Duplex Sonography of the Temporal and Occipital Artery in the Diagnosis of Temporal Arteritis. A Prospective Study

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ABSTRACT. Objective. Evaluation of the diagnostic contribution of color coded duplex sonography (CCDS) of the superficial temporal (STA) and the occipital artery (OCCA) in biopsy-controlled patients suspected of having temporal arteritis (TA).

Methods. Prospective study in 67 patients suspected of having TA who underwent CCDS of the STA in all cases and the occipital arteries if involvement of the OCCA was suspected clinically. The final diagnosis, based on biopsy results in 67 cases and standard criteria, were compared to the ultrasonographic findings to determine their diagnostic contribution.

Results. TA was diagnosed in 40 patients, other diseases in 27 patients. In the STA periarterial hypoechogenic tissue, the so-called halo, halo and stenoses, and occlusions were found in 83% of TA patients and 11% of patients with other diseases. In the OCCA, these abnormalities were found in 65% of TA patients and in no patient with other diseases. Taking both STA and OCCA together, halo, stenosis, and widespread abnormalities were found in patients with TA, but not in patients with other diseases.

Conclusion. CCDS of the STA and OCCA clearly contributes to the diagnosis of TA, with a high rate of perivascular hypoechogenic abnormalities (so-called halos) and stenosis and a low rate of these abnormalities in the control patients. However, CCDS cannot differentiate between inflammatory and degenerative artery disease and has spatial resolution limitations. (J Rheumatol 2003;30:2177–81)

Key Indexing Terms: TEMPORAL ARTERITIS ULTRASONOGRAPHY HALO

Temporal arteritis (TA) is a systemic vasculitis with a particular affinity to the superficial temporal artery (STA) and the extraocular parts of the central retinal, posterior ciliary, and ophthalmic arteries. Less common is involvement of other branches of the external carotid arteries, the internal carotid, the vertebral and coronary arteries, and the aorta1. Rapidly progressing visual disturbances and even bilateral blindness are serious complications of the disease2.

Currently, histological diagnosis of temporal artery biopsy is the diagnostic gold standard. Previously, arteriography3 and cw-Doppler sonography4-10 and recently color coded duplex sonography (CCDS)11-16 have been used for the direct demonstration of arteritis of the ophthalmic and supratrochlear arteries, the superficial temporal, and the carotid and vertebral arteries, but also to exclude collateral flow via the superficial temporal artery in severe stenosis of the internal carotid artery before biopsy. Ho, et al17 were able to observe occlusions and stenoses of the orbital arteries by means of CCDS.

CCDS of the STA was able to reveal not only stenosis and occlusion of the artery but also perivascular hypoechogenic areas, so-called halos, that were considered to represent inflammatory edema of the arterial wall14. However, there are discrepancies concerning sensitivity and specificity among the studies11-16.

In this prospective study, we evaluated the diagnostic contribution of CCDS of the STA and occipital artery (OCCA) in patients in whom the diagnosis of TA was suspected clinically.

MATERIALS AND METHODS

Patients. Over a period of 51 months, we included into this prospective study 67 (51 women, 16 men, median age 69 yrs) of 115 patients for whom the diagnosis of TA was suspected clinically. The STA was examined in all cases and the occipital arteries if involvement of the OCCA was suspected clinically. The final diagnosis, based on biopsy results in 67 cases and standard criteria, were compared to the ultrasonographic findings to determine their diagnostic contribution.

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Patients with TA. The final diagnosis was TA in 40 patients. Thirty-two patients had steroid treatment before CCDS (median 2 days, range 1–7 days). On examination, no temporal artery abnormalities were found in 3 patients; halos could be observed by CCDS in 2 of them. A unilateral biopsy was taken from the OCCA in one patient and from the STA in 39 patients. Corticosteroid treatment was highly effective in all patients. Neuroophthalmological complications were found in 12 patients (30%) [9 anterior ischemic optic neuropathy (one of them bilateral), one central retinal artery occlusion, one amaurosis fugax, one ocuulmotor nerve palsy]. Followup examinations were done in 5 patients undergoing steroid treatment.

Patients with other diagnoses. Other diagnoses were given to 27 patients: PMR (n = 10), autoimmune diseases [10, including Wegener’s disease 2, polyarteritis nodosa one, systemic lupus erythematosus (SLE) one, rheumatoid arthritis (RA) 3, other 3], degenerative vascular diseases (4), and other diseases (3). Unilateral biopsies from the STA were taken in 27 patients. Six patients (22%) had neuroophthalmological complications [5 anterior ischemic optic neuropathy (one bilateral), one central retinal artery occlusion]. Tenderness related to the temporal arteries and to the frontotemporal scalp was found in 13 patients.

