Vertebral Artery Halo Sign in Patients With Stroke
A Key Clue for the Prompt Diagnosis of Giant Cell Arteritis

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Background and Purpose—The purpose of this study was to describe typical ultrasonographic findings of vertebral arteries in patients affected by giant cell arteritis.

Methods—Color duplex sonography was used to assess the cervical arteries within 24 hours from stroke onset in 1237 patients. Vertebral arteritis was considered if concentric, homogeneous, and smooth hypoechoic mural thickening (the so-called halo sign) was present in at least 1 cervical segment of the vertebral artery. If the patient showed such findings, an ultrasound examination of the temporal artery was also performed. Patients with probable giant cell arteritis were treated with high-dose intravenous methylprednisolone in association with antiplatelet therapy. Temporal artery biopsy was carried out by a vascular surgeon in the site targeted by the ultrasonographer.

Results—Five patients were diagnosed as having vertebral arteritis according to ultrasound criteria. All of them had initial neurological deficit due to infarctions affecting the vertebrobasilar territory. One patient died due to aspiration pneumonia, whereas the others were independent at discharge. All patients had a positive biopsy for giant cell arteritis.

Conclusions—Vertebral artery involvement in giant cell arteritis may be suspected by color duplex sonography. This fact would allow a prompt diagnosis and treatment of this otherwise fatal disease. Because duplex ultrasonography is a usual test performed on patients with stroke, the recognition of the halo sign in vertebral arteries may be of crucial interest in selected cases. (Stroke. 2011;42:3287-3290.)

Key Words: acute stroke ■ etiology ■ imaging ■ neurosonology ■ steroids ■ ultrasound ■ vasculitis ■ vertebral artery ultrasound

Giant cell arteritis (GCA) is an immune-mediated vasculitis of large- and medium-sized vessels affecting especially arteries arising from the aorta, in particular superficial temporal artery.1 Stroke happens in just 3% to 7% of the cases, but it is the leading cause of death in patients with GCA.2 Clinical and pathological findings suggest that cerebral ischemic events in patients with GCA are due to the involvement of extradural vertebral and carotid arteries rather than to intracranial vasculitis.3 Overall, GCA is a very rare cause of stroke, being diagnosed in only 0.15% of brain infarctions but to identify them properly is important because these patients have a high mortality rate and a specific and potentially curative treatment exists.

Temporary artery (TA) biopsy is still the gold standard for GCA diagnosis.4 Nevertheless, in recent years, color duplex ultrasonography has been proposed as a useful noninvasive diagnostic tool as a screening test in case of GCA suspicion.5,6 These studies have shown that a dark area (the so-called halo sign) around the TA lumen is a quite common feature of GCA and is indicative of vasculitic mural edema.

The halo sign is not exclusive of TA and has been observed in other vessels affected by GCA like vertebral, occipital, or axillary arteries. However, ultrasound diagnosis in vessels other than the TA is more difficult and positive findings are less frequent.7

We report 5 cases attended in our neurology department in the last 2 years in which vertebral artery (VA) halo sign was the clue for the early diagnosis of GCA as the primary cause of brain infarction.

Patients and Methods

From March 2008 to January 2010, all patients admitted to our neurology department due to acute ischemic stroke were prospectively studied. None of these patients had a prior diagnosis of GCA. We recorded in all cases demographic characteristics, vascular risk factors, clinical and neurological examination, and results of laboratory tests, chest radiography, electrocardiography monitoring in the stroke unit, cranial CT, and/or MRI.

We used transcranial Doppler and cervical and cranial duplex as the initial test to evaluate the intra- and extracranial arteries; this evaluation was always performed within 24 hours after stroke onset. Color duplex sonography was done by the same examiners (J.G., O.A., T.S.) who were aware of the clinical characteristics of patients. A sonography device (Esaote MyLab 70) with a multifrequency linear array transducer (3 to 9 MHz) was used to assess vessels morphology. The pars transversaria (V2) and the prevertebral (V1)
segment of the VA were imaged on both sides. Diameter, flow, direction, and flow velocity within the V2 segment were routinely documented. We considered vertebral arteritis if homogeneous, hypoechoic, concentric, or circumferential mural thickening was present in at least 1 cervical segment of the artery. If the patient showed such findings in the VA, an ultrasound examination of the TA was then performed using a 6- to 18-MHz multifrequency linear array transducer. This artery was depicted distally as far as possible under local anesthetic by a vascular surgeon at the site targeted by the ultrasonographer according to the localization of a presumed area of stenosis or occlusion. VA signal appeared in at least 1 segment of the artery regardless of the direction, and flow velocity within the V2 segment were routinely documented. When GCA was suspected, intravenous methylprednisolone (500 mg/day for 5 days) and aspirin were immediately started. In our cases, the clinical response to corticosteroids was dramatic, except for 1 patient who died due to aspiration pneumonia. In survivors, oral aspirin and steroids were prescribed at discharge.

