

Involvement of peripheral arteries in giant cell arteritis: A color Doppler sonography study

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Abstract

Objective

To investigate the involvement of arteries other than the temporal arteries in active giant cell arteritis using color Doppler sonography.

Methods

The occipital, facial, vertebral, carotid, subclavian, axillary, brachial, ulnar, radial, femoral, popliteal, posterior tibial, and dorsal pedal arteries, and the abdominal aorta of 33 consecutive patients with acute giant cell arteritis and 33 age- and sex-matched controls were investigated.

Results

In 10 patients (30%), but in none of the controls, a characteristic inflammatory mural thickening (halo) could be demonstrated in these arteries. The subclavian, external carotid, and/or facial arteries were involved in 4 patients, the occipital and/or axillary arteries in 3 patients, the brachial and/or ulnar arteries in 2 patients, and the common carotid, vertebral, popliteal, and/or radial arteries in 1 patient each. Two patients had symptomatic large vessel giant cell arteritis with arm claudication. The other patients were asymptomatic concerning the involved arteries. Furthermore the ulnar artery was occluded in 3 cases, the posterior tibial artery in 2 cases, and the dorsal pedal and the vertebral artery in 1 case each. No occlusions were found in the controls. Occlusion of the temporal arteries occurred more frequently in patients with peripheral artery involvement than in those without peripheral involvement (60% versus 26%). In most of the non-stenotic, small arteries the halo disappeared within 9 to 21 days. Mural thickening remained in large, stenotic arteries.

Conclusion

Peripheral artery involvement occurs more frequently in acute temporal arteritis than has been assumed up to now. Color Doppler sonography offers a new method to evaluate this peripheral involvement.

Key words

Temporal arteritis, giant cell arteritis, ultrasonography, color Doppler sonography, large vessel giant cell arteritis, polymyalgia rheumatica, carotid artery, brachial artery, subclavian artery, popliteal artery.

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Introduction

Giant cell (temporal) arteritis is a vasculitis that most commonly involves the temporal arteries. The American College of Rheumatology (ACR) requires three of the following five criteria to be met for its classification: age > 50 years, new onset of localized headache, temporal artery tenderness or decreased pulse, ESR > 50 mm/h, and histologic findings (1). Inflammation of the vasculature of the eye occurs less frequently, but is potentially disabling. Symptomatic involvement of other arteries has been described.

Color Doppler sonography represents a new, non-invasive method to diagnose temporal arteritis. We have described a characteristic dark (hypoechoic) circumferential wall thickening of the temporal arteries (halo) that occurs in the acute phase of the disease (2). It disappears with corticosteroid treatment. This phenomenon is due to an edema of the artery wall in active arteritis. Additionally, stenoses and occlusions of the temporal arteries are frequently detected in acute disease. The sensitivity and specificity of this imaging technique for the diagnosis and histologic findings are high (3, 4).

In the present study we used this method to examine the involvement of the peripheral arteries in consecutive patients with acute disease.

Patients and methods

Patients

All consecutive patients with acute, newly diagnosed giant cell arteritis presenting at the Medical Center for Rheumatology, Berlin-Buch between January 1, 1998 and March 1, 2001 were investigated. A total of 33 patients were investigated: 23 females and 10 males with a mean age of 73 years (range 58 - 88). Fifteen patients met 5 of the ACR classification criteria for temporal arteritis, 12 patients met 4 criteria, 4 patients met 3 criteria, and 2 patients met 2 criteria. One of the patients who only met 2 criteria showed positive findings on temporal artery histology. The other had large vessel giant cell arteritis (5) based on the clinical investigation and findings of angiography and sonography. The clinical diagnosis of temporal

arteritis was confirmed by at least 2 rheumatologists in all patients.

Sixteen patients (49%) complained of jaw claudication, and 11 patients (33%) had symptoms of polymyalgia rheumatica. All patients were investigated by an ophthalmologist. Ocular involvement was diagnosed in 15 patients (45%): 7 with anterior ischemic optic neuropathy, 3 with amaurosis fugax, 2 with anterior segment ischemic lesion, 1 with central retinal artery occlusion, 1 with abducens paralysis and amaurosis fugax, and 1 with cotton-wool exudates.

Twenty patients had not received corticosteroids before undergoing sonography, 5 patients had received corticosteroids for less than 24 hours beforehand, 1 had received corticosteroids for 2 days, 3 for 3 days, 2 for 4 days, and 1 for 5 days. One patient had already been taking 5 mg prednisolone for a much longer time due to a clinical diagnosis of polymyalgia rheumatica. Despite this therapy she developed acute temporal arteritis with positive findings on both temporal artery histology and sonography (including halo and stenoses), as well as amaurosis fugax.

The average ESR was 71 mm/h (range 8-110). One patient with an ESR of 8 mm/h had stenoses documented by sonography and positive histologic findings. Two patients with an ESR of 38 mm/h had both a halo seen on sonography and positive histologic findings.

The results of sonography of the temporal arteries and temporal artery histology are shown in Table I. In all patients color duplex sonography of the temporal arteries was performed. Twenty-six of the 33 patients had a temporal artery biopsy. The biopsy was unilateral in 5 patients and bilateral in 21 patients. All patients with unilateral biopsy had a positive result. Histology was positive in 18 of the 21 patients with bilateral biopsy. All patients who did not undergo temporal artery biopsy or who had a negative biopsy showed positive sonographic findings delineating a halo as well as stenoses or occlusions of the temporal arteries. The only exception was one of the 2 patients with the clinical appearance of large

Table I. Results of sonography of the temporal arteries and temporal artery histology in the patients with giant cell arteritis.

