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Temporal artery biopsy for suspected giant cell arteritis: a retrospective analysis

Döndü Üsküdar Cansu[1] · Hava Üsküdar Teke[2] · Cengiz Korkmaz[1]

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Abstract

Temporal artery biopsy (TAB) is one of the diagnostic criteria of giant cell arteritis (GCA) according to 1990 ACR criteria and remains a tool for diagnosis. Although clinicians perform TAB with an intent to confirm suspected GCA, some biopsies result in negative and some lead to non-GCA diagnoses. We aim to review the diagnoses after TAB biopsy performed for suspected GCA and also wanted to evaluate the diagnostic changes and concomitant diseases that develop over time. The patients who had undergone TAB for suspected GCA were identified using the record entry code for TAB. Patients meeting the classification criteria for GCA were designated as the GCA group and not meeting criteria were designated as a non-GCA group. Other classification criteria were implemented for the non-GCA group diseases. A total of 51 patients (Female: 62.7%, median age: 72.1 ± 7.4 years) who had undergone TAB for suspected GCA were evaluated. TAB was positive in 23 (69.6%) of the 33 patients who met the GCA classification criteria. No significant difference was found between TAB-positive and TAB-negative GCA patients in terms of clinical and laboratory parameters. In the non-GCA group, 12 patients had isolated polymyalgia rheumatica (PMR), and the diagnoses of the remaining six patients were as follows: four large vessel vasculitis (LVV) not satisfying GCA diagnostic criteria, one chronic myelomonocytic leukemia (CMML), and one amyloidosis. TAB was negative in all patients with isolated PMR. TAB showed primary amyloidosis in one patient. Out of 33 GCA patients, 21 had "isolated" GCA, four had GCA + Rheumatoid arthritis (RA), seven had GCA + PMR, and one had GCA + polymyositis. RA was diagnosed antecedent to GCA in two patients, and after GCA in the other two patients. One of the patients had developed GCA 20 years after polymyositis had been diagnosed. TAB was found to be positive in two-thirds of patients with suspected GCA. Late-onset RA and rarely other inflammatory rheumatic diseases may develop in the course of GCA.

Keywords Giant cell arteritis · Biopsy · Rheumatoid arthritis · Polymyositis · Arthritis

Introduction

Although the importance of temporal artery biopsy (TAB) has decreased in recent years due to the more frequent use of radiological imaging methods, TAB is still used as a diagnostic tool for giant cell arteritis (GCA). Temporal arteries might be inflicted by other diseases that are likely to mimic GCA. Therefore, TAB is useful not only to establish GCA diagnosis but also to identify other diseases. Systemic amyloidosis, antineutrophil cytoplasmic antibody-positive or negative vasculitis, and lymphoproliferative diseases might be diagnosed by TAB [1–4]. Even in the absence of typical GCA symptoms, TAB has proven applicable for unexplained hyperinflammatory conditions of the elderly [5, 6]. Of note, some patients remain asymptomatic although their temporal artery has indeed been inflamed (occult GCA) whereby TAB helps us determining inflammation involving the temporal arteries [7–9]. It is well known that GCA and polymyalgia rheumatica (PMR) are related to each other. In addition to the close relationship between GCA and PMR, they may have some clinical similarities with late-onset rheumatoid arthritis (RA) which requires taking RA into account in

✉ Döndü Üsküdar Cansu
ducansu@hotmail.com

Hava Üsküdar Teke
havaus@yahoo.com

Cengiz Korkmaz
ckorkmaz@ogu.edu.tr

[1] Division of Rheumatology, Department of Internal Medicine, Eskişehir Osmangazi University, Eskişehir 26480, Turkey

[2] Division of Hematology, Department of Internal Medicine, Eskişehir Osmangazi University, Eskişehir, Turkey

differential diagnosis [10–14]. To this end, understanding the cross-sectional clinical picture corresponding to the biopsy results in the patients who had undergone TAB is essential in addition to be cognizant of whether any other inflammatory rheumatic diseases will emerge over time. We aimed to review the diagnoses after TAB biopsy performed for suspected GCA and would also like to evaluate concomitant diseases that develop over time.

