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Comparisons of clinical manifestations and prognosis between giant cell arteritis patients with or without sensorineural hearing loss

A retrospective study of Chinese patients

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Abstract

Auditory manifestations has rarely been mentioned in studies concerning giant cell arteritis (GCA). This study explores the proportion of hearing loss (HL) in Chinese GCA patients and investigates the differences in clinical features between GCA patients with and without HL.

The study retrospectively reviewed the clinical records of 91 patients diagnosed with GCA at Peking Union Medical College Hospital (PUMCH) from November 1998 to October 2017. GCA diagnoses were reconfirmed according to the American College of Rheumatology 1990 criteria. Diagnosis of HL was made based on a patient's symptoms combined with physical examination or ear-nose-throat (ENT) audiometry tests. Subgroup analysis was conducted according to the occurrence of HL.

Totally 23 patients (25.3%) had HL. A higher percentage of males (65.2% vs 38.2%, $p=0.025$) was seen in HL group. Symptoms such as headache (91.3% vs 61.2%, $p=0.011$), visual loss (56.5% vs 32.4%, $p=0.039$) and CNS symptoms (39.1% vs 17.6%, $p=0.035$) were more frequent in HL group. Moreover, they were more likely to have smoking history ($p=0.019$), lower lymphocyte count ($p=0.049$), positive ANA or APL ($p=0.047$, $p=0.017$) or negative biopsy results ($p=0.015$). Symptom like myalgia (26.1% vs 66.2%, $p=0.001$) as well as comorbid disease like coronary artery disease ($p=0.037$) and hypertension ($p=0.040$) was more frequent in patients without HL. Either C-reactive protein (90.91 ± 65.86 vs 76.05 ± 61.15 mg/L, $p=0.347$) or erythrocyte sedition rate (83.04 ± 29.61 vs 93.69 ± 26.78 mm/h, $p=0.136$) was high in both groups but the differences were not significant. Meanwhile, no significant differences were found in age, disease course, vascular involvement or prognosis between the two groups. Unilateral HL tended to happen at the same side with unilateral headache, visual loss, scalp tenderness or jaw claudication.

HL is probably not rare in GCA patients and is more frequently to be seen in patients presented with headache, visual loss or CNS symptoms. Differentiation of HL is necessary for specialists and GCA should be considered as a potential diagnosis especially in HL patients with high inflammatory markers. Auditory assessment should be conducted in GCA management.

Abbreviations: ENT = ear-nose-throat, GCA = giant cell arteritis, HL = hearing loss, SSHL = sensorineural hearing loss.

Keywords: giant cell arteritis, hearing loss

1. Introduction

Giant cell arteritis (GCA), also known as temporal arteritis or granulomatous arteritis, is the most common type of arteritis found in western countries. GCA usually affects females over 50 years old, and its incidence increases with age.[1] Large or medium-sized arteries such as aorta and its primary branches, as well as cranial vessels, are frequently involved,[2] causing typical symptoms such as temporal headache, jaw claudication, scalp tenderness, or temporal artery abnormalities (tenderness, decreased pulsation, etc). Clinical manifestations of GCA can vary in different patients. Transient or permanent visual loss caused by ischemia of vessels supplying the eyes is considered one of the most serious symptoms.[3–5] Hearing loss (HL) was reported in GCA cases from time to time while previous research has seldom focused on the symptom. The mechanism underlying HL remains unclear. Previous studies have suggested that HL is caused by the involvement of vertebrobasilar or the terminal cochleovestibular vessels, and by narrowing or the occlusion of related vessels.[6,7] Also, the exact incidence of HL in GCA patients still remains controversial. Hausch et al have conducted a 10-year retrospective review on 271 GCA patients from Geisinger Medical Center[8] and have found that only 4 patients had HL symptoms. Their data suggested that HL can occur at the onset of GCA or develop during the course of the disease, and it

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can recover completely or partly after treatment. Moreover, Amor-Dorado et al have conducted a prospective study on 44 GCA patients and discovered that vestibular disorders were present in 39 (90%) cases; however, the exact number of patients with HL was not mentioned in the article while it demonstrated 12 (27.3%) patients had hearing improvement after treatment.[9] The above data implied that HL is not a rare event in patients with GCA, and that it might get ignored due to poor recognition.

