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A typical case of giant cell arteritis with vision loss due to delayed diagnosis

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A 75-year-old woman suffered from masseter pain for 1 year, occipital and left-sided temporal scalp pain for 3 months, and an enlarged temporal artery for 1 month. The etiology was unknown despite consultations by several specialists, including oral surgeons and neurosurgeons. She developed visual disturbance in the right and left eyes 7 and 5 days, respectively, prior to admission. She visited an ophthalmologist and underwent follow-up observation. One day prior to admission, her best-corrected visual acuity (BCVA) deteriorated to 0.08 in the right eye and light perception in the left eye. With suspected giant cell arteritis (GCA), she was eventually referred to the internal medicine department at our hospital. She denied weight loss, fever, or fatigability. On physical examination, the temporal arteries appeared enlarged bilaterally (Figure 1) and there was a weak pulse on the left. The direct light reflex was attenuated in the left. Fundoscopy showed bilateral indistinct optic disk boundaries with pallor and swelling in the left eye, indicating ischemic optic neuropathy (Figure 2). Considering her symptoms of headache, absence of a temporal artery pulse, and elevated erythrocyte sedimentation rate (113 mm/h), she was clinically diagnosed with GCA. Intravenous pulse methylprednisolone therapy (500 mg/d for 3 days) was started immediately. The therapy was switched to 60 mg of oral prednisolone for 4 weeks and was then tapered. Pathological analysis of a biopsy sample of the left temporal artery confirmed GCA. Subsequently, headache and masseter



FIGURE 1 Right temporal scalp: Enlargement of the right temporal artery is visible (arrow)

pain improved. Her BCVA ultimately stabilized at 1.5 in the right eye, but remained counting fingers in the left eye.

Giant cell arteritis is a large-vessel vasculitis that results in complete vision loss as a serious sequela; however, emergency cases are rare. If left untreated, 15%-20% of patients experience vision loss; however, the rate of vision loss can be reduced to 1% with glucocorticoid treatment.¹ Moreover, patients with visual symptoms due to GCA may lose sight in the other eye within days if left untreated.²

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Photographic consents: Consent was obtained from the patient.



FIGURE 2 Fundoscopy findings: Boundaries of the optic disk are indistinct bilaterally, with pallor and swelling observed in the left eye

These findings underscore the importance of early diagnosis as well as urgent and adequate glucocorticoid therapy.

This case indicates that visual disturbance may be caused by systemic diseases, even if the patient does not have other systemic symptoms. The diagnosis was delayed probably because specialists evaluated single organs only, and because the patient showed no systemic symptoms. Although not all patients with GCA show all symptoms of the disease, clinicians should consider nonspecific findings such as fever, fatigability, muscle pain, and inflammatory biomarkers as indicators of vasculitis. Furthermore, the existence of any temporal artery abnormality, such as beading or tenderness, increases the likelihood of GCA.³ Jaw claudication, in particular, is a risk factor for visual disturbance and is a specific symptom;⁴ thus, the chewing gum test might be useful.⁵ In conclusion, physicians must evaluate the entire body and consider combinations of associated symptoms.

We should address preventable visual loss to reduce the occurrence of such cases of GCA and should consider the urgency of GCA treatment.

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CONFLICT OF INTEREST

The authors have stated explicitly that there are no conflicts of interest in connection with this article.

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