Materials and methods

Patient selection

This cross-sectional cohort study included patients who were examined in the rheumatology department and underwent TAB for suspected GCA between 1998 and 2019 (in our center, all patients pre-diagnosed with GCA undergo TAB). Patients were selected using the TAB entry code in hospital records. Patients who met the American College of Rheumatology 1990 Criteria for the Classification of GCA were called a GCA group and those who fail to meet those criteria were accepted as a non-GCA group. Within the non-GCA group, those fulfilling the Bird classification criteria were further stratified as PMR groups, and those left as a group of other diseases. Any other inflammatory rheumatic diseases (e.g. RA) co-existing with GCA or PMR were identified as per their relevant diagnostic criteria [15–18].

The clinical and physical examination findings of the patients were retrospectively reviewed. Steroid initiation time and its dosage were recorded. TAB was performed unilaterally in all patients. The size of TAB materials was not evaluated in the study. Biopsy materials were stained with hematoxylin–eosin and evaluated in terms of the presence of inflammatory cell infiltration, fragmentation, giant cell, and other histologic findings.

Laboratory testing and imaging

Data collected included complete blood count (CBC), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and alkaline phosphatase (ALP) values; positron emission tomography-computed tomography (PET-CT) findings, if available; and rheumatoid factor (RF) and anti-cyclic citrullinated peptide antibody (anti-CCP) status and findings on plain hand X-ray for peripheral arthritis patients.

Our study was approved by the local ethics committee with decision number 2018-279.

Statistical analysis

Continuous variables are presented as Average ± Standard Deviation. Categorical variables are presented in percentages (%). Shapiro Wilk's test was used to investigate whether a set of variables has a normal distribution. In the event of non-normal distribution, groups were compared using the Mann–Whitney *U* test, in the case of two groups. Cross tabulation was implemented and analyzed using Pearson exact chi-square, Yates' chi-square, and Fisher's exact chi-square tests. Analyses were carried on using the software IBM SPSS Statistics 21.0 (IBM Corp. Released 2012. IBM SPSS Statistics for Windows, Version 21.0. Armonk, NY: IBM Corp.). A p value of < 0.05 was considered to have statistical significance.

Results

Features of the TAB patient group

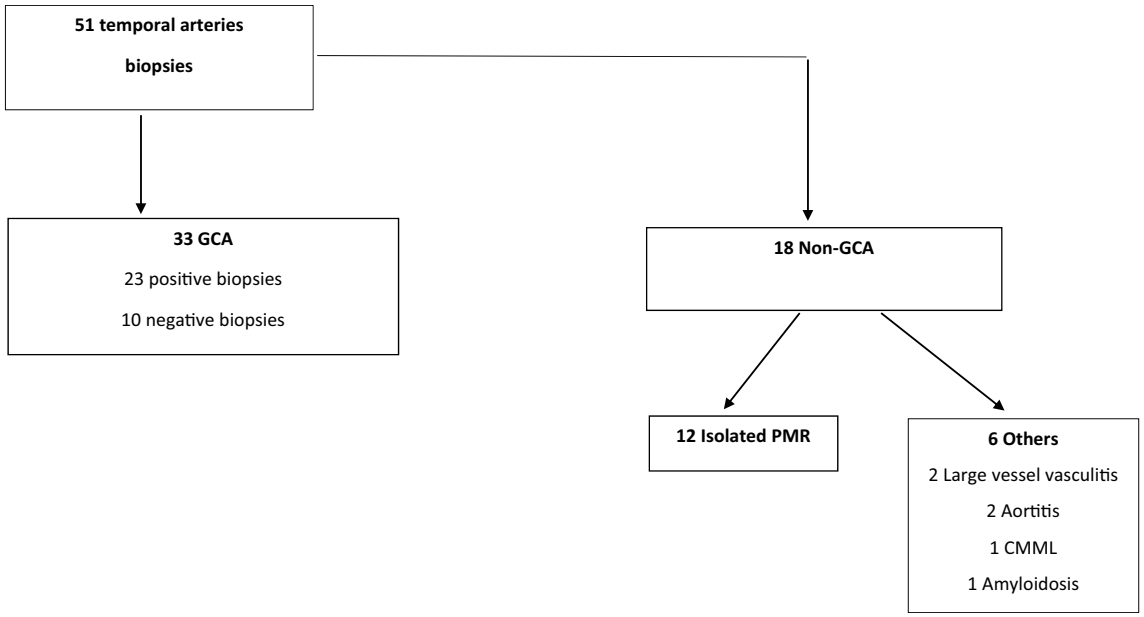
We have reviewed a total of 51 patients who had undergone TAB. In this cohort, 32 patients were female, and the average age was 72.1 ± 7.4 (66–78) years. Patients were divided into two groups based on GCA classification criteria, 33 in the GCA group, and 18 in the non-GCA group. In the GCA group, 21 patients had isolated GCA, four had GCA + RA, seven had GCA + PMR, and one had GCA + polymyositis. The Non-GCA group is composed of 12 isolated PMR and six other diseases. (The study design and disease groups by TAB results are given in Fig. 1).

