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Ultrasound of proximal upper extremity arteries to increase the diagnostic yield in large-vessel giant cell arteritis

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Objective. To describe characteristic ultrasound findings and clinical features of patients with newly diagnosed cranial and large-vessel (LV) GCA in a specialized ultrasound clinic.

Methods. This case–control study includes all consecutive patients between 1997 and 2006 with newly diagnosed GCA. Duplex ultrasound of the temporal, subclavian, axillary and proximal brachial arteries was performed in all patients with suspected temporal arteritis, PMR, arm claudication, unclear inflammation or pyrexia of unknown origin (PUO).

Results. In 53 of 176 patients, ultrasound depicted characteristic vasculitic homogeneous wall swelling of the axillary, subclavian and/or proximal brachial arteries. These were affected in 98, 61 and 21%, respectively, in the 53 patients. The findings were bilateral in 79%. Axillary arteries were stenotic or occluded in 51 and 2% and temporal artery ultrasound and histology were positive in 62 and 67% of LV-GCA cases, respectively. A significantly greater number of LV-GCA patients were female (83 *vs* 65%) and younger (mean 66 *vs* 72 yrs) as compared with those without proximal arm involvement. Headaches (38 *vs* 75%), jaw claudication (24 *vs* 48%) and anterior ischaemic optic neuropathy (4 *vs* 19%) occurred significantly less frequently. The median time until diagnosis was significantly longer (31 *vs* 8 weeks). ESR and presence of PMR were similar in both groups.

Conclusions. Performing axillary artery ultrasound in all patients with suspected temporal arteritis, PMR, arm claudication, unclear inflammation or PUO increases the diagnostic yield for LV-GCA. Patients with LV-GCA differ from those without arm involvement.

Key words: Temporal arteritis, Giant cell arteritis, Large-vessel giant cell arteritis, Ultrasonography, Colour Doppler ultrasonography, Angiography, Magnetic resonance imaging.

Introduction

Giant cell arteritis (GCA) is a primary vasculitis that frequently involves the temporal arteries and the vasculature of the eye. Within the last few years a greater number of patients have been identified with involvement of other arteries because of increased use and higher quality of imaging studies.

The systemic nature of GCA has been recognized for a long time [1, 2]. The term 'large-vessel GCA' (LV-GCA) has been introduced between 1995 and 1999 describing patients with extracranial artery involvement, in particular with vasculitis of the subclavian, axillary and proximal brachial arteries [3, 4]. Patients typically complain of intermittent arm claudication, paraesthesias and Raynaud's phenomenon. The most important distinguishing feature between LV-GCA and Takayasu arteritis is the age at disease onset with LV-GCA occurring almost exclusively in an age-group of $\geq$50 yrs and Takayasu arteritis $\leq$40 yrs [5]. Axillary arteritis is more common in LV-GCA than in Takayasu arteritis [6]. Furthermore, aortitis has been increasingly recognized in GCA with a higher incidence of aortic aneurysm or aortic dissection than in an age-matched population [7, 8]. Thoracic aortic dissection in GCA is associated with increased mortality [9]. Vasculitis of other vessels like carotid, femoral and popliteal arteries is less common [3].

Several imaging techniques, such as angiography, MRI, CT, PET and duplex ultrasound allow to assess LV-GCA. Although the greatest experience exists with angiography, there is no golden standard for the diagnosis of LV-GCA. Angiography displays smooth stenoses [10]. It is invasive, but it allows for measuring central blood pressure and for performing procedures such as angioplasty and stenting. Ultrasound, MRI and CT depict characteristic homogeneous wall swelling [11]. PET displays vascular fluorodeoxyglucose uptake of larger arteries with a diameter >4 mm [12, 13].