Sonography	Histology positive	Histology not done	Histology negative	Total
Halo + stenosis + occlusion	2	2	0	4
Halo + stenosis	8	3	3	14
Halo + occlusion	6	1	0	7
Halo	2	0	0	2
Stenosis + occlusion	1	0	0	1
Stenosis	4	0	0	4
Normal	0	1*	0	1
Total	23	7	3	33

*Patient with the typical clinical, sonographic, and angiographic findings of large vessel giant cell arteritis.

vessel giant cell arteritis. Angiography was performed in the 2 patients with symptomatic peripheral artery involvement; it showed typical changes of vasculitis at the same arteries that had been identified by sonography before.

Six patients had a history of smoking, 5 had a history of diabetes mellitus, 8 had a history of hypercholesterolemia, 11 had a history of arterial hypertension, 1 had a history of myocardial infarction, 2 had a history of occlusive arterial disease of the lower limbs, and none had a history of stroke.

Controls

Thirty-three persons without clinical signs of temporal arteritis or polymyalgia rheumatica were matched for age and sex to the case patients. There was no significant difference in the risk factors for arteriosclerotic complications compared to the 33 patients with temporal arteritis. Five of the controls had a history of smoking, 6 had a history of diabetes mellitus, 8 had a history of hypercholesterolemia, 12 had a history of arterial hypertension, 2 had a history of myocardial infarction, 1 had a history of occlusive arterial disease of the lower limbs, and 1 had a history of stroke.

Ultrasonographic evaluation

Simultaneous color Doppler and duplex sonography was performed with a linear transducer (L 10-5; 10 to 5 MHz, length of the probe, 38 mm; ATL Ultramark 9 HDI, Advanced Technology Laboratories, Bothell, WA, USA). For the abdominal aorta a 3.5 MHz curved

array transducer of the ATL Ultramark 9 HDI was used.

We examined following arteries as completely as possible in both a longitudinal and a transverse plane: the occipital, facial, extracranial vertebral, common carotid, extracranial internal carotid, external carotid, subclavian, axillary, brachial, radial, ulnar, femoral, popliteal, posterior tibial, and dorsal pedal arteries, and the abdominal aorta. We looked for arteriosclerotic lesions, stenoses, occlusions, aneurysms, and inflammatory artery wall pathologies with or without stenoses. Inflammatory changes are characterized by a dark (hypoechoic), homogenous wall thickening, as has already been described at the temporal arteries in temporal arteritis (2) and at the subclavian, axillary, and brachial arteries in large vessel giant cell arteritis (6). The inflammatory changes are different from arteriosclerotic changes of the vessel wall, which are brighter (mid-echoic or hyperechoic), have an irregular surface, and frequently coexist with calcifications.

Furthermore, both supratrochlear arteries were investigated by cw-Doppler sonography to determine perfusion and to exclude collateral flow from the external to the internal carotid artery before biopsy in the patients with temporal arteritis. A Kranzbühler Logidop 1 (Kranzbühler, Solingen, Germany) with an 8 MHz transducer (KMS 9186) was used for this investigation.

The two experienced physicians who carried out the ultrasonography examinations were blinded to the clinical and

histologic diagnoses of the patients. Nineteen of the patients with temporal arteritis were investigated by both investigators who were not aware of the results of the other investigator. If the results of both investigators disagreed, they examined the results together and reached a consensus on the findings. One investigation takes 60 to 90 minutes.

If sonography detected a halo at peripheral arteries, follow-up investigations were performed in 8 of 10 patients 9 to 21 days after the first investigation to evaluate if this phenomenon persisted. The two patients with classic large vessel giant cell arteritis were followed for 6 and 24 months, respectively.

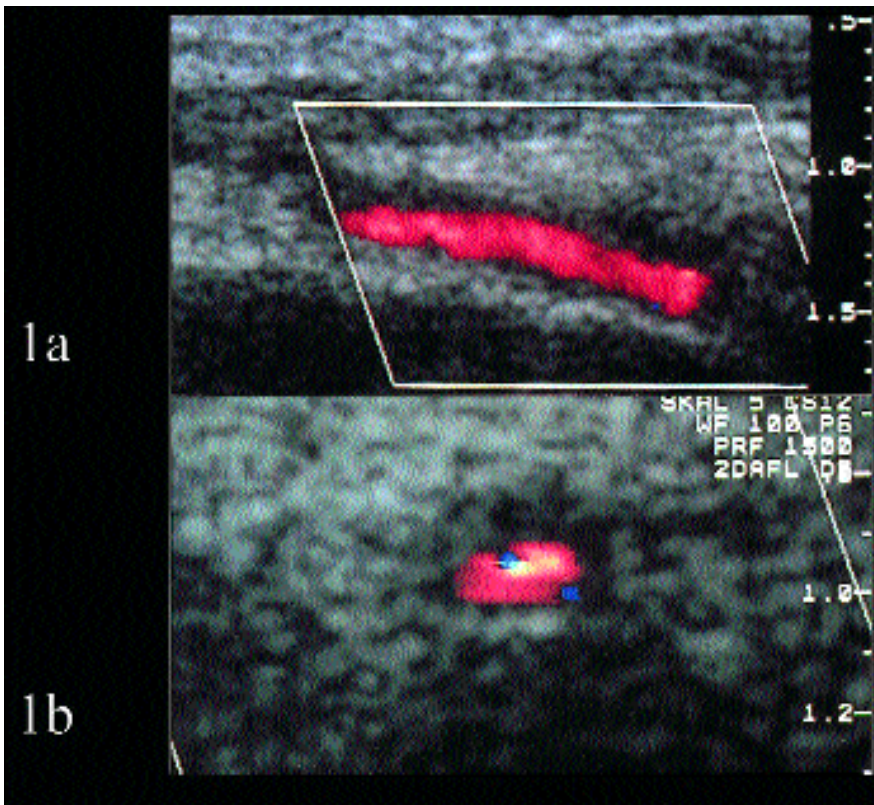
Statistical analysis

The SPSS statistical package was used for statistical analysis. The Mann-Whitney U test or the chi-square test were used to compare the results between groups.

