Comment on: Carotid artery ultrasonography and shear wave elastography in Takayasu's arteritis: a comparative analysis with diabetes mellitus

Sirs,

We read with great interest the article by Uysal *et al*. comparing carotid morphology and stiffness between patients with Takayasu's arteritis (TAK) and those with type 2 diabetes mellitus (T2DM) (1). The study is noteworthy for applying shear-wave elastography (SWE) to characterise vascular changes in large-vessel vasculitis. However, several methodological aspects deserve clarification and further consideration. First, while the authors excluded subjects with overt cardiovascular disease, they did not fully address treatment-related confounding factors that could influence arterial stiffness and intima-media thickness (IMT). Glucocorticoids, biologic therapies (such as anti-TNF agents, infliximab, adalimumab, etanercept, and anti-IL-6 therapy, tocilizumab), statins and antihypertensives can each alter vascular inflammation and elasticity, thereby affecting SWE values (2, 3). Multivariable models should therefore include treatment exposure, dose and duration, and ideally employ propensity adjustments to isolate disease-specific effects. Second, disease activity was assessed using ITAS2010 but not explicitly incorporated into regression analyses. Active vasculitis may transiently increase stiffness independent of structural atherosclerosis; stratification according to validated activity indices (*e.g*. ITAS2010/DEI.Tak) would clarify whether SWE predominantly reflects inflammatory or fibrotic wall change (4). Third, although the authors acknowledge that intra- and inter-observer variability was not assessed, other technical aspects of SWE measurement also require attention. SWE readings depend on multiple variables, including probe pressure, region-of interest depth, cardiac cycle phase, and arterial wall heterogeneity (5). Comprehensive reporting of methodological details, such as standardisation of the measurement site, averaging over several cardiac cycles, and control of probe compression, would improve reproducibility and inter-study comparability. In particular, assessing reproducibility using intraclass correlation coefficients (ICC) or coefficients of variation (CoV), as suggested in recent methodological studies, would further validate the reliability of SWE data (5). To ensure consistency and comparability, ensure intima-media thickness measurement should also adhere to the Mannheim consensus (plaque-free distal wall, lateral averagin).

Finally, interpretation of SWE findings in the context of vascular pathology requires caution. Lesions in TAK exhibit heterogeneous wall composition, including fibrosis and residual inflammation-each exerting distinct effects on stiffness. In the absence of segmental analysis, adjunctive imaging, or histopathologic correlation, it remains uncertain whether elevated SWE values predominantly reflect chronic fibrotic remodeling or ongoing subclinical inflammation. Future studies integrating SWE with PET/CT or high-resoltion MRI vessel wall imaging may help clarify this distinction.

In conclusion, Uysal *et al*. provided valuable data on carotid remodeling in TAK using an innovative imaging technique. However, controlling for treatment-related confounders, incorporating disease activity into statistical models, standardising the SWE protocol, and ensuring reproducibility are essential steps for validating SWE as a reliable vascular biomarker in vasculitis.

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