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Echoes of Change: How Ultrasound Has Transformed Giant Cell Arteritis Detection

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Introduction

Giant cell arteritis (GCA) is the most common form of vasculitis affecting adults. The diagnosis of GCA is suspected in patients older than 50 years of age with a new headache and elevated inflammatory markers. Once the diagnosis of GCA is suspected, patients require urgent treatment with glucocorticoids to prevent ischemic complications such as blindness and stroke. As there are many causes for headache, diagnosing GCA can be a 'headache' for many rheumatologists. For years, rheumatologists have relied on the temporal artery biopsy (TAB) as the gold standard for diagnosing GCA, despite the 33–92% sensitivity.[1] As patients with suspected GCA remain on high doses of glucocorticoids, which have multiple side-effects and potential adverse events, rapid access to tests that have a greater impact on clinical decision-making is essential.[2] Vascular imaging is a non-invasive tool that can help diagnose, monitor, and predict the course of GCA. This article will focus on how ultrasound has transformed the detection of GCA and its potential to reduce some of the 'headaches' faced by both rheumatologists and patients.

Epidemiology

GCA is a large vessel (LV) vasculitis that has a predilection for the temporal artery and its branches but it can also affect the aorta, its branches, and the orbital arteries.[3] The incidence of GCA increases with age, and women are more commonly affected by GCA than men, with a ratio of 3:1.[4,5] Polymyalgia rheumatica (PMR) is a disease that significantly overlaps with GCA; approximately 50% of patients with GCA also have PMR, while approximately 20% of patients with PMR also have GCA.[6,7]

Pathophysiology

Disease induction and progression of GCA are due to a failure of immune tolerance. Factors including age-related loss of regulatory T-cells and/or genetic deficiencies contribute to unopposed T-cell activation.[5] Increased endothelial permeability, partly related to aging, allows inflammatory T-cells to enter the otherwise immune-privileged blood vessel wall, triggering a cascade of events that leads to the infiltration of pro-inflammatory mediators. This inflammatory milieu leads to vascular inflammation, which results in changes that can be observed on ultrasound as concentric intima and media thickening, creating a 'halo' around the lumen of the blood vessel.[8] While ultrasound reliably shows vessel wall edema, it does not provide ultrasonographic features specific for the location of granulomatous inflammation, the presence of giant cells, or regions of disruption of the internal elastic lamina, which explains the limited efficacy of ultrasound for guiding the TAB site in GCA.[9]

Clinical Presentation

GCA is classically recognized in patients presenting with new onset headache, jaw claudication, visual symptoms, scalp tenderness, and temporal artery abnormalities.[10] However, vascular imaging has expanded our understanding of GCA and its clinical manifestations. We now better appreciate that GCA can be stratified into clinical subsets based on the site of inflammation, including cranial GCA, LV-GCA, and LV-GCA with cranial involvement.[11] As such, not all patients with GCA present with cranial symptoms; those with LV involvement are more likely to have vascular abnormalities such as bruits, blood pressure asymmetry, abnormal pulses, and/or constitutional symptoms, while

those with overlapping PMR can have pain and stiffness in the shoulder and hip girdles.[6,11]

Diagnosis

The diagnosis of GCA is clinical, involving a combination of patient history, physical examination, laboratory investigations, and imaging parameters. Traditionally, TAB has been the gold standard for confirming a GCA diagnosis.[12] While TAB can investigate suspected cranial GCA and LV-GCA with cranial involvement, it has several shortcomings, including variable sensitivity (range from 33%–92%), skip lesions, and a focus on cranial GCA.[13] These drawbacks highlight the appeal of using imaging to guide GCA diagnosis.

Ultrasound assessment of the cranial arteries in GCA involves scanning the common temporal arteries and the frontal and parietal branches. Patients with occipital headaches may benefit from assessing the vertebral and occipital arteries, while those with jaw claudication may benefit from having the maxillary and facial arteries scanned. Ultrasound assessment of the extra-cranial arteries most commonly includes the axillary arteries but can also include the subclavian arteries, parts of the ascending aorta and aortic arch, as well as the femoral and popliteal vessels.[14] A 2023 systematic review and meta-analysis concluded that the pooled sensitivities and specificities for using ultrasound to assess the cranial arteries for the diagnosis of GCA are 88% (95% CI 82%–92%) and 96% (95% CI 86%–99%), respectively.[15] The sensitivity increases to 93% (95% CI 88%–96%) when ultrasound is used to assess both the cranial and extra-cranial arteries, without any loss in specificity.[15] As such, the recommended minimal GCA scan is currently the length of the common superficial temporal arteries bilaterally, the frontal and parietal branches of the superficial temporal arteries bilaterally, and the axillary arteries bilaterally.[16]