Results

In 10 of 33 patients with acute giant cell arteritis but in none of the controls a hypoechoic wall thickening (halo) of arteries other than the temporal arteries was detected by sonography ($P < 0.001$). Table II summarizes the ultrasound findings of the patients and the controls. The halo was demonstrated at the occipital arteries (Fig. 1), the facial arteries (Fig. 2), the external carotid arteries, the common carotid arteries, the vertebral arteries, the subclavian arteries, the axillary arteries, the brachial arteries, the radial arteries (Fig. 3), the ulnar arteries, and the popliteal arteries (Fig. 4).

If stenoses occurred at the subclavian/proximal vertebral arteries, at the axillary/proximal brachial, or at the popliteal arteries, corresponding murmurs could always be heard. The stenoses of the facial and radial arteries could not be detected by auscultation. No cord-like thickening and no tenderness on palpation were found at any of the inflamed arteries. The 2 patients with stenoses of the subclavian/axillary/brachial arteries had arm claudication, decreased pulse, and decreased blood pressure. The patient who developed inflamma-



tory stenoses of the popliteal arteries had claudication of the right leg with a walking distance of 200 meters. Table III describes the 10 patients with sonographic findings of peripheral artery involvement in more detail.

Table IV compares the patients with and without sonographic findings of peripheral artery inflammation. In significantly more patients with peripheral artery involvement, sonography detected occlusions of the temporal arteries. No significant differences were found for other parameters, such as the finding of a halo or stenoses on temporal artery sonography, positive histology of the temporal arteries, the number of ACR classification criteria for the diagnosis of temporal arteritis, ocular involvement, polymyalgia rheumatica,

Fig. 1. Color Doppler sonography image of the left occipital artery in acute temporal arteritis in a longitudinal (a) and transverse plane (b). A dark (hypoechoic) area is found around the perfused lumen of the artery (patient 3).

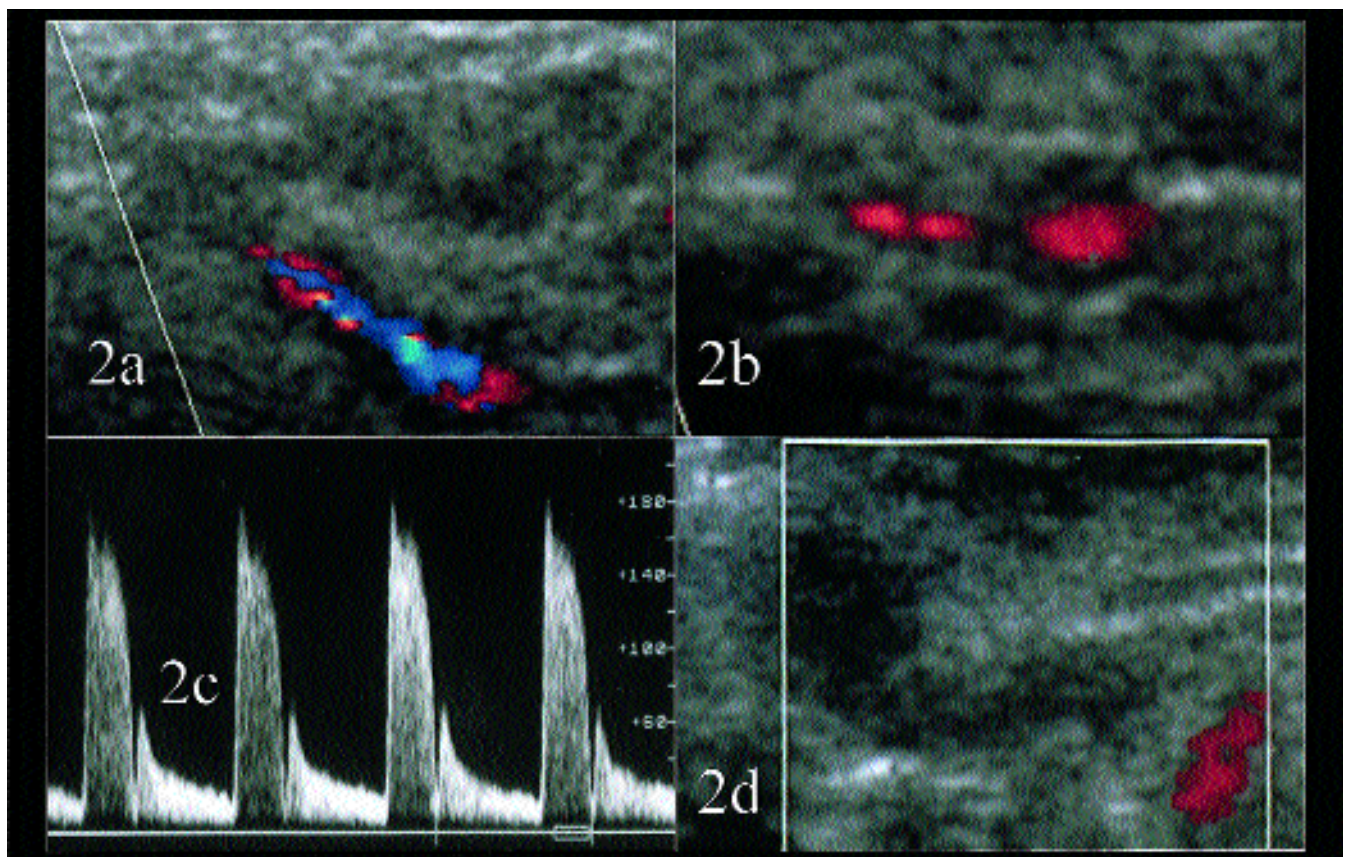


Fig. 2. Color Doppler sonography image of the right facial artery in acute temporal arteritis in a longitudinal (a) and transverse plane (b). A dark (hypoechoic) area is found around the perfused lumen of the artery. The non-homogenous pattern of colors suggests increased and turbulent flow (patient 7). This is demonstrated by the Doppler curves showing an increased blood flow velocity of up to 180 cm/s (c). An occlusion of the left facial artery in patient 10 is shown in (d).

