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# The ultrasound compression sign to diagnose temporal giant cell arteritis shows an excellent interobserver agreement

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M. Aschwanden<sup>1</sup>, S. Imfeld<sup>1</sup>, D. Staub<sup>1</sup>, T. Baldi<sup>1</sup>, U.A. Walker<sup>2</sup>, C.T. Berger<sup>3</sup>,  
C. Hess<sup>3</sup>, T. Daikeler<sup>2</sup>

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<sup>1</sup>Department of Angiology, University Hospital, Basel, Switzerland;

<sup>2</sup>Department of Rheumatology, University Hospital, Basel, Switzerland;

<sup>3</sup>Medical Outpatient Department, University Hospital, Basel, Switzerland.

Markus Aschwanden, MD\*

Stephan Imfeld, MD, PhD\*

Daniel Staub, MD

Thomas Baldi, MD

Ulrich A. Walker, MD

Christoph T. Berger, MD

Christoph Hess, MD, PhD

Thomas Daikeler, MD

\*These authors made an equal contribution to this study.

Reprints will not be available from the author.

Please address correspondence to:

Markus Aschwanden, MD,

Department of Angiology,

University Hospital Basel,

Petersgraben 4,

CH-4031 Basel, Switzerland.

E-mail: markus.aschwanden@usb.ch

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## ABSTRACT

**Objective.** To compare the diagnostic performance between a vascular specialist and a rheumatologist not familiar with vascular ultrasound when applying the compression sign for the diagnosis of temporal arteritis.

**Methods.** Sixty consecutive patients with suspicion of giant cell arteritis were examined by both examiners. Compression of the temporal artery on both sides (stem and both branches) was performed to define whether signs of vasculitis, no vasculitis or an indefinite result were present. Each examiner was blinded to the result of the other.

**Results.** In 59/60 patients, the examiners found an identical result. The interobserver agreement (Krippendorff alpha) was 0.92.

**Conclusion.** The new compression sign for the diagnosis of temporal arteritis is a simple and robust sonographic marker with an excellent interobserver agreement.

## Introduction

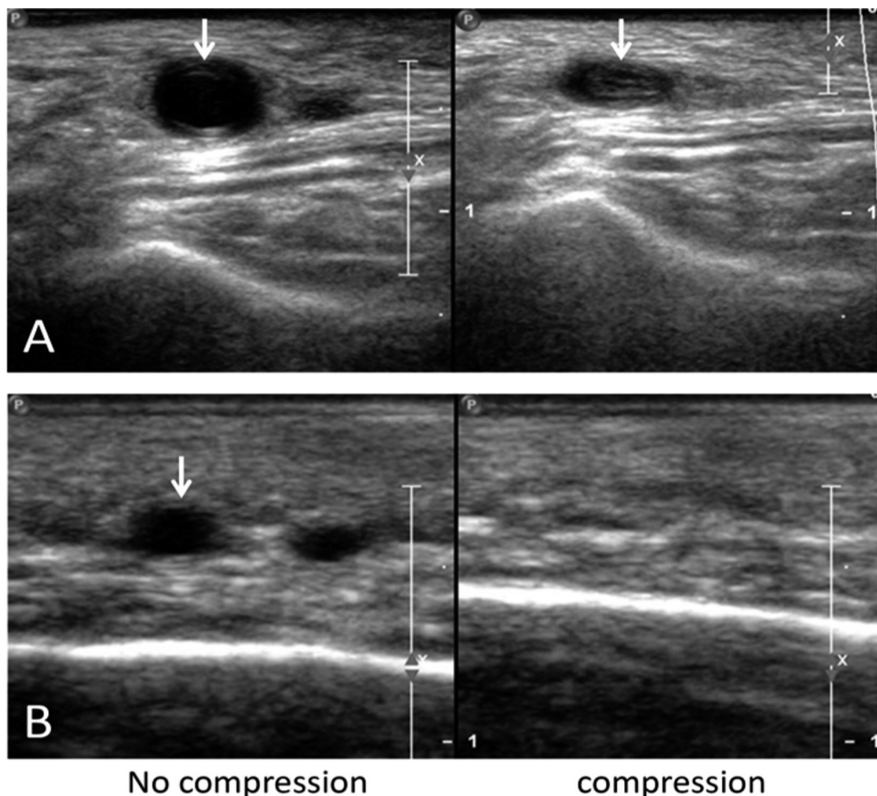
Giant cell arteritis (GCA) is the most prevalent primary vasculitis (1) and temporal artery (TA) biopsy is still considered the gold standard for diagnosis. However, the invasive nature and the risk of false negative biopsies due to segmental or unilateral affection of the TA highlight the need for alternative diagnostic approaches. Sonography is considered the most reliable non-invasive technique to detect TA vasculitis (2, 3). Sensitivity and specificity of existing ultrasound (US) criteria such as the halo-sign, stenosis and occlusions are reported to be high and have been used in everyday practice for many years (4). However, the diagnostic accuracy of these TA-US signs is dependent on the examiners' experience, thus results vary considerably from publication to publication

