

case report

Diagnosis and Management of Temporal Arteritis: A Review and Case Report

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ABSTRACT

Temporal arteritis is an insidious disease which, if not recognized and treated with high-dosage oral prednisone or intravenous prednisolone, can result in unilateral or even total blindness due to anterior ischemic optic neuropathy (AION) or closure of the central artery of the retina. Unfortunately, the symptoms and clinical signs of temporal arteritis mimic those of a number of other conditions including angle-closure glaucoma, hypertension, migraine, trigeminal neuralgia, temporomandibular joint syndrome, carotid artery occlusive disease, Foster-Kennedy syndrome, and nonarteritic AION. When a patient complains of a severe pain in the temporal region, along with scalp tenderness and a feeling of malaise or depression—with or without episodes of transient loss of vision—he or she should be referred for a diagnostic work-up which includes an erythrocyte sedimentation rate and a temporal artery biopsy. We present here a review of the recent literature concerning temporal arteritis, followed by a report of an unusual case in which high-dosage prednisone therapy was effective in relieving the patient's symptoms and lowering the sedimentation rate in spite of a negative temporal artery biopsy.

Key Words: temporal arteritis, cranial arteritis, giant cell arteritis, polymyalgia rheumatica

LITERATURE REVIEW

Temporal arteritis, also known as giant cell arteritis and cranial arteritis, has been described by Henkind and Chambers¹ as "the most frequently misdiagnosed extraocular cause of preventable visual loss," having an exceedingly high incidence of ocular involvement. Most temporal arteritis sufferers are people beyond the age of 55 or 60 years

(with a slight female preponderance) who have a history of auto-immune disease such as rheumatoid arthritis. The pathological process is a patchy granulomatous inflammation in the walls of the large and medium sized arteries, with fragmentation of the internal elastic lamina and the presence of giant cells. The annual incidence of giant cell arteritis has been estimated by Fledelius and Nissen² as in excess of 1 per 1000 for people over the age of 50 years; however, they reported that marked visual loss was found in only 10 of 264 patients diagnosed as having giant cell arteritis.

Symptoms

Symptoms of temporal arteritis include a severe headache, usually in the temporal and occipital regions; scalp tenderness, noted particularly when combing the hair; a feeling of malaise or depression; a loss of appetite; claudication of the jaw while chewing; and possibly a low-grade fever.³⁻¹¹ If the inflammatory process affects the ophthalmic artery and its branches, the patient may report one or more episodes of transient loss of vision.¹ In addition, there may be complaints of pain in the neck, shoulders, upper arms, and trunk—a condition known as polymyalgia rheumatica.³ Many patients who have experienced the symptoms of temporal arteritis fail to volunteer them in a routine history. Smith⁴ has commented that the doctor has to "pull the symptoms out of the patient." Keltner⁵ said that "the most common reason for failure to make the diagnosis may be the failure to ask the appropriate questions." In a discussion of the systemic symptoms of giant cell arteritis, Hayreh¹¹ stated:

"I find widespread lack of awareness among physicians of this important, typical symptom-complex of giant cell arteritis. It is not at all uncommon in our experience to find patients who have repeatedly given a classical history of this disease to their physicians and the diagnosis was totally missed and the patient went blind in one or both eyes" (p. 33).

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Ocular Involvement

The most common ocular involvement in temporal arteritis is anterior ischemic optic neuropathy, which is responsible for the episodes of transient loss of vision and may even cause permanent loss of vision. The cause of the anterior ischemic optic neuropathy (AION) is a loss of blood supply to the branches of the short posterior ciliary arteries supplying the circle of Zinn, which surrounds the optic nerve. It is seen ophthalmoscopically as a pseudo-papilledema, or pallid papilledema. The disk appears to be pale, with narrowing of the retinal arteries and small peripapillary nerve-fiber-layer hemorrhages (Fig. 1). The optic nerve ischemia may be accompanied by an afferent pupillary defect and may cause an altitudinal field defect, most commonly in the inferior field.³ In some cases the optic nerve ischemia occurs behind the globe, causing retrobulbar optic neuropathy, resulting in a normal appearing disk, but with the characteristic altitudinal field defect.⁵ If fluorescein angiography is performed, there may be a delayed choroidal filling.¹² A less common ocular involvement of temporal arteritis is occlusion of the central retinal artery resulting in complete and usually irreversible loss of vision in the involved eye. Ocular involvement may be unilateral or bilateral.