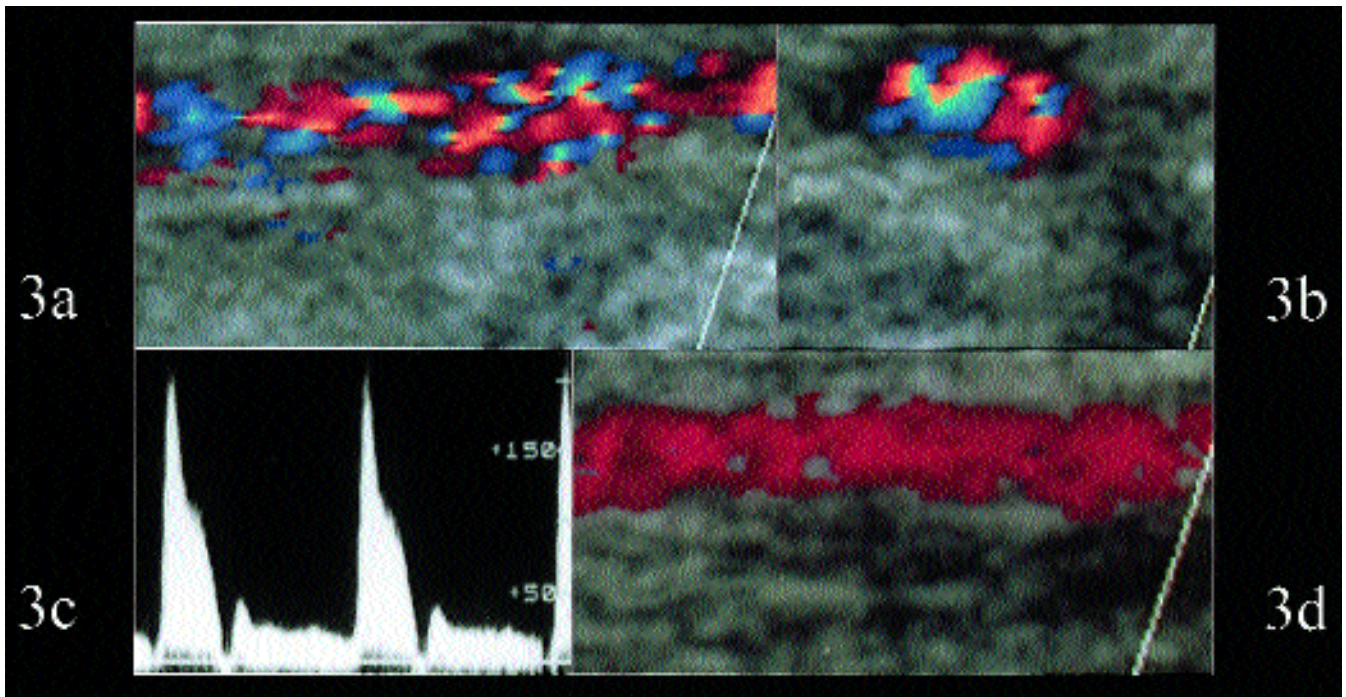


Fig. 3. Color Doppler sonography image of the right radial artery in acute temporal arteritis in a longitudinal (a) and transverse plane (b). A dark (hypoechoic) area is found around the perfused lumen of the artery, and the non-homogenous pattern of colors suggests increased and turbulent flow (patient 10). (c) Doppler curves show an increased blood flow velocity of up to 210 cm/s. Twelve days later with corticosteroid treatment both the hypoechoic wall thickening and the stenosis disappeared (longitudinal view: d).

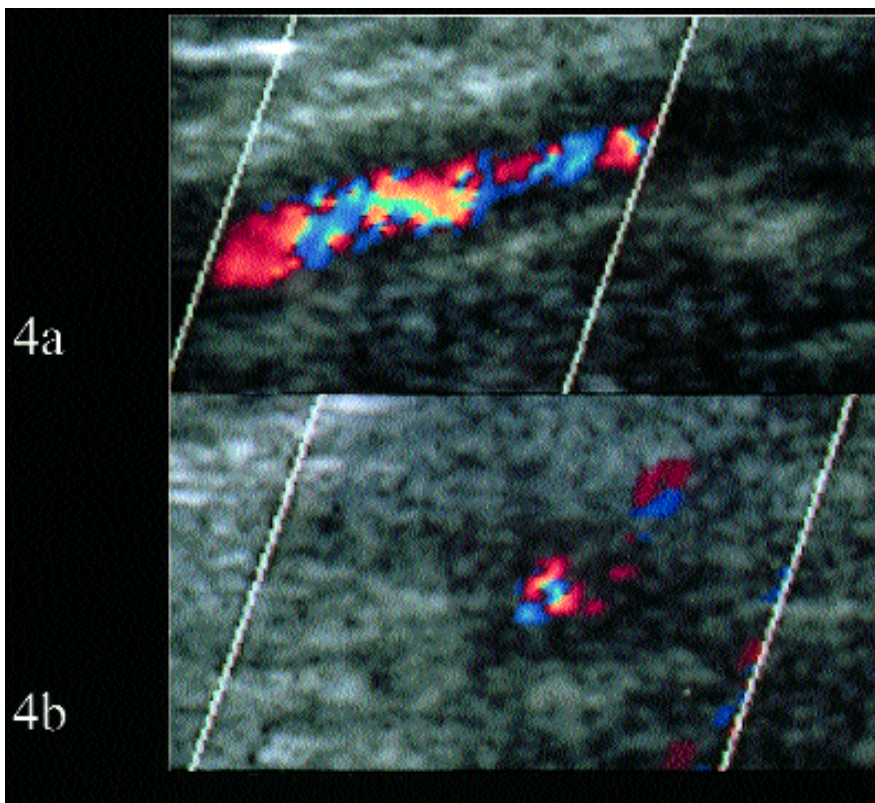


Fig. 4. Color Doppler sonography image of the right popliteal artery that developed in patient 5 despite corticosteroid treatment, in a longitudinal (a) and transverse plane (b). A dark (hypoechoic) area is found around the perfused lumen of the artery. The non-homogenous pattern of colors suggests increased and turbulent flow.