(5). We recently demonstrated that in GCA the compression of an affected TA elicits contrasting echogenicity between the diseased artery wall and the surrounding tissue (Fig. 1). This 'compression sign' performs excellently in the hand of specialised vascular physicians (6). Here, we aimed to evaluate the practicability and robustness of the compression sign in daily practice by comparing the performance of a rheumatologist unfamiliar with vascular ultrasound to the one of vascular physicians who routinely deal with sonography of blood vessels.

## Patients and methods

After giving informed consent, consecutive patients referred to our clinic with suspected GCA were prospectively examined in a sub-study of the ethics committee – approved Basel GCA cohort (BARK) by a vascular specialist and a non-specialist (rheumatologist) who had received a short training for the method. For this purpose the rheumatologist examined five patients under supervision and instruction of an experienced vascular physician (MA). For the study both examiners were blinded to each other's findings. They assessed the TA at its trunk, parietal and frontal branches for the presence or absence of the compression sign as previously described in detail (6). In short, the compression sonography was conducted as follows: slight pressure was applied via the transducer until the lumen was occluded and no arterial pulsation remained visible in B-mode imaging. If the vessel lumen contrast disappeared, temporal artery segments were graded as 'normal'. If a thickened contrasting vessel wall remained visible they were considered as 'vasculitis', or as 'inconclusive' in the case of suspicious but unclear findings (Fig. 1). If one of the six segments was

Competing interests: none declared.



**Fig. 1.** Illustration of the compression sign. **A.** Remaining visibility upon transducer imposed compression in a temporal arteritis. **B.** Loss of visibility upon transducer imposed compression in a normal temporal artery. Arrow indicates temporal artery.

**Table I.** Patient characteristics.

	GCA (n=24)	non-GCA (n=36)	p-value
Female n (%)	14 (58)	26 (72)	NS*
Mean age in years (SD)	73 ± 8	69 ± 13	NS‡
<i>Clinical presentation n (%)</i>			
Fever	2 (8)	2 (6)	NS*
Vision disorder	9 (38)	7 (19)	NS*
Headache	11 (46)	18 (50)	NS*
Jaw claudication	11 (46)	4 (11)	<0.004*
Tenderness on scalp	11 (46)	4 (11)	0.002*
Prominent TA	7 (29)	2 (6)	0.019‡
Polymyalgia	7 (29)	19 (53)	0.071*
Weight loss	5 (21)	5 (14)	NS*
<i>Laboratory values</i>			
Median ESR mm/h [IQR]	54 [44,74]	42 [18,60]	0.024‡
Median CRP mg/l [IQR]	40 [28,93]	12 [5,41]	0.002‡
Mean Lc x10 <sup>9</sup> /l (SD)	11 ± 4	27 ± 101	NS‡
Mean Hb g/l (SD)	131 ± 13	120 ± 16	0.037‡
Mean Tc x10 <sup>9</sup> /l (SD)	405 ± 108	338 ± 174	0.018‡
Biopsy performed n (%)	21** (88)	5 (14)	<0.001*
positive	13	0	
Median days on steroids before CDU [IQR]	2 [1,20]	2 [0,15]	NS‡
Median days on steroids before biopsy [IQR]	4 [3,21]	5 [0,-]	NS‡
Median days between CDU and biopsy [IQR]	3 [1,5]	2 [1,2]	NS‡