In the entire cohort, 7.8% of the patients have a visual loss, 19.6% had a fever, and 51% had joint pain. When we compared the GCA group to the isolated PMR subgroup, there were significant differences in terms of headache, visual loss, joint pain, tenderness in the temporal artery, PMR prevalence, and steroid dosing, whereas no significant difference was determined in laboratory findings or any other findings. As expected, the steroid dosage used in the GCA group was higher than that used in the isolated PMR subgroup.

Features of GCA patients

GCA group composed of 33 patients, of whom 19 were women. Average age and disease duration were 73.7 ± 7.7 years and 6.46 ± 13.5 years, respectively. The chief complaint on admission was headache, at a rate of 69.6%. Of the patients, 42% had joint pain and 93.9% had temporal artery abnormalities. TAB was positive in 23 of 33 patients, the other ten had negative results. Within the GCA group, patients with positive vs negative biopsy results had no significant difference in terms of their clinical or laboratory findings.

All patients received steroid treatment, at an average dose of 40.7 mg/day. Three of the patients were administered pulse steroids due to visual loss. The steroid was given in six patients before TAB (median 2.5 (2–3.75) and given in



GCA; Giant cell arteritis, PMR; Polymyalgia rheumatica, CMML; Chronic myelomonocytic leukemia

Fig. 1 Study design and results of temporal artery biopsies.

27 patients after TAB (median 2 (1–5) days). Methotrexate was used in 66.7% ($n=22$) of the patients while azathioprine was used in 15.2% ($n=5$) of the patients. Tocilizumab was prescribed in only one patient (Case 5), because high-dose steroids were contraindicated due to steroid-induced osteoporotic fracture. (The patients' demographics and laboratory findings for 51 TAB cases who were diagnosed with GCA and isolated PMR are given in Table 1).

Features of non-GCA patients

Non-GCA group composed of 12 isolated PMR patients and six other patients with neither GCA nor PMR. All of those six patients were female, at an average age of 65.5 ± 4.7 years. PET-CT revealed that two patients had aortitis and two had large vessel vasculitis (LVV) which does not meet GCA diagnostic criteria. One of the patients had CMML and for the last one, the TAB result indicated amyloidosis. (The clinical and laboratory findings for six TAB cases who were diagnosed with a non-GCA and non-PMR disease are given in Table 2).

Concomitant disorders associated with GCA or PMR

Among 33 GCA patients, 4 (12%) had RA and one had polymyositis. On average, patients in RA subgroup were 80 ± 4.9 years old, with an average age at GCA diagnosis of 72 ± 7.7 years. RF and anti-CCP were positive in two of these patients, and joint X-rays showed erosion in all four cases. Diagnosis of RA was made in the two patients before GCA (9 years earlier and 6 months earlier), whereas in the other two patients after GCA (9 years later and 11 years later). In the sole polymyositis patient, GCA developed 20 years after polymyositis had been diagnosed. (The features of five patients who were diagnosed with RA or polymyositis in addition to GCA are given in Table 3).

