Diagnostic value of ultrasound halo count and Halo Score in giant cell arteritis: a retrospective study from routine care

We read with great interest the paper published by van der Geest *et al*[1] on 'Novel ultrasonographic Halo Score for giant cell arteritis (GCA): assessment of diagnostic accuracy and association with ocular ischaemia'. The authors aimed to quantify the extent of vascular inflammation by ultrasound (US) in patients with GCA and developed two novel US scoring systems, the halo count and Halo Score, including the assessment of the three temporal artery (TA) segments and axillary arteries. First, we would like to congratulate them for the novelty of their work that opens up new perspectives in the use of US in the assessment of GCA. According to recent EULAR recommendations, US is recommended as the first imaging modality in patients with suspected predominantly cranial GCA.[2] The halo sign is the most relevant US finding in GCA and is defined as a homogeneous, hypoechoic wall thickening, well delineated towards the luminal side, visible in two perpendicular planes, most commonly concentric in transverse scan.[3] The halo count and Halo Score constitute the first quantitative tools to assess the extent of vascular inflammation by US in GCA.[1] According to their findings, a high volume of vascular inflammation on US might strongly support the diagnosis of GCA, is linked to systemic markers of inflammation and identifies patients at risk for ocular ischaemia. On the other hand, a modified Halo Score has been recently proposed by Chattopadhyay *et al*[4] including the assessment of three vascular territories (bilateral temporal, subclavian and axillary arteries) instead of two, as the original Halo Score may underestimate the burden of the inflammation in large-vessel GCA and Takayasu arteritis.

We aim to assess the diagnostic value of both scoring systems and its association with systemic inflammation in patients with GCA seen in routine care. This was a retrospective observational study including patients suspected of having GCA over a 9-month period. All patients underwent bilateral US examination of the three TA segments (common superficial TA, its parietal and frontal branches) and extracranial (carotid, subclavian and axillary) arteries as part of a diagnostic fast track pathway (FTP)[5] where US is undertaken within 24 hours. The extent of vascular inflammation was quantified according to the halo count (number of TA segments and axillary arteries with a halo) ranging from 0 to 8 and the Halo Score (a composite index that incorporates both the number of halos and the maximum halo thickness in each region) ranging from 0 to 48.[1] TA biopsy was performed according to the treating clinician criteria. The gold standard for GCA was the clinical diagnosis after 6 months of follow-up. Validity was analysed by receiver operating characteristic (ROC) curves and correlations were determined by Spearman's rank correlation coefficient (rho).

Fifty-eight patients were evaluated in the FTP (mean age 74.7 years, 65.5% females). Clinical and US variables of patients with and without GCA are shown in table 1. A clinical diagnosis of GCA was established in 15 (25.9%) patients. Only 4.7% patients without GCA versus 86.7% with GCA had positive US findings according to the ultrasonographer criteria (sensitivity (Sens) 86.7%, specificity (Spec) 95.3%, positive likelihood ratio (LR+) 18.4 and negative likelihood ratio (LR−) 0.14). Halo count and Halo Score showed similar diagnostic accuracy for a clinical diagnosis of GCA (area under the ROC

Table 1 Clinical, laboratory and ultrasound findings of patients included in the fast track pathway

	Total, n=58	Patients with GCA, n=15	Patients without GCA, n=43	P value
Age, mean (SD)	74.7 (10.9)	76.5 (10.2)	74 (11.3)	0.431
Sex, no. of female	38 (65.5%)	8 (53.3%)	30 (69.8%)	0.249
Baseline use of steroids, no. of patients	28 (49.1%)	6 (40%)	22 (52.4%)	0.410
TA biopsy positive n=11, no. of patients	3 (27.3%)	3 (37.5%)	0 (0%)	0.491
TA biopsy length (mm) n=11, mean (SD)	5.5 (3.1)	5.9 (3.6)	4.7 (1.5)	0.6
^{18}F-FDG-PET/CT positive n=10, no. of patients	5 (50%)	4 (66.7%)	1 (25%)	0.197
Fulfilling 1990 GCA criteria, no. of patients	13 (22.4%)	5 (33.3%)	8 (18.6%)	0.239
Headache, no. of patients	30 (51.7%)	11 (73.3%)	19 (44.2%)	0.052
Scalp tenderness, no. of patients	4 (6.9%)	2 (13.3%)	2 (4.7%)	0.273
Jaw claudication, no. of patients	10 (17.2%)	7 (46.7%)	3 (7%)	**0.002**
Visual symptoms, no. of patients	10 (17.2%)	5 (33.3%)	5 (11.6%)	0.055
Fever, no. of patients	7 (12.1%)	2 (13.3%)	5 (11.6%)	1
Polymyalgia, no. of patients	27 (46.6%)	10 (66.7%)	17 (39.5%)	0.07
Ocular ischaemia, no. of patients	3 (5.2%)	1 (6.7%)	2 (4.7%)	1
Abnormal TA clinical examination, no. of patients	4 (6.9%)	2 (13.3%)	2 (4.7%)	0.273
CRP (mg/dL), mean (SD)	4.5 (6.7)	9.3 (8.8)	2.7 (4.6)	**0.001**
ESR (mm/h), mean (SD)	51.2 (33.7)	65.7 (33.2)	46.1 (33.1)	0.075
Haemoglobin (g/dL), mean (SD)	12.6 (1.7)	11.9 (1.6)	12.9 (1.6)	0.05
Platelets 10^9/L, mean (SD)	266.8 (96)	307.5 (104.1)	252.2 (89.7)	0.081
Positive US findings, no. of patients	15 (25.9%)	13 (86.7%)	2 (4.7%)	**<0.001**
TA positive US findings, no. of patients	11 (19%)	10 (66.7%)	1 (2.3%)	**<0.001**
Axillary positive US findings, no. of patients	8 (13.8%)	7 (46.7%)	1 (2.3%)	**<0.001**
TA+axillary positive US findings, no. of patients	4 (7%)	4 (26.7%)	0 (0%)	**0.003**
Halo sign positive, no. of patients	15 (25.9%)	13 (86.7%)	2 (4.7%)	**<0.001**
Compression sign positive, no. of patients	8 (13.8%)	7 (46.7%)	1 (2.3%)	**<0.001**
Halo count, mean (SD)	0.7 (1.4)	2.5 (1.9)	0.04 (0.2)	**<0.001**
Halo Score, mean (SD)	4.5 (8.7)	15.8 (9.9)	0.5 (2.7)	**<0.001**

CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; 18F-FDG-PET/CT, Fluorine-18 Fluorodeoxyglucose Positron Emission Tomography/Computed Tomography; GCA, giant cell arteritis; TA, temporal artery; US, ultrasound.

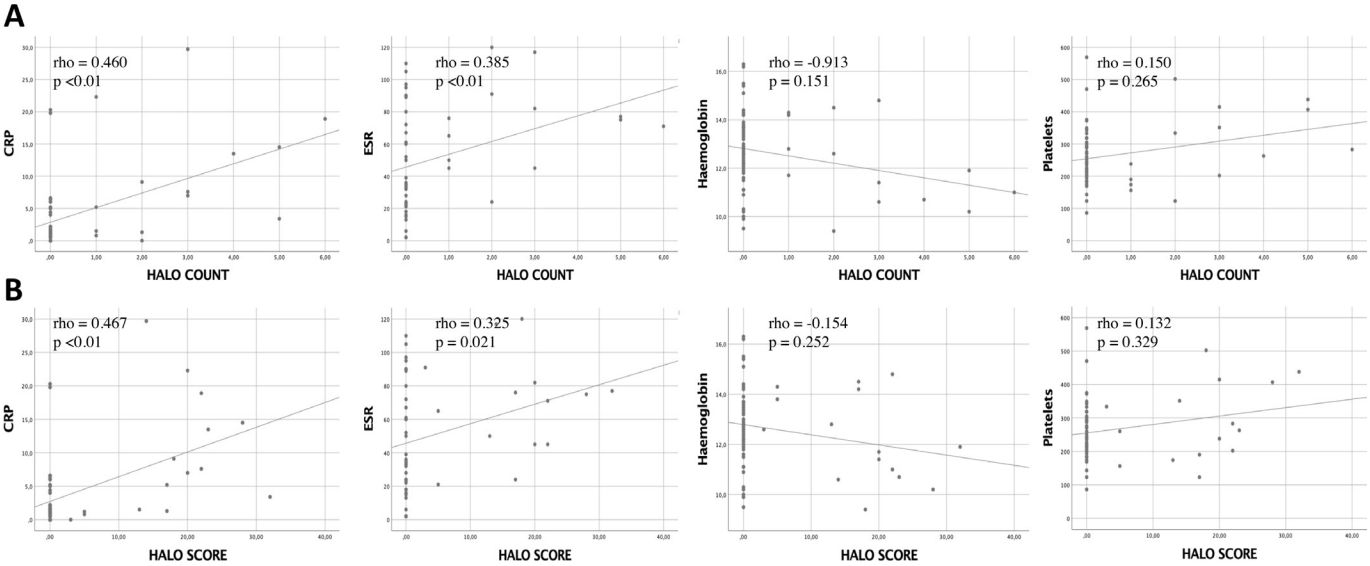

Figure 1 Correlations between halo count (A) and Halo Score (B) with markers of systemic inflammation (CRP, ESR, haemoglobin and platelets). CRP, C-reactive protein; ESR, erythrocyte sedimentation rate.

curve of 0.892 and 0.921, respectively). The optimal cut-off point for halo count was ≥1 (Sens 80%, Spec 95.3, LR+ 17.02, LR− 0.21) and for Halo Score ≥2 (Sens 86.7%, Spec 95.3%, LR+18.4, LR− 0.14). Statistically moderate positive correlations were found between halo count and Halo Score and ESR (rho 0.385 and 0.325, $p<0.05$) and C-reactive protein (CRP) (rho 0.460 and 0.467, $p<0.01$), but not with haemoglobin and platelet count ($p>0.05$) (figure 1).

To our knowledge, this is the first study to assess the diagnostic value of the halo count and Halo Score in a routine clinical setting, after its first description by van der Geest *et al*.[1] They first demonstrated that the Halo Score correlated positively with CRP levels and platelet counts and negatively with haemoglobin levels, but they found no correlation with ESR. Our findings confirm the link between both scoring systems with systemic inflammation in GCA, both with CRP and ESR, and show a good diagnostic accuracy in a clinical setting. In summary, the extent of vascular inflammation by US halo count and Halo Score can help to support the diagnosis of GCA in routine care as they correlate with laboratory markers of systemic inflammation. In the future, they may also have a role in monitoring disease activity. Although both scoring systems needs further validation, they can be easily implemented in FTP of patients with GCA.

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