Ultrasonographic investigation. CCDS of the temporal and occipital artery. All examinations were done before biopsy by one investigator (K.P.) using a Siemens Sonoline Elegra ultrasound system (Siemens, Erlangen, Germany). A linear array high resolution 7.5 MHz transducer (Siemens L40, 5, 1–9 MHz) was used. Standard parameters for the B-mode were emission frequency 9 MHz, dynamic range 50 dB, gain 70 dB. Axial resolution was 0.45 and lateral resolution 0.48 mm.

For Doppler sonography a wall filter of 50 Hz and a pulse repetition frequency of 3125 Hz were used. The arteries were examined as extensively as possible in transverse and longitudinal sections. A complete examination of the STA and OCCA took 20–30 min.

Absent flow in the STA was considered to be an occlusion of the artery; segmental increase of blood flow velocity with wave forms indicating turbulence was classified as stenosis if it was not attributable to other abnormalities like kinking of the artery. Periarterial hypoechogenic areas were classified as “halo” according to suggestions from Schmidt, et al.14. Widespread TA was assumed when the main trunk and both rami of the STA and proximal and distal segments of the OCCA were abnormal.

Anatomy. The trunk of the STA can easily be found when the artery passes through the parotid gland anterior to the tragus. From there the artery can be followed to the bifurcation and to the frontal and parietal rami. Both rami are embedded between the 2 layers of the fascia temporalis, which can be visualized clearly in the B-mode as 2 parallel hyperechogenic lines. The ramii are embedded between the 2 layers of the fascia temporalis, which can be followed to the bifurcation and to the frontal and parietal rami. Both rami are embedded between the 2 layers of the fascia temporalis, which can be visualized clearly in the B-mode as 2 parallel hyperechogenic lines. The ramii are embedded between the 2 layers of the fascia temporalis, which can be visualized clearly in the B-mode as 2 parallel hyperechogenic lines. The ramii are embedded between the 2 layers of the fascia temporalis, which can be visualized clearly in the B-mode as 2 parallel hyperechogenic lines.
Table 1. Summary of the final diagnoses, histopathological findings, and CCDS findings of the occipital arteries (OCCA) and superficial temporal arteries (STA) in 67 biopsy-controlled patients.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Histopathology</th>
<th>CCDS, STA</th>
<th>CCDS, OCCA</th>
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<tbody>
<tr>
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<td>N = 40</td>
<td>N = 40</td>
<td>N = 26</td>
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<tr>
<td></td>
<td>Positive, 33</td>
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<tr>
<td></td>
<td>Negative, 7</td>
<td>Halo, 24</td>
<td>Halo, 13</td>
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<td></td>
<td>Halo stenosis, 7</td>
<td>Halo stenosis, 3</td>
<td>Halo stenosis, 3</td>
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<td></td>
<td>Occlusion, 2</td>
<td>Occlusion, 1</td>
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<td>Bilateral, 24</td>
<td>Bilateral, 9</td>
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<td></td>
<td>CCDS, STA and OCCA</td>
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<td>3 arteries, 6</td>
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<td>2 arteries, 5</td>
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<td></td>
<td>Normal, 24</td>
<td>N = 12</td>
<td>N = 12</td>
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<tr>
<td></td>
<td>Halo, 3</td>
<td>Normal, 12</td>
<td>Normal, 12</td>
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</table>

Figure 1. Color coded duplex sonography of the superficial temporal artery in a 75-year-old healthy woman (C, D) and in a 73-year-old patient with temporal arteritis (A, B). CCDS longitudinal sections from the anterior ramus of the superficial temporal artery show normal perivascular tissue (a) and hypoechogenic perivascular tissue corresponding to active giant cell arteritis. Corresponding pulsed-wave Doppler spectra from the superficial temporal arteries (b, d) show normal results.
found a high rate of abnormalities in 83% of our 40 patients who received a final diagnosis of TA, and a low rate of 11% in patients with other diseases, all examined for suspected TA. Thus the sensitivity of CCDS in our study is as high as that of histology. Previously, Doppler sonography has been used to investigate hemodynamic changes due to stenosis or occlusion in the STA and OCCA in TA patients. The main advantage of CCDS is the additional detection of perivascular hypoechogenic inflammatory tissue changes, which clearly were much more frequent in our TA patients compared to stenosis and occlusion (60% vs 23%). Pathological proof of TA may be difficult in some patients, mainly because of the segmental characteristic of the disease. In contrast to biopsy, CCDS allows full-length examination of the STA and other cranial arteries and monitoring of disease activity in response to therapy over time. The main problems of CCDS are the restrictions due to limited spatial resolution and the lack of specificity for the differentiation of TA from other vascular diseases. Both are important advantages of histological analysis of biopsy specimens.