Discussion

As European League Against Rheumatism (EULAR) outlined in their updated recommendations, TAB remains to be a tool for GCA diagnosis, although imaging of the temporal arteries by experienced experts by ultrasound conducted at the early period may be used as an alternative [19, 20]. The diagnostic value of TAB depends on the length of the collected specimen and whether TAB was taken in a unilateral or contralateral fashion [21, 22]. Among our patients who were fulfilling GCA criteria, TAB was positive for GCA in 69.6%. On the other hand, we have figured out that TAB was requested in one-fourth of the patients with isolated PMR. According to the literature data, 25% of PMR patients present with headache. PMR patients are prone to suffer headaches due to interspinous bursitis or pain radiating from the pectoral girdle both of which may emerge during disease. In such cases, TAB may be performed to rule out GCA [23].

In our case series, four patients of advanced age met the classification criteria neither for GCA nor for PMR

Table 1 Patient demographics and laboratory results in temporal artery biopsy study cohort; groups of giant cell arteritis and polymyalgia rheumatica

	All study group undergoing temporal artery biopsy	GCA group	Isolated PMR group	*P* value
N	51	33	12	–
Average age, mean ± SD, year Median (Q1–Q3), year	72.1 ± 7.4 73 (66–78)	73.7 ± 7.7 74 (68–79)	71.4 ± 6.2 73.5 (65–76)	0.315
Gender, woman,%	32, 62.7%	19, 57.6%	7, 58.3%	1
GCA, PMR diagnosis age, mean ± SD, year (Temporal artery biopsy age) Median (Q1–Q3), year	68.9 ± 6.9 69 (64–69)	69.7 ± 6.9 (65–73)	69.3 ± 7.1 69 (64–74)	0.929
GCA or PMR disease duration, mean ± SD, year Median (Q1–Q3), year	4.81 ± 11 1.5 (0.5–5)	6.46 ± 13.5 3 (0.4–7)	2.1 ± 1.2 1.7 (1–3)	0.658
Headache, *n*,%	34, 66.7%	27, 81.8%	3, 25%	0.001
Vision loss, *n*, %	4, 7.8%	4, 12%	0	< 0.0001
Joint pain, *n*, %	26, 51%	14, 42%	11, 91.6%	0.009
Presence of PMR, *n*, %	19, 37.3%	7, 21%	12, 100%	< 0.0001
Problem in temporal artery examination, *n*, %	31, 60.8%	31, 93.9%	0	< 0.0001
Temporal artery sensitivity, *n*,%	26, 51%	26, 78.7%	0	< 0.0001
Laboratory findings				
Hb, mean ± SD, gr/dl Median (Q1–Q3)	11.5 ± 1.51 11.8 (10.6–12.9)	11.5 ± 1.4 11.6 (10.6–12.9)	12.1 ± 1.2 12.2 11.7–13.1)	0.226
WBC, mean ± SD, /10^9/L Median (Q1–Q3)	9.6 ± 2.6 8.8 (8–10.4)	9.5 ± 2.9 8.5 (8–10.4)	9.8 ± 2 9.6 (8–10.3)	0.424
PLT, mean ± SD, /10^9/L Median (Q1–Q3)	374 ± 121 366 (279–469)	379 ± 126 366 (279–469)	379 ± 118 387 (298–464)	0.990
ESR, mean ± SD, mm/h Median (Q1–Q3)	93 ± 26 101 (83–111)	97 ± 22 101 (85–111)	80 ± 35 80 (46–107)	0.115
CRP, mean ± SD, mg/dl Median (Q1–Q3)	9.7 ± 8.9 7.39 (3.1–13.6)	10 ± 9 9.5 (3.8–15)	7.2 ± 5.5 5.4 (4.1–10)	0.371
ALP, mean ± SD Median (Q1–Q3)	206 ± 144 (43–613) 173 (96–230)	248 ± 163 203 (127–338)	143 ± 54 137 (95–190)	0.082
Steroid use, *n*,%	51, 100%	33, 100%	12, 100%	NA
Steroid dose, mean ± SD, mg/day Median (Q1–Q3)	36.4 ± 19 40 (15–60)	40.7 ± 18.6 40 (30–60)	19.3 ± 12.7 13.8 (0–22)	0.001

