the celiac, renal and mesenteric arteries, are visible, particularly in slim patients, with abdominal longitudinal and transverse scans.[41]

The pedal pulses should be palpated in newly diagnosed LVV. If absent, the **iliac, femoral and popliteal arteries** should be examined with longitudinal and transverse scans. As arteriosclerosis is common in the elderly, minor changes should be approached with caution. Table 4 outlines indications, clinical correlation and probes for arteries most involved in LVV.

Other arteries are accessible with ultrasound. Abdominal ultrasound can access **renal, celiac, mesenteric and iliac arteries**. Although a halo of the superior mesenteric artery may be visible in slim patients,[41] only stenoses of the celiac and

Table 4. Indications, clinical correlation and probes for arteries involved in LVV.

Artery	Indication/clinical correlation	Probe	Compressible	Reference
Temporal artery	Routine	Linear >18 MHz	+	15
Facial artery	Jaw claudication, visual loss	Linear ≥15 MHz	+	53
Occipital artery	Retroauricular pain	Linear 7–20 MHz	+	53
Maxillary artery	Only case reports	Linear 5–15 MHz	−	54
Lingual artery	Only case reports	Linear >15 MHz	−	55
Axillary artery	Routine, arm claudication	Linear 7–15 MHz	−	15
Subclavian artery	Arm claudication, common in TAK	Linear 7–15 MHz	−	50
Carotid artery	Common in TAK	Linear 7–15 MHz	−	28
Vertebral artery	Stroke	Linear 5–15 MHz	−	56
Brachiocephalic artery	Extracrananial GCA	Linear, convex <10 MHz	−	44
Ascending aorta	Follow-up for aneurysm	Sector, convex <7 MHz	−	28
Aortic arch	Fever of unknown origin	Convex, sector, linear <7 MHz	−	28
Abdominal aorta	Fever of unknown origin	Convex, linear <7 MHz	−	41
Femoral artery	Leg claudication	Linear 5–15 MHz	−	41
Popliteal artery	Leg claudication	Linear 5–15 MHz	−	57

GCA, giant cell arteritis; LVV, large-vessel vasculitis; TAK, Takayasu arteritis.

Table 5. Pooled sensitivities of low-risk of bias studies on ultrasound, MRI and PET-CT for the diagnosis of GCA.[43]

Imaging modality	Number of studies	Pooled sensitivity (%)	Pooled specificity (%)
Ultrasound	8	88	96
MRI	3	81	98
PET-CT	4	76	95

GCA, giant cell arteritis; MRI, magnetic resonance imaging; PET-CT, positron emission tomography-computed tomography.

renal arteries can be detected through Doppler curves due to their non-parallel orientation to the probe.[58]

Advantages and disadvantages of ultrasound and other imaging techniques

Many studies report a high accuracy of ultrasound. A meta-analysis, which informed the 2023 EULAR recommendations on imaging in LVV, demonstrated also high specificity for MRI and PET, with ultrasound exhibiting greater sensitivity (Table 5).[43] The sensitivity of PET may further increase with newer technology concerning access to cranial arteries.

Some studies have directly compared ultrasound with other imaging techniques in GCA. One study observed a very good agreement between ultrasound and PET-CT.[45] Another study

described comparable diagnostic accuracy for GCA. However, PET/CT and ultrasound were often discrepant within single vascular regions.[57] High agreement of ultrasound and MRI was observed in another study.[59]

In TAK, fewer studies have been conducted. A meta-analysis of mostly small retrospective studies, comparing imaging with clinical criteria or conventional angiography, yielded pooled sensitivities of 81%, 81% and 92% for ultrasound, PET and MRI, respectively. Pooled specificities were >90% for ultrasound and MRI and 74% for PET.[60]

Despite ultrasound being recommended as the preferred diagnostic test for GCA, alternative diagnostic techniques can be applied in the absence of expertise or adequate equipment. Table 6 summarizes the advantages and disadvantages of diagnostic techniques.[61]

Ultrasound is often perceived as highly investigator dependent. However, all diagnostic modalities require training, standard operating procedures and adequate equipment.[15] Exercises with ultrasound have demonstrated good reliability in suspected GCA with trained examiners.[42,62] Currently, ultrasound machines can be connected to Picture Archiving and Communication Systems. Findings should be archived in two planes. For temporal arteritis at least one video showing an incompressible halo sign together with a longitudinal still image should be stored.

Transcranial ultrasound can visualize intracerebral arteries, but its resolution is too low for detecting a halo sign. High-resolution MRI could identify at least one vasculitic intracranial artery in 15% of GCA patients, a prevalence higher than previously suspected.[63]

How rapidly should the diagnosis be confirmed?

How rapidly does pathology normalize with glucocorticoid treatment? This depends on the diagnostic method, equipment quality and artery size (Table 7). PET normalizes most rapidly. In a study with repeated PET scans 100% remained positive after 3 days, but only 36% remained positive after 10 days.[64] PET should preferably be performed within the first 3 days of glucocorticoid treatment.

Temporal arteries normalize more rapidly than extracranial arteries. In the first study on ultrasound in GCA, the halo sign persisted for a mean of 16 days.[10] With newer equipment, pathology may be remain visible for a longer duration. Other studies comparing the length of glucocorticoid treatment in cohorts with suspected GCA noted a decrease in sensitivity already after 2 days.[68,69] In this regard, MRI and ultrasound of temporal arteries exhibit similar trends.[68] Consequently, ultrasound or MRI of temporal arteries should be performed within the initial 7 days of glucocorticoid treatment.

Histological pathology of TAB persists for a longer time. In a study with sequential TAB, 75% remained positive after 6 months, and 44% were positive after 9 and 12 months, respectively. Notably, none of the positive specimens after 12 months exhibited giant cells any more.

Axillary artery wall thickening persists even longer, with pathology still visible in 70% of patients after a mean of 50 months.[67] In another cohort, IMT of axillary arteries exceeded the cut-off for chronic vasculitic axillary artery changes of 0.9 mm in half of the patients after 4 years.[70,71]

Nevertheless, diagnosis becomes more challenging with increasing length of glucocorticoid treatment, leading to a decrease of diagnostic certainty. Slight wall changes of vessel wall morphology observed through ultrasound, MRI or CT, a slight increase in glucose metabolism in PET, or the absence of giant cells in histology bear a risk of misinterpretation. Therefore, confirming the suspected diagnosis of LVV, particularly of GCA, is crucial as soon as possible. However, diagnostic procedures should not delay the initiation of treatment.[15]

Pitfalls

Several pitfalls may be encountered during ultrasound examinations:

- Poor quality of colour Doppler signals when sound waves are perpendicular to the artery. Therefore, the colour box should be angled in longitudinal scans, and the probe should be slightly tilted in transverse scans [Figure 6(a)].

Table 6. Advantages and disadvantages of imaging techniques for LVV.

