



The role of colour doppler ultrasonography of facial and occipital arteries in patients with giant cell arteritis: A prospective study



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ABSTRACT

Objective: Colour Doppler Sonography (CDS) in giant cell arteritis (GCA) allows the study of involvement of cranial arteries other than the temporal arteries, which are inconvenient to biopsy, such as the facial (FaA), and occipital (OcA) arteries. We aimed to estimate the frequency of the FaA, and OcA involvement in GCA; and to explore the clinical characteristics of these subgroups of patients.

Methods: From 1 January 2014 to 31 December 2016 we prospectively performed a CDS of the FaA, and OcA in addition to the temporal (TA), and the extracranial supra-aortic arteries in all newly diagnosed patients suspected of having GCA. All the arteries were evaluated in two planes for the highly specific *halo* sign.

Results: During the 36-month observation period we performed a CDS of the cranial and extra-cranial arteries in 93 GCA patients. We observed the *halo* sign on the FaA, and OcA in 38 (40.9%), and 29 (31.2%) cases, respectively. The FaA, or OcA were affected in 4/22 (18.2%) patients with a negative TA CDS. FaA involvement significantly correlated with jaw claudication and with severe visual manifestations, including permanent visual loss.

Conclusions: A fifth of patients with a negative CDS of the TAs had signs of vasculitis on the CDS of the FaA, or OcA. The addition of FaA and OcA CDS to the routine CDS of the TAs could identify 4.3% more patients and thus further improve the sensitivity of the CDS in the suspected GCA.

1. Introduction

Giant cell arteritis (GCA) is the most common systemic vasculitis in Western countries with an annual incidence of 1.6–32.8 cases per 10⁵ adults over the age of 50 years. [1,2] Due to severe ischemic complications like permanent visual loss, GCA represents a medical emergency. The diagnostics have improved a lot over the past few years. The fast-track clinical pathways and imaging modalities like Colour Doppler sonography (CDS), computed tomography angiography (CTA), and positron emission tomography (PET) have greatly improved patients' odds for early disease recognition and contributed to a better long-term outcome. The diagnostic value of the temporal artery (TA) CDS, proven in several studies, is commonly used in daily practice. [3] A recent multicentre study found that the TA CDS is more sensitive than the TA biopsy (TAB). [4] As a systemic vasculitis of large-, and medium-sized arteries, the GCA can affect cranial arteries other than the TAs such as facial arteries (FaA), or occipital arteries (OcA). The FaA, and OcA are easily accessible to ultrasonographic examination, but inconvenient to biopsy. Nevertheless, data on the systematic CDS evaluation of the FaA or OcA are scarce. The involvement of the OcA was previously assessed

by Pfaenhauer et al. in patients with an occipital and nuchal pain suspected of having GCA. [5] A *halo* sign, stenosis, or an occlusion of the OcA were present in 63% of GCA patients. Schmidt et al., evaluated the peripheral arteries in 33 GCA patients and found an involvement of the FaA, and OcA in 12%, and 9%, respectively. [6] Aside from these two CDS studies, there are case reports of OcA involvement detected by the PET, and histologically proven GCA of the FaA. [7,8]

The usefulness of routinely evaluating the FaA, and OcA by CDS in daily clinical practice is unknown. Our primary goal was to study the involvement of the FaA, and OcA in newly diagnosed GCA cases using CDS, and the secondary goal was to explore how the involvement of these two arteries influenced the clinical characteristics of the patients.

2. Materials and methods

2.1. Setting

This prospective, observational study was performed at author's department, which is a part of an integrated teaching hospital serving approximately 1,000,000 adult residents on the tertiary level, and is the

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Table 1
Characteristics of GCA patients as a group and of GCA subgroups according to the CDS of temporal, facial and occipital arteries.

| | A All patients | A1 Temporal artery halo | B Facial artery halo | | p ^s | C Occipital artery halo | | |
|-----------------------------|-------------------|----------------------------|-------------------------|------------------|----------------|----------------------------|------------------|----------------|
| | | | yes | no | | yes | no | p ^s |
| | | | | | | | | |
| Number of patients | 93 | 71 | 38 | 55 | | 29 | 64 | |
| Female (%) | 66.7 | 60.6 | 60.5 | 70.9 | ns | 62.1 | 68.8 | ns |
| Age (years) [#] | 73.5 (66.1–79.1) | 76.5 (67.6–79.6) | 78.1 (74.7–81.3) | 67.8 (64.1–76.1) | ns | 76.0 (72.2–79.3) | 69.9 (64.5–79.0) | ns |
| General symptoms (%) | 81.7 | 81.7 | 81.6 | 83.6 | ns | 75.9 | 84.4 | ns |
| Polymyalgia rheumatica (%) | 18.3 | 19.7 | 15.8 | 20.0 | ns | 13.8 | 20.3 | ns |
| New headache (%) | 67.7 | 76.1 | 89.5 | 52.7 | 0.002 | 82.8 | 60.9 | ns |
| Occipital headache (%) | 32.3 | 33.8 | 42.1 | 25.5% | ns | 37.9 | 29.7 | ns |
| Scalp tenderness (%) | 28.0 | 32.4 | 39.5 | 20.0% | ns | 41.4 | 21.9 | ns |
| Jaw claudication (%) | 45.2 | 53.5 | 71.1 | 27.3 | < 0.001 | 69.0 | 34.4 | 0.028 |
| Visual disturbances (%) | 21.5 | 28.2 | 34.2 | 12.7 | ns | 20.7 | 21.9 | ns |
| Permanent visual loss (%) | 10.8 | 14.1 | 23.7 | 1.8 | 0.004 | 10.3 | 10.9 | ns |
| Clinically abnormal TAs (%) | 55.9 | 70.4 | 78.9 | 40.0 | 0.002 | 89.7 | 40.6 | < 0.001 |
| Dry cough (%) | 25.8 | 25.4 | 23.7 | 29.1 | ns | 27.6 | 26.6 | ns |
| Stroke (%) | 5.1 | 4.2 | 5.3 | 1.8 | ns | 6.9 | 1.6 | ns |
| TAB positive (%) | 82.1 | 85.0 | 84.4 | 79.4 | ns | 87.5 | 78.6 | ns |
| TA CDS positive (%) | 76.3 | | 92.1 | 67.3 | 0.019 | 93.1 | 68.8 | ns |
| LVV CDS positive (%) | 45.2 | 36.6 | 39.5 | 50.9 | ns | 27.6 | 54.7 | ns |
| ESR (mm/h) [#] | 80 (61–109) | 80 (56–110) | 72 (62–94) | 86 (59–115) | ns | 83 (65–108) | 80 (55–109) | ns |
| CRP (mg/l) [#] | 73 (45–126) | 73 (46–129) | 72 (49–118) | 76 (35–132) | ns | 105 (55–134) | 70 (38–118) | ns |

