

Giant Cell Arteritis Misdiagnosed as Temporomandibular Disorder: A Case Report and Review of the Literature

Shoshana Reiter, DMD

Teacher
Department of Oral Rehabilitation

Ephraim Winocur, DMD

Lecturer
Department of Oral Rehabilitation

Carole Goldsmith, DMD

Instructor
Department of Oral Rehabilitation

Alona Emodi-Perlman, DMD

Teacher
Department of Oral Rehabilitation

Meir Gorsky, DMD

Professor
Department of Oral Pathology and Oral
Medicine

The Maurice and Gabriela Goldschleger
School of Dental Medicine
Tel Aviv University, Israel

Correspondence to:

Dr. Shoshana Reiter
Department of Oral Rehabilitation
The Maurice and Gabriela Goldschleger
School of Dental Medicine
Tel Aviv University
Tel Aviv, Israel
Fax: 972-3-6409250
Email: shosh5@bezeqint.net

Giant cell arteritis (GCA) is a systemic vasculitis involving medium and large-sized arteries, most commonly the extracranial branches of the carotid artery. Early diagnosis and treatment are essential to avoid severe complications. This article reports on a GCA case and discusses how the orofacial manifestations of GCA can lead to misdiagnosis of GCA as temporomandibular disorder. GCA should be included in the differential diagnosis of orofacial pain in the elderly based on the knowledge of related signs and symptoms, mainly jaw claudication, hard end-feel limitation of range of motion, and temporal headache. J OROFAC PAIN 2009;23:360-365

Key words: Giant cell arteritis, jaw claudication, temporomandibular disorders, trismus

Giant cell arteritis (GCA) is a systemic vasculitis involving the large and medium-sized vessels, particularly the extracranial branches of the carotid artery. It is more common in women (M:F ratio 2:5) and usually affects patients older than 50 years with an increased risk with age.¹ The highest prevalence of GCA has been reported in Scandinavian populations and in those with a strong Scandinavian ethnic background.²

Permanent visual loss may result from GCA due to ischemia of the optic nerve secondary to vascular occlusion. Therefore, GCA is considered a medical emergency. The clinician's awareness can reduce the prevalence of visual loss associated with GCA (from 30% to 15%).³ Additional life-threatening conditions including myocardial infarction, aneurysm of the aorta, infarction of the intestine, renal insufficiency, pulmonary embolism, transient ischemic attacks, and stroke may also be related to GCA.⁴ Therefore, early diagnosis and appropriate management are essential to avoid further complications.⁵

The most prevalent signs and symptoms of GCA include temporal headache, jaw claudication, polymyalgia rheumatica (PMR) (neck, shoulder, hip pain, morning stiffness), constitutional syndrome (asthenia, anorexia, and weight loss) and scalp tenderness.^{6,7} In addition to jaw claudication, orofacial manifestations of GCA include trismus, throat pain developing while chewing, changes in tongue sensation and tongue claudication, odontogenic pain, dysphagia, dysarthria, submandibular mass, lip and chin numbness, macroglossia, glossitis, lip and tongue necrosis, and facial swelling.⁸ Jaw claudication is caused by arteritis of the maxillary artery which leads to an ischemia of the masticatory muscles. Approximately 40% of patients with GCA have jaw claudication,^{8,9} but its prevalence has been reported as high as 65%.¹⁰

Jaw claudication is considered one of the predictors for an increased risk of permanent vision loss⁵ and could also represent a manifestation of other diseases, such as amyloidosis/multiple myeloma,¹¹⁻¹³ atherosclerosis of the main blood vessels,^{14,15} and Wegener granulomatosis.^{16,17} Therefore, these diseases should be included in the differential diagnosis when jaw claudication is suspected.

Limited range of motion (ROM) of the mandible associated with GCA is a less frequently reported sign compared to jaw claudication.¹⁸⁻²⁵ In a retrospective study,²⁴ 6.8% of the patients presented with jaw "trismus." These patients presented with a more aggressive disease, a higher percentage of eye involvement, and a shorter time required for diagnosis. The authors concluded that the prevalence of trismus is likely underestimated due to the physician's lack of awareness of this complaint that is easily confused with jaw claudication. In another study,²⁵ 36% of GCA patients reported limited ROM. However, there was no association between the limitation of ROM and severity of the disease, eye involvement and delay in diagnosis. The need to become familiar with trismus as a sign of GCA was emphasized.^{24, 25}

It must be pointed out that jaw claudication and its associated hard end-feel limitation of ROM (no increase in maximum mouth opening when applying mild steady downward pressure to the lower incisors) may lead the clinician to misdiagnose GCA as a temporomandibular disorder (TMD), especially in the elderly.^{26,27} The purpose of this article was to present a case of GCA and to review the orofacial manifestations of GCA which can lead to misdiagnosis of GCA as TMD.