jaw claudication, ESR, perfusion of the supratrochlear arteries, and risk factors for arteriosclerosis. Three of the 4 patients with facial artery involvement had jaw claudication.

In none of the areas with a halo demonstrated by sonography were calcifications or other signs suspicious of arteriosclerosis found. Most of the inflammatory changes were detected at areas that are usually less frequently involved by arteriosclerosis (see Table II).

Arterial occlusions can be due either to inflammation or arteriosclerosis. Occlusions were found in 6 patients, 2 of whom also had sonographic findings of inflammation. The ulnar artery was involved in 3 cases, the posterior tibial artery in 2 cases, and the vertebral and dorsal pedal artery in 1 case each. No occlusion was found in the controls ($P < 0.01$). If the ulnar, posterior tibial, or dorsal pedal arteries were occluded palpation of these vessels was not possible.

The halo disappeared in all patients with non-stenotic involvement of the occipital, external carotid, axillary, or radial arteries within 9 to 21 days. This interval is comparable to our previous

Table II. Sonography findings at investigated arteries.

Artery	Inflammatory changes				Arteriosclerotic changes				Occlusion		Aneurysm	
	No stenosis		Stenosis		No stenosis		Stenosis		TA	Controls	TA	Controls
	TA	Controls	TA	Controls	TA	Controls	TA	Controls				
Occipital	3	-	-	-	-	-	-	-	-	-	-	-
Facial	3	-	1	-	-	-	-	-	*	-	-	-
External carotid	4	-	-	-	5	4	-	1	-	-	-	-
Internal carotid	-	-	-	-	12	13	-	-	-	-	-	-
Common carotid	1	-	-	-	19	13	-	-	-	-	-	-
Subclavian	3	-	1	-	-	-	-	-	-	-	-	-
Vertebral	-	-	1	-	-	-	-	-	1	-	-	-
Axillary	1	-	2	-	-	1	-	-	-	-	-	-
Brachial	-	-	2	-	-	-	-	-	-	-	-	-
Radial	-	-	1	-	-	-	-	-	-	-	-	-
Ulnar	1	-	1	-	-	-	-	-	3	-	-	-
Aorta	-	-	-	-	25	24	-	-	-	-	2	1
Femoral	-	-	-	-	25	25	-	-	-	-	-	-
Popliteal	-	-	1**	-	19	25	1	-	-	-	-	-
Posterior tibial	-	-	-	-	1	3	-	1	2	-	-	-
Dorsal pedal	-	-	-	-	1	2	-	1	1	-	-	-
Total	16	-	10	-	107	110	1	3	7	-	2	1

TA: temporal arteritis

* an occlusion occurred after the start of treatment in an artery that had a halo without stenosis at presentation (patient 10, see Table III)

** a halo was detected after 6 months despite treatment in an artery which appeared normal at presentation (patient 5, see Table III).

findings. The halo of the temporal arteries disappeared after a mean of 16 days (3). A small halo of an ulnar artery and an acute occlusion of the facial artery were found in one patient after 12 days of corticosteroid treatment (patient 10; see Table III).

In the 2 patients with classic large vessel GCA and hemodynamically relevant stenoses of the larger arteries, the mural thickening remained but became brighter, as described earlier (6). In patient 4 (Table III) a stent was implanted into the left axillary artery. The other patient with large artery involvement (patient 5) developed intermittent claudication of the right leg despite treatment with corticosteroids. A stenotic inflammatory infiltrate could be detected at both popliteal arteries.

Investigation of the supratrochlear arteries with cw-Doppler sonography revealed orthograde flow in 30 patients. In 3 patients with peripheral arteritis, the results were pathologic: patient 3 (Table III) had no detectable flow of the left supratrochlear artery, patient 4 had no detectable flow of the right supratrochlear artery, and patient 6 had no detectable flow in either supratrochlear artery. Retrograde flow was not found in any of the other patients.

The rates of agreement between the two ultrasonographers were 89% for inflammatory vascular lesions (17 of 19 tests), 95% for arteriosclerotic lesions (18 of 19 tests), and 100% for occlusions (19 of 19 tests).

No significant difference was found between patients and controls concerning arteriosclerotic lesions with and without stenosis and aneurysms (Table II).

Discussion

To our knowledge involvement of the extracranial arteries has not yet been investigated in a prospective study of consecutive patients with newly diagnosed, active giant cell arteritis.

Involvement of the large, peripheral arteries in temporal arteritis has been described as large vessel giant cell arteritis. These patients usually present with symptoms related to upper extremity vascular insufficiency. A series of 74 patients with large vessel giant cell arteritis seen at the Mayo Clinic over a 36-year period have been described. Temporal artery biopsy findings were negative in 42% of these patients, the percentage of females was greater (88% versus 78%), the presence of arm claudication was more common (51%

versus 0%), and headaches were less frequent (14% versus 57%), compared to patients with classic, biopsy-proven temporal arteritis (5). A meta-analysis of 8 studies involving 837 temporal arteritis patients found that 6% (3-15%) could be classified as cases of large vessel giant cell arteritis (7). In our cohort of 80 consecutive German patients with newly diagnosed temporal arteritis seen since 1994, three (4%) had symptomatic large vessel giant cell arteritis, two are mentioned in this study, and the other one has been described in an earlier report (6). We use the term "peripheral arteries" to describe all arteries except for the temporal arteries, and include in this term both large vessels such as the aorta and small vessels such as the occipital, facial, or ulnar arteries.