Oculo-motor anomalies, which may or may not be accompanied by diplopia, occur in some cases of temporal arteritis. In one report,⁶ a patient complained of transient vertical diplopia followed by horizontal diplopia occurring on extreme rightward gaze. According to Riech et al.,¹⁰ ptosis accompanying ophthalmoplegia may suggest temporal arteritis, and ophthalmoplegia associated with difficulty in upward gaze may precede the more classical symptoms of giant cell arteritis such as headache or blindness.

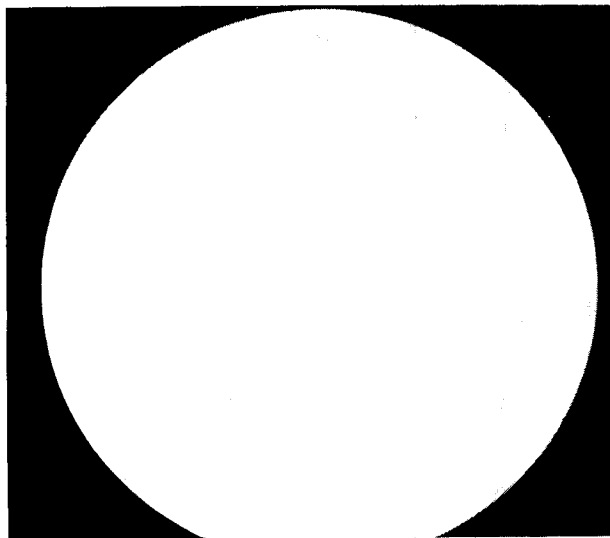


Figure 1. Acute pallid disk edema commonly occurring in temporal arteritic ischemic optic neuropathy. Retinal nerve fiber layer edema and hemorrhage can also be present.

Possibility of Blindness in Untreated Cases. In untreated cases of temporal arteritis, the possibility of blindness or other severe visual loss is astonishingly high. In a compilation of data taken from 13 studies, Keltner⁵ found that blindness occurred in 36% of untreated cases. In a study comparing outcomes of patients treated with glucocorticoids to those treated in the preglucocorticoid era, a 3-fold increase in bilateral blindness was found for those patients who did not receive glucocorticoids; furthermore, of those giant cell arteritis patients who presented with visual symptoms and were not treated with glucocorticoids, 50% developed bilateral blindness.³

Systemic Manifestations

Systemic manifestations have been reported in a small percentage of patients diagnosed as having giant cell arteritis including transient ischemic attacks, cerebral hemorrhage, stroke, aortic arch occlusive disease, ataxia, dementia, and hearing loss.¹⁰ Keltner⁵ has suggested that many cerebral vascular accidents in the elderly that have been attributed to arteriosclerosis—as well as peripheral neuropathy of the feet, carpal tunnel syndrome, and sciatic neuropathy—may be secondary to giant cell arteritis. Riech et al.¹⁰ stated that cerebrovascular events are a frequent cause of fatalities in giant cell arteritis, but their specific prevalence is unknown. These authors reported that both the vertebrobasilar artery system and the carotid artery system have been found to be responsible for transient ischemic attacks and strokes.

Diagnosis

A tentative diagnosis of temporal arteritis can be made on the basis of the patient's symptoms and ocular signs. The diagnosis is confirmed by the presence of an elevated erythrocyte sedimentation rate and by a positive temporal artery biopsy.