Case Presentation

History

A 67-year-old woman who was referred by her dentist to an Orofacial Pain Clinic complained of left temporal headache, limitation of mouth opening, and pain upon chewing over the previous 2 weeks. The pain was initially controlled with paracetamol and dipyrone. However, the pain progressively worsened and the patient presented to the emergency room (ER) 5 days after the onset of pain. Since a computer tomography (CT) brain scan was within normal limits and neurological and ear, nose and throat (ENT) examinations were nondiagnostic, she was discharged. Five days later the patient returned to the ER and complained that her headache had severely increased. A blood

test revealed an elevated erythrocyte sedimentation rate (ESR) of 70 mm/h. The differential diagnosis at that point included GCA due to the elevated ESR and temporal headaches, and a possibility of osteoarthritis of the temporomandibular joint (TMJ) based on the limitation of mouth opening, pain upon chewing, and ear/TMJ area which were included in the description of the location of the pain. However, she was discharged from the ER with a final diagnosis of left TMJ osteoarthritis and was referred to an oral surgery department for further treatment. Three days later she consulted with her dentist who suspected a dental cause for her pain and extracted her mandibular left first molar. Following the extraction, the patient reported no relief of pain and therefore was referred the following day to the Orofacial Pain Clinic for further evaluation and treatment 14 days after the initial onset of her chief complaint.

Her medical history included high blood pressure (which was controlled by medications), vitamin B12 deficiency, and myocardial infarction (MI) 17 years previously. The patient was taking disothiazide (25 mg/day), aspirin (100 mg/day), and vitamin B12 injections once a month. The patient denied any history of headaches, trauma to the head and neck area, or TMD.

Pain Characteristics

The patient described the onset of the pain as sudden and pointed at the left ear/left TMJ area and left temple as the sites of pain. The pain was described as pressing and pulsating in quality, continuous, and gradually worsening. On a 0 to 10 visual analog scale (VAS), pain intensity was graded between 7 and 8 with at least 3 daily episodes, lasting from several seconds to 90 minutes, of stronger pain (grade 10). The severe pain was accompanied by nausea, vomiting, and photophobia and could awaken the patient from sleeping. Any attempt to chew, open the mouth, or lie down aggravated the pain. The pain was not aggravated by physical activity and no autonomic signs were apparent. The malaise, depression, and inability to chew resulted in a 5 kg weight loss for the patient within a period of 2 weeks.

Clinical Examination

No unusual findings were noted on extraoral and intraoral examinations or on panoramic radiography. The TMJ examination was based on the Research Diagnostic Criteria for TMD examination guidelines (RDC/TMD).²⁸ The mandibular

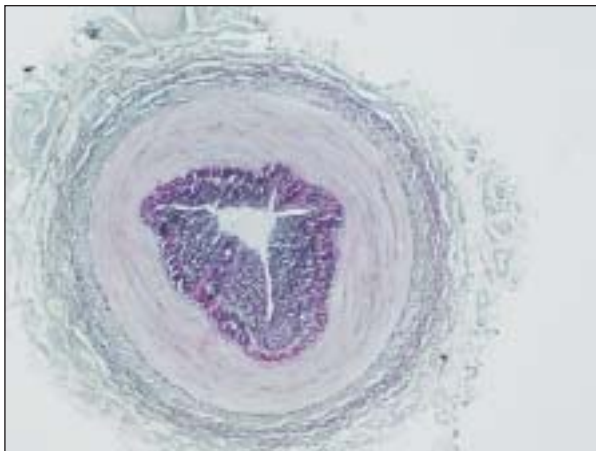


Fig 1 Biopsy of the left superficial temporal artery showing thickening of the intima layer, narrowing of the artery lumen, and no disruption of the internal elastic lamina (elastin staining by resorcin fuchsin method, original magnification $\times 40$).

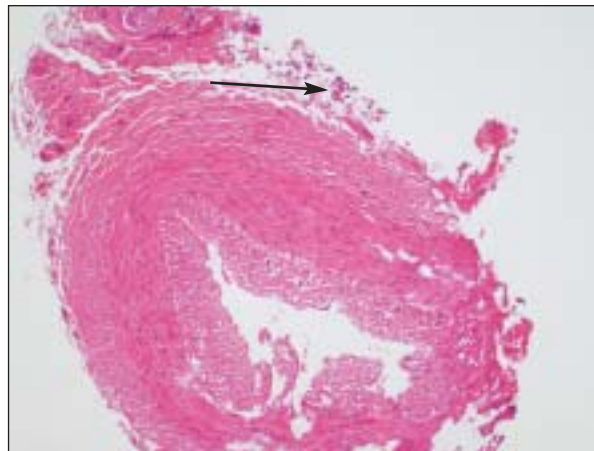


Fig 2 Another section of the same artery as in Fig 1 showing intima thickening, disruption of internal elastic lamina, most probably due to an artifact, and scattered plasma cells in the periphery (see *arrow*) (hematoxylin and eosin stain, original magnification $\times 40$).

opening pattern was straight but unassisted opening without pain was limited to 30 mm. Maximum unassisted opening and maximum assisted opening were limited to 32 mm and were accompanied by pain at the left TMJ and left masseter areas.