Diagnostic modality	Advantages	Disadvantages
Ultrasound	• Available in rheumatology practice • Mostly performed by rheumatologists themselves • No additional referral • Physician can communicate with patient directly during examination • Findings can be directly explained to the patient • Focused exam depending on patient's complaints. • No radiation • No contrast agent • Easily repeatable • Pathology of extracranial arteries persists for a long time • Inexpensive • Very high resolution • Excellent evidence level	• Thoracic descending aorta inaccessible • Time-consuming for a whole-body scan
MRI	• Good overview of cranial and extracranial arteries • Accesses the entire aorta • Accesses intracranial arteries • Strong evidence for cranial arteries • No radiation	• Few expertise for cranial arteries • Weak evidence for extracranial arteries • Limitation for claustrophobia • Limitation for metal • Limitation for pacemakers • Long acquisition time • Optional contrast agent • More expensive than ultrasound
CT	• Good overview for extracranial arteries • High sensitivity for arteriosclerosis • Fast acquisition time • Widely available	• No data on cranial arteries • Radiation • Optional contrast agent • More expensive than ultrasound
PET, PET-CT	• Good overview for extracranial arteries • New technology for cranial arteries • Sensitive to disease activity • Sensitive for differential diagnoses • High evidence for extracranial arteries • Can detect silent GCA in PMR • Shows typical distribution of inflammation paravertebral in the pelvic and shoulder girdle.	• Limited availability • Long examination time • Radiation (up to 20 mSv) • Normal serum glucose needed • Sensitivity decreases rapidly with treatment • Infusion needed • Very expensive
Conventional angiography	• Good overview • Indicated for interventions	• Not recommended for diagnosis • High complication rate (needle placement, contrast agent) • Does not show artery wall • Temporal arteries not accessible • Radiation • Expensive
Temporal artery biopsy	• Good evidence • By far the highest resolution • Detects minor pathology • Detects other reasons for inflammation • Remains positive for a long time	• Long waiting time for results • Invasive with potential complications • Expensive

CT, computed tomography; GCA, giant cell arteritis; LVV, large-vessel vasculitis; MRI, magnetic resonance imaging; PET, positron emission tomography; PMR, polymyalgia rheumatic.

Table 7. Estimation of time intervals for pathology normalization for PET-CT, ultrasound, MRI and histology.

Imaging modality	Positive findings at follow-up	Reference
PET-CT	36% after 10 days	64
Ultrasound of temporal arteries	31% after 24 weeks	65
Histology	44% after 9-12 months	66
Ultrasound of axillary arteries	70% after 50 months	67
MRI, magnetic resonance imaging; PET-CT, positron emission tomography-computed tomography.		

- Pseudo-halo: Colour Doppler signals appearing only in the middle of the artery lumen may leave an anechoic area around this signal, creating a false impression of vasculitis [Figure 6(b)]. This issue can be resolved by compressing the temporal artery, increasing the Doppler gain, and focusing on the B-mode image showing hypoechoic, but not anechoic wall swelling.
- Colour signals extend over the artery walls because the colour gain is too high, leading to overlooking the halo-sign [Figure 6(c)].
- No colour signal and poor visibility of the arteries below hair. This challenge can be overcome by using more ultrasound gel or focusing on compression sonography in B-mode [Figure 6(d)].
- Arteriosclerosis: Arteriosclerotic vessel wall changes may also cause wall thickening of the temporal and other arteries. Unlike vasculitis, arteriosclerotic plaques are hyperechoic, irregular and asymmetric. Arteriosclerosis is rare in temporal arteries [Figure 6(e)]. However, it can be misinterpreted, particularly in the carotid arteries and at the root of the right subclavian artery [Figure 6(e)].
- Skip lesions: Vasculitis may be limited to a few segments, leading to potential oversight if arteries are not thoroughly scanned [Figure 6(f)].
- Normal findings due to previous glucocorticoid treatment. This is particularly relevant to the temporal, facial and occipital arteries. Ultrasound examination can be extended to extracranial arteries where pathology may persist for a longer duration.
- True halo sign due to a diagnosis other than GCA or TAK. This aspect is further explained in the next paragraph.

Differential diagnosis

An experienced sonologist using modern technology and adhering to standard operating procedures can hardly overlook or misdiagnose vasculitis. However, other conditions causing arterial wall thickening may **mimic the halo sign**. For instance, granulomatosis with polyangiitis, may affect temporal arteries or the aorta.[72] Eosinophilic granulomatosis with polyangiitis of temporal arteries has also been described.[73] Additionally, diseases like amyloidosis, angiolymphoid hyperplasia or lymphoma may induce the same morphological changes in temporal arteries.[74–76] This occurs only in approximately 2–3% of patients.[76] Therefore, all available clinical information is essential for correctly diagnosing LVV.

Aortitis and periaortitis appear differently. Aortitis typically correlates with the characteristic halo sign. In case of arteriosclerosis, hypoechoic material extends around the hyperechoic rim of plaques.[28] Chronic periaortitis due to Ormond's disease or IgG4 syndrome is less distinct, appearing hyperechoic with often poorly defined boundaries.[77] Especially in younger TAK patients, infectious disease such as lues or mycobacterial infection should be considered. Furthermore, TAK may co-occur with axial spondyloarthritis, Cogan syndrome, relapsing polychondritis and other autoimmune diseases.[78]

Ultrasound fast-track clinics

Rheumatologists increasingly offer fast-track clinics for suspected GCA, providing a service within 24 h on working days for patients in whom a physician suspects GCA. The fastest route is to contact the nurse of the fast-track clinic by telephone. Initially, an experienced rheumatologist conducts a brief, structured history and a focused clinical
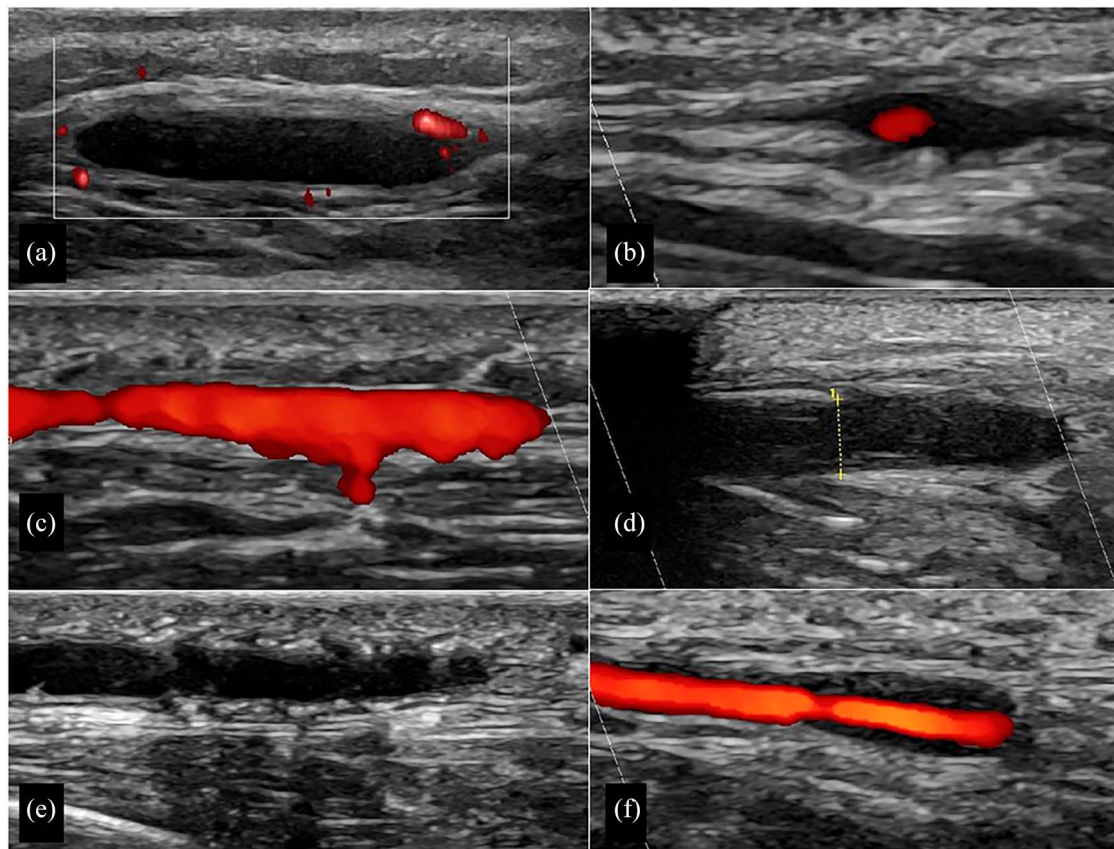
Figure 6. Pitfalls: (a) The colour box is not angled resulting in poor colour Doppler signals. (b) Pseudo-halo because colour gain is too low. (c) Colour gain is too high, while colour signals extend over the artery wall. (d) Hair impairs sound waves on the left side of the image, but vasculitic artery is not compressible on its right side. The callipers indicate a thickness of 2.2 mm for the compressed artery, meaning that each IMT is 1.1 mm. (e) Arteriosclerosis of a temporal artery, displayed as hyperechoic (white) irregular spots in the IMT of both sides. (f) Focal vasculitis on right side with normal temporal artery segment on left side.