The aim of this research was to investigate the occurrence of HL and the clinical manifestations of GCA in patients with and without HL.

2. Materials and methods

2.1. Patients

One hundred seventeen patients diagnosed with GCA at Peking Union Medical College Hospital, Beijing, China (PUMCH) between November 1998 and October 2017 were enrolled. Patients' clinical data, including medical records, and follow up, were retrospectively reviewed. After excluding 26 cases due to obviously incomplete data, 91 patients were included in the study. GCA diagnoses were reconfirmed according to The American College of Rheumatology 1990 classification criteria[10]:

(1) 50 years of age;
(2) localized headache of new onset or new type;
(3) tenderness or decreased pulsation of the temporal artery;
(4) elevated erythrocyte sedimentation rate (erythrocyte sedition rate [ESR] ≥50 mm/h);
(5) histologic changes of arteritis: either granulomatous lesions, usually with multinucleated giant cells, or diffuse mononuclear cell infiltration.

The presence of 3 or more criteria were enough to make a diagnosis of GCA (sensitivity 93.5%, specificity 91.2%). Temporal artery biopsy is still the gold standard for the diagnosis of GCA.

2.2. Methods

We reviewed and summarized the medical history of patients, including symptoms and signs, physical examinations, comorbid diseases, previous medical history, laboratory results and imaging results, histological results as well as follow-up data. Ninety-one patients were divided into 2 groups, that is, those with HL and those without. HL was diagnosed by ear–nose–throat (ENT) specialists based on obvious hearing impairment complained by the patients or ENT audiometry tests such as pure tone audiometry test. Stabilization was defined as relief after treatment with no relapsing symptoms after reduction of glucocorticoid; recurrence was defined as relief after treatment while symptoms relapsed or treatment-related complications such as infection occurred during the reduction of glucocorticoid.

This study has been approved by the Ethics Board of PUMCH.

2.3. Statistical analysis

Data analysis was performed using SPSS 19.0 software (IBM SPSS Statistics 19). General information was analyzed by descriptive statistics. Mean value and standard deviation were calculated for continuous variables. Categorical variables were analyzed using frequency and percentage. Independent-sample t test was applied for analyzing continuous variables. Chi-square test was applied for categorical variables where frequencies of either group are no less than 5 (≥5) and total frequencies are no less than 40 (≥40), otherwise, Fisher exact test will be conducted. All tests were 2-sided, and $P < .05$ was considered statistically significant.

3. Results

3.1. Sociodemographic data and clinical manifestation

One hundred seventeen GCA patients were reviewed and 91 met the inclusion criteria. There were 23 patients (25.3%) with HL; among these, 4 patients had HL at the onset of disease. Sixty-eight patients experienced no HL during the disease course. In addition, 8 patients were diagnosed with GCA comorbid with HL during the last 5 years; among them, only 2 of them underwent pure tone audiometry test and the results both revealed sensorineural hearing loss (SSHL), 4 patients had mastoiditis features in magnetic resonance imaging, and 3 patients had unilateral HL at the same side with unilateral headache, visual loss, scalp tenderness or jaw claudication or consistent with the more severe side of those symptoms. The reason why many patients did not undergo pure tone audiometry test results was that most physicians did not realize the HL symptom may be related to GCA, and they usually advised patients to go to ENT clinic after being discharged from hospital; nonetheless, many patients did not follow the physician's advice due to various reasons.