The intima of normal arteries is very thin. In GCA, concentric intima and media thickening occur due to inflammatory infiltrates and edema in the vessel wall, known as the halo sign.[8] According to Outcome Measures in Rheumatology (OMERACT), the halo sign is defined as a homogenous, hypoechoic wall swelling visible in both longitudinal and transverse planes (**Figure 1c/d/g**).[17] The compression sign, also defined by OMERACT, occurs when the thickened arterial wall remains visible upon compression (**Figure 1e/f**).[17] A non-compressible halo sign in the temporal arteries and their branches is highly suggestive of GCA.[18] The intima-media thickness (IMT) can also be measured using ultrasound and compared to standard cut-offs for healthy age-matched controls, with higher IMT being more suggestive of GCA.[19] Recently, the slope sign of the axillary artery has been described as a feature of GCA.[20] The slope sign describes a smooth transition from normal to increased IMT in the axillary artery in patients with GCA and can help differentiate vasculitis from other causes of arterial wall thickening, such as atherosclerosis (**Figure 1g**).[20]

In 2022, the American College of Rheumatology (ACR)/European Alliance of Associations for Rheumatology (EULAR) updated the classification criteria for GCA to include the role of ultrasound.[21] This classification criteria uses a point system that includes ten items, requiring a score of at least six points to classify a patient with GCA. According to these recommendations, a patient with a positive halo sign or a positive TAB, along with features from the history, and/or physical examination, laboratory tests, and/or imaging parameters in various combinations can be classified as having GCA (**Table 1**). Using these criteria, both TAB and ultrasound evidence of the halo sign are equally weighted (five points), while ultrasound carries the most weight (seven points) due to

Recommended minimal GCA scan
• Common superficial temporal arteries bilaterally
• Frontal and parietal branches of the superficial temporal arteries bilaterally
• Axillary arteries bilaterally
Technical Requirements:
• High frequency (preferably >18 MHz) linear probe for temporal arteries
• A 7–15 MHz probe for extra-cranial supra-aortic arteries
• Ultrasound machine with both B-mode and colour Doppler and/or power Doppler

Box 1. GCA Scan Requirements; *courtesy of Maria Powell, MD, MSc (Med Ed), CIP, FRCPC and Mohammad Bardi, MD, FRCPC.*