Ultrasound of inflamed temporal arteries depicts a hypoechoic (dark) wall swelling ('halo') that disappears within 2–3 weeks with corticosteroid therapy. Furthermore, most patients exhibit temporal artery stenoses in case of inflammation, and, less commonly, have acutely occluded temporal arteries [14]. Data from our institution on 101 patients with newly diagnosed GCA plus 751 controls showed a sensitivity of 88% and a specificity of 96% with regard to the clinical diagnosis and a sensitivity of 95% with regard to positive temporal artery histology. The specificity for the presence of a hypoechoic temporal artery wall swelling ('halo') was 99.5% [15]. A meta-analysis of 23 studies that investigated the diagnostic accuracy of Doppler, colour Doppler and duplex ultrasound for the presence of temporal arteritis showed that several centres reached similar results, while others arrived at less sensitive or less specific results. Sensitivities and specificities were higher in centres that used higher quality equipment and duplex ultrasound [16].

Ultrasound displays the same findings at the subclavian, axillary and proximal brachial arteries in LV-GCA (Fig. 1), but wall swelling remains for a longer period with therapy [17]. In acute vasculitis, the wall swelling is hypoechoic (dark) because of oedema. It becomes more echoic (brighter) over months with corticosteroid therapy [18].

The aim of this study is to investigate if proximal upper extremity artery ultrasound picks up vasculitis of the proximal arm arteries in patients with suspected active GCA, PMR, arm claudication, inflammation of unknown origin or pyrexia of unknown origin (PUO), furthermore, to compare clinical parameters from patients with GCA arteritis with and without proximal arm artery involvement.

Patients and methods

Patients

This is a case–control study from a specialized clinic. We established this clinic as a new project after having demonstrated

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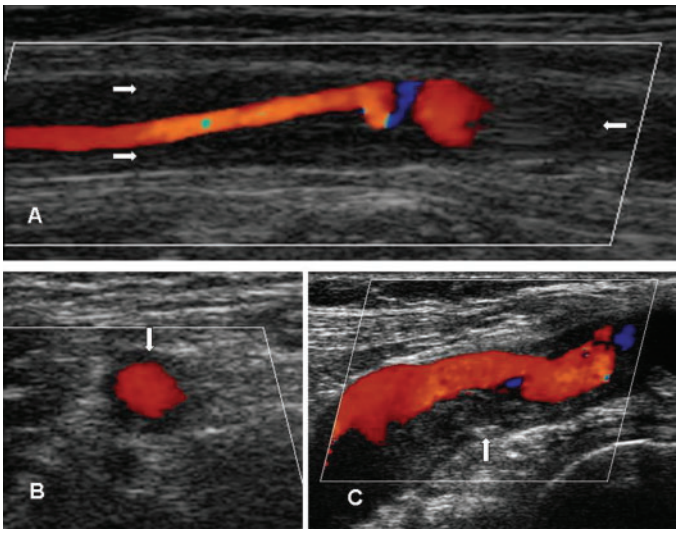

FIG. 1. Ultrasound images of pathological axillary arteries. (**A**) Longitudinal view of hypoechoic wall swelling (right arrow) with >50% stenosis in acute LV-GCA, on the right side the artery is occluded (left arrow). (**B**) Transverse view of concentric hypoechoic wall swelling (downward arrow) with <50% stenosis in acute LV-GCA. (**C**) Longitudinal view of rare finding with major atherosclerotic axillary disease. The wall swelling is localized only on one side of the artery, it is non-homogeneous and displays a calcification (upward arrow).

characteristic sonographic features of temporal arteries in acute GCA [19] to provide a fast and systematic diagnostic process in patients with suspected GCA.

Physicians suspecting GCA in a patient are invited to call our ultrasound department. The patients usually receive an appointment for the same or the next working day. In case of highly suspected temporal arteritis, we encourage the referring physician to initiate corticosteroid treatment already before the appointment. The ultrasound clinic is part of a large rheumatological tertiary care institution with 35 000 outpatient visits and 2000 inpatients per year serving a population of about 2 million in the north-east of Berlin, Germany and its suburbs.