In most cases of large vessel giant cell arteritis, the distal subclavian, the axillary and the proximal brachial arteritis are involved (5-9). Vasculitis in giant cell arteritis has been less frequently reported in the femoropopliteal arteries (9). Less than 10 cases have been reported with the involvement of other arteries such as the common carotid (6, 10), the internal carotid (11, 12), the external carotid (13), and the vertebral

Table III. Clinical, sonographic and histologic findings of patients with giant cell arteritis and peripheral artery involvement

No.	Sex, age	Sonography of temporal arteries	Histology	No. ACR criteria	Eye involvement	PMR	Jaw claudication	ESR mm/h	Arteries with ultrasound finding of a halo	Side	Stenosis	Length of halo/stenosis (mm)	Results of control investigations
1	f. 84	HO	ND	4	AION	-	-	70	Occipital Ext. carotid Ulnar	r, l l r, l	- - -	25 20 5	halo disappeared after 9 days halo disappeared after 9 days halo disappeared after 21 days
2	f. 78	HS	+	5	-	+	-	82	Ext. carotid	r, l	-	20	no follow-up
3	m. 83	HSO	+	5	AION	-	+	90	Occipital Facial Ext. carotid	r, l r, l l	- - -	30 10 20	halo disappeared after 10 days halo disappeared after 10 days halo disappeared after 10 days
4	m. 58	S	+	4	AF	+	-	100	Subclavian Axillary Axillary Brachial	r, l l r l	- + + +	30 50/30 40/3 20/20	remained, became brighter remained, became brighter remained, became brighter remained, became brighter
5	f. 63	normal	ND	2	-	+	-	82	Com. carotid Subclavian Axillary Vertebral Brachial Popliteal	l r, l r, l r l r, l	- + + + + +	50 30/20 30/10 30/5 10/10 60/40	remained, became brighter remained, became brighter remained, became brighter remained, became brighter remained, became brighter remained, became brighter
6	f. 79	HS	+	5	-	-	+	86	Facial	r	-	15	halo disappeared after 14 days
7	f. 73	HSO	ND	3	AION	+	+	45	Facial	r, l	+	15/10	no follow-up
8	f. 81	HO	+	5	-	-	+	72	Ext. carotid Subclavian Axillary	l l r	- - -	15 15 40	halo disappeared after 21 days halo disappeared after 21 days halo disappeared after 21 days
9	f. 67	HO	+	5	-	+	+	80	Subclavian	r	-	10	halo disappeared after 10 days
10	f. 88	HO	+	5	-	-	-	70	Occipital Facial Radial Ulnar Ulnar	l l r r l	- - + + -	15 15 15 15 10	disappeared after 12 days disappeared after 12 days disappeared after 12 days disappeared after 12 days small halo remained

Sonography: H = Halo; S = Stenosis; O = Occlusion.

Histology: ND, not done.

Eye involvement: AION = anterior ischemic optic neuropathy; AF = amaurosis fugax.

side: r = right; l = left.

Table IV. Comparison of patients with and without sonographic findings of peripheral artery involvement in acute giant cell arteritis.

	Peripheral artery involvement		No peripheral artery involvement		Statistical significance
	N = 10	%	N = 23	%	
Age	75 (58-88)		72 (58-88)		n.s.
Sonography: halo of temporal arteries	8	80	19	83	n.s.
Sonography: stenosis of temporal arteries	5	50	18	78	n.s.
Sonography: occlusion of temporal arteries	6	60	6	26	0.035
Temporal artery histology: positive	7 of 7	100	16 of 19	84	n.s.
No flow in the supratrochlear artery	3	30	0	0	n.s.
Average number of ACR-criteria	4.3 (2-5)		4.1 (2-5)		n.s.
Eye involvement	4	40	11	48	n.s.
Polymyalgia rheumatica	5	50	6	26	n.s.
Jaw claudication	5	50	11	48	n.s.
Days of corticosteroid treatment	0.5 (0-3)		1.0 (0-5)		n.s.
ESR	78 (45-100)		68 (8-110)		n.s.
Smoking	3	30	3	13	n.s.
Diabetes mellitus	3	30	2	9	n.s.
Hypercholesterolaemia	2	20	6	26	n.s.
Arterial hypertension	3	30	8	35	n.s.
Myocardial infarction	0	0	1	4	n.s.
Stroke	0	0	0	0	n.s.
Arterial occlusive disease of legs	1	10	0	0	n.s.

(11,12), occipital (14,15), facial (16), and radial arteries (17), respectively. Involvement of the ulnar arteries as described in our study has, to our knowledge, not yet been reported. In the course of the disease patients with giant cell arteritis are 17.3 times more likely to develop thoracic aortic aneurysm and 2.4 times more likely to develop isolated abdominal aortic aneurysm (18). A study using positron emission tomography showed that 58% of the patients with temporal arteritis or polymyalgia rheumatica had an increased fluorodeoxyglucose uptake in their thoracic vessels compared to 2% of the controls (19). This finding indicates that a generalized vasculitis occurs more frequently in giant cell arteritis than assumed up to now.