There were no joint sounds upon opening, lateral and protrusive mandibular movements. Right lateral excursion was 9 mm, accompanied by pain in the left masseter muscle. Left lateral excursion was 7 mm, accompanied by pain in the left masseter muscle. Protrusive movement was symmetrical and measured 2 to 3 mm. Both TMJs were not tender to palpation at the lateral poles, nor the posterior attachment areas. The left temporal muscle (posterior, middle and anterior zones; 2 to 3 on a 1 to 3 scale) and the left masseter muscle (superior, middle and inferior zones; 3 on a 1 to 3 scale) were sensitive to palpation. No nodules or pulsation of the temporal artery were found on palpation of the left temporalis muscle.

Diagnosis

Based on the patient's age, pain characteristics, sudden headache onset, lack of history of TMD, and the clinical findings including the elevated ESR, the patient was urgently referred to the ER with a provisional diagnosis of GCA. On examination performed then in the ER, sensitivity to palpation of the left temple was noted. Ultrasonography

of the left superficial temporal artery was within normal limits. Blood tests, taken the same day, showed an elevated ESR (80 mm/h) and C reactive protein (CRP) (3.5 mg/dL).

Therapy and Follow-up

Prednisone (60 mg/day) was prescribed. The headache disappeared within 24 to 48 hours of commencing steroid treatment and the ESR was reduced to 50 mm/hour 3 days later. However, 4 days after initiating steroid therapy, pain upon chewing was still reported and the results of a repeated TMJ examination were essentially the same as on the day of diagnosis.

The left superficial temporal artery was biopsied 10 days after initiating prednisone therapy. The biopsy was negative for GCA. However, the specimen showed thickening of the intima layer of the temporal artery and narrowing of the lumen, without disruption of the internal elastic lamina (Fig 1). In one section, scattered plasma cells were also observed in the periphery of the artery (Fig 2). It should be noted that a positive biopsy result for GCA reveals inflammatory infiltrate of lymphocytes, macrophages, or multinucleated giant cells within the intima or media layers, disruption or loss of the internal elastic lamina, and intimal thickening. The presence of multinucleated giant cells is not necessary for the diagnosis of GCA.⁶

Table 1 Diagnostic Criteria for GCA from the International Headache Society (IHS) 2004 and the American College of Rheumatology (ACR) 1990

IHS diagnostic criteria for GCA ³³	ACR diagnostic criteria for GCA ^{*34}
(1) Any new persisting headache fulfilling criteria C and D	(1) Age at disease onset \geq 50 years
(2) At least one of the following: (a) Swollen tender scalp artery with elevated erythrocyte sedimentation rate (ESR) and/or C reactive protein (CRP) (b) Temporal artery biopsy demonstrating giant cell arteritis	(2) New onset of or new type of localized headache
(3) Headache develops in close temporal relation to other symptoms and signs of giant cell arteritis	(3) Temporal artery abnormality: temporal artery tenderness to palpation or decreased pulsation, unrelated to arteriosclerosis of cervical arteries
(4) Headache resolves or greatly improves within 3 days of high-dose steroid treatment	(4) Erythrocyte sedimentation rate \geq 50 mm/hr
	(5) Abnormal artery biopsy: biopsy specimen with artery showing vasculitis characterized by a predominance of mononuclear cell infiltration or granulomatous inflammation, usually with multinucleated giant cells

* For purposes of classification, a patient shall be said to have GCA if at least three of these five criteria are present.

One month later, the patient was still taking prednisone (40 mg/day) and was followed up by a rheumatologist and an ophthalmologist. Although the headache had disappeared, the patient occasionally felt a heavy sensation in the left temporal area with no pain on chewing and an increased ROM of 44 mm. Two months later, the patient experienced a relapse. Interestingly, jaw claudication was the first symptom, followed by blurry vision and temporal headache. At 15 months after the initial diagnosis, the patient was taking a maintenance dose of prednisone (10 mg/day) and methotrexate (10 mg once a week). The patient reported no headaches, facial pain, or limitation of ROM.