Among 91 GCA cases, 50 (54.9%) were female (male: female ratio 1:1.22). However, more males were observed in the HL group (65.2% males vs 38.2% females, $P = .025$). In addition, no age differences were found between the 2 groups. Considering clinical manifestations, out of 91 GCA patients, 74 and 64 presented with fever and headache, respectively, which were also the most frequent symptoms. Patients with HL experienced more headache, visual loss and central nervous system (CNS) symptoms ($P = .011$, $P = .039$, $P = .035$), reported a lower ratio of myalgia ($P = .001$) and were also more likely to have smoking history ($P = .019$) compared to patients without HL. With respect to comorbid diseases, patients with HL had lower percentage of hypertension ($P = .040$), coronary heart disease (CHD) ($P = .037$), diabetes mellitus, cerebrovascular disease, malignancy, and dyslipidemia history, while the differences were not significant in diabetes mellitus, cerebrovascular disease, malignancy, and dyslipidemia history (Table 1).

3.2. Laboratory results

Comparisons of blood cell counts between the 2 groups illustrated that patients with HL had lower lymphocyte counts than patients without HL ($P = .049$). Eight out of 23 and 5 out of 13 GCA patients with HL showed positive antinuclear antibody (ANA) and antiphospholipid antibody (APL) (lupus anticoagulant, anticardiolipin antibody, anti-β2 glycoprotein 1 antibody). Ten out of 65 and 7 out of 61 GCA patients without HL had positive ANA and APL, respectively. The percentage of positive ANA and APL were significantly higher in HL group ($P = .047$, $P = .017$, respectively). Meanwhile, the level of C-reactive protein and ESR in both groups were higher than normal range, but there was no significant difference between the 2 groups (Table 2).

3.3. Artery involvement and biopsy results

No remarkable differences of artery distributions were found between the 2 groups when evaluating their imaging results. The number of patients receiving temporal artery biopsy was 11 out

Table 1

Clinical features and comorbid diseases of the patients with hearing loss and without hearing loss.

	GCA with HL n = 23 (%)	GCA without HL n = 68 (%)	*P*-value
Age, yr (diagnosis)	63.70 ± 7.58	65.87 ± 7.67	.244
Disease course, mo	10.72 ± 16.00	8.09 ± 17.81	.511
Sex			
Male	15 (65.2)	26 (38.2)	.025*
Female	8 (34.8)	42 (61.8)	
Headache	21 (91.3)	43 (63.2)	.011*
Fever	19 (82.6)	55 (80.9)	.854
Scalp tenderness or pain	6 (26.1)	18 (26.5)	.971
Tenderness and abnormal pulsation of temporal artery	6 (26.1)	14 (20.6)	.582
Visual loss	13 (56.5)	22 (32.4)	.039*
Myalgia	6 (26.1)	45 (66.2)	.001*
CNS symptoms	9 (39.1)	12 (17.6)	.035*
Jaw claudication	7 (30.4)	19 (27.9)	.819
Arthralgia	8 (34.8)	39 (57.4)	.061
GI symptoms	3 (13.0)	9 (13.2)	.963
Constitutional symptoms	16 (69.6)	45 (66.2)	.765
Weight loss*	16 (69.6)	34 (50.0)	.103
Atherosclerosis	5 (21.7)	28 (41.2)	.094
Smoking	13 (56.5)	20 (29.4)	.019*
Tumor family history	4 (17.4)	12 (17.6)	.972
Diabetes	3 (13.0)	11 (16.2)	.694
Cerebrovascular disease	1 (4.3)	7 (10.3)	.309
Coronary artery disease	1 (4.3)	12 (17.6)	.037*
Hypertension	4 (17.4)	28 (41.2)	.040*
Dyslipidemia*	5 (21.7)	26 (38.2)	.149

CNS symptoms: vertigo, transient ischemia attack, stroke; GI involvement: abdominal pain, abdominal distention; Constitutional symptom: fatigue, night sweat, anorexia.

CNS = central nervous system, GCA = giant cell arteritis, GI = gastrointestinal, HL = hearing loss.

* Significantly different.

of 23 and 36 out of 68, respectively. Five patients (45.5%) in HL group and 4 patients without HL (11.1%) were negative for biopsy results and no remarkable difference was found between the 2 groups ($P = .015$) (Table 3).