An experienced rheumatologist (W.A.S. or A.N.) is performing a standardized history and a clinical assessment focusing on the diagnosis of GCA. Subsequently, the same rheumatologist performs duplex ultrasound of the temporal, subclavian, axillary and proximal brachial arteries. Since 1994, more than 1800 patients have been examined in this clinic.

In this study, we included all consecutive patients between 1997 and 2006 who had been newly diagnosed with active GCA aiming at comparing patients with and without upper arm artery involvement. We distinguished cranial from LV-GCA as has been done earlier in the study from Brack and colleagues [4]. Some of the patients with LV-GCA had upper extremity vasculitis without any clinical signs and symptoms of temporal arteritis.

The diagnosis of GCA was either established histologically or clinically. The clinical diagnosis was defined by fulfilling three or more of the five ACR classification criteria for temporal arteritis [20] together with good response to corticosteroid therapy. Furthermore, no other diagnosis related to the patient's symptoms would have been made after at least 6 months [21].

The diagnostic workup included chest radiography, urinalysis, ophthalmological and ENT examinations, and analysis of ANCA to exclude temporal artery involvement of other vasculitides. Thus, we excluded one patient with ANCA-positive necrotizing glomeruloneprhitis from this study [22]. The study was approved by the local ethics committee. Patients gave informed consent.

Ultrasound examination

Until July 2003, we performed ultrasound examinations with a linear transducer (L10-5; 5–10 MHz; ATL Ultramark 9 HDI; Advanced Technology Laboratories, Bothell, WA, USA). The scanner settings for all examinations were as follows: dynamic range, 50 dB; signal-processing characteristic, G6; dynamic-contrast gain, K4; colour gain, 78%; type of colour gain, V; colour scale, 4; colour sensitivity, 10; colour wall filter, 100 Hz; colour persistence, 4; pulse-repetition frequency, 2.5 kHz; dynamic-movement differentiation D3; one focus point position just below the region of interest [14].

Since August 2003, we have been using different equipment: Esaote Technos MPX; Esaote SP, Genua, Italy. For the examination of the temporal arteries we employed a linear probe (LA 424; 8–14 MHz; length of probe, 38 mm). Scanner settings were uniform for all examinations: B mode frequency, 13 MHz; B mode gain, 100%; one focus point position just below the region of interest; procession parameter, PB 3; scan correlation parameter, SCC 5; enhancing parameter, ENH 5; colour Doppler frequency, 10 MHz; pulse repetition frequency, PRF 2.5 kHz; colour gain, 171%.

For the examination of the proximal arm arteries we employed another linear probe (LA 523; 4–13 MHz, length of probe, 45 mm). Scanner settings were uniform for all examinations: B-mode frequency, 10 MHz; B mode gain, 100%; one focus point position just below the region of interest; procession parameter, PB 3; scan correlation parameter, SCC 6; enhancing parameter, ENH 2; colour Doppler frequency, 4.5 MHz; pulse repetition frequency, PRF 2.5 kHz; colour gain, 68%.

Ultrasound examination of the temporal arteries included longitudinal and transverse views of the common superficial temporal arteries with its frontal and parietal branches on both sides as completely as possible [23–25]. Biopsies were preferably performed at temporal artery branches that displayed pathological findings by ultrasound. In addition, we examined the distal subclavian, the axillary and the proximal brachial arteries with colour duplex ultrasound in two planes.

We defined vasculitis if a homogeneous wall swelling of at least 1.5 mm was present (Fig. 1). This finding clearly differs from non-homogeneous and, in part, hyperechoic wall thickening in arteriosclerosis. We regarded lesions as stenotic if the artery lumen was <50% of the original lumen together with characteristic Doppler curves showing turbulences and increased systolic and diastolic blood flow velocities. Arteries were defined as occluded if ultrasound was unable to delineate colour in the former artery lumen that showed a hypoechoic or mid-echoic appearance.

Statistical analysis

We applied the SPSS V.14 statistical package for statistical analysis. The *t*-test, the Mann–Whitney U-test or the chi-square test was used to compare results between groups.