GCA Giant cell arteritis, *PMR* Polymyalgia rheumatica, *Hb* Hemoglobin, *WBC* White blood cell, *PLT* platelet count; *ESR* Erythrocyte sedimentation rate, *CRP* C-reactive protein, *ALP* Alkaline phosphatase

but had elevated acute phase reactants and constitutional symptoms. PET scans of these patients showed inflammation along the aortic wall. It is well known that large vessel involvement had been reported in almost half of the TAB-positive GCA patients [24]. According to the 2018 Draft Classification Criteria (DCC) for GCA presented at the 2018 ACR [25]., FDG-PET activity throughout the aorta was considered as imaging evidence for GCA. In line with this information, we can claim that these four cases can be included in the GCA group. In addition to FDG-PET activity, in two of these four patients, TAB showed fragmentation of internal elastic lamina and intimal hyperplasia without inflammatory cell infiltration. These findings may be suggestive of vasculitis according to the 2018 DCC criteria. However, we could not include these four patients in the GCA group, because DCC has not been approved by ACR and EULAR. The most important conclusion we can draw here is that we should use imaging methods more frequently in the diagnosis of GCA.

Besides GCA, temporal arteries are vulnerable to effects arising from other diseases. Therefore, it is possible to come up with non-GCA diagnoses upon TAB. In our series, in collaboratively similar terms, one patient was diagnosed with amyloidosis and another one with CMML. Reports in the literature have mentioned non-GCA-related inflammatory involvement identified through TAB such as chronic lymphocytic leukemia, eosinophilic granulomatosis with polyangiitis, and amyloidosis [2, 3]. Although there was a headache in patients with isolated PMR patients who underwent TAB, there was no temporal artery sensitivity or abnormalities. TAB was not positive in any of these patients. In line with previous literature data, this result may suggest that

Table 2 Clinical and laboratory features in temporal artery biopsy study cohort; remaining 6 patients without giant cell arteritis and without polymyalgia rheumatica

N	6
Gender, *n*,%	6, 100% female
Average age, mean ± SD, year	65.5 ± 4.7 (59–72)
Average age of diagnosis, mean ± SD, year	64.1 ± 4.7 (59–72)
Diagnosis	Aortitis, 2 Large vessel vasculitis, 2 CMML, 1 Amyloidosis, 1
PET-CT positivity, *n*,%	4, 66.7%
Hb, mean ± SD (min–max), gr/dl	10.2 ± 1.7 (8–12.6)
WBC, mean ± SD (min–max) /10^9/L	9.7 ± 2.5 (7.5–13.4)
PLT, mean ± SD (min–max) /10^9/L	338 ± 116 (224–543)
ESR, mean ± SD (min–max), mm/h	98 ± 21 (65–118)
CRP, mean ± SD (min–max), mg/dl	8.9 ± 11.7 (0.62–32)
ALP, mean ± SD (min–max),	116 ± 61 (43–206)
Number of patients receiving steroids, *n*,%	6, 100%

PET–CT Positron Emission Tomography–Computed Tomography, *CMML* Chronic myelomonocytic leukemia, *Hb* Hemoglobin, *WBC* White blood cell, *PLT* platelet count; *ESR* Erythrocyte sedimentation rate, *CRP* C-reactive protein, *ALP* Alkaline phosphatase

there is no need for TAB in patients without TA sensitivity [26].