Erythrocyte Sedimentation Rate. Of the two methods used to measure the sedimentation rate—the Westergren method and the Wintrobe method—the Westergren method is preferred.^{4, 5, 13-15} A sedimentation rate below 20 mm/h is usually considered to be normal. Some controversy exists concerning the sedimentation rate diagnostic of temporal arteritis: a sedimentation rate of 50 mm/h or greater has been specified by Henkind and Chambers¹ and by the American College of Rheumatology.¹³ However, Smith⁴ has stated that a patient having a sedimentation rate no higher than 30 mm/h may have temporal arteritis; Keltner⁵ has reported that the sedimentation rate may be normal early in the course of the illness, but becomes elevated later in the disease process. Cases of giant cell arteritis with normal sedimentation rates have been reported.^{16, 17} Other possible laboratory findings may be normochromic and normocytic anemia, and elevation of alkaline phosphatase.

Hayreh¹¹ commented that for the past few years, in addition to erythrocyte sedimentation rate, he

has used C-reactive protein to evaluate and manage giant cell arteritis patients: a rise in C-reactive protein level in serum and other body fluids is produced by inflammatory processes in the body; combined information on C-reactive protein and erythrocyte sedimentation rate is very useful in diagnosis and evaluation of systemic corticosteroid treatment.

Temporal Artery Biopsy. Even though giant cell arteritis involves many large and medium sized arteries throughout the body, the temporal artery is in an exposed position and is therefore easily excised. Albert et al.¹⁴ have listed four pathological changes seen in temporal arteritis: (1) occlusion of the vessel lumen; (2) fragmentation of the internal elastic lamina; (3) a patchy degeneration and drop-out of smooth muscle cells in the media; and (4) the characteristic granulomatous inflammation, containing various kinds of inflammatory cells including giant cells. However, giant cells are not always seen and are not necessary for the diagnosis.¹⁴

Additional Diagnostic Procedures. Fundus fluorescein angiography and Doppler sonographic examination have been recommended for the diagnosis of temporal arteritis. Mack et al.¹² found delayed choroidal filling (mean filling time of 69 s) for 13 patients with visual disturbances due to biopsy-proven giant cell arteritis, 11 of whom also had AION, as compared to a mean filling time of 5.5 s for 33 patients with nonarteritic AION. Vollrath and Gloor¹⁸ have recommended that a Doppler sonographic examination should be performed, to rule out the possibility of carotid artery stenosis, before temporal artery biopsy. They made the point that in the presence of stenosis of the internal carotid artery, a temporal artery biopsy could result in sudden death.

Management

Before the availability of glucocorticoids, there was no effective treatment for temporal arteritis. These agents mimic the action of cortisol, which is produced by the human adrenal cortex in response to the release of the adrenocorticotropic hormone by the anterior lobe of the pituitary gland. When a glucocorticoid agent is administered, it inhibits the release of cortisol by the adrenal cortex and when it is discontinued, the adrenal cortex must resume its excretory function. Because cortisol is necessary for the continuation of life, sudden withdrawal of glucocorticoid therapy can have disastrous consequences. Glucocorticoid therapy must be initiated at a sufficiently high dosage to control the inflammatory process; once the inflammatory process has been controlled, the dose must be tapered gradually so that the adrenal cortex can be reactivated.¹⁹

All authorities agree that the treatment for temporal arteritis is the oral or intravenous administration of high doses of glucocorticoids. Henkind and Chambers¹ recommend an initial dosage of oral prednisone in the range of 60 to 120 mg/day, and

suggest that the dosage be increased, if indicated by a sedimentation rate taken after the first day's dosage. Smith⁴ recommends that after an initial dosage of 50 mg/day, the sedimentation rate should be checked on a regular basis, and the dosage should be sufficiently high to keep the sedimentation rate below 30 mm/h. Hollenhorst (in the Wang and Henkind report)⁹ stated that he uses about 80 mg of prednisone per day, in divided doses, for the first week, and then reduces the dosage to once daily, aiming at 40 mg/day by the end of the first month and 20 mg/day by the end of the second month, and discontinuing treatment by the end of the fourth month. He stated that as far as he is aware, none of his patients have lost vision after the fourth month of the disease. However, other practitioners find that treatment can be necessary for a period of several years.^{11, 21}