examination, followed by an ultrasound examination of at least the temporal and axillary arteries. Ideally, two to three experts should be trained within a centre.

The implementation of fast-track clinics correlated with a substantial **reduction in permanent vision loss**, attributed to the earlier establishment of diagnosis and treatment.[77,79–81] In Berlin-Buch, the first fast-track clinic has examined over 1000 newly diagnosed GCA patients with GCA since the late 1990s.

In Europe, mostly the **same clinician performs both the clinical and the ultrasound examinations**. After the examination, the patient receives a written report clearly stating whether LVV has been confirmed or excluded.[25,82] The authors strongly favour this approach. Conversely,

some institutions, particularly in the United States, have established fast-track clinics where the patients are referred to an ultrasound technologist after the first consultation with the expert rheumatologist. Images and videos are stored and later evaluated by a vascular specialist, who then reports the results to the rheumatologist.[83,84] Although this approach may encounter challenges in terms of organization, speed and cost, differences in political, legal, administrative and economic issues may exist between countries.

A GCA **Probability Score** has been developed, relying on a structured history.[85] While this score may be informative, it should not replace direct communication between the patient and the experienced rheumatologist. Distinct information, such as the quality of headache, is mandatory.

For patients with a high clinical suspicion of GCA and positive ultrasound findings, an immediate diagnosis of GCA can be established. Likewise, low clinical probability combined with negative ultrasound excludes GCA. TAB or further imaging is warranted if findings are inconclusive, a situation encountered in less than 5% in our fast-track clinics.

Vascular ultrasound in PMR

PMR and GCA are closely related conditions characterized by systemic inflammation, a predominant interleukin-6 signature, an excellent response to glucocorticoids, a tendency to a chronic and relapsing course and older age of the affected population.[86] Despite these commonalities, differences exist in pathogenesis. For instance, inflamed arteries in GCA exhibit extensive infiltrates of T helper-1 and -17 cells, whereas T-cell infiltrates in PMR synovium are limited, predominantly consisting of T helper-1 cells.[87] However, a significant clinical connection between PMR and GCA exists. This relationship has implications for disease stratification,[88] leading to introduction of the term 'GCA-PMR Spectrum Disease'.[86] New treat-to-target Recommendations in GCA and PMR emphasize that clinical management of GCA and PMR should be driven by the awareness that they are closely interrelated conditions in a common spectrum of inflammatory diseases and can occur separately, simultaneously, or in temporal sequence to each other.[89]

In a cohort of over 1000 newly diagnosed GCA patients in Berlin-Buch and in other cohorts, approximately 50% exhibited symptoms of PMR characterized by shoulder or pelvic girdle pain and morning stiffness.[86,87] 'Pure PMR' is characterized by PMR symptoms without clinical features of GCA, and PMR may precede, coincide with or follow the clinical manifestations of GCA.[87]

How many PMR patients have subclinical GCA? A meta-analysis of 13 cohorts, utilizing various diagnostic tests such as histology, ultrasound and PET in PMR, found that 22% of PMR patients with and without GCA symptoms were ultimately diagnosed with GCA.[90] In a monocentre study involving ultrasound examinations of temporal, facial, axillary and carotid arteries, 22% of 60 patients with newly diagnosed 'pure' PMR had subclinical GCA.[91] In a multicentre study, examining temporal, axillary, common carotid and subclavian arteries with ultrasound in 346 patients with newly diagnosed PMR, and subclinical GCA was found in 23%. PMR patients with subclinical GCA more frequently exhibited an extracranial large vessel pattern compared to classical GCA.[92] Follow-up assessments of 100 patients with non-vasculitic PMR and 50 patients with subclinical GCA revealed that the latter experienced more relapses (62% *versus* 16%) and required higher doses of prednisone equivalent after 12 months (mean dose, 7.5 *versus* 2.4 mg/day).[93] In conclusion, PMR patients with subclinical GCA necessitate treatment as GCA, highlighting the importance of searching for GCA in PMR for effective disease management.

Should all patients with suspected new PMR be referred to a rheumatologist? An international survey involving 394 general practitioners (GPs) and 937 rheumatologists concluded that GPs only referred 25% of their patients to rheumatologists.[94] A systematic literature review found only sparse evidence on early management and referral strategies. Four studies on shared care and fast-track clinics revealed promising results including reduced hospitalization rates, lower starting glucocorticoid doses and faster PMR diagnosis.[95] Based on these outcomes, an international GCA/PMR study group recommends early referral of individuals with suspected PMR to specialist care. Patients should also be informed about the potential overlap with GCA.[96]

Ultrasound for disease monitoring

Ultrasound can be applied for monitoring disease activity or damage in both GCA and in TAK. Disease activity may correlate with IMT. Damage refers to stenoses, occlusions and particularly to aortic aneurysms.

For monitoring IMT, normal and cut-off IMT values have been established for GCA. However, such values have not yet been determined for TAK. Younger individuals reveal lower IMT. Several studies reveal consistent results for suspected GCA (Table 8).[97–101]

For diagnosing GCA or TAK, the presence or absence of the halo sign, which is uncompressible in cranial arteries, is crucial. IMT scores were not developed for diagnosis, but for disease monitoring, although cut-off values may be sometimes helpful.

Table 8. Normal and cut-off values in the age of patients with suspected GCA.[28]

Artery	Normal (mm)	Cut-off (mm)	Reference	Other references[a]
Common superficial temporal artery	0.23	0.42	97	100–101
Frontal branch	0.19	0.34	97	98–101
Parietal branch	0.20	0.29	97	101
Facial artery	0.24	0.37	97	101
Axillary artery	0.59	1.0	97	100–101
Subclavian artery	0.61	1.0	101	100
Common carotid artery	0.73	1.0	101	100
Vertebral artery	0.42	0.7	101	
Aorta[b]	1.76	2.2	102	

[a]With similar results.
[b]Assessed by CT.102.
CT, computed tomography; GCA, giant cell arteritis.