Legend: [#] median (IQR); CDS colour Doppler sonography; LVV large vessel vasculitis; TA temporal artery, TAB temporal artery biopsy; ESR erythrocyte sedimentation rate; CRP C-reactive protein; ^sp value, adjusted using the Benjamini-Hochberg procedure.

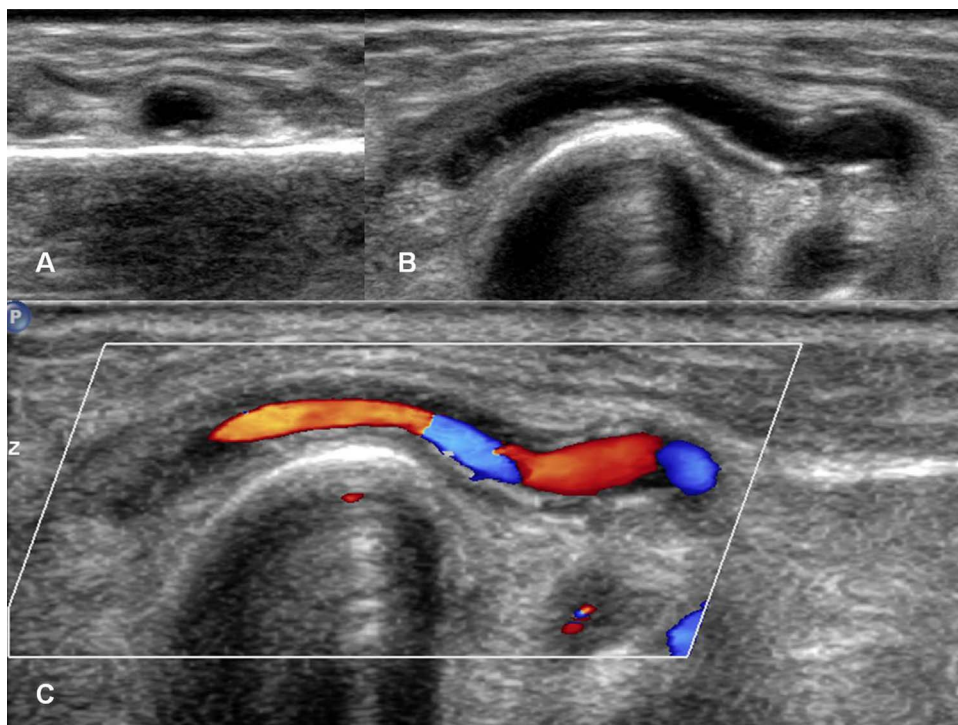


Fig. 1. A halo sign on a facial artery: B mode transverse (A) and longitudinal (B) scan; CDS of a facial artery (C).

only secondary level referral hospital in the region, serving approximately 500,000 adult residents. Consequently, most of the patients suspected of having GCA are referred to this early interventional clinic.

2.1.1. Patients and diagnosis

We included all consecutive patients with cranial GCA (c-GCA) or extracranial large vessel GCA (lvv-GCA), newly diagnosed at our fast-track GCA clinic between 1 January 2014 and 31 December 2016.

We classified patients as c-GCA based on the 1990 ACR classification criteria, and a positive TA biopsy (TAB) or the presence of the halo sign on the TA CDS. To diagnose lvv-GCA, we performed a CDS of the

supra-aortic large arteries (carotid, vertebral, subclavian, axillary and brachial arteries), or PET/computer tomography (PET/CT), or both.

2.1.2. Ultrasonographic evaluation of the facial and occipital arteries

A single, experienced ultrasonographer (AH) performed the CDS of the TAs, supra-aortic large arteries, the FaA, and OcA in all the patients prior to Table We used a Philips IU22 with a 5–17.5 MHz multi-frequency linear probe from January 2014 to August 2016 and a Philips Epiq 7 with a 5–18 MHz multi-frequency linear probe from September 2016 to December 2016. The adjustable settings of the ultrasonographic machines were uniform for all examinations. The focus position

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