Juchet et al.²⁰ recommend administration of a bolus of methylprednisolone, with the aim of reducing the side effects which may otherwise occur. Their protocol is to use intravenous methylprednisolone, 500 mg/day, for 3 consecutive days, followed by low doses (an average of 22.5 mg/day) of prednisone or an equivalent drug. The method of treatment used by Hayreh¹¹ is as follows: for a patient with no visual symptoms or visual loss, he starts with at least 80 mg of oral prednisone daily, or even up to 120 mg, depending on the severity of the systemic complications, but for a patient who has had transient loss of vision or more severe visual loss, he uses megadose prednisolone therapy, beginning with the equivalent of 1 g of prednisone slowly by intravenous drip, every 6 to 8 h, with 3 to 4 such doses. This is followed by 120 mg of oral prednisone daily. Initially, sedimentation rate and C-reactive protein level are assessed daily, to evaluate the response of the patient to the therapy. Hayreh stressed that some patients react very differently to the therapy than others, stating that determination of the maintenance dose is a slow, laborious, and painstaking job over many months.

Adverse Effects of Prednisone Therapy. Temporal arteritis is one of a small number of conditions in which the possible adverse effects of high-dosage prednisone therapy (see Table 1) are outweighed by the consequences of withholding therapy or of pro-

TABLE 1. Adverse effects of prednisone therapy.

Effects of short-term therapy	
Edema of the hands and face (typical "moon-face" appearance)	
Gastrointestinal tract involvement, including peptic ulcer	
Muscular weakness, including loss of muscle mass	
Immunosuppression (at risk for fungal and other infections)	
Increased intraocular pressure (IOP)	
Effects of long-term therapy	
Sodium and fluid retention; elevated blood pressure	
Elevated blood sugar	
Osteoporosis	
Posterior subcapsular cataracts	
Glaucoma	

TABLE 2. Conditions to be ruled out in the differential diagnosis of temporal arteritis.

Condition	Similarities in Symptoms or Signs	Additional Diagnostic Features
Angle-closure glaucoma	Unilateral head pain	Gonioscopic evidence of angle closure; elevated IOP
Hypertension	Early morning headaches	Elevated blood pressure
Migraine	Unilateral headache	Prodromal visual aura; nausea
Trigeminal neuralgia	Facial pain; "trigger zone"	Pain in area served by maxillary or mandibular branch of trigeminal ganglion
Temporo-mandibular joint syndrome	Pain in temporal area	Malocclusion or other mechanical problem
Carotid artery occlusive disease	Transient, unioocular, painless loss of vision	Transient ischemic attacks; cholesterol plaques in retinal arterioles
Foster-Kennedy syndrome	Papilledema	Optic atrophy in contralateral eye; due to frontal lobe tumor or abscess
Nonarteritic anterior ischemic optic neuropathy (AION)	Papilledema	An absence of the signs and symptoms of temporal arteritis

viding inadequate dosage.^{4, 19} Smith⁴ has stressed the importance of continuing high-dosage prednisone therapy in spite of the presence of side effects such as black, tarry stools. Fortunately, the more serious complications of prednisone therapy such as diabetes, hypertension, and osteoporosis are not likely to occur with relatively short-term therapy.

Course and Prognosis

According to Raskin and Appenzeller,³ temporal arteritis is a self-limited disease that usually persists for 1 or 2 years, but spontaneous remission may occur as soon as 6 months. These authors state that the purpose of the high dosage prednisone therapy is not to shorten the course of the disease, but to prevent the occurrence of complications such as blindness and aortic rupture, by suppressing the inflammatory arterial component of the disease until it spontaneously enters its quiescent stage. Hayreh¹¹ warned that the concept of giant cell arteritis burning itself out in 9 to 12 months is not at all true: his experience over a period of 15 years has been that these patients require maintenance dosage for years. In a review of the records of 98 patients who had had temporal artery biopsy during a 5-year period, Chmielewski et al.²² reported that mild flare-ups were common after 1 year of therapy, and that almost one-half of the 98 patients were still on steroid therapy after 2 or 3 years.

When vision has been lost in one eye, aggressive prednisone treatment is recommended in order to prevent a similar loss in the fellow eye. Smith⁴ points out that even with prednisone treatment, there is a possibility that the vision in the second eye will be lost, but the treatment greatly reduces the chances of this occurring. In a case reported by Cohen and Damaske,⁶ a patient, already having lost light perception in her right eye, was treated with prednisone for several weeks until she discontinued the treatment on her own volition: 10 days later, she lost all vision in her left eye, and was thus totally blind.