IMT measurements should be taken at the level of greatest wall thickness. The thickest wall should be measured. Measuring the lower wall is usually easier. The IMC is measured from the top of the thin hyperechoic line representing the interface between lumen and intima to the top of the thicker hyperechoic line, representing the interface between media and adventitia. Alternatively, both walls can be measured with compression, dividing the result by 2.[99] Measurements in grey scale images may be more accurate since colour signals may either cover portions of the wall or fail to completely fill the lumen. It is advisable to conduct measurements in longitudinal planes and include two decimals after the comma. Preferably, the same method should be applied at baseline and follow-up. Biopsied and missing segments should be excluded from analyses.[103] IMT should be measured outside arteriosclerotic plaques or areas of localized eccentric wall thickening suspected of arteriosclerosis. IMT may be slightly greater in patients with arteriosclerosis even when outside arteriosclerotic plaques.[98,104]

Scores for disease monitoring, particularly designed for research studies, were developed. The **Southend Halos Score**, which converts IMT of six temporal artery segments and two axillary artery segments into grades[105] has primarily been used for disease stratification.[88] In another study, the score decreased from 11 significantly to 0 in the temporal arteries, but was stable in the axillary arteries within 12 months in GCA patients treated with glucocorticoids and tocilizumab.[106]

A comparable score based on IMT of the same segments, known as the Outcome Measures in Rheumatology (OMERACT) Giant Cell Arteritis Ultrasonography Score (**OGUS**) was developed based on the OMERACT Instrument Selection Algorithm,[107] as illustrated in Figure 7.[103] The OGUS exhibited good reliability in an online exercise with ultrasound images on a Digital Imaging and Communications in Medicine reader. Results from a patient-based reliability exercise will soon be available.

Sensitivity to change and convergent construct validity of the OGUS were tested in a prospective cohort of new GCA patients undergoing ultrasound-based IMT measurements at baseline, weeks 1, 3, 6, 12 and 24. This study found a continuous and significant reduction of the halo size of temporal arteries, whereas the reduction in the axillary arteries was less pronounced and insignificant. The reduction in the temporal artery halo size correlated with ESR, CRP, Birmingham Vasculitis Score and cumulative glucocorticoid dose.[65]

Alternative to the OGUS, the number of vasculitic segments can be counted (**halo count**).

GCA ultrasound score (OGUS)	Rounded cut-off	Intima–media thickness (IMT) measured in every segment is divided by the rounded cut-off values of IMTs in every segment
1. Common superficial temporal artery	0.4 mm	
Right side in mm □.□□ Left side in mm □.□□		
2. Frontal branch of temporal artery	0.3 mm	The sum is divided by the number of available segments (maxium 8)
Right side in mm □.□□ Left side in mm □.□□		
3. Parietal branch of temporal artery	0.3 mm	Interpretation: 0–<1: mean intima-media thickness below cut-off
Right side in mm □.□□ Left side in mm □.□□		>1: mean intima-media thickness above cut-off
4. Axillary artery	1.0 mm	
Right side in mm □.□□ Left side in mm □.□□		Cave: A value of 1 does not represent a cut-off for the diagnosis of GCA!

A calculator is available at http://scoring.multimedium.at/OMERACT

Figure 7. Calculation of the OGUS.
OMERACT, Outcome Measures in Rheumatology; OGUS, OMERACT Giant Cell Arteritis Ultrasonography score.

In an ultrasound substudy of the GUSTO trial from Switzerland, a score based on normal IMT was applied, easily convertible to the OGUS. IMT in both temporal and axillary arteries closely correlated with disease activity in a cohort of GCA patients treated with glucocorticoid pulse therapy for only 3 days followed by tocilizumab.[108]

Another independent cohort from Denmark, comprising 47 newly diagnosed GCA patients tested several scores. Similar to the other cohorts, the OGUS exhibited a large magnitude of change. It was considered the score least prone to potential bias. All scores demonstrated the potential to discriminate between remission and relapse.[109,110]

Another study revealed a decrease of abnormal temporal and extracranial artery IMT in 85% and 45%, respectively, within 24 months.[111]

The IMT decreased more rapidly in the temporal arteries, particularly in the first 24 weeks. A study investigating GCA patients for up to 12 years identified also a substantial decrease in IMT in the axillary arteries.[70] Relapses correlated with a significant increase of axillary artery IMT.[31]

Confirming new drug's positive on structural vascular pathology, not just on inflammation, is crucial. The most significant score reductions occur in the first few weeks of treatment. Therefore, trials should retrospectively include IMT measurements of the initial diagnostic ultrasound scan if available.[110]

Although primarily developed for trials, the OGUS may be useful in clinical practice. The benefit of regular IMT monitoring for all LVV patients is yet unclear. However, monitoring appears relevant in uncertain clinical situations, such as tocilizumab treatment, unclear symptoms

and normal CRP. The interpretation of ultrasound findings both at diagnosis and follow-up should always consider clinical and laboratory features.

Only few prospective studies exist on **monitoring TAK** with ultrasound. Carotid artery IMT was higher in active compared to inactive patients.[112–114]

Contrast-enhanced ultrasound (CEUS) can visualize the vasculature within inflamed walls of large extracranial arteries, showing increased artery wall perfusion in active GCA and TAK.[114–118] Most studies focused on the common carotid arteries. CEUS cannot yet be applied to smaller arteries like the temporal arteries. Costs are higher due to contrast agent and needle placement. Two arteries can be examined at the time of contrast agent injection, possibly more with a second injection. CEUS is currently mainly used for research.[28]

Ultrasound can detect the development of structural damage. New arterial stenoses develop particularly in TAK and, to some extent, in GCA, due to active disease.

A study using PET-CT at baseline and yearly CT scans found a tenfold increase in thoracic aorta aneurysms in GCA patients with aortitis compared to those without.[119] Nevertheless, it remains unclear how many patients need to be regularly screened with which imaging technique in which intervals for preventing severe complications or death caused by aortic dissection or rupture. Therefore, the EULAR recommendations on imaging in LVV do not yet advocate routinely screening all patients for aneurysm development, but decide on an individual basis.[15]

In Berlin-Buch, we annually determine the diameter of the ascending aorta with ultrasound in patients with extracranial GCA.[28,41] This examination takes about 10 s when performed by an experienced sonologist with a sector probe available for echocardiography. The first 6–8 cm of the ascending aorta are well visible with ultrasound. The cut-off value for dilatation depends on body size and patient age. As a rule of thumb, MRI or CT should be initiated if the diameter of the ascending aorta increases above 38 mm to provide information on the whole thoracic aorta. Further studies are needed to evaluate this very simple concept.