In 1995 we described the phenomenon of a black area (halo) delineated by color duplex ultrasonography around the perfused lumen of the temporal arteries in acute giant cell arteritis (2). This finding proved to be highly specific (3,4). We only found this halo in patients with temporal arteritis and in 7% of patients with clinically pure polymyalgia rheumatica (20). The halo is due to an edema of the vessel wall. Nevertheless the halo has also been described in one case of temporal ar-

tery vasculitis caused by polyarteritis nodosa (21). Thus sonography cannot differentiate between temporal vasculitis due to classic giant cell arteritis and other vasculitides as a cause of temporal arteritis. Additionally sonography can detect stenoses and acute occlusions of the temporal arteries in acute stages of the disease. Other studies have found similar results (22,23).

As color Doppler sonography is an effective means to detect inflammation of the temporal arteries in giant cell arteritis, we applied this method to other vessels that may be easily investigated.

In studies comparing sonography with angiography in patients with Takayasu's arteritis sonography had been found to be superior in detecting smaller inflammatory lesions, because it delineates not only the lumen of the artery but also the vessel wall (26,27). On the other hand sonography is unable to completely evaluate the thoracic aorta unless performed via the oesophagus and may be inferior at the abdominal vessels due to intestinal air and the longer distance between the transducer and the vessel. Thus sonography and angiography are complementary methods.

The abdominal aorta and the renal, the

superior mesenteric, and the celiac arteries can be investigated with sonography in a non-invasive way. Studies have not yet been published that compare sonography with angiography or any other imaging methods for these vessels in vasculitis. It is definitely more difficult to see inflammatory changes of the abdominal arteries than of the carotid and the limb arteries with sonography. The distance between the transducer and the arteries is larger, and intestinal air may interfere with the ultrasound image.

In our study stenoses were found in four of the patients who were classified as having peripheral involvement. In two patients angiography was performed that confirmed the sonographic findings. The sonographic images of the two other patients with stenoses of the smaller arteries are shown in Figures 2 and 3.

Large vessel giant cell arteritis can be distinguished from Takayasu's arteritis. According to Michel and co-authors the age barrier of 40 years at disease onset is almost absolute (28). In more patients with large vessel giant cell arteritis the distal subclavian, the axillary, and the proximal brachial arteries are involved, whereas vasculitis of the proximal subclavian and the common

carotid arteries occurs more frequently in Takayasu's arteritis. Furthermore the mural thickening appears to be darker in large vessel giant cell arteritis than in Takayasu's arteritis due to the more acute nature of giant cell arteritis (6, 26). The wall thickening is homogeneous with a regular surface in both entities, but the echogenicity of the mural thickening is different. It is mid-echoic or hyperechoic (bright) in Takayasu's arteritis and hypoechoic (dark) in large vessel giant cell arteritis, even if the same arteries like the subclavian or carotid arteries are involved (6,26). The dark wall thickening in the acute phase of temporal arteritis and large vessel giant cell arteritis is due to a mural edema. The brighter wall in Takayasu's arteritis may be due to the more chronic nature of the disease. Nevertheless we recently described a patient with acute vasculitis of the internal carotid artery in Wegener's granulomatosis. The wall swelling in this patient was hyperechoic as in Takayasu's arteritis, but we additionally found a large perivasculitis which is uncommon in Takayasu's arteritis (29).

Ocular manifestations occurred more frequently in our patients than in other cohorts investigated by rheumatologists. Ten of our 15 patients with ocular manifestations had been referred by an ophthalmologist. Furthermore, in this study with consecutive patients, all had been investigated by an ophthalmologist in the acute phase of the disease, and even minor findings were defined as ocular involvement. In the 214 patients of the ACR cohort 28% had visual impairment (1). In cohorts investigated by ophthalmologists the incidence of ocular manifestations is higher. Hayreh described a 50% incidence of ocular involvement in 170 patients (30). Could the high incidence of ocular manifestations have biased the results of our study? There was no significant difference of eye involvement between those patients with or without signs of peripheral arteritis. The risk of cranial ischemic complications has been found to be even lower in patients with large vessel giant cell arteritis (5). Could the hypoechoic wall thickening seen in our patients be due to arterio-

sclerotic lesions? We could not find a halo in any of the control patients. The morphology of arteriosclerotic lesions is different. The wall is irregular, calcifications are frequently found, and most lesions are localized in arteries in which arteriosclerotic involvement is far less frequent. Further studies should be performed using ultrasound integrated backscatter to investigate whether this method, which represents a novel technical option offered by the new high-end ultrasound machines, can help to differentiate between vasculitic and non-vasculitic changes of the artery wall, as has been shown for arteriosclerotic lesions (31, 32).

Our color Doppler sonography study of the peripheral arteries reveals that a generalized vasculitis occurs more frequently in acute giant cell arteritis than has been assumed up to now. This technique offers a new method to evaluate such peripheral involvement.