Spatial resolution of CCDS may not be sufficient in some cases: the different extent of arterial wall inflammation has been recognized and described by Horton and Jennings. The main advantage of CCDS is the additional detection of perivascular hypoechogenic inflammatory tissue changes, which clearly were much more frequent in our TA patients compared to stenosis and occlusion (60% vs 23%). Pathological proof of TA may be difficult in some patients, mainly because of the segmental characteristic of the disease. In contrast to biopsy, CCDS allows full-length examination of the STA and other cranial arteries and monitoring of disease activity in response to therapy over time.

Confounding factors such as age, body build, and vessel tortuosity may lead to false positive halos. In our study, only circumferential halos in the intima and media layer, leaving the arterial lumen unchanged. There may be different types of inflammation that also affect echogenicity of the inflamed arterial wall. Reinecke and Kuwabara found different histological types of inflammation, ranging from severe occlusive necrotizing panarteritis with involvement of the adventitia to more restricted manifestations in the intima and media layer, leaving the arterial lumen unchanged.

Important factors affecting diagnostic sensitivity and specificity as well include patient selection, diagnostic criteria, examination technique, and ultrasound equipment, as follows.

Patient selection. The numbers of biopsy-controlled patients in the different groups studied vary from 7 to 86. As well, the selection of patients was very different for TA and control patients. Neuroophthalmological complications in TA patients as a possible indicator for the severity of illness were found in 100%, 43%, and 30% in our study. While most studies describe patients in active stages of TA, one study also included patients in more chronic stages of the disease undergoing steroid treatment. False positive halos and stenoses and occlusions are mainly caused by arteriosclerosis, arteritis due to other diseases, and anatomical abnormalities such as accompanying veins and other hypoechogenic tissue. Patient groups used as controls were highly selected by including only patients suspected to have TA (present study and others), patients with mainly arteriosclerotic eye disease, PMR, and selected rheumatic disease. Of particular interest is the 80% rate of stenoses/occlusions in the study by Schmidt, et al, which corresponds to previous studies using cw-Doppler sonography. In contrast to this are the 22% rate in our study and missing stenoses/occlusions in other studies. Most probably this is due to examination technique and diagnostic criteria and not to patient selection.

Diagnostic criteria. Diagnostic criteria may affect sensitivity and specificity: adhering to criteria like halo thickness of 1 mm and more and widespread disease involving more than 2 arteries may reduce sensitivity by excluding TA patients with less severe and more focal disease. Selecting only circumferential halos may improve specificity by excluding accompanying veins and other hypoechogenic anatomical abnormalities, but again may exclude TA patients with eccentric involvement of the arterial walls.

Examination technique. False negative findings may result from overlooking halos by using color intensity that is too strong (color bleeding) so that it covers the inflamed arterial wall. Occlusion of one ramus of the STA can be mistaken as an anatomical variant. False positive findings may be due to artificial stenosis or occlusion caused by too firm pressure of the transducer on the underlying artery.

Ultrasound equipment of adequate resolution was used in all studies, except one reporting the lowest sensitivity.
Followup examinations of the STA, OCCA, and supratrochlear artery using cw-Doppler sonography\textsuperscript{4,6,7,10} and CCDS\textsuperscript{14} have demonstrated the disappearance of hemodynamic abnormalities under adequate steroid treatment over several weeks to months, and this could be observed in our patients even in completely occluded STA. This is of great importance for the differentiation of TA from arteriosclerosis.

In summary, CCDS of the STA and OCCA clearly contributes to the diagnoses of TA, with a high rate of perivascular hypoechoic abnormalities, so-called halos, and stenoses/occlusions, a low rate of these abnormalities in the control patients. The detection of a halo in combination with stenosis/occlusion, widespread bilateral abnormalities of the STA, and involvement of more than 2 arteries were specific findings occurring only in patients with TA. CCDS can show abnormalities even in patients with negative biopsy results and negative findings on physical examination, and helps in the selection of ideal STA/OCCA segments for biopsy. The main advantage of CCDS is the full-length visualization of the STA. However, CCDS cannot differentiate precisely between inflammatory and degenerative artery disease and has limitations in spatial resolution. In contrast to arteriosclerosis, CCDS abnormalities resolve within a few weeks in patients with TA undergoing steroid treatment. Before CCDS will be able to replace biopsy in clinical practice, further experience has to be gathered in larger cohorts of patients using our recommended diagnostic criteria.

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REFERENCES