Ultrasound for prediction of outcome

Predicting the outcome of GCA and TAK remains challenging. Ultrasound findings poorly correlate with disease outcome,[65,111,120] and treatment recommendations for cranial and extracranial GCA have not yet differentiated, despite slightly longer and higher-dose glucocorticoid treatment in extracranial GCA patients.[16]

Conclusion

Ultrasound is recommended as the first diagnostic tool in GCA and proves valuable in TAK. Ultrasound in suspected GCA should at least include the temporal and axillary arteries. Nearly all other arteries are accessible with ultrasound except the descending thoracic aorta. Fast-track clinics led to a reduction of vision loss in GCA. If clinical and ultrasound findings align, no further confirmatory tests are needed. Ultrasound detects subclinical GCA in over 20% of PMR patients. These 'silent' GCA patients need to be treated as GCA. Ultrasound is also useful for follow-up with IMT measurements. Scores have been developed primarily for research studies.

Declarations

Author contributions
Wolfgang A. Schmidt: Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Writing – original draft; Writing – review & editing.

Valentin S. Schäfer: Resources; Validation; Writing – review & editing.

ORCID iDs

Wolfgang A. Schmidt https://orcid.org/0000-0001-7831-8738

Valentin S. Schäfer https://orcid.org/0000-0002-6591-5936

References

1. Jennette JC, Falk RJ, Bacon PA, *et al.* 2012 revised international chapel hill consensus conference nomenclature of vasculitides. *Arthritis Rheum* 2012; 65: 1–11.

2. Marozoff S, Mai A, Dehghan N, *et al.* Increased risk of venous thromboembolism in patients with granulomatosis with polyangiitis: a population-based study. *PLoS One* 2022; 17: e0270142.

3. Alibaz-Oner F, Ergelen R, Yıldız Y, *et al.* Femoral vein wall thickness measurement: a new diagnostic tool for Behçet's disease. *Rheumatology (Oxford)* 2021; 60: 288–296.

4. Kissin EY, Niu J, Balint P, *et al.* Musculoskeletal ultrasound training and competency assessment program for rheumatology fellows. *J Ultrasound Med* 2013; 32: 1735–1743.

5. Inanc N, Jousse-Joulin S, Abacar K, *et al.* The novel OMERACT Ultrasound Scoring System for salivary glands changes in patients with Sjögren's syndrome is associated with MRI and salivary flow rates. *J Rheumatol* 2024; 51: 263–269.

6. Di Battista M, Delle Sedie A, Romei C, *et al.* Lung ultrasound and high-resolution computed tomography quantitative variations during nintedanib treatment for systemic sclerosis-associated interstitial lung disease. *Rheumatology (Oxford)*. Epub ahead of print 4 December 2023. DOI: 10.1093/rheumatology/kead642.

7. Santiago T, Santos EJF, Ruaro B, *et al.* Recommendations for the execution and reporting of skin ultrasound in systemic sclerosis: an international collaboration under the WSF skin ultrasound group. *RMD Open* 2022; 8: e002371.

8. Weiß K, Schmidt WA, Krause A, *et al.* A study on echocardiographic findings in hospitalized patients with connective tissue diseases. *Scand J Rheumatol* 2022; 51: 142–151.

9. Schmidt WA, Kraft HE, Völker L, *et al.* Colour Doppler sonography to diagnose temporal arteritis. *Lancet* 1995; 345: 866.

10. Schmidt WA, Kraft HE, Vorpahl K, *et al.* Color duplex ultrasonography in the diagnosis of temporal arteritis. *N Engl J Med* 1997; 337: 1336–1342.

11. Schmidt WA. Role of ultrasound in the understanding and management of vasculitis. *Ther Adv Musculoskelet Dis* 2014; 6: 39–47.

12. Ponte C, Grayson PC, Robson JC, *et al.* 2022 American College of Rheumatology/EULAR classification criteria for giant cell arteritis. *Ann Rheum Dis* 2022; 81: 1647–1653.

13. Grayson PC, Ponte C, Suppiah R, *et al.* 2022 American College of Rheumatology/EULAR classification criteria for Takayasu arteritis. *Ann Rheum Dis* 2022; 81: 1654–1660.

14. Dejaco C, Ramiro S, Duftner C, *et al.* EULAR recommendations for the use of imaging in large vessel vasculitis in clinical practice. *Ann Rheum Dis* 2018; 77: 636–643.

15. Dejaco C, Ramiro S, Bond M, *et al.* EULAR recommendations for the use of imaging in large vessel vasculitis in clinical practice: 2023 update. *Ann Rheum Dis* 2024; 83: 741–751.

16. Hellmich B, Agueda A, Monti S, *et al.* 2018 update of the EULAR recommendations for the management of large vessel vasculitis. *Ann Rheum Dis* 2020; 79: 19–30.

17. Mollan SP, Paemeleire K, Versijpt J, *et al.* European Headache Federation

recommendations for neurologists managing giant cell arteritis. *J Headache Pain* 2020; 21: 28.

18. Mackie SL, Dejaco C, Appenzeller S, *et al.* British Society for Rheumatology guideline on diagnosis and treatment of giant cell arteritis: executive summary. *Rheumatology (Oxford)* 2020; 59: 487–494.

19. Turesson C, Börjesson O, Larsson K, *et al.* Swedish Society of Rheumatology 2018 guidelines for investigation, treatment, and follow-up of giant cell arteritis. *Scand J Rheumatol* 2019; 48: 259–265.

20. Haaversen AB, Brekke LK, Bakland G, *et al.* Norwegian society of rheumatology recommendations on diagnosis and treatment of patients with giant cell arteritis. *Front Med (Lausanne)* 2023; 9: 1082604.

21. Schirmer JH, Aries PM, Balzer K, *et al.* S2k guideline: management of large vessel vasculitides. *Z Rheumatol* 2020; 79(Suppl. 3): 67–95.

22. Pfeil A, Krusche M, Vossen D, *et al.* Model curriculum of the German Society for Rheumatology for advanced training in the discipline internal medicine and rheumatology. English version. *Z Rheumatol* 2021; 80(Suppl. 2): 64–67.

23. Watanabe R, Berry GJ, Liang DH, *et al.* Pathogenesis of giant cell arteritis and Takayasu arteritis – similarities and differences. *Curr Rheumatol Rep* 2020; 22: 68.

24. Gribbons KB, Ponte C, Carette S, *et al.* Patterns of arterial disease in Takayasu's arteritis and giant cell arteritis. *Arthritis Care Res (Hoboken)* 2020; 72: 1615–1624.

25. Schmidt WA and Nielsen BD. Imaging in large-vessel vasculitis. *Best Pract Res Clin Rheumatol* 2020; 34: 101589.

26. Monti S, Águeda AF, Luqmani RA, *et al.* Systematic literature review informing the 2018 update of the EULAR recommendation for the management of large vessel vasculitis: focus on giant cell arteritis. *RMD Open* 2019; 5: e001003.

27. Águeda AF, Monti S, Luqmani RA, *et al.* Management of Takayasu arteritis: a systematic literature review informing the 2018 update of the EULAR recommendation for the management of large vessel vasculitis. *RMD Open* 2019; 5: e001020.

28. Schmidt WA. Vascular ultrasound in rheumatology practice. *Best Pract Res Clin Rheumatol* 2023; 37: 101847.

29. Schmidt WA, Seifert A, Gromnica-Ihle E, *et al.* Ultrasound of proximal upper extremity arteries to increase the diagnostic yield in large-vessel giant cell arteritis. *Rheumatology (Oxford)* 2008; 47: 96–101.