Results

The diagnosis of GCA was established in 176 patients according to the aforementioned criteria within 1997 and 2006. Table 1 provides detailed information on these patients.

GCA patients with and without proximal artery involvement differ with regard to age, sex, headaches, jaw claudication, clinical findings at the temporal arteries, eye involvement, time until diagnosis, ultrasound and histological findings as shown in Table 1. Arm claudication occurred in 21% of LV-GCA patients and in none of the cases with classic cranial GCA. Eye involvement was not significant between the groups, but it was less severe. Anterior optic ischaemic neuropathy occurred in significantly more patients with classic cranial GCA but amaurosis fugax tended to be more common in those with LV-GCA

Table 1. Comparison of GCA patients with and without proximal arm artery vasculitis (LV-GCA *vs* classic temporal arteritis)

	All patients	Classic temporal arteritis	LV-GCA	Significance
n	176	123	53	
Age	70	72	66	<0.001
Females (%)	70	65	83	0.019
PMR (%)	43	42	45	0.74
Headaches (%)	64	75	38	<0.001
Jaw claudication (%)	41	48	24	0.004
Swollen, hard temporal arteries (%)	53	64	29	<0.001
Plus reduced temporal artery pulse (%)	66	77	42	<0.001
Eye involvement (all features) (%)	29	33	19	0.070
Anterior optic ischaemic neuropathy (%)	14	19	4	0.008
Median time until diagnosis (weeks)	15	8	31	<0.001
ESR (mm/h)	75	74	76	0.44
Temporal artery ultrasound: pathological (%)	85	95	62	<0.001
Temporal artery ultrasound: halo (%)	73	82	53	<0.001
Temporal artery ultrasound: stenosis (%)	59	70	34	<0.001
Temporal artery ultrasound: occlusion (%)	18	20	15	0.53
Temporal artery histology pathological (%)	75	78	67	0.38

Table 2. Symptoms and diagnoses of 53 patients before establishing the diagnosis of LV-GCA

	n	%
Temporal arteritis	21	40
PMR	24	45
Arm claudication	11	21
Leg claudication	4	8
Pyrexia of unknown origin (PUO)	5	9
Unclear inflammation (except PUO)	4	8
Stroke	2	4

Table 3. Pathological findings in proximal arm arteries in 53 patients with LV-GCA

Involvement	Wall swelling without stenosis (%)	Wall swelling with stenosis (%)	Occlusion (%)	Normal (%)
Axillary artery	45	51	2	2
Subclavian artery	40	21	0	40
Proximal brachial artery	6	13	2	79

(13 *vs* 2%; $P = 0.38$). CRP values were similar in both groups (79 mg/l in LV-GCA *vs* 70 mg/l in cranial GCA). When applying the Bonferroni correction for the 16 parameters in Table 1 all significant parameters remain significant except those for sex, jaw claudication and anterior optic ischaemic neuropathy ($P > 0.003$). In both groups, 72% of patients had no corticosteroid therapy or corticosteroid therapy for <24 h before the ultrasound examination, 2% of LV-GCA patients and 6% of patients without LV-GCA had long-term corticosteroid therapy for more than 1 month with up to 7 mg of prednisolone because of suspected PMR before the diagnosis of GCA was established. Results between both groups were not significant.

Establishing the diagnosis of LV-GCA is more difficult compared with classic cranial GCA, as LV-GCA patients present with a greater variety of symptoms. Table 2 describes the symptoms and diagnoses that finally led to the diagnosis of LV-GCA.

Table 4. Detection of vasculitis of temporal arteries in patients with LV-GCA

	All LV-GCA *n* (%)	LV-GCA without signs and symptoms of temporal arteritis *n* (%)	LV-GCA with signs and symptoms of temporal arteritis *n* (%)
Temporal artery ultrasound +	33/53 (62)	15/28 (54)	18/25 (72)
Halo	28/53 (53)	12/28 (42)	16/25 (64)
Stenosis	18/53 (34)	9/28 (32)	9/25 (36)
Occlusion	8/53 (15)	3/28 (11)	5/25 (20)
Histology +	14/21 (67)	9/15 (60)	5/6 (83)

Less than half of the patients presented with clinical symptoms of temporal arteritis. Only 20 patients (38%) had headaches. Fourteen of the 24 patients with PMR presented with 'pure' PMR. They did not exhibit any clinical signs of temporal arteritis.