Problems in Differential Diagnosis

Some of the symptoms and signs of temporal arteritis are sufficiently similar to those of other

disease conditions to bring about a delay in making the diagnosis, and therefore in initiating the prednisone treatment. Many cases have been reported in the literature in which irreversible vision loss has occurred due to delays caused by inappropriate diagnostic and treatment procedures.^{4-7, 11}

Conditions having one or more of the same symptoms or signs as temporal arteritis are shown in Table 2. Angle-closure glaucoma is obviously of particular interest to primary eye care practitioners: in our case report, involving a patient with narrow anterior chamber angles, a diagnosis of angle closure was initially considered but was ruled out. Hypertension is also of importance, because patients in the age-range at risk for temporal arteritis are also likely to have hypertension. Although migraine and cluster headache are usually considered to affect a relatively young age group, Solomon et al.¹⁵ have reported a high prevalence of these conditions among headache patients over the age of 65 years and it has been reported that some patients with biopsy-proven temporal arteritis presented with occipital (rather than temporal) head pain.²¹ Because of the presence of a "trigger zone," the facial pain of trigeminal neuralgia may be confused with that of temporal arteritis. Temporo-mandibular joint syndrome may be confused with temporal arteritis inasmuch as both conditions involve a claudication of the jaw while eating. Carotid artery occlusive disease, although not causing a headache, like temporal arteritis is a major cause of transient loss of vision.¹ The appearance of the optic nerve head in AION, sometimes referred to as pallid papilledema, can be responsible for a pseudo-Foster-Kennedy syndrome which, according to Miller et al.,⁷ is much more common than the true Foster-Kennedy syndrome.

When AION is present, it is imperative that it be diagnosed correctly as either the arteritic or the nonarteritic form. Hayreh¹¹ has stressed the fact that giant cell arteritis is an ocular emergency, and has listed the following criteria which indicate that AION, in a given case, is the arteritic rather than the nonarteritic form: systemic and visual symptoms of giant cell arteritis; elevated erythrocyte sedimentation rate and C-reactive protein level;

early massive visual loss; chalky-white disk swelling (occurring exceedingly rarely in the nonarteritic form); AION associated with cilio-retinal artery occlusion; and massive choroidal nonfilling on fundus fluorescein angiography.

Because the consequences of untreated temporal arteritis are so severe, eye care practitioners should suspect its presence whenever an older patient complains of temporal headaches, scalp tenderness, a feeling of malaise or depression, and particularly if the symptoms include transient visual disturbance with amaurosis fugax, diplopia, ptosis, or blurred vision. Valuable time should not be lost in pursuing incorrect diagnoses. Prompt treatment with high dosage of prednisone is imperative, if catastrophic vision loss is to be avoided.

CASE REPORT

Our patient, a 65-year-old woman, was examined at the Indiana University School of Optometry's Atwater Eye Care Center on October 17, 1991. She complained of blurred vision at near, and headaches which were relieved by pressing on her right eye. She reported a history of high intraocular pressure (IOP), hypertension, and osteoarthritis. She also had a history (although not reported at the initial visit) of shooting pains in the neck, shoulders, upper arms, and hips (polymyalgia rheumatica) which had been treated by chiropractic therapy for the past 7 months.

Her refractive error was found to be: right eye +3.75 D, resulting in 6/6- (20/20-) visual acuity, and left eye +3.25 D, resulting in 6/6 (20/20) acuity. The slight reduction in acuity for the right eye was due to lens vacuoles. All pupillary reflexes were found to be present and normal: there was no indication of an afferent pupillary defect. Using the Van Herrick angle grading method, both eyes were found to have grade 1 anterior chamber angles. IOP's, measured by applanation tonometry, were right eye 28 mm Hg and left eye 25 mm Hg before dilation and right eye 29 mm Hg and left eye 28 mm Hg 1 h postdilation. Cup/disk ratios were right eye 0.35/0.45, left eye 0.3/0.3. A small superior-nasal notch of the right disk was found upon stereoscopic fundus examination. Gonioscopy showed unoccludable angles with the trabecular meshwork visible in three of the four quadrants for each eye, no peripheral anterior synechiae were found, and the scleral spur was seen in the remaining quadrant.