30. Gribbons KB, Ponte C, Craven A, *et al.* Diagnostic assessment strategies and disease subsets in giant cell arteritis: data from an international observational cohort. *Arthritis Rheumatol* 2020; 72: 667–676.

31. Bosch P, Dejaco C, Schmidt WA, *et al.* Association of ultrasound-confirmed axillary artery vasculitis and clinical outcomes in giant cell arteritis. *Semin Arthritis Rheum* 2022; 56: 152051.

32. Molina-Collada J, Castrejón I, Monjo I, *et al.* Performance of the 2022 ACR/EULAR giant cell arteritis classification criteria for diagnosis in patients with suspected giant cell arteritis in routine clinical care. *RMD Open* 2023; 9: e002970.

33. van Nieuwland M, van Bon L, Vermeer M, *et al.* External validation of the 2022 ACR/EULAR classification criteria in patients with suspected giant cell arteritis in a Dutch fast-track clinic. *RMD Open* 2023; 9: e003080.

34. Tomelleri A, Padoan R, Kavadichanda CG, *et al.* Validation of the 2022 American College of Rheumatology/EULAR classification criteria for Takayasu arteritis. *Rheumatology (Oxford)* 2023; 62: 3427–3432.

35. Schmidt WA, Dasgupta B, Luqmani R, *et al.* A multicentre, randomised, double-blind, placebo-controlled, parallel-group study to evaluate the efficacy and safety of sirukumab in the treatment of giant cell arteritis. *Rheumatol Ther* 2020; 7: 793–810.

36. Cid MC, Unizony SH, Blockmans D, *et al.* Efficacy and safety of mavrilimumab in giant cell arteritis: a phase 2, randomised, double-blind, placebo-controlled trial. *Ann Rheum Dis* 2022; 81: 653–661.

37. Venhoff N, Schmidt WA, Bergner R, *et al.* Safety and efficacy of secukinumab in patients with giant cell arteritis (TitAIN): a randomised, double-blind, placebo-controlled, phase 2 trial. *Lancet Rheumatol* 2023; 5: e341–e350.

38. Schmidt WA, Dasgupta B, Sloane J, *et al.* A phase 3 randomized, double-blind, placebo-controlled study to evaluate the efficacy and safety of sarilumab in patients with giant cell arteritis. *Arthritis Res Ther* 2023; 25: 199.

39. Kreis L, Dejaco C, Schmidt WA, *et al.* The meteoritics trial: efficacy of methotrexate after

remission-induction with tocilizumab and glucocorticoids in giant cell arteritis-study protocol for a randomized, double-blind, placebo-controlled, parallel-group phase II study. *Trials* 2024; 25: 56.

40. Sundholm JKM, Paetau A, Albäck A, *et al*. Non-invasive vascular very-high resolution ultrasound to quantify artery intima layer thickness: validation of the four-line pattern. *Ultrasound Med Biol* 2019; 45: 2010–2018.

41. Schmidt WA. How extensively should ultrasound be performed in suspected giant cell arteritis? *Rheumatology (Oxford)* 2023; 62: 1733–1735.

42. Chrysidis S, Duftner C, Dejaco C, *et al*. Definitions and reliability assessment of elementary ultrasound lesions in giant cell arteritis: a study from the OMERACT Large Vessel Vasculitis Ultrasound Working Group. *RMD Open* 2018; 4: e000598.

43. Bosch P, Bond M, Dejaco C, *et al*. Imaging in diagnosis, monitoring and outcome prediction of large vessel vasculitis: a systematic literature review and meta-analysis informing the 2023 update of the EULAR recommendations. *RMD Open* 2023; 9: e003379.

44. Skoog J, Svensson C, Eriksson P, *et al*. The diagnostic performance of an extended ultrasound protocol in patients with clinically suspected giant cell arteritis. *Front Med (Lausanne)* 2022; 8: 807996.

45. Nielsen BD, Hansen IT, Keller KK, *et al*. Diagnostic accuracy of ultrasound for detecting large-vessel giant cell arteritis using FDG PET/CT as the reference. *Rheumatology (Oxford)* 2020; 59: 2062–2073.

46. Chrysidis S, Moller-Dohn U, Terslev L, *et al*. Diagnostic accuracy of vascular ultrasound in patients with suspected giant cell arteritis (EUREKA): a prospective, multicentre, non-interventional, cohort study. *Lancet Rheumatol* 2021; 3: e865–e873.

47. Diamantopoulos AP, Haugeberg G, Hetland H, *et al*. Diagnostic value of color Doppler ultrasonography of temporal arteries and large vessels in giant cell arteritis: a consecutive case series. *Arthritis Care Res (Hoboken)* 2014; 66: 113–119.

48. Prearo I, Dekorsy FJ, Brendel M, *et al*. Diagnostic yield of axillary artery ultrasound in addition to temporal artery ultrasound for the diagnosis of giant cell arteritis. *Clin Exp Rheumatol* 2022; 40: 819–825.

49. Bull Haaversen AC, Brekke LK, Kermani TA, *et al*. Extended ultrasound examination identifies more large vessel involvement in patients with giant cell arteritis. *Rheumatology (Oxford)* 2023; 62: 1887–1894.

50. Oshinsky C, Bays AM, Sacksen I, *et al*. The usefullness of subclavian artery ultrasound assessment in giant cell arteritis evaluation. *J Clin Rheumatol* 2023; 29: 43–46.

51. Molina-Collada J, Castrejón I, Monjo-Henry I, *et al*. Impact of ultrasound limitation to assess aortitis in patients with giant cell arteritis: comparative study with FDG-PET/CT. *RMD Open* 2023; 9: e003329.

52. Brilakis L, Tsakotos G, Lykoudis PM, *et al*. Prevalence of axillary artery variants and their clinical significance: a scoping review. *Cureus* 2023; 15: e47809.

53. Jese R, Rotar Z, Tomsic M, *et al*. The role of colour Doppler ultrasonography of facial and occipital arteries in patients with giant cell arteritis: a prospective study. *Eur J Radiol* 2017; 95: 9–12.

54. Chen-Xu M, Coath FL, Ducker G, *et al*. Maxillary artery involvement in giant cell arteritis demonstrated by ultrasonography. *J R Coll Physicians Edinb* 2021; 51: 366–368.

55. Burg LC, Schmidt WA, Brossart P, *et al*. A 78-year-old female with severe tongue pain: benefit of modern ultrasound. *BMC Med Imaging* 2021; 21: 55.

56. Kargiotis O, Psychogios K, Safouris A, *et al*. Cervical duplex ultrasound for the diagnosis of giant cell arteritis with vertebral artery involvement. *J Neuroimaging* 2021; 31: 656–664.

57. Imfeld S, Aschwanden M, Rottenburger C, *et al*. [18F]FDG positron emission tomography and ultrasound in the diagnosis of giant cell arteritis: congruent or complementary imaging methods? *Rheumatology (Oxford)* 2020; 59: 772–778.

58. Wang Y, Wang Y, Zhang L, *et al*. The performance of duplex ultrasonography for the assessment of renal artery stenosis in Takayasu's arteritis patients. *Arthritis Res Ther* 2023; 25: 139.

59. Lecler A, Hage R, Charbonneau F, *et al*. Validation of a multimodal algorithm for diagnosing giant cell arteritis with imaging. *Diagn Interv Imaging* 2022; 103: 103–110.

