

Use of occipital artery ultrasound to diagnose and monitor response to therapy in a patient with giant cell arteritis

Sirs,

Temporal artery ultrasound (TAUS) has an increasingly important role in the diagnosis of giant cell arteritis (GCA) and is included as a key criterion in the updated ACR/EULAR 2022 classification for GCA (1). Furthermore, EULAR imaging recommendations for large-vessel vasculitis suggest that TAUS be the first-line investigation for those with suspected GCA, and in the appropriate clinical context, abnormal findings can be used to confirm diagnosis, *in lieu* of biopsy (2). While the classic “halo” and “compression” signs suggestive of vessel wall inflammation are more often observed in the temporal artery, these features can be seen in occipital arteries (OA) in GCA (3). Although some groups have advocated for the inclusion of occipital arteries in the evaluation of potential GCA cases

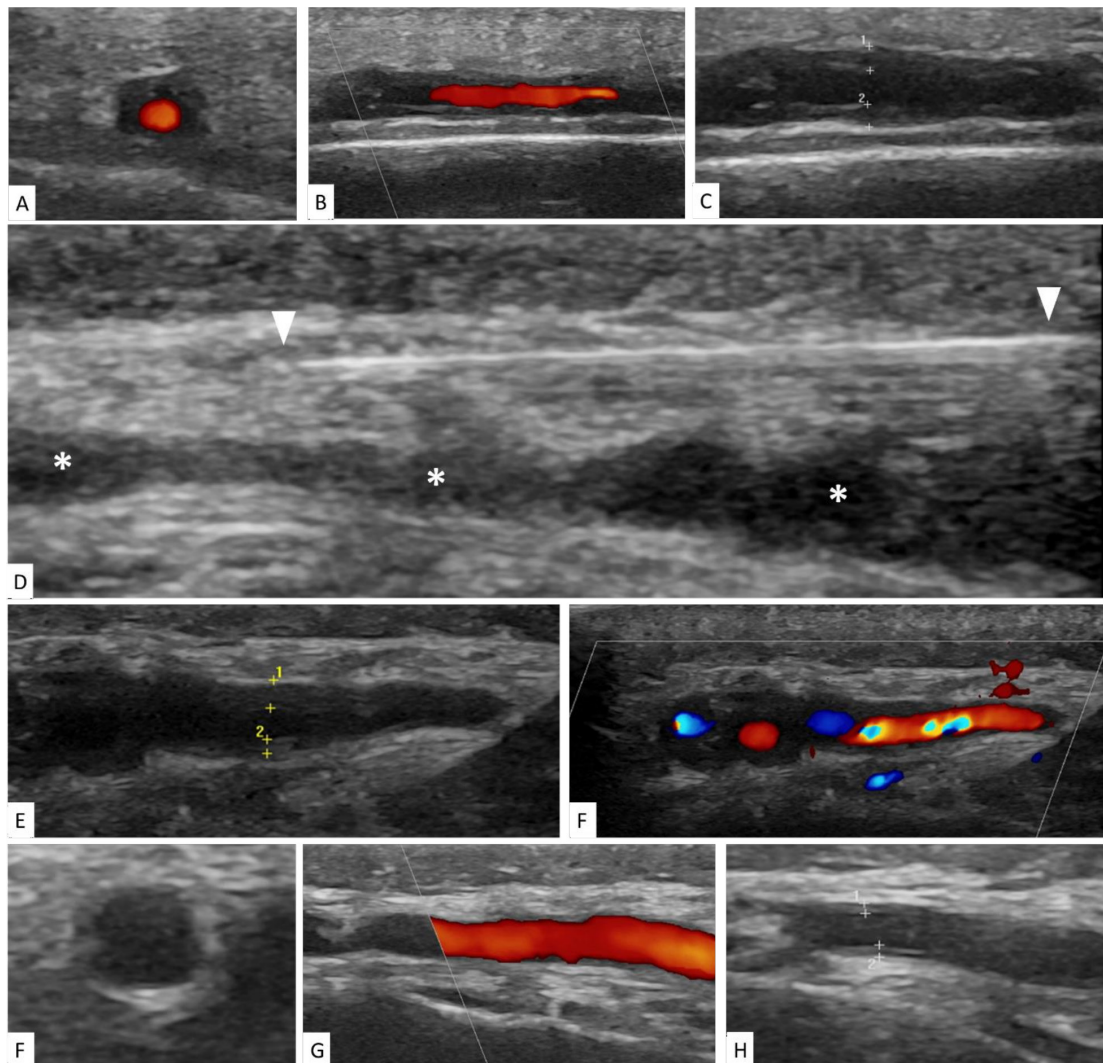
in fast-track clinics (4), definitions of ultrasound pathology consistent with GCA and provisional scoring systems proposed for monitoring disease activity have focused primarily on TAUS (4-6). Herein, we present a case that highlights not only the diagnostic utility of occipital artery ultrasound (OAUS) for diagnosing GCA but also for assessing treatment response.