Four patients who did not complain of headaches had reduced or missing temporal artery pulse, and one patient had temporal artery thickening, when being assessed just before the ultrasound examination. Two of the four patients with reduced or missing temporal arteries presented with PMR and two with arm claudication.

Other patients presented with PUO, which is defined as with fever >38.3°C for ≥3 weeks and no prior diagnosis despite intensive diagnostic workup [26] and without clinical signs of temporal arteritis. Patients with unclear inflammation were similar. These patients complained of malaise, weight loss and night sweats, but temperature was lower.

Some patients presented with a combination of features, such as arm claudication/PMR (five patients), arm claudication/temporal arteritis, arm claudication/PUO, leg claudication/temporal arteritis, stroke/temporal arteritis, PMR/PUO (two patients, respectively), arm and leg claudication, and leg claudication/PUO (one patient, respectively).

One patient with arm claudication had finger necroses that healed after angioplasty. Ultrasound of the popliteal arteries delineated homogeneous wall swelling with stenoses in all patients with leg claudication.

Ultrasound demonstrated either non-stenotic wall swelling, stenotic wall swelling or occlusion of the proximal arm arteries. Table 3 shows the distribution of pathological changes at the proximal arm arteries in the patients with LV-GCA.

The axillary arteries were most frequently and most severely involved. We found subclavian artery wall swelling without axillary vasculitic changes in only one patient. The artery involvement was bilateral in 79%.

We performed other imaging techniques in 28% of the patients, angiography in 17% and MR-angiography in 11%. The results correlated with the ultrasound findings in 100%.

Some patients with LV-GCA did not exhibit any signs and symptoms of temporal arteritis like headache, scalp tenderness, thickened and hard temporal arteries or reduced temporal artery pulse. Other patients in whom we could detect LV-GCA had concurrent symptoms and clinical signs of temporal arteritis. Table 4 compares both groups and their relation to sonographic and histological findings.

In 11 patients with LV-GCA we succeeded in demonstrating temporal arteritis both by ultrasound and histology, four patients had a positive histology although ultrasound was negative. Histology displayed only few inflammatory cells in two of these four patients, and it was positive on one side only in another patient. In four patients, we found inflammatory temporal artery wall swelling on ultrasound although histology was negative. Inflammation visualized by ultrasound was confined to the common superficial temporal artery in these cases whereas biopsy was performed at the distal frontal branch that appeared normal on ultrasound. In three patients, both ultrasound and histology were negative.

TABLE 5. Number of cases with newly diagnosed GCA and LV-GCA between 1997 and 2006

Year	Diagnosis of GCA	Diagnosis of LV-GCA	Percentage of LV-GCA
1997	9	1	11
1998	11	0	0
1999	8	1	13
2000	15	1	7
2001	26	5	19
2002	13	3	23
2003	21	10	48
2004	22	8	36
2005	29	13	45
2006	22	11	50
1997–2006	176	53	30

Table 5 describes the annual incidence of newly diagnosed GCA and LV-GCA in our clinic.

We recognized only few patients with LV-GCA at the beginning. Since an increasing number of patients with newly diagnosed PMR, arm claudication, unclear inflammation and PUO were referred to our clinic, we detected LV-GCA more frequently. We found proximal upper-extremity arteritis in 45% GCA patients within the last 4 yrs of the study.

We could delineate the axillary arteries including changes both with the old and the new equipment. Nevertheless, this development displays a learning curve with regard to the indication for examining the proximal arm arteries, to the awareness of LV-GCA, to the experience with the examination technique, and to the use of newer technology.