60. Barra L, Kanji T, Malette J, *et al*. Imaging modalities for the diagnosis and disease activity assessment of Takayasu's arteritis: a systematic review and meta-analysis. *Autoimmun Rev* 2018; 17: 175–187.

61. Schäfer VS, Jin L and Schmidt WA. Imaging for diagnosis, monitoring, and outcome prediction

of large vessel vasculitides. *Curr Rheumatol Rep* 2020; 22: 76.

62. Schäfer VS, Chrysidis S, Dejaco C, *et al.* Assessing vasculitis in giant cell arteritis by ultrasound: results of OMERACT patient-based reliability exercises. *J Rheumatol* 2018; 45: 1289–1295.

63. Guggenberger KV, Vogt ML, Song JW, *et al.* High-resolution magnetic resonance imaging visualizes intracranial large artery involvement in giant cell arteritis. *Rheumatology (Oxford)*. Epub ahead of print 10 January 2024. DOI: 10.1093/rheumatology/keae010.

64. Nielsen BD, Gormsen LC, Hansen IT, *et al.* Three days of high-dose glucocorticoid treatment attenuates large-vessel 18F-FDG uptake in large-vessel giant cell arteritis but with a limited impact on diagnostic accuracy. *Eur J Nucl Med Mol Imaging* 2018; 45: 1119–1128.

65. Ponte C, Monti S, Scirè CA, *et al.* Ultrasound halo sign as a potential monitoring tool for patients with giant cell arteritis: a prospective analysis. *Ann Rheum Dis* 2021; 80: 1475–1482.

66. Maleszewski JJ, Younge BR, Fritzlen JT, *et al.* Clinical and pathological evolution of giant cell arteritis: a prospective study of follow-up temporal artery biopsies in 40 treated patients. *Mod Pathol* 2017; 30: 788–796.

67. Schmidt WA, Moll A, Seifert A, *et al.* Prognosis of large-vessel giant cell arteritis. *Rheumatology (Oxford)* 2008; 47: 1406–1408.

68. Hauenstein C, Reinhard M, Geiger J, *et al.* Effects of early corticosteroid treatment on magnetic resonance imaging and ultrasonography findings in giant cell arteritis. *Rheumatology (Oxford)* 2012; 51: 1999–2003.

69. Ponte C, Serafim AS, Monti S, *et al.* Early variation of ultrasound halo sign with treatment and relation with clinical features in patients with giant cell arteritis. *Rheumatology (Oxford)* 2020; 59: 3717–3726.

70. Bosch P, Dejaco C, Schmidt WA, *et al.* Ultrasound for diagnosis and follow-up of chronic axillary vasculitis in patients with long-standing giant cell arteritis. *Ther Adv Musculoskel Dis* 2021; 13: 1759720X21998505.

71. Schäfer VS, Chrysidis S, Schmidt WA, *et al.* OMERACT definition and reliability assessment of chronic ultrasound lesions of the axillary artery in giant cell arteritis. *Semin Arthritis Rheum* 2021; 51: 951–956.

72. Schmidt WA. The ultrasound halo sign of temporal arteries: is it always giant cell arteritis? *Rheumatology (Oxford)* 2019; 58: 1898–1899.

73. Chrysidis S, Lewinski M and Schmidt WA. Temporal arteritis with ultrasound halo sign in eosinophilic granulomatosis with polyangiitis. *Rheumatology (Oxford)* 2019; 58: 2069–2071.

74. Daoussis D, Geropoulou C, Kargiotis O, *et al.* Painless, eosinophilic infiltration of temporal arteries. *Rheumatology (Oxford)* 2019; 58: 2065–2067.

75. Molina Collada J, Ruíz Bravo-Burguillos E, Monjo I, *et al.* Positive ultrasound halo sign of temporal arteries due to amyloidosis. *Rheumatology (Oxford)* 2019; 58: 2067–2069.

76. Fernández-Fernández E, Monjo-Henry I, Bonilla G, *et al.* False positives in the ultrasound diagnosis of giant cell arteritis: some diseases can also show the halo sign. *Rheumatology (Oxford)* 2020; 59: 2443–2447.

77. Schmidt WA. Ultrasound in the diagnosis and management of giant cell arteritis. *Rheumatology (Oxford)* 2018; 57(Suppl. 2): ii22–ii31.

78. Seitz L, Seitz P, Pop R, *et al.* Spectrum of large and medium vessel vasculitis in adults: primary vasculitides, arthritides, connective tissue, and fibroinflammatory diseases. *Curr Rheumatol Rep* 2022; 24: 352–370.

79. Patil P, Williams M, Maw WW, *et al.* Fast track pathway reduces sight loss in giant cell arteritis: results of a longitudinal observational cohort study. *Clin Exp Rheumatol* 2015; 33(Suppl. 89): S-103–S-106.

80. Diamantopoulos AP, Haugeberg G, Lindland A, *et al.* The fast-track ultrasound clinic for early diagnosis of giant cell arteritis significantly reduces permanent visual impairment: towards a more effective strategy to improve clinical outcome in giant cell arteritis? *Rheumatology (Oxford)* 2016; 55: 66–70.

81. Monti S, Bartoletti A, Bellis E, *et al.* Fast-track ultrasound clinic for the diagnosis of giant cell arteritis changes the prognosis of the disease but not the risk of future relapse. *Front Med (Lausanne)* 2020; 7: 589794.

82. Hernández P, Al Jalbout N, Matza M, *et al.* Temporal artery ultrasound for the diagnosis of giant cell arteritis in the emergency department. *Cureus* 2023; 15: e42350.

83. Tedeschi SK, Sobiesczyzk PS, Ford JA, *et al.* Clinical experience with a multidisciplinary model of vascular ultrasound for the evaluation for giant cell arteritis. *ACR Open Rheumatol* 2021; 3: 147–153.

84. Oshinsky C, Bays AM, Sacksen I, *et al.* Vascular ultrasound for giant cell arteritis: establishing a protocol using vascular sonographers in a

fast-track clinic in the United States. *ACR Open Rheumatol* 2022; 4: 13–18.

85. Sebastian A, Tomelleri A, Kayani A, *et al*. Probability-based algorithm using ultrasound and additional tests for suspected GCA in a fast-track clinic. *RMD Open* 2020; 6: e001297.

86. Tomelleri A, van der Geest KSM, Khurshid MA, *et al*. Disease stratification in GCA and PMR: state of the art and future perspectives. *Nat Rev Rheumatol* 2023; 19: 446–459.

87. Salvarani C, Padoan R, Iorio L, *et al*. Subclinical giant cell arteritis in polymyalgia rheumatica: concurrent conditions or a common spectrum of inflammatory diseases? *Autoimmun Rev* 2024; 23: 103415.

88. Tomelleri A, van der Geest KSM, Sebastian A, *et al*. Disease stratification in giant cell arteritis to reduce relapses and prevent long-term vascular damage. *Lancet Rheumatol* 2021; 3: E886–E895.

89. Dejaco C, Kerschbaumer A, Aletaha D, *et al*. Treat-to-target recommendations in giant cell arteritis and polymyalgia rheumatica. *Ann Rheum Dis* 2024; 83: 48–57.

