# Beyond axillary arteries: The role of extended ultrasound in characterizing extracranial involvement in giant cell arteritis

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Dear Editor,

EULAR recommendations [1] identify ultrasound of the temporal and axillary arteries as a first-line imaging modality for suspected giant cell arteritis (GCA). Systematic inclusion of axillary arteries increases diagnostic sensitivity and provides a baseline for follow-up [1]. However, GCA often affects other extracranial large vessels, which may be missed if the assessment is limited to temporal and axillary arteries.

We report two cases of GCA in which extended ultrasound revealed large-vessel involvement, despite normal axillary findings.

A 75-year-old woman presented with amaurosis fugax, new-onset headache, weight loss and shoulder/pelvic girdle inflammatory pain for 3 months. Laboratory findings revealed elevated inflammatory markers (ESR 117 mm/h; CRP 124 mg/L), anemia (hemoglobin 10.4 g/dL) and thrombocytosis (771 $\times 10^9$/L). Ultrasound showed hypoechoic halo and increased intima-media thickening (IMT) in temporal and vertebral arteries both sides (vertebral IMT 1.2 mm), while axillary arteries were unremarkable (Figure 1 A-C). She was treated with intravenous methylprednisolone and oral prednisolone at 1 mg/kg, but tocilizumab was added 3 months later due to GCA relapse.

A 77-year-old woman with prior polymyalgia rheumatica and steroid-associated psychosis developed headaches, malaise, jaw claudication and tongue pain during steroid tapering. Laboratory findings revealed elevated inflammatory markers (ESR 131 mm/h; CRP 71.5 mg/L), and anemia (hemoglobin 9.8 g/dL). Ultrasound showed hypoechoic halo and increased intima-media thickening in temporal and vertebral arteries (vertebral IMT 1.1 mm) (Figure 1 D-F). Axillary arteries were unremarkable. Given the previous steroid-associated adverse event, she was treated with tocilizumab, allowing faster glucocorticoid tapering.

In both patients, temporal artery involvement was unequivocal, so the diagnosis would have been reached with the EULAR-recommended protocol, but extracranial large-vessel involvement would have gone unnoticed should only axillary artery ultrasound have been performed. Its documentation was based on vertebral artery IMT exceeding the cut-off proposed by Jese et al of 0.7 mm, which showed high diagnostic accuracy[2]. Atherosclerosis was ruled unlikely due to hypoechogenicity of the thickening and absence of signs of vascular calcifications in these or other sides (Figure 1). This is highly relevant, as extracranial disease appears to carry prognostic impact in GCA[3, 4], making its documentation important in different clinical settings.

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Our cases suggest that reliance on the limited EULAR-recommended protocol may fail to document the full extent of disease. A more extended ultrasound protocol, encompassing additional cranial (such as the facial or occipital arteries) and extracranial territories (such as the vertebral, brachiocephalic, subclavian and carotid arteries) has been reported to detect additional arterial involvement, potentially increasing diagnostic sensitivity for GCA [5-7], at the cost of reduced specificity and greater technical complexity [7, 8]. This approach may allow non-invasive, ultrasound-based diagnosis, sparing patients more invasive or resource-intensive investigations.

On the other hand, the therapeutic relevance of detecting large-vessel involvement is still debated, as current guidelines are not fully aligned: ACR recommendations [9] support immunomodulator use (tocilizumab or methotrexate) in addition to steroid therapy in case of large-vessel involvement, whereas EULAR [10] and BSR recommendations [3] restrict their use to relapsing disease or to patients at higher risk of glucocorticoid-related toxicity. Although the therapeutic impact of detecting extracranial involvement remains undefined and requires further study, this recognition may support earlier consideration of biologic steroid-sparing strategies in selected patients. In neither case did the vertebral findings themselves influence therapeutic decisions, as the use of tocilizumab was driven by disease relapse and prior glucocorticoid toxicity, respectively. However, given the prognostic impact of large-vessel involvement, particularly its possible association with relapses[4], documenting it may contribute to risk stratification in GCA.

In conclusion, our cases underscore that normal axillary findings do not exclude extracranial involvement in other territories, and that an extended protocol, beyond what is currently recommended, can better characterize disease distribution in GCA, which may contribute to risk stratification. Whether this translates into clinical or therapeutic benefit remains to be established.

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## Tables and Figures

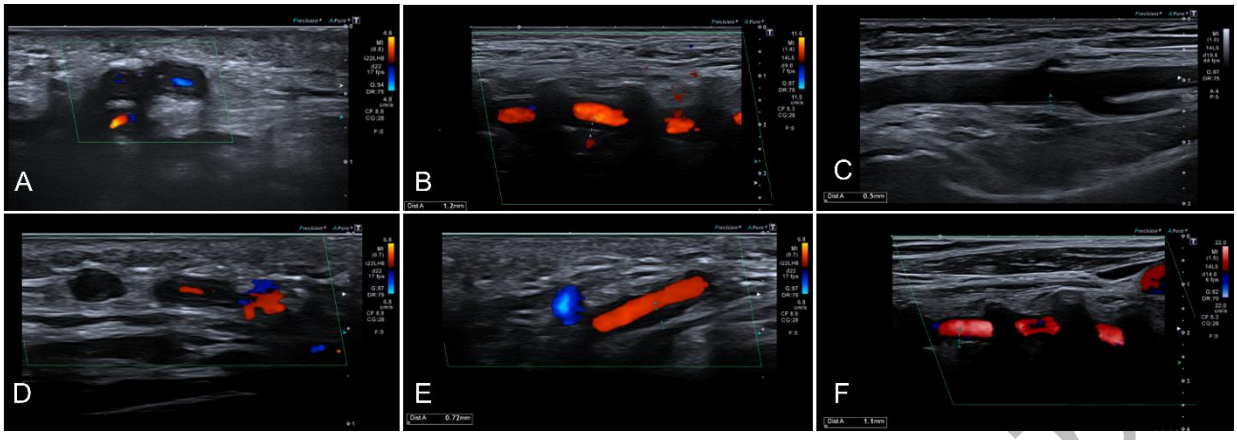



**Figure 1. Ultrasound findings in case 1 (A-C) and case 2 (D-F). (A)** Transverse view showing a hypoechoic halo in the common superficial temporal artery; **(B)** Longitudinal view of the vertebral artery, showing increased IMT (1.2 mm); (**C)** Longitudinal view of an unremarkable axillary artery (IMT 0.5 mm); **(D)** Transverse view of the parietal (left) and frontal (right) branches of the superficial temporal artery; **(E)** Longitudinal view of the frontal branch of the superficial temporal artery with increased IMT (0.72 mm); **(F)** Longitudinal view of the vertebral artery with increased vertebral IMT (1.1 mm). All images were collected with Canon Aplio i800 system: temporal arteries with a hockey-stick probe (iL22LH8, 22 MHz) and axillary and vertebral arteries with a linear probe (14L5, 5-14 MHz). *IMT, intima-media thickness.*

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