3.4. Treatment

All 23 GCA patients with HL received glucocorticoid treatment and 22 received immunosuppressive drugs. Only 2 patients

Table 3

Artery involvement and biopsy results in patients with or without hearing loss.

	With HL n (%)	Total	Without HL n (%)	Total	*P*
Intracranial vessels	11 (57.9)	19	23 (48.9)	47	.510
Extracranial vessels above aorta	17 (89.5)		41 (87.2)		.801
Extracranial vessels below aorta	11 (57.9)		33 (70.2)		.336
Biopsy negative	5 (45.5)	11	4 (11.1)	36	.015*

HL = hearing loss.

* The difference in proportion of patients who were biopsy negative are significant between two groups.

comorbid with atherosclerotic plaque and high risk of thrombosis were prescribed aspirin as an antiplatelet treatment. In the 4 patients who experienced HL at the onset of disease, no specific treatment was applied to deal with HL. Moreover, the conditions of HL after application of those treatment were described in only a small number of GCA patients when they were discharged from the hospital. Most of the patients did not deal with or pay attention to HL after they got out of the hospital, and this was mostly because that the doctors neglected to emphasize the importance of examination and follow-up of hearing.

3.5. Follow-up information

The average follow-up time was 86.04 (3–218) months. Five patients in the HL group and 9 patients without HL were lost during follow-up. No statistically significant difference was found between the 2 groups regarding the percentage of patients who were stable, unstable, developed a malignancy or died (Table 4).

4. Discussion

GCA is the most common systematic arteritis in European and American populations, with the approximate prevalence of 17/100,000 among people from North America and North Europe who are over 50 years of age.[1] Previously, it has been thought that GCA is uncommon in Asian populations, and according to the research published by Kobayashi in 2003, occurrence of GCA in Japan was only 1.47/100,000.[8,11,12] The incidence of GCA in China is still unknown, and limited research has been published. From January 1998 to December 2017, only 117 patients were

Table 2

Laboratory findings of GCA patients with or without hearing loss.

	GCA With HL			GCA without HL			
	Mean ± SD	n (%)	Total n	Mean ± SD	n (%)	Total n	*P*-value
ESR, mm/h	83.04 ± 29.61		23	93.69 ± 26.78		68	.136
CRP, mg/L	90.91 ± 65.86		23	76.05 ± 61.15		68	.347
ALB, g/L	32.26 ± 5.11		23	32.67 ± 4.47		66	.737
WBC, $\times 10^9$/L*	9.09 ± 4.55		23	9.18 ± 4.14		66	.939
LYM, $\times 10^9$/L	1.29 ± 0.51		23	1.55 ± 0.71		66	.049*
HGB, g/L	110.13 ± 21.02		23	105.80 ± 19.01		66	.390
PLT, $\times 10^9$/L	401.48 ± 141.94		23	372.11 ± 155.65		66	.410
ANA positive		8 (34.8)	23		10 (15.4)	65	.047*
ANCA positive		5 (22.7)	22		8 (12.9)	62	.274
APL positive*		5 (38.5)	13		7 (11.5)	61	.017*

ALB = albumin, ANA = antinuclear antibody, ANCA = antineutrophil cytoplasmic antibody, APL = antiphospholipid antibody, CRP = C-reactive protein, ESR = erythrocyte sedition rate, GCA = giant cell arteritis, HGB = hemoglobin, HL = hearing loss, LYM = lymphocyte, PLT = platelet, SD = standard deviation, WBC = white blood cell.

* Significantly different.

Table 4

Follow-up information of GCA patients with or without hearing loss.