Discussion

This study shows that LV-GCA is more common than previously assumed. Ultrasound detects characteristic wall swelling of the axillary arteries in nearly all cases with upper extremity involvement. Patients with LV-GCA differ from those with no proximal arm artery involvement.

Several retrospective studies described patients with LV-GCA. An early study from the Mayo Clinic in Rochester, MN, USA, reported on 23 patients with definite vasculitis of the aorta or its major branches [27]. The sample size was too small to reach clear significant differences between patients with cranial and LV-GCA.

A study from France described 10 female patients with a mean age of 67 yrs with upper-extremity ischaemia [28]. Temporal artery histology was positive for GCA in seven of nine biopsied cases. Angiograms showed bilateral smooth stenoses of post-vertebral subclavian and/or axillary arteries. Four patients had cephalic symptoms, five had PMR, mean time to diagnosis was 12 months and one patient had visual symptoms. None of the patients required reconstructive surgery.

A large study described 72 cases from the United States, consisting of 19 autopsy and 53 biopsy specimens [3]. The latter were obtained at surgical interventions. A total of 35 specimens were taken from the aorta, 22 from subclavian/axillary/brachial arteries, 13 from femoral/popliteal arteries and 2 from carotid/vertebral arteries. The mean age was 69 yrs. Seventy-one per cent of patients were female. The diagnosis of temporal arteritis had preceded that of LV-GCA in 63% of cases. Typical symptoms of temporal arteritis were absent in 25% of these cases.

Aneurysms of the thoracic aorta are 17 times more likely in patients with GCA than in age-matched controls [7]. Twenty-seven per cent of a prospectively followed cohort of 168 GCA patients developed large-artery complication, mostly aortic aneurysm and/or aortic dissection, [8]. Only 4% of this cohort presented with proximal arm artery stenosis. Yearly chest radiograph, transthoracic echocardiography and abdominal ultrasound in search for aneurysm formation have been suggested for all GCA patients [29].

Another retrospective study compared 74 patients who had been diagnosed by angiography in the Mayo Clinic with upper arm artery vasculitis between 1960 and 1998 with the same number of patients with temporal artery biopsy-proven GCA without LV involvement [4]. Most patients presented with symptoms of upper extremity blood flow impairment. Patients with LV-GCA were significantly younger than controls (66 *vs* 72 yrs), the number of females was significantly greater (88 *vs* 78%), ESR was significantly lower (61 *vs* 84%), and the time between onset of symptoms and diagnosis was significantly longer (8.1 *vs* 2.6 months). A significantly smaller number of patients with LV-GCA had headaches, jaw claudication, visual symptoms, abnormal temporal arteries and constitutional symptoms. The prevalence of PMR was similar between groups (41 *vs* 46%). Only 33/58 LV-GCA patients (58%) had positive temporal artery biopsies for the presence of GCA, which indicates that this procedure cannot be the gold standard for establishing the diagnosis. The arterial involvement was almost always bilateral but not usually symmetric.

The study designs of the aforementioned publications differ considerably from the present study. Most of the other studies were retrospective, and they mostly described symptomatic patients with regard to upper extremity ischaemia and/or autopsy cases. Ours is a case–control study of consecutive patients, many of whom are expressing less-severe LV involvement as only every other patient exhibited arterial stenoses or occlusions of the proximal arm arteries.

Nevertheless, the results of all studies arrive at surprisingly similar results with regard to patient characteristics. LV-GCA compared with classic temporal arteritis occurs more frequently in females, patients are slightly younger, time to diagnosis is longer, headaches and severe visual complications are less common, the prevalence of PMR is similar and only about 60% of cases have positive temporal artery biopsy results.

Other studies examined consecutive patients with GCA and PMR with PET. They found an even higher incidence of large-artery involvement predominantly at the subclavian arteries. Extracranial vascular fluorodeoxyglucose uptake was noted in 83% of 35 patients with GCA [30] and in 31% of 35 patients with PMR [31]. Areas with fluorodeoxyglucose uptake correlate well with homogeneous artery wall thickening at ultrasound [32]. The use of PET is limited because of low availability and high costs. PET does not directly depict artery wall morphology and imaging of the temporal arteries is impossible.