Patients with probable GCA according to ultrasound examination were treated with intravenous prednisolone in association with antiplatelet therapy. In these patients, TA biopsy was carried out under local anesthetic by a vascular surgeon at the site targeted by the ultrasonographer according to the localization of a presumed arteriopathy (halo sign presence). Degree of clinical disability at discharge from the hospital was evaluated by means of modified Rankin Scale score. We repeated ultrasonography 3 months after initiation of corticosteroid therapy.

Results
A total of 1237 consecutive patients with stroke were prospectively evaluated. Five of them (0.4%) were diagnosed of vertebral arteritis due to GCA according to the ultrasound criteria previously described. The main clinical data of these 5 patients are presented in the Table.

The neurological clinical picture of our patients reflected a predominant involvement of the vertebrobasilar vascular territory and neuroimaging revealed ischemic infarctions in the posterior brain circulation. All patients showed a halo sign in the V2 segment of VA (Figure 1A) associated with a variable degree of flow stenosis and exhibited concentric hypoechoic mural thickening in the TA (Figures 2A and 2C; Table). Histological examination confirmed the diagnosis in all cases. When GCA was suspected, intravenous methylprednisolone (500 mg/day for 5 days) and aspirin were immediately started. In our cases, the clinical response to corticosteroids was dramatic, except for 1 patient who died due to aspiration pneumonia. In survivors, oral aspirin and steroids were prescribed at discharge.

In the ultrasonographic follow-up, the TA dark halo had disappeared with marked flow improvement (Figures 2B and 2D). However, VA wall thickening persisted with a narrow color signal flowing in the arterial center (Figure 1B), although we also observed hemodynamic improvement with a lesser degree of flow stenosis in the VA (Figure 1D–E).

Discussion
We discuss 5 patients with posterior circulation stroke in which the characteristics of ultrasound imaging of VA allowed diagnosis of GCA as the cause of brain infarction.

Early-onset headache, jaw claudication, or anterior ischemic optic neuropathy in a patient aged >50 years with elevated inflammatory markers may obviously raise suspicion of GCA.3,8 However, in our patients, an early clinical diagnosis of GCA was challenging because stroke was the main clinical manifestation and the classical symptoms of the disease were scarce in almost all cases. However, cervical ultrasound examination and recognition of the VA halo sign

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**Table. Clinical Presentation, Vascular Risk Factors, ESR Values, Ultrasonography Findings, Brain MRI Findings, and Outcome at Discharge of Patients Diagnosed With Vertebral Arteritis**

<table>
<thead>
<tr>
<th>Case Number</th>
<th>Age, y/Gender</th>
<th>Clinical Presentation</th>
<th>Vascular Risk Factors</th>
<th>ESR, mm/h</th>
<th>Color Duplex Sonography of VA</th>
<th>Color Duplex Sonography of TA</th>
<th>Brain MRI</th>
<th>Outcome at Discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>79/female</td>
<td>Sudden loss of consciousness with right hemiparesis. Fever in the last week</td>
<td>Hypertension</td>
<td>60</td>
<td>Halo sign in right V2</td>
<td>Halo sign and segmentary stenosis in both TA</td>
<td>Both middle cerebellar peduncles acute infarctions</td>
<td>mRS 6</td>
</tr>
<tr>
<td>2</td>
<td>82/female</td>
<td>Right hemiataxia, dysarthria, and ophthalmoparesis Headache, fever, and muscle pains in the last month</td>
<td>Diabetes mellitus</td>
<td>118</td>
<td>Halo sign in both V2</td>
<td>Halo sign in left TA</td>
<td>Right cerebellar acute infarction</td>
<td>Survival</td>
</tr>
<tr>
<td>3</td>
<td>81/male</td>
<td>Dizziness, gait imbalance, and diplopia No other symptoms or signs in previous days</td>
<td>Hypertension</td>
<td>47</td>
<td>Halo sign in right V2 and stenosis in left V2 to V3</td>
<td>Halo sign in left TA and thrombosis of right TA</td>
<td>Left pons acute infarction</td>
<td>Survival</td>
</tr>
<tr>
<td>4</td>
<td>75/female</td>
<td>Dizziness, blurred vision, and gait imbalance No other symptoms or signs in previous days</td>
<td>Dyslipidemia</td>
<td>67</td>
<td>Halo sign in both V2</td>
<td>Halo sign in right TA</td>
<td>Bilateral cerebellar acute infarctions</td>
<td>Survival</td>
</tr>
<tr>
<td>5</td>
<td>79/male</td>
<td>Monocular blindness and later vertigo, diplopia, and gait ataxia Headache in the last 5 d</td>
<td>None</td>
<td>34</td>
<td>Halo sign and stenosis in both V2</td>
<td>Halo sign in both TA</td>
<td>Left cerebellar acute infarction</td>
<td>Survival</td>
</tr>
</tbody>
</table>