	GCA with HL n (%)	Total	GCA without HL n (%)	Total	*P*
Stable	8 (44.4)	18	29 (49.2)	59	.726
Unstable	4 (22.2)		22 (37.3)		.252
Tumor	1 (5.6)		1 (1.7)		.648
Death	5 (27.8)		7 (11.9)		.103

HL = hearing loss.

diagnosed with GCA in PUMCH, which illustrated the rarity of this disease in China. This single-centered research included a total of 91 GCA patients, who were younger at the onset of the disease (63.70, 65.87, respectively) compared to the reported average age (76.7 years old).[13] Researchers from Xiangya Hospital, Hunan, China have reported 13 cases of GCA in 2003, and the average age they reported at the onset of disease was 43.1,[14] which was obviously younger than published data reported by previous studies and our data. The sample size in our study, or study conducted at Xiangya Hospital was not large enough to verify whether the observed age difference was due to ethnic difference. Further epidemiologic research should be conducted in the Chinese population, so as to improve the recognition, diagnosis, and treatment of GCA.

HL has rarely been reported in previous research. To the best of our knowledge, this is the largest research that addressed HL in Chinese GCA patients. In 1998, Hausch reported in a retrospective study that among 271 patients with GCA, only 4 patients reported HL.[8] Another prospective research by Amor-Dorado in 2003 revealed that 39 out of 44 patients (90%) had vestibular disorders, and 12 patients (27.3%) experienced hearing improvement after treatment; nonetheless, the exact number of patients with HL was not mentioned in the article.[9] This study illustrated that HL was not so rare as we thought, and this symptom can occur at any period of disease. In our study, 23 out of 91 patients (25.3%) had HL, and 4 patients experienced it at the onset of disease. The prevalence is consistent in general with the data published by Amor-Dorado, which demonstrates that HL is not rare in Chinese GCA patients. The manifestations of GCA at disease onset varies a lot in different patients while HL has seldom been regarded as a typical symptom of GCA. HL can be an underestimated symptom of GCA. The reason why the prevalence of HL varies in different research may be due to the neglect of the symptom in medical history taking and the small sample size of research. These results urge physicians to pay more attention to HL symptom and highlight the importance of ENT consultation, which is necessary in gaining a comprehensive understanding of the disease in treatment of GCA patients. On the other hand, some patients have HL at disease onset and they initially seek help from ENT clinic. According to our research, GCA patients with HL are more likely to experience symptoms such as headache, visual loss or CNS symptoms, and thus ENT specialists should take GCA into consideration when an old patient presents HL symptoms as well as headache, visual loss, CNS symptoms or fever. In our study, patients from the HL group reported a lower proportion of myalgia and history of diabetes mellitus, cerebrovascular disease, CHD, hypertension, and dyslipidemia. The underlying mechanism remained unclear.

The mechanism of HL is still unclear. Published research has suggested that it is caused by involvement of vertebrobasilar vessels or terminal branches of cochleovestibular vessels.[6,7] Three possible mechanisms of sudden SSHL have been suggested by Greco et al in their research, including virus infection, vascular occlusion, and immunologic factors. SSHL can be an isolated problem or the presenting symptom of a systemic disease, such as systemic lupus, or Wegner's granulomatosis.[15] Vascular hypothesis suggests that thrombosis or occlusion of vessels is a possible cause of SSHL. The immunologic hypothesis is based on the theory that circulating antibodies cross-react with inner ear antigens or activated T cells, thereby damaging the inner ear.[16,17] Several inner ear antigens have been detected; reductions in the T lymphocyte subpopulations, C3, C4, and C8 and an increase in the level of C3bc complement factor have been found in patients with SSHL.[18–20] Virus infection is another possible mechanism which suggests that viruses damage inner ear tissues or bones, thus directly causing SSHL. Nevertheless, no histopathological or infection evidence was found to support the hypothesis. Another hypothesis is that viruses can trigger antibodies targeting inner ear antigens such as type 2 collagen, causing tissue damage and HL.[19] Meanwhile, viruses can induce the production of anti-phospholipids (PLs) antibodies, causing acquired thrombophilia and SSHL. This mechanism is related to the vascular hypothesis and is consistent with the findings that SSHL has an association with anti-PLs.[21–24] Accordingly, the authors concluded that immunological theory is a more plausible one. This study demonstrated that GCA patients with HL have higher percentage of positive anti-PLs ($P = .017$), and higher prevalence of visual loss and CNS symptoms ($P = .039$, $P = .035$). These results were consistent with the suggested mechanism that anti-PLs antibodies and immune-mediated injury played an important role in HL. The percentage of positive ANA was also higher in HL group ($P = .047$), though no specific reports concerning the association between ANA and HL were found. The significant difference observed in ANA between the 2 groups could just be due to a bias caused by relatively small sample size, or there could be some potential relationship between the antibody and HL.