An interesting aspect of our study is that we have encountered other inflammatory rheumatic diseases during GCA. We have determined seropositive erosive RA in four patients and polymyositis in another one. Both GCA and PMR occur during advanced decades of life, and hence differential diagnosis is inevitable to rule out late-onset RA. From another perspective, it is possible to recognize GCA, PMR, and late-onset RA as an intermingled state of illness. They may convert into each other at various rates or may have similar manifestations.[27, 28]. Musculoskeletal symptoms in GCA are common and varied. Although PMR stands out as the most well-known, studies have also investigated co-occurrence of peripheral arthritis, pitting edema, tenosynovitis, and, albeit rarely, RA [10–14]. We have identified three previous studies with different methodologies and patients' numbers where the prevalence of peripheral arthritis and RA varied at a range of 4–15 and 2–4%, respectively, among patients followed-up with GCA [10–12]. In our study, peripheral arthritis was similarly frequent, in 15%, but the prevalence rate of RA was as high as 12%. (Data from previous studies on GCA and RA association and our data are given in Table 4). However, if we add four patients to the GCA group considering the DCC, the frequency of RA is determined as 10.8%. Our higher rate of RA prevalence is attributable to our smaller number of GCA patients in comparison to the other three studies or might be related to "Berksonian bias". All three studies mentioned above were conducted before the introduction of anti-CCP for routine use, and as a consequence, our study is likely to include more true RA patients. That being said, given the rate of RA prevalence among the normal population lies in the range of 0.5–1%, the mentioned figure between 2 and 4% is still higher than the worldwide prevalence [29]. When our study and three former studies are collectively evaluated, post-GCA RA occurred after 2 months, at the earliest, and after 6 years, at the latest, while pre-GCA RA occurred 4 months in advance, at the earliest, and 19 years in advance, at the latest. Putting together, the association between GCA and RA may take place over a long period. Therefore, our results suggest not a random coincidence but rather a set of common genetic and environmental associations are responsible for the relationship among RA, PMR, and GCA. Of note, patients of advanced age who have RA may in a later period develop GCA, and, similarly, RA may arise at an early or late stage during GCA. This association has a further interesting aspect; such that, small and medium-sized arteries may get inflamed and large-vessel involvements such as aortitis and Takayasu arteritis-like vascular involvement may occur throughout RA [30–34]. The reason for the aforementioned vascular inflammatory association is yet unknown. Nevertheless, it seems that a possible cause is the non-specific arterial response against

Table 3 Features of four giant cell arteritis + rheumatoid arthritis patients and one giant cell arteritis + polymyositis patient

Case	Diseases	Age	Gender	GCA diagnosis age	RA/PM diagnosis age	Time between two diseases	RF	Anti-CCP	Erosion	PMR	Extraarticular involvement
1	GCA + RA	78	M	65	74	RA after 9 years	−	−	+	−	Neuropathy
2	GCA + RA	85	M	67	78	RA after 11 years	+	+	+	−	SC nodule
3	GCA + RA	83	M	82	73	GCA after 9 years	+	+	+	−	−
4	GCA + RA + PMR	74	F	74	74	GCA after 6 months	−	−	+	+	−
5	GCA + PM + PMR	65	F	65	46	GCA after 20 years	−	−	−	+	−

GCA Giant cell arteritis, *RA* Rheumatoid arthritis, *PMR* Polymyalgia rheumatica, *PM* Polymyositis, *M* Male, *F* Female, *RF* Rheumatoid factor, *anti-CCP* Anti-cyclic-citrullinated peptide antibodies, SC; Subcutaneous

Table 4 Literature studies on association of giant cell arteritis and rheumatoid arthritis