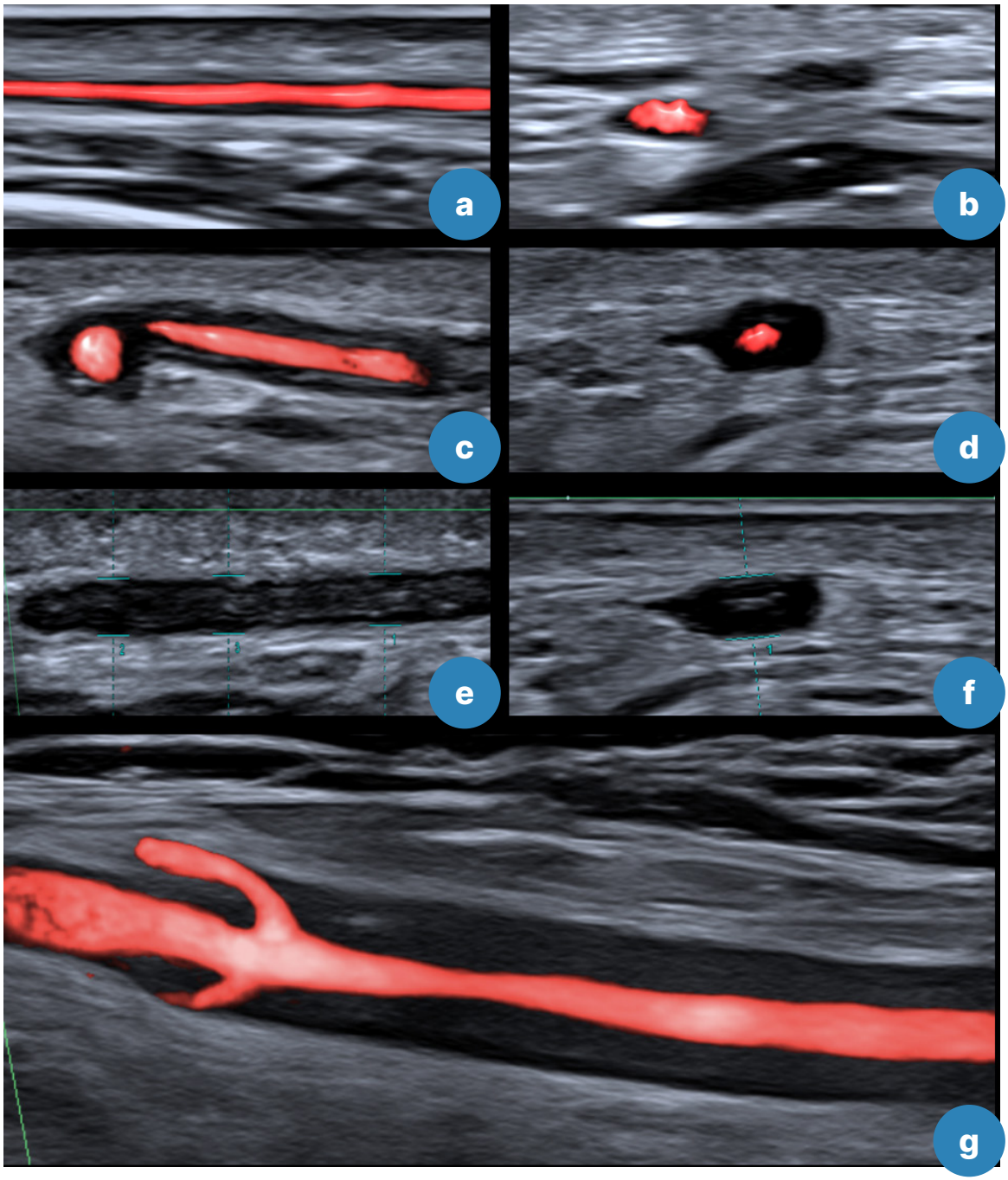

Figure 1. Normal artery in a healthy patient versus abnormal artery in a patient with giant cell arteritis (GCA). Normal common temporal artery in uncompressed longitudinal **(a)** and transverse **(b)** views. Abnormal common temporal artery in uncompressed longitudinal **(c)** and transverse **(d)** views with evidence of a halo sign (homogenous, hypoechoic wall swelling seen in the image as a dark area around the vessel). Abnormal common temporal artery in compressed longitudinal **(e)** and transverse **(f)** views with evidence of compression sign (thickened arterial wall remains visible upon compression). Large vessel vasculitis seen in an axillary artery in longitudinal view with evidence of slope sign (smooth transition from normal to increased intima-media thickness (seen in the image as a dark area around the vessel) **(g)**; *courtesy of Maria Powell, MD, MSc (Med Ed), CIP, FRCPC and Mohammad Bardi, MD, FRCPC.*

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Absolute Requirement	
Age >50 years at the time of diagnosis	
Additional Clinical Criteria	
Morning stiffness in the shoulders/neck	**+2**
Sudden visual loss	**+3**
Jaw or tongue claudication	**+2**
New temporal headache	**+2**
Scalp tenderness	**+2**
Abnormal examination of the temporal artery	**+2**
Laboratory, Imaging, and Biopsy Criteria	
Maximum ESR >50 mm/hour or maximum CRP >10 mg/liter	**+3**
Positive temporal artery biopsy or halo sign on temporal artery ultrasound	**+5**
Bilateral axillary involvement	**+2**
FDG-PET activity throughout the aorta	**+2**

Table 1. 2022 American College of Rheumatology/European Alliance of Associations for Rheumatology classification criteria for giant cell arteritis.[21]

Sum the scores for the 10 items, if present.
A score of >6 points is needed for the classification of GCA

Abbreviations: ESR: Erythrocyte sedimentation rate, **CRP:** C-reactive protein, **FDG-PET:** Fluorodeoxyglucose-Positron Emission Tomography

the inclusion of LV imaging. While TAB is still preferred by the 2021 ACR/vasculitis foundation guidelines, likely due to GCA ultrasound education being in the early development phases in the United States, the role of ultrasound in the classification of GCA is recognized.