GCA effects mainly large or medium-sized arteries such as aorta and its primary branches as well as cranial vessels. Auditory dysfunction is frequently asymmetrical, and it is in keeping with the asymmetrical cranial manifestations reported in many GCA patients.[8,9] The results of our research are consistent with these findings. Currently, temporal artery biopsy is still the golden standard for GCA biopsy. In HL group, GCA patients with negative biopsy accounted for nearly half of the patients (5/11, 45.5%), while in patients without HL, only 11.1% (4/36) had negative results. A previous study has revealed that small-vessel vasculitis, which is defined as aggregates of mononuclear inflammatory cells surrounding a capillary, distant from an uninflamed temporal artery, is strongly associated with symptoms of polymyalgia rheumatica (PMR) in GCA patients with or without PMR.[25] So, the pathological features of GCA patients with HL may be different from the typically acknowledged histological findings, indicating another kind of GCA. The

difference is so impressive that physicians and pathologists should pay more attention to this phenomenon that HL in GCA is potentially associated with different biopsy result.

40% to 50% or more patients experience relapse in application of glucocorticoids or during the reduction of glucocorticoids, and long-term, large dose of glucocorticoids has been related to relapse.[26,27] According to the follow-up information, the ratio of stable patients, relapse, tumor occurrence, or death revealed no remarkable difference between 2 groups, while the percentage of stable patients was less than 50% in both groups and the relapse proportion was quite considerable (22.2% vs 37.3%). These data are consistent with the previous study where almost half of the patients experienced relapse, thus suggesting that more attention should be paid to this problem.

This study was conducted retrospectively, and the conditions of HL after glucocorticoid treatment were described in only a small number of GCA patients when they were discharged from the hospital due to the poor recognition of this symptom. According to our study, some GCA patients did not experience improvement in hearing, while other patients totally or partly recovered from HL after glucocorticoid treatment, which was consistent with previous research.[8,9] It is not clear whether the time from the onset of HL to glucocorticoid therapy influences recovery, or it is the kind of HL that matters. Previous research has revealed that early use of glucocorticoid in patients with idiopathic sudden SSHL leads to a better prognosis.[28] Accordingly, ENT specialists may offer glucocorticoid treatment early in patients diagnosed with idiopathic sudden SSHL after making comprehensive assessment of the patients and administering audiometric follow-up during treatment.[29]

The challenge of GCA diagnosis is that the disease is rare in China and the clinical manifestations vary in different patients. In our study, we retrospectively reported 91 patients among which 25.3% reported HL, which illustrates that HL in GCA patients is not rare. Rheumatologists should pay more attention to this symptom and should seek ENT consultation to ensure a more comprehensive diagnosis and management of patients with GCA. Meanwhile, ENT specialists should be more alert and should consider GCA when dealing with an older patient presenting HL symptom as well as headache, visual loss, CNS symptoms or fever. Early recognition and steroid treatment may lead to a better prognosis of GCA and HL. Glucocorticoid could be offered early as an option and follow-up could be performed in patients diagnosed with idiopathic sudden SSHL.

5. Conclusions

This study demonstrated that HL was not rare in Chinese GCA patients. Rheumatologists, neurologists, ophthalmologists, and ENT clinicians should consider GCA when patients present HL as well as headache, visual loss, CNS symptoms or fever.

The results from the present study should be interpreted with caution. First, this is a retrospective study that had some missing clinical data. Second, the patients recruited in this study are all hospitalized patients with severe clinical symptoms which makes the clinical bias and lower estimated GCA prevalence. However, there are some strengths including a relatively large sample size from China mainland, and specific clinical focus on HL.

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