Study	Number of patients	Study design/Follow-up time/ Cohort features	Frequency of peripheral arthritis	Frequency of RA	RA patient characteristics	Comment
Our study	33 GCA	21-year cohort	15% ($n=5$)	12% ($n=4$) RA	* 2 of 4 patients before GCA diagnosis (6 months and 9 years ago) 2 after (9 years and 11 years later) * RF (+) in 2 patients, anti-CCP (+) in 2 patients *Erosion in all 4 * Age of diagnosis of RA 74.75 ± 3.3 years (73–78)	Frequency of RA in patients with GCA is higher than expected
Gran JT 2000 [13]	44 GCA *(29 GCA, 15PMR + GCA)*	Patients diagnosed between 1987–1983	6% ($n=3$)	2% ($n=1$) RA	* 1 female patient * Age of diagnosis 70 years * The time between GCA and RA diagnosis 43 months * RF negative * No erosion	Aetiopathogenic differences may exist between PMR and GCA as peripheral arthritis and the development of RA was observed among the former patient group only
Salvarani C 1999 [14]	128 GCA	42-year cohort (91% positive biopsy)	13% ($n=17$)	2% ($n=6$) RA	* RA in 3 of 6 patients before GCA (4 months-2.5 years-4 years) RA in 3 of patients after GCA (2 months-6 months-6 years) * All seronegative * 1 has erosion	Musculoskeletal symptoms in GCA are common and varied. Most appear linked temporally to the underlying GCA, indicating that the nature of this illness and its clinical expression are broader than often considered
Ginsburg WW 1985 [15]	520 GCA	Patients diagnosed with biopsy-proven GCA between 1970–1982, patients with polyarthritis and seronegative were included (3 patients who were not seropositive)	4% ($n=22$)	4% for 22 patients 3.6% if 19 patients are admitted	* Seropositive erosive 3 patients * They were diagnosed with RA on average 19 years ago from GCA * 19 patients seronegative * 10 of 19 patients have radiological erosion or narrowing of the joint space * RA developed approximately 1 year after GCA diagnosis in 7 patients	In patients over the age of 50 who have onset of seronegative polyarthritis and a high erythrocyte sedimentation rate, GCA should be considered as a possible cause of the clinical symptoms

GCA Giant cell arteritis, *RA* Rheumatoid arthritis, *PMR* Polymyalgia rheumatica; *RF* Rheumatoid factor, *anti-CCP* Anti-cyclic-citrullinated peptide antibodies

the chronic inflammatory condition created by common genetic, environmental, and hormonal factors.

Our study has some limitations. Its retrospective design may have caused some missing data. TAB was performed unilaterally in all our patients. Although care was taken to take the biopsy sample at least 2 cm, this could not be achieved in every patient. This situation may have negatively affected the TAB results. A relatively small patient number is another limitation of this study. On top of that, it is also limited by the lack of a RA control group. A study cohort based on the reverse point of view, which includes late-onset RA patients and searches for any emergence of GCA might be necessary to determine the frequency of overlaps between two entities. However, while following RA patients, the involvement of temporomandibular joint might readily be blamed for the indeed GCA-induced symptoms such as temporal headache and jaw pain when eating. Similarly, occipital pain could be explained by cervical vertebral involvement which also takes place during RA, misleading GCA to be overlooked. Moreover, long-term steroid use in RA patients is likely to suppress GCA symptoms. For these reasons, the determination of the GCA prevalence among RA patients allocated in such a study will require extensive clinical and radiological examinations.

After all, in addition to GCA identification of two-thirds of suspected GCA, TAB is also important for demonstrating non-GCA diseases, although rarely. Moreover, the course of GCA may accommodate various inflammatory rheumatic diseases. Taking into account the results from our study and former studies available in the literature, the co-existence of GCA, RA, and PMR may be concurrent or sequential. For this particular reason, while managing seronegative arthritis patients and specifically those of advanced age, clinicians have to keep such overlaps in mind to ensure proper and timely diagnosis and treatment. As an aid to further elaborate the possible association of these diseases, it would be interesting to assess RA patients from the GCA perspective.

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Authors contribution Study concept and design: DUC, HUT and CK; analysis and interpretation of data: DUC, HUT, and CK; drafting of the manuscript: DUC and CK, critically revision of the manuscript for important intellectual content: DUC, HUT and CK; final approval of the version to be published: DUC, HUT and CK.

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Compliance with ethical standards

Conflict of interest We have no conflict of interest.

Ethical approval Ethical approval was obtained from Eskisehir Osmangazi University Ethics Committee (20 November 2018, No.279).

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