90. Nielsen AW, Frølund LL, Våben C, *et al*. Concurrent baseline diagnosis of giant cell arteritis and polymyalgia rheumatica – a systematic review and meta-analysis. *Semin Arthritis Rheum* 2022; 56: 152069.

91. Burg LC, Karakostas P, Behning C, *et al*. Prevalence and characteristics of giant cell arteritis in patients with newly diagnosed polymyalgia rheumatica – a prospective cohort study. *Ther Adv Musculoskelet Dis* 2023; 15: 1759720X221149963.

92. De Miguel E, Macchioni P, Conticini E, *et al*. Prevalence and characteristics of subclinical giant cell arteritis in polymyalgia rheumatica. *Rheumatology (Oxford)* 2024; 63: 158–164.

93. De Miguel E, Karalilova R, Macchioni P, *et al*. Subclinical giant cell arteritis increases the risk of relapse in polymyalgia rheumatica. *Ann Rheum Dis* 2024; 83: 335–341.

94. Donskov AO, Mackie SL, Hauge EM, *et al*. An international survey of current management practices for polymyalgia rheumatica by general practitioners and rheumatologists. *Rheumatology (Oxford)* 2023; 62: 2797–2805.

95. Nielsen AW, Hemmig AK, de Thurah A, *et al*. Early referral of patients with suspected polymyalgia rheumatica – a systematic review. *Semin Arthritis Rheum* 2023; 63: 152260.

96. Keller KK, Mukhtyar CB, Nielsen AW, *et al*. Recommendations for early referral of individuals with suspected polymyalgia rheumatica: an initiative from the international giant cell arteritis and polymyalgia rheumatica study group. *Ann Rheum Dis*. Epub ahead of print 1 December 2023. DOI: 10.1136/ard-2023-225134.

97. Schäfer VS, Juche A, Ramiro S, *et al*. Ultrasound cut-off values for intima-media thickness of temporal, facial and axillary arteries in giant cell arteritis. *Rheumatology (Oxford)* 2017; 56: 1479–1483.

98. De Miguel E, Beltran LM, Monjo I, *et al*. Atherosclerosis as a potential pitfall in the diagnosis of giant cell arteritis. *Rheumatology (Oxford)* 2018; 57: 318–321.

99. Czihal M, Schröttle A, Baustel K, *et al*. B-mode sonography wall thickness assessment of the temporal and axillary arteries for the diagnosis of giant cell arteritis: a cohort study. *Clin Exp Rheumatol* 2017; 35(Suppl. 103): 128–133.

100. López-Gloria K, Castrejón I, Nieto-González JC, *et al*. Ultrasound intima media thickness cut-off values for cranial and extracranial arteries in patients with suspected giant cell arteritis. *Front Med (Lausanne)* 2022; 9: 981804.

101. Jese R, Rotar Z, Tomsic M, *et al*. The cut-off values for the intima-media complex thickness assessed by colour Doppler sonography in seven cranial and aortic arch arteries. *Rheumatology (Oxford)* 2021; 60: 1346–1352.

102. Berthod PE, Aho-Glélé S, Ornetti P, *et al*. CT analysis of the aorta in giant-cell arteritis: a case–control study. *Eur Radiol* 2018; 28: 3676–3684.

103. Dejaco C, Ponte C, Monti S, *et al*. The provisional OMERACT ultrasonography score for giant cell arteritis. *Ann Rheum Dis* 2023; 82: 556–564.

104. Molina-Collada J, López Gloria K, Castrejón I, *et al*. Impact of cardiovascular risk on the diagnostic accuracy of the ultrasound halo score for giant cell arteritis. *Arthritis Res Ther* 2022; 24: 232.

105. Van der Geest KS, Borg F, Kayani A, *et al*. Novel ultrasonographic halo score for giant cell arteritis: assessment of diagnostic accuracy and association with ocular ischaemia. *Ann Rheum Dis* 2020; 79: 393–399.

106. Sebastian A, Kayani A, Prieto-Pena D, *et al*. Efficacy and safety of tocilizumab in giant cell arteritis: a single centre NHS experience

using imaging (ultrasound and PET-CT) as a diagnostic and monitoring tool. *RMD Open* 2020; 6: e001417.

107. Terslev L, Naredo E, Keen HI, *et al*. The OMERACT stepwise approach to select and develop imaging outcome measurement instruments: the musculoskeletal ultrasound example. *J Rheumatol* 2019; 46: 1394–1400.

108. Seitz L, Christ L, Lötscher F, *et al*. Quantitative ultrasound to monitor the vascular response to tocilizumab in giant cell arteritis. *Rheumatology (Oxford)* 2021; 60: 5052–5059.

109. Nielsen BD, Therkildsen P, Keller KK, *et al*. Ultrasonography in the assessment of disease activity in cranial and large-vessel giant cell arteritis: a prospective follow-up study. *Rheumatology (Oxford)* 2023; 62: 3084–3094.

110. Schmidt WA. Monitoring giant cell arteritis with ultrasound. *Rheumatology (Oxford)* 2023; 62: 2948–2950.

111. Aschwanden M, Schegk E, Imfeld S, *et al*. Vessel wall plasticity in large vessel giant cell arteritis: an ultrasound follow-up study. *Rheumatology (Oxford)* 2019; 58: 792–797.

112. Kenar G, Karaman S, Çetin P, *et al*. Imaging is the major determinant in the assessment of disease activity in Takayasu's arteritis. *Clin Exp Rheumatol* 2020; 38(Suppl. 124): 55–60.

113. Svensson C, Eriksson P and Zachrisson H. Vascular ultrasound for monitoring of inflammatory activity in Takayasu arteritis. *Clin Physiol Funct Imaging* 2020; 40: 37–45.

114. Ma LY, Li CL, Ma LL, *et al*. Value of contrast-enhanced ultrasonography of the carotid artery for evaluating disease activity in Takayasu arteritis. *Arthritis Res Ther* 2019; 21: 24.

115. Bergner R, Splitthoff J and Wadsack D. Use of contrast-enhanced ultrasound sonography in giant cell arteritis: a proof-of-concept study. *Ultrasound Med Biol* 2022; 48: 143–148.

116. Ding J, Wu D, Han Q, *et al*. Follow-up contrast-enhanced ultrasonography of the carotid artery in patients with Takayasu arteritis: a retrospective study. *J Rheumatol* 2022; 49: 1242–1249.

117. Dong Y, Wang Y, Wang Y, *et al*. Ultrasonography and contrast-enhanced ultrasound for activity assessment in 115 patients with carotid involvement of Takayasu arteritis. *Mod Rheumatol* 2023; 33: 1007–1015.

118. Schmidt WA. Contrast-enhanced ultrasound for monitoring Takayasu arteritis. *J Rheumatol* 2022; 49: 1185–1187.

119. Moreel L, Coudyzer W, Boeckxstaens L, *et al*. Association between vascular ^{18}F-fluorodeoxyglucose uptake at diagnosis and change in aortic dimensions in giant cell arteritis: a cohort study. *Ann Intern Med* 2023; 176: 1321–1329.

120. Monti S, Ponte C, Pereira C, *et al*. The impact of disease extent and severity detected by quantitative ultrasound analysis in the diagnosis and outcome of giant cell arteritis. *Rheumatology (Oxford)* 2020; 59: 2299–2307.