An 80-year-old female presented with refractory scalp nodules. Dermatopathology, obtained one year prior, noted a subcutaneous artery with giant cells and mixed inflammation suggestive of GCA. The patient was started on high-dose prednisone and intravenous tocilizumab (4 mg/kg/month). Notably, her inflammatory markers before initiating immunosuppression were normal. When she presented to our facility, she had discontinued tocilizumab one month prior due to ongoing posterior scalp tenderness but remained on chronic daily 5 mg prednisone. Repeat inflammatory markers were again normal. Computed tomography angiography showed minimal thickening of the ascending aorta without delayed contrast

enhancement. Bilateral TAUS was negative. OAUS, however, demonstrated circumferential wall thickening and halo signs in the bilateral OAs (Fig. 1A-C). A trial of periarterial betamethasone injection to the left OA (Fig. 1D) was performed due to the patient’s hesitancy to restart systemic therapy and focal nature of symptoms. Ultrasound re-evaluation one month after injection (Fig. 1E) found worsening of left OA thickening despite ongoing normal inflammatory markers. Oral prednisone was increased from 5 mg to 10 mg. Methotrexate 15 mg weekly was started, and tocilizumab monthly infusions were resumed at a higher dose (8 mg/kg/month).

Four months after escalating her immunosuppression, she returned for reassessment. Repeat OAUS showed slight improvement in the OA vessel wall thickening, corresponding with clinical reduction in scalp nodule size and number and improvement of posterior scalp tenderness. Methotrexate and tocilizumab were continued, and prednisone was tapered. Eleven months after the reinitiating immunosuppression, the

Fig. 1. Left occipital artery ultrasound with Doppler demonstrating the characteristic ‘halo sign’ indicative of wall oedema (A, transverse view). Baseline longitudinal view with Doppler noted reduced flow (B) and increased intima-media thickness [IMT] (C) (1–0.07 cm, 2–0.06 cm). Periarterial betamethasone injection of the left occipital artery (D) with arrow heads outlining the needle path and asterisks highlighting the occipital artery course. Repeat grey-scale (E) and colour Doppler occipital artery ultrasound, one month after periarterial betamethasone injection with increase in the IMT (1–0.09 cm, 2–0.06 cm). Occipital artery ultrasound 11 months after reinitiation and escalation of immunosuppression showed resolution of halo sign (F, transverse view), improvement in Doppler flow (G) and normalisation of IMT (H; 1–0.02 cm, 2–0.03 cm).



patient denied any active scalp nodules and had symptomatic resolution of posterior scalp discomfort. Repeat OAUS (Fig. 1F-H) demonstrated resolution of arterial wall thickening and halo sign.

With normal initial temporal artery findings, negative inflammatory markers and scant evidence of extra-cranial inflammation, this case highlights the strengths of OAUS in diagnosing GCA. Indeed, Jese *et al.* observed that 20% of patients with negative TA findings may have other facial or OA pathology on ultrasound (7). As such, it is reasonable to evaluate additional facial arteries if TAUS is negative and large-vessel imaging is equivocal.

The utility of ultrasound for GCA monitoring has not yet been fully established. However, Ponte *et al.* have demonstrated the utility of TAUS for disease monitoring in GCA, showing dynamic changes with treatment and recurrence of findings on relapse (8). The case presented herein highlights that longitudinal monitoring of other cranial vessels, including OAs, can be considered both for diagnosis and assessment of treatment response, particularly in patients with atypical presentations such as ours, and in those for whom other objective laboratory or radiographic parameters are absent or unavailable.

D. MONTES¹, MD
J.J. SCHMITZ², MD
T. PAISANSINSUP³, MD
K.J. WARRINGTON⁴, MD
M.J. KOSTER⁴, MD

¹Department of Internal Medicine, and

²Department of Radiology, Mayo Clinic, Rochester, MN;

³Division of Rheumatology, Health Partners, Saint Louis Park, MN;

⁴Division of Rheumatology, Mayo Clinic, Rochester, MN, USA.

Please address correspondence to:

Daniel Montes

Department of Internal Medicine,
Mayo Clinic,

200 1st Street SW,

Rochester, MN 55905, USA.

E-mail: montes.daniel@mayo.edu

Competing interests: K.J. Warrington has received clinical trial support from BMS and consulting fees from Amgen and Sanofi. M.J. Koster has received consulting fees from Amgen. The other authors have declared no competing interests.

© Copyright CLINICAL AND EXPERIMENTAL RHEUMATOLOGY 2025.

References

1. 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(12): 1647-53. <https://doi.org/10.1136/ard-2022-223480>

2. 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(5): 636-43. <https://doi.org/10.1136/annrheumdis-2017-212649>
3. PFADENHAUER K, WEBER H: Duplex sonography of the temporal and occipital artery in the diagnosis of temporal arteritis. A prospective study. *J Rheumatol* 2003; 30(10): 2177-81.
4. 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(1): 13-18. <https://doi.org/10.1002/acr.2.11346>
5. 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(1): e000598. <https://doi.org/10.1136/rmdopen-2017-000598>
6. TREPPO E, MONTI S, DELVINO P *et al.*: Systemic vasculitis: one year in review 2024. *Clin Exp Rheumatol* 2024; 42(4): 771-81. <https://doi.org/10.55563/clinexprheumatol/gkve60>
7. JESE R, ROTAR Z, TOMSIC M, HOCEVAR A: 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. <https://doi.org/10.1016/j.ejrad.2017.07.007>
8. 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(11): 1475-82. <https://doi.org/10.1136/annrheumdis-2021-220306>