Using ultrasound to diagnose GCA is non-invasive, involves no radiation or contrast, allows for real-time imaging, and can be performed at the bedside. Ultrasound is more sensitive than TAB for GCA diagnosis because it evaluates more than the 1.5 cm of the temporal artery sampled with TAB and can evaluate both the cranial and extra-cranial arteries.[15] Ultrasound assessment of the temporal and axillary arteries is more cost-effective than TAB for diagnosing GCA, even when accounting for additional factors such as training and equipment.[22] Compared to TAB, using ultrasound to diagnose GCA can decrease the length of inpatient admission (from 3.6 days to 0.6 days), reduce steroid comorbidity, and lower the risk of GCA complications such as vision-loss.[23,24]

Disease Monitoring

Traditional disease monitoring in GCA relies on measuring acute phase reactants such as c-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) in conjunction with the clinical assessment. However, there are limitations to consider when monitoring laboratory

parameters for GCA. Inflammatory markers can increase for reasons other than disease relapse such as infections, cancer, and other inflammatory disorders, and patients can experience disease relapse with normal inflammatory markers.[25] This is further complicated by treatment with interleukin (IL)-6 inhibitors, which make CRP measurements unreliable.[26] Additionally, patients with GCA can experience headaches, jaw pain, vision changes, and other symptoms compatible with GCA without a true disease relapse. Ultrasound can assist with disease monitoring in GCA by confirming improvement and/or normalization of wall thickening in involved vascular territories after treatment. On ultrasound assessment, regression of the halo sign of the temporal arteries occurs as early as 48 hours, while regression of the vessel wall edema in the axillary arteries can take several months.[27] By using ultrasound to assess the IMT and/or the number of vessel segments with a halo sign compared to the last measured value, rheumatologists can make crucial decisions to escalate or de-escalate immunosuppressive therapy.[28] This approach may be particularly valuable when there is discordance between a patient's clinical assessment and laboratory markers.

Prognosis

The ACR/vasculitis foundation recommends obtaining non-invasive vascular imaging for patients newly diagnosed with GCA to help capture the full extent of vascular involvement and predict the disease phenotype (cranial GCA versus LV-GCA versus both).[11,29] Ultrasound can be used to tabulate a halo score, which includes an assessment of the thickness of the halo sign in eight segments of the temporal and axillary arteries.[30] The halo score can help identify a subset of GCA patients with increased intimal hyperplasia who are at a higher risk of ischemic complications such as visual-loss and stroke.[30,31] The OMERACT GCA Ultrasonography Score (OGUS) includes measurement of the IMT of these same 8 arterial segments, divided by the normal IMT values for each segment, and can be used to predict early relapses during the first 6 months after treatment initiation.[32,33] Thus, using ultrasound to risk-stratify patients with GCA can be informative for rheumatologists as they counsel patients on treatment options.

Limitations and Considerations

Despite its advantages, there are limitations and considerations when using ultrasound to guide the diagnosis of GCA. First, to accurately use ultrasound to assess for evidence of GCA, a high frequency (preferably >18 MHz) linear probe with a small footprint is recommended for imaging the temporal arteries and a 7–15 MHz probe is recommended for imaging the extra-cranial supra-aortic arteries. The ultrasound machine must be capable of using both B-mode and colour Doppler and/or power Doppler. These machines can be expensive and have limited portability. Second, while select rheumatologists are learning how to perform vascular ultrasound, ultrasound training is not currently a mandatory part of the Canadian rheumatology training objectives (listed as an optional competency).[34] Thus, performing vascular ultrasound carries a high upfront cost for interested rheumatologists, who will require the proper equipment and training before using it to guide GCA diagnosis. We as authors prefer that rheumatologists perform the ultrasound assessment, as conducting the imaging separately from the clinical assessment reduces the reliability of the examination and increases the variability of treatment initiation. However, other health professionals, such as radiologists, can participate in the scanning depending on the centre and their expertise.[35] Third, although ultrasound is excellent for assessing the cranial and supra-aortic arteries, even when using low-frequency probes, it can currently only reliably assess the first 4 cm of the ascending aorta and aortic arch, and the assessment of the thoracic aorta is limited.[14] As such, other imaging modalities such as [18F]-fluorodeoxyglucose positron emission tomography (FDG-PET), MRI or CT must be used if involvement of these vascular territories is suspected.[16] Fourth, a false positive halo sign can be observed in other conditions such as amyloidosis, lymphoma, infection, and small vessel vasculitis.[36] Thus, the sonographer must be trained to differentiate vasculitis from infection, malignancy, and other primary rheumatic disorders. Finally, while ultrasound assessment of the cranial arteries is highly accurate, there is a rapid reduction in accuracy after initiation of glucocorticoids. The cranial arteries remain positive for only 3–7 days, with 50% remaining positive at three weeks.[14] To preserve the accuracy of ultrasound assessments for suspected GCA, the establishment of a GCA ultrasound fast-track clinic is recommended.[37]

Conclusions

Using ultrasound to guide the diagnosis of GCA is non-invasive, highly accurate, cost-effective, and improves patient outcomes. It has transformed our ability for early detection, disease stratification, and prognostication in GCA, providing rheumatologists with more confidence as they evaluate patients with headaches and suspected LV vasculitis.

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