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**A CASE REPORT**

**JAW CLAUDICATION: A SIGN OF  
GIANT CELL ARTERITIS**

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**A B S T R A C T**

**Jaw claudication is an often unrecognized sign of giant cell arteritis. The significance of this symptom has been underemphasized in the dental literature.**

**Dentists should consider jaw claudication when making the differential diagnosis of jaw pain, especially in the elderly patient.**

**Early diagnosis and treatment of giant cell arteritis may help save a patient's vision.**

**G**iant cell arteritis (GCA) is a polysymptomatic disease with protean manifestations, including headaches, joint pains, scalp tenderness, fever, malaise and weight loss. This case report describes a patient with jaw claudication as the initial sign of giant cell arteritis. Visual loss, usually due to ischemic optic neuropathy, is the major complication of the disorder, and early diagnosis and treatment are critical.

**CASE REPORT**

A 75-year-old white woman complained to her dentist of a two-week history of intermittent pain in her jaw. Her medical history included malaria, an appendectomy, kidney stones, open-angle glaucoma and uncomplicated bilateral cataract extraction. She had been administering optipranolol drops twice a day in the left eye to treat glaucoma. She had smoked two packs of cigarettes per day for 50 years and drank alcohol in moderate amounts. She was in her usual state of good health until early September 1994 when she developed intermittent pain in her left jaw, which became worse whenever she chewed food.

The results of the patient's dental examination were normal, and her dentist recommended that she put a hot washcloth on her face as needed; however, her symptoms persisted. She had no fatigue, fever, night sweats or arthralgias but had lost 4 pounds over the previous two months. Several weeks later, the patient developed new, moderately severe temporal and retrobulbar headaches on a daily basis. Later that week, she had several transient episodes of binocular vertical diplopia. She saw an ophthalmologist in early January 1995; the results of the ophthalmological examination were normal.

Two weeks later, the patient returned to the ophthalmologist complaining of acute visual loss in her right eye. The ophthalmological examination revealed a visual acuity of 20/50 in the right eye and 20/25 in the left eye. The right pupil reacted sluggishly to light and there was a right afferent pupillary defect. Visual field testing of the right eye revealed a superior altitudinal defect. The slit-lamp examination revealed anterior basement-membrane changes in each eye and bilateral posterior-chamber intraocular

lenses. An ophthalmoscopic examination revealed a pale and swollen optic nerve in the right eye and a normal optic nerve in the left eye.

A diagnosis of anterior ischemic optic neuropathy (AION) was made. The patient's erythrocyte sedimentation rate (ESR) was elevated at 96 millimeters per hour. The patient was admitted to the hospital and treated with 80 milligrams of prednisone per day. A temporal artery biopsy specimen was positive for GCA. The patient received 1,000 mg of intravenous methylprednisolone for three days and was discharged from the hospital with instructions to follow a regimen of 80 mg per day of oral prednisone. Her visual function remained stable in the right eye, and her normal eye did not become involved.

#### DISCUSSION

GCA is a systemic, inflammatory vasculopathy of unknown etiology that affects large- and medium-sized arteries. The clinical manifestations of the disorder vary, but include headaches; arthralgias and myalgias; scalp or temporal tenderness; swollen, nodular or tender temporal arteries; fever; anorexia; malaise; weight loss; an elevated ESR; and jaw claudication.<sup>1-5</sup> GCA occurs most commonly in whites and is rare in blacks. It is a disease of elderly people, rarely seen in patients younger than 50 years of age.<sup>1-3,5</sup>

The most common ophthalmologic manifestation of GCA is AION, but transient loss of vision, retinal and choroidal ischemia and diplopia also may

occur.<sup>1</sup> Early diagnosis and treatment of GCA are critical because severe visual loss, usually due to AION, occurs in a mean of 36 percent (range, 7 to 60 percent) of untreated patients, and contralateral eye involvement develops in 65 percent of these patients, often within a period of a few days to several weeks.<sup>1</sup> Visual loss in GCA tends to be severe; 27 percent of patients become totally blind, and the majority of the rest suffer debilitating visual loss.

The diagnosis of GCA depends on the clinical signs and symptoms, with or without an elevated ESR, and is confirmed by a positive temporal artery biopsy specimen.<sup>1-5</sup> Systemic steroids are the standard treatment for GCA. Prompt treatment may prevent further visual loss.

The dentist's link to GCA is jaw claudication. This condition is manifested by pain when the jaw is used and is believed to be due to ischemia of the facial artery and the muscles of mastication. It occurs in 50 to 75 percent of patients with GCA and may be difficult to differentiate from temporomandibular joint syndrome or other types of dental pain.<sup>4</sup>

Hollenhorst and others<sup>5</sup> reported "mandibular joint arthritis" as the referring diagnosis in

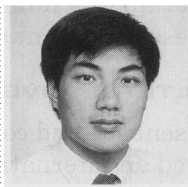
eight of 175 patients who eventually received the diagnosis of GCA. Patients with this symptom often complain of jaw weakness, fatigue and pain when chewing or talking, all of which diminish with rest. Patients may chew their food on the contralateral side or may avoid solid food altogether to avoid pain. Horton<sup>2</sup> believed that jaw claudication could be differentiated from other types of head and facial pain in that it was an "exercise phenomenon," and some authors believe it to be an almost pathognomonic symptom of GCA.<sup>3</sup>

#### CONCLUSION

Jaw claudication is often unrecognized or misdiagnosed by general physicians and dentists. Dentists, in particular, should be aware of the clinical implications of true jaw claudication, since they may be the first health care providers consulted by patients with jaw pain. GCA should be considered in the differential diagnosis for any elderly patient with jaw pain. Early diagnosis, prompt referral to ophthalmologists and other physicians and treatment with steroids may help save the vision of patients with GCA. ■

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