TA: temporal artery, ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, Lc: leucocytes, Hb: haemoglobin, Tc: thrombocytes, CDU: colour duplex ultrasound, IQR: inter-quartile range. \*Chi-square test; ‡Mann-Whitney U-test; †Fisher's exact test; \*\*Three biopsies revealed only a venous segment.

graded as ‘vasculitis’, the US diagnosis of vasculitis was made. All examinations were performed with an iU22 duplex ultrasound machine (Philips, Best, Netherland) using a high frequency (5-17MHz) broadband linear transducer. The sequence of examination (rheumatologist or vascular specialist first) was dependent on the availability of the rheumatologist. All examinations took place before TA biopsy. Interobserver agreement between vascular specialists and the rheumatologist was calculated using Krippendorff’s alpha (7).

**Results**

Sixty consecutive referrals with suspected GCA were included between October 2011 and December 2012. In 24 (40%) of these subjects, the diagnosis of GCA was established based on the ACR criteria (8). In the remaining 36 patients (non-GCA patients) GCA was excluded. Main diagnoses amongst the non GCA patients were polymyalgia rheumatica in 19 patients, headache in 4 patients, arthralgia, optic neuritis, maxillary sinusitis, trigeminal neuralgia and bronchial carcinoma, each in one case. In the GCA group 58% of patients were female compared to 72% in the non GCA group. The mean age at presentation was 73±8 years and 69±13 years, respectively. GCA patients presented significantly more often with jaw claudication ( $p<0.004$ ), scalp tenderness ( $p=0.002$ ), and a prominent TA ( $p=0.019$ ). Furthermore, C-reactive protein (CRP) and ESR were higher in the GCA, than in the non GCA patients ( $p=0.002$  respectively  $p=0.024$ ). We found no significant differences between groups with respect to vision impairment, headache, polymyalgia or weight loss. From a total of 18 TA biopsies performed in the GCA patients, 13 revealed vasculitic changes, whereas none of the 5 biopsies performed in the non GCA patients did so. The median number of days [IQR] on corticosteroids before US and biopsy was similar for both groups (Table I). The overall interobserver agreement for ‘vasculitis’ versus ‘no vasculitis’ was very high with a Krippendorff’s alpha of 0.92. Detailed segmental analysis revealed best agreement in the frontal branch

with an  $\alpha=0.92$  (0.89 right and 0.89 left separately), with slightly weaker agreement in the trunk with an  $\alpha=0.88$  (0.78 right, 0.86 left separately) and parietal branch with  $\alpha=0.73$  (0.82 right, 0.78 left separately). An overall discrepancy in the grading occurred in one patient only. The findings were judged as inconclusive by the vascular specialist and 'vasculitis' by the rheumatologist. The calculated diagnostic sensitivity for the final diagnosis of GCA was 75% for vascular specialists and 79% for the rheumatologist, and specificity was 100% for both.

The compression sonography was performed quickly, with a mean sonographic examination time (start to definite decision) for the rheumatologist of  $6.4 \pm 2.1$  minutes as determined in a subset of 30 study subjects.

### Discussion

The novel compression sign to detect vasculitic changes in the temporal artery is known to perform well in the hand of trained vascular specialists with reported specificity and positive predictive values of 100% (6). The current data now demonstrate that the TA com-

pression sign can be assessed with an equal diagnostic accuracy even without extensive expertise in vascular US. Interestingly the best interobserver agreement was found in the frontal branch, despite the fact that the parietal branch runs more favourably for compression (the trunk and the frontal branch are more "hidden" by bony structures) and is therefore the easiest to examine. Nevertheless a positive sign at any location can contribute to the diagnosis of the disease, independent of which and how many segments are affected.

The current study is limited by the relatively modest number of sixty patients. However the results are a strong indication that a short training of an unexperienced examiner can result in a reliable application of the method, even without specific experience in vascular ultrasound. In addition, the excellent specificity and positive predictive value reported earlier was repeated, which highlights the validity of the method in the diagnosis of GCA.

The simplicity of the technique may thus allow a more widespread, yet reliable use of US to rapidly substantiate the diagnosis of GCA.

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