References

- HUNDER GG, BLOCH DA, MICHEL BA, *et al.*: The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. *Arthritis Rheum* 1990; 33:1122-8.
- SCHMIDT WA, KRAFT HE, VÖLKER L, VORPAHL K, GROMNICA-IHLE EJ: Colour Doppler sonography to diagnose temporal arteritis. *Lancet* 1995; 345: 866.
- SCHMIDT WA, KRAFT HE, VORPAHL K, VÖLKER L, GROMNICA-IHLE EJ: Color duplex ultrasonography in the diagnosis of temporal arteritis. *N Engl J Med* 1997; 337: 1336-42.
- SCHMIDT WA: Doppler ultrasonography in the diagnosis of giant cell arteritis. *Clin Exp Rheumatol* 2000; 18 (Suppl. 20):40-2.
- BRACK A, MARTINEZ-TABOADA V, STANSON A, GORONZY JJ, WEYAND CM: Disease pattern in cranial and large vessel giant cell arteritis. *Arthritis Rheum* 1999; 42: 311-7.
- SCHMIDT WA, KRAFT HE, BORKOWSKI A, GROMNICA-IHLE EJ: Colour duplex ultrasonography in large vessel giant cell arteritis. *Scand J Rheumatol* 1999; 28: 374-6.
- NINET JP, BACHET P, DUMONTET CM, DU COLOMBIER PB, STEWART MD, PASQUIER JH: Subclavian and axillary involvement in temporal arteritis and polymyalgia rheumatica. *Am J Med* 1990; 88: 13-20.
- KLEIN RG, HUNDER GG, STANSON AW, SHEPS SG: Large artery involvement in giant cell (temporal) arteritis. *Ann Int Med* 1975; 83: 806-12.
- LIE JT: Aortic and extracranial large vessel giant cell arteritis: a review of 72 cases with histopathologic documentation. *Semin Arthritis Rheum* 1995; 24: 422-31.
- CHAMBERS BR, DONNAN GA, RIDDELL RJ, BLADIN PF: Carotidynia: Aetiology, diagnosis and treatment. *Clin Exp Neurol* 1981; 17: 113-23.
- THIELEN KR, WIJDECKS EF, NICHOLS DA: Giant cell (temporal) arteritis: Involvement of the vertebral and internal carotid arteries. *Mayo Clin Proc* 1998; 73: 444-6.
- WILKINSON IM, RUSSELL W: Arteries of the head and neck in giant cell arteritis. A pathological study to show the pattern of arterial involvement. *Arch Neurol* 1972; 27: 578-91.
- GONZALEZ-GAY MA, GARCIA-PORRUA C: Carotid tenderness: an ominous sign of giant cell arteritis? *Scand J Rheumatol* 1998; 27: 154-6.
- KATTAH JC, CUPPS T, MANZ HJ, EL KHODARY A, CAPUTY A: Occipital artery biopsy: a diagnostic alternative in giant cell arteritis. *Neurology* 1991; 41: 949-50.
- WEEMS JJ: Diagnosis of giant cell arteritis by occipital artery biopsy. *Am J Med* 1992; 93: 231-2.
- ACHKAR AA, LIE JT, GABRIEL SE, HUNDER GG: Giant cell arteritis involving the facial artery. *J Rheumatol* 1995; 22: 360-2.
- HAMRIN B: Polymyalgia arteritica. *Acta Med Scand* 1972; 533 (Suppl.): 1-131.
- EVANS J, O'FALLON WM, HUNDER GG: Increased incidence of aortic aneurysm and dissection in giant cell (temporal) arteritis. A population-based study. *Ann Intern Med* 1995; 122: 502-7.
- BLOCKMANS D, STROOBANTS S, MAES A, MORTELMANS L: Positron emission tomography in giant cell arteritis and polymyalgia rheumatica: evidence for inflammation of the aortic arch. *Am J Med* 2000; 108: 246-9.
- SCHMIDT WA, GROMNICA-IHLE E: Incidence of temporal arteritis in patients with polymyalgia rheumatica - a prospective study using colour Doppler ultrasonography of the temporal arteries. *Rheumatology* 2002; 41: 46-52.
- FRANCOIS M, KOUSSA D, DECLERCK D: Halo péri-artériel temporal en écho-Doppler au cours d'une panartérite nouvelle. *Presse Med* 1999; 23: 133.
- VENZ S, HOSTEN N, NORDWALD K, *et al.*: Einsatz der hochauflösenden Farb-Doppler-Sonographie in der Diagnostik einer Arteriitis temporalis. *Fortschr Roentgenstr* 1998; 169: 605-8.
- STAMMLER F, YSERMANN M, MOHR W, KUHN C, GOETHE S: Stellenwert der farbkodierten Duplexsonographie bei Patienten mit Polymyalgia rheumatica ohne klinische Zeichen einer Arteriitis temporalis. *Dtsch Med Wschr* 2000; 125: 1250-6.
- DUHAUT P, PINEDE L, BORNET H, *et al.*: Biopsy proven and biopsy negative temporal arteritis: differences in clinical spectrum at the onset of the disease. *Ann Rheum Dis* 1999; 58: 335-41.
- SALVARANI C, HUNDER GG: Musculoskeletal manifestations in a population-based cohort of patients with giant cell arteritis. *Arthritis Rheum* 1999; 42: 1259-66.
- MAEDA H, HANDA N, MATSUMOTO M, *et al.*: Carotid lesions detected by B-mode ultrasonography in Takayasu's arteritis: "Macaroni sign" as an indicator of the disease. *Ultrasound Med Biol* 1991; 17: 695-701.

27. TANIGUCHI N, ITOH K, HONDA M, *et al.*: Comparative ultrasonographic and angiographic study of carotid arterial lesions in Takayasu's arteritis. *Angiology* 1997; 48: 9-20.
28. MICHEL BA, AREND WP, HUNTER GG: Clinical differentiation between giant cell (temporal) arteritis and Takayasu's arteritis. *J Rheumatol* 1996; 23: 106-11.
29. SCHMIDT WA, SEIPELT E, MOLSSEN HP, POEHLS C, GROMNICA-IHLE E: Vasculitis of the internal carotid artery in Wegener's granulomatosis: comparison of ultrasonography, angiography, and MRI. *Scand J Rheumatol* 2001; 30: 48-50.
30. HAYREH SS, PODHAJSKY PA, ZIMMERMAN B: Ocular manifestations of giant cell arteritis. *Am J Ophthalmol* 1998; 125: 509-20.
31. TAKIUCHI S, RAKUGI H, HONDA K, *et al.*: Quantitative ultrasonic tissue characterization can identify high-risk atherosclerotic alteration in human carotid arteries. *Circulation* 2000; 102: 766-70.
32. KAWASAKI M, TAKATSU H, NODA T, *et al.*: Noninvasive quantitative tissue characterization and two-dimensional color-coded map of human atherosclerotic lesions using ultrasound integrated backscatter: comparison between histology and integrated backscatter images. *J Am Coll Cardiol* 2001; 38: 486-92.