MRI of the aorta displays characteristic wall swelling and oedema in LV-GCA [33]. In a study with 15 patients, it was slightly less sensitive than PET [34]. In another study with six patients, oedema had improved significantly with corticosteroid treatment but wall thickness remained invariable [35]. It has been recently demonstrated that contrast-enhanced MRI displays inflamed temporal arteries. Sensitivities and specificities are comparable with those for temporal artery duplex ultrasound, particularly for 3 tesla high-resolution equipment [36].

Ultrasound is widely available. An increasing number of rheumatologists are using this technique. Ultrasound examination of both axillary arteries takes about 2 min if an experienced sonographer performs the test and if the anatomy is normal and ~5 to 10 min if it is pathological. Extending the ultrasound examination to both subclavian and both proximal brachial arteries takes another 5 min, but there are only few patients in which the axillary arteries are spared according to our findings.

This short and cheap examination offers a lot of relevant information in patients with temporal arteritis, PMR, pyrexia or inflammation of unknown origin and/or arm claudication. The drawback of ultrasound is its poor performance at the thoracic aorta. Therefore, some cases in whom the aorta is exclusively involved will be missed. Furthermore, as GCA may involve nearly

every large artery, ultrasound limited to the temporal and axillary arteries misses some cases. Nevertheless, the examination can be easily extended to symptomatic areas. Therefore, this protocol is quick and effective in the diagnosis of GCA. In another study, we detected temporal arteritis with temporal artery ultrasound in 7 of 102 patients with 'pure' PMR [37]. MRI, MR-angiography, CT, CT-angiography or PET can be performed in those patients whose diagnosis remains ambivalent even after ultrasound examination.

This is an open study in a clinical setting. As the same rheumatologist performed history, clinical assessment and ultrasound examination the results of ultrasound examination may have been influenced by the clinical appearance. On the other hand, this setting provides an excellent diagnostic approach. Both rheumatologists (W.A.S. and A.N.) have a long experience both in rheumatology and ultrasound.

This study does not provide information on sensitivities, specificities or rates of agreement between sonographers as this has been shown before. Previously published data from our institution indicated that even with a more blinded approach, sensitivities, specificities, and rates of agreement between sonographers reached high levels. The rate of agreement for temporal artery vasculitis was 95% [14] and for the detection of vasculitis in other arteries than the temporal arteries 89% [17]. Therefore, we have not performed temporal artery biopsy routinely any longer in patients with suspected GCA, but we have continued to biopsy ambivalent cases.

We have been using two different ultrasound machines over the 10-yr period of this study. Both higher image quality and increasing experience with the examination technique may have contributed to the greater number of patients with LV-GCA since 2003, although the number of LV-GCA patients had already increased before we started to use the new equipment.

It will be interesting to investigate the appearance of the proximal arm artery wall changes with long-term corticosteroid therapy. The wall swelling of the temporal arteries disappears after a mean of 2–3 weeks in most patients [14], but it remains much longer at the proximal arm arteries. The echogenicity increases because of less oedema and increasing fibrosis but none of our patients developed organ-threatening occlusions at follow-up [18].

We conclude that LV-GCA occurs more frequently than thought before. Axillary artery ultrasound is a simple, cheap and quick examination that provides characteristic findings in LV-GCA. Consequently, use of this test in patients with PMR, temporal arteritis, arm claudication, unclear inflammation or PUO leads to a greater number of cases diagnosed with LV-GCA. This study with consecutive patients confirms the results of previous retrospective studies that show differences between patients with cranial and with LV-GCA.

Rheumatology key messages

- LV-GCA occurs more frequently than previously assumed.
- Axillary artery ultrasound aids in diagnosing LV-GCA.
- Patients with LV-GCA differ from those GCA patients without proximal arm involvement.

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