Discussion

While a positive temporal artery biopsy is considered the gold standard for diagnosis of GCA, its sensitivity is only 87.1%.²⁹ A false-negative biopsy can occasionally result from a short specimen length (due to skip lesions),³⁰ technique, number of biopsies taken, and the time between initial steroid treatment and biopsy procedure.³¹ Thus, to avoid false-negative results, it is recommended that the biopsy specimen should be at least 2 to 2.5 cm in length³² and the biopsy performed within a week of the initial steroid treatment. In the current case, the biopsy was taken 10 days after initial steroid treatment, specimen length was 1.1 cm, and no contralateral biopsy was taken, all of which could have contributed to possible negative results. However, a negative biopsy result does not preclude a diagnosis of GCA. The current case almost fulfilled the International Headache Society

(IHS) diagnostic criteria for GCA,³³ (no swollen tender artery was palpated), and fulfilled the diagnostic criteria of the American College of Rheumatology (ACR) for GCA (Table 1).³⁴

The patient's jaw pain was closely related to mastication, a feature that could easily be attributed to TMD-related musculoskeletal pain, but actually represented jaw claudication, eg, pain in the jaw that begins while chewing and eases when chewing is discontinued. The pain associated with jaw claudication can mimic musculoskeletal pain associated with TMD, odontogenic pain, or osteoarthritis of the TMJ.^{26,35-37} In fact, jaw claudication and the additional finding of hard-end feel limitation of ROM and TMJ/ear pain location were the main reasons for the initial misdiagnosis of TMD. However, a careful consideration of the following signs, symptoms, and pain characteristics should have prompted the clinician to exclude a TMD: the sudden onset of pain; the quality of the pain which was described as pressing and pulsating, continuous, and gradually worsening. Pain intensity was graded between 7 and 8 with at least 3 daily episodes of grade 10 pain intensity. The accompanying symptoms of nausea, vomiting, and photophobia, and increasing pain when the patient was lying down could not be attributed to TMD. In addition, contrary to musculoskeletal TMD pain, jaw claudication usually appears as facial pain associated directly with eating in combination with a recent onset of headaches; pain associated with chewing any food, or even licking an ice cream cone or drinking,²⁶ and subsiding within a few minutes after chewing is stopped is not a feature of musculoskeletal TMD pain. Nonetheless, even when these unique features are considered,

differences between jaw claudication and TMD-related musculoskeletal pain can be insubstantial. Therefore, the differential diagnosis must consider, in addition to a precise pain history, the patient's age, headache characteristics, constitutional symptoms, and blood test results. For instance, the highest prevalence of TMD occurs in the age range of 25 to 44 years.³⁸⁻⁴¹ Therefore, when an elderly patient is referred with a provisional diagnosis of TMD, vascular, neurogenic, and space-occupying lesions should be ruled out before confirming the diagnosis of TMD.

The simultaneous appearance of headaches and TMD is well documented in clinical and epidemiological studies.⁴²⁻⁴⁵ The prevalence of headaches among TMD patients can be as high as 70% compared to 20% in the normal population,⁴⁶ and the headache may be secondary to the TMD. Headache is also a common complaint in the elderly. However, one has to keep in mind that approximately 15% of headaches in patients over 65 years of age are due to emergency life-threatening conditions.^{47,48} For instance, the 67-year-old patient's headache did not support the diagnosis of a TMD-related headache, but fulfilled several of what is known as the "red flags for headaches"⁴⁹: a new headache in a patient over 50 years, sudden appearance of a new type of headache, most severe headache that the patient recalls, gradual worsening of the headache, headache aggravated by physical activity or movement, headache accompanied by systemic signs and symptoms, such as nausea or vomiting, and headache awakening the patient from sleep. Finally, the elevated ESR and CRP cannot be explained by TMD-related pathologies (excluding systemic arthritis) and should have raised suspicion of a GCA.

The clinical finding which helped to diagnose this patient as a non-TMD muscle related hard end-feel limitation of ROM was the symmetry in mandibular movements (opening, lateral, and protrusive) which allowed exclusion of a unilateral intra-articular pathology, such as a disc displacement without reduction, as this normally results in asymmetric opening, protrusion, and laterotrusions.

In summary, GCA should be considered in the differential diagnosis of facial pain, especially in the elderly with concomitant complaints of jaw claudication and new headache. It may be difficult to differentiate jaw claudication from a TMD-related muscle pain, therefore a thorough history that takes into account all pain characteristics, the presence of accompanying signs and symptoms, such as a concomitant sudden onset of temporal headache, and the patient's age are all mandatory to consider

for a correct differential diagnosis that will eventually also require a blood test and temporal artery biopsy. Early diagnosis of GCA is essential to prevent serious consequences, such as loss of vision and other ischemic complications.

Acknowledgments

The authors wish to thank Dr Marilena Vered for her assistance in acquiring the photomicrographs and their interpretation, and Dr David Cohen for his assistance in writing and editing the article.

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