ESR indicates erythrocyte sedimentation rate; VA, vertebral artery; TA, temporal artery; V2, pars transversalis of vertebral artery; V3, atlas loop of vertebral artery; mRS, modified Rankin Scale; MRI, magnetic resonance imaging.

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suggested the diagnosis of GCA. In our cases, we also found a typical TA halo when VA halo was present in the early investigation of stroke etiology.

The identification of the VA halo sign is of great importance given that duplex ultrasonography of carotid and VAs, but not of TAs, is a common test performed in most patients who have a stroke. So, if we are able to recognize the VA halo sign, then we can also look for it in TAs, suspecting GCA in an appropriate clinical context. The prompt diagnosis of GCA as the primary cause of stroke in these patients can lead to early treatment with intravenous steroids, avoiding an otherwise probable fatal prognosis.

Figure 1. Longitudinal cervical Duplex ultrasonography of a patient with giant cell arteritis with involvement of vertebral arteries; before (A, C) and after (B, D) corticoid treatment. There is a long straight homogeneous hypoechoic halo (A) surrounding the vertebral artery, which is still present after treatment (B). This halo (vertebral inflammation) promotes a severe vessel stenosis (peak systolic velocity 218 cm/s; C) that is still present after corticoid treatment, although it exits a lower hemodynamic degree of stenosis (peak systolic velocity 75 cm/s; D).

Figure 2. Duplex ultrasonography of the temporal artery in axial (A–B) and longitudinal (C–D) projections of a patient with giant cell arteritis. A and C were obtained before corticoid treatment, showing a homogeneous hypoechoic area (halo) around the lumen of the artery. In B and D, it is shown how the temporal artery becomes normal after treatment.
Hypoechogenic mural thickening described in vertebral arteritis could be misdiagnosed as a wall hematoma caused by vessel wall dissection. In the latter case, an intramural hematoma may look hypoechoic with a residual channel of color flow, yielding a sonographic appearance similar to wall thickening due to transmural inflammation (halo sign). However, in the case of GCA, ultrasound examination commonly reveals a concentric hypoechoic halo around the lumen, whereas the hypoechoic area resulting from a wall hematoma is usually eccentric and crescent-shaped with a spiraling course. Moreover, in the case of vertebral arteritis, but not in vertebral dissection, temporal arteritis signs coexist.

Another potential pitfall for ultrasound may be the presence of hypoechogenic atherosclerotic plaques in the VA, although these are frequently short, irregular in shape, and eccentric. Bilateral artery involvement is also common both in vasculitis and atherosclerosis; however, although atherosclerosis classically involves V1 (prevertebral) or V4 (intracranial) segments, most of the lesions due to vasculitis affect V2 or V3 segments (atlas loop). This fact facilitates the diagnosis of vertebral arteritis because the V2 segment can be insolated in all cases, whereas the origin of the VA or the distal segments is hardly explored by ultrasound.

GCA of the VA is a rare cause of vertebrobasilar disease, but 1 with a serious prognosis. Most reported patients either died or had severe disability at discharge. However, Sutter et al reported 2 cases with favorable outcomes, highlighting the role of the ultrasound in early diagnosis of GCA. Although it has not been proved that prompt intervention with immunosuppressive treatment improves the outcome, in Sutter’s patients, like ours, early treatment with intravenous methylprednisolone was associated with a good recovery.

In summary, diagnosis of GCA may be challenging in patients with stroke if brain infarction is the only clinical manifestation of the disease. Given that ultrasonographic study of VAs is very common in patients with stroke, the recognition of the halo sign in these vessels is of paramount interest to achieve a proper and early diagnosis of GCA.

Disclosure
None.

References
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http://stroke.ahajournals.org/content/suppl/2011/09/22/STROKEAHA.111.625152.DC1
This MRI image showed a circumferential enhancement of the vertebral artery wall after gadolinium injection, suggesting an inflammatory origin of the artery thickening.