The patient was rescheduled for follow-up visits for Humphrey full-threshold fields and measurement of diurnal IOP variation. Full-threshold fields were found to be normal. Diurnal IOP variation was found to be within normal limits, varying no more than 1 or 2 mm Hg for findings taken at 8:30 a.m., 12:00 noon, and 4:30 p.m.

On December 3, 1991—6 weeks after the initial examination—the patient came to the Eye Care Center and was seen as a walk-in patient. She reported that she had awakened that morning with a severe, persistent headache in the right temporal

area, and she thought she was having a glaucoma attack. In addition to the temporal headache, she complained that she was sensitive to light, that her right eye and face felt numb, and that she had recently had three similar but milder attacks. Corrected acuities at that visit, and at all subsequent visits, were right eye 6/6- (20/20-) and left eye 6/6 (20/20). IOP's were found to be right eye 24 mm Hg, left eye 19 mm Hg (in the same range as her previous IOP findings). Because her IOP's were not elevated and gonioscopic findings were negative, in spite of the severe temporal headaches, a diagnosis of angle-closure glaucoma was ruled out and a tentative diagnosis of temporal arteritis was made. An erythrocyte sedimentation rate was ordered, and an appointment was made for the patient to see her primary care physician. The sedimentation rate, measured the following day, was found to be 34 mm/h. Against the wishes of the optometric staff, the primary care physician was unwilling to make an emergency appointment for the patient, believing that it was not warranted by the symptoms and the sedimentation rate. The patient later reported that during the 6-day period before her appointment with her physician, the temporal headaches, light sensitivity, and feeling of numbness persisted. In addition, she experienced what seemed to be dizzy spells, in which her vision would swim out of focus several times each day. Her vision did not completely black out during these out-of-focus periods, which lasted less than 1 s.

When the patient was examined by her physician, on December 9, 1991, the sedimentation rate was tested again and was 58 mm/h. The physician immediately prescribed oral prednisone, 40 mg (two 20 mg tablets)/day, and arranged for a temporal artery biopsy, which unfortunately could not be performed until 10 days later. After taking the 40 mg of prednisone for 2 days, the patient found that this dosage failed to completely control her temporal headaches, light-sensitivity, and facial numbness; she continued to have the transient episodes of blurred vision. She telephoned her physician, who instructed her to increase the prednisone dosage to 60 mg (3 tablets)/day. The 60 mg dosage controlled the temporal headaches and the transient losses of vision more effectively, and she no longer was aware of the neck, shoulder, and arm pains. However, after 3 more days, because of the persistence of mild headaches, light sensitivity, and numbness, the dosage was increased to 80 mg/day. This dosage controlled all of her symptoms and was continued for the next 12 days, at which time she began to experience adverse effects of prednisone therapy including indigestion, soreness of the mouth, nausea, swelling of the hands and face, and weakness in the arms and legs. The dosage was therefore decreased to 60 mg/day, and after an additional 7 days it was reduced to 40 mg/day.

The temporal artery biopsy was performed, as scheduled, 10 days after the prednisone therapy was begun. The surgeon reported that the right tem-

poral artery, upon palpation, was constricted and in spasm. However, the pathologist's report was negative for the presence of giant cells in sections of a branch of the temporal artery—the normal procedure is for the surgeon to excise a portion of a branch of the temporal artery rather than excising the temporal artery itself. Doppler sonographic studies were not considered necessary in this case, inasmuch as there was no reason to suspect carotid artery stenosis; fluorescein angiography was not considered necessary because there were no ophthalmoscopic signs of anterior ischemic optic neuropathy.

Because of the negative temporal artery biopsy, the primary care physician instructed the patient to begin tapering the prednisone dosage. At this point she asked to be referred to an internist, whom she first visited on January 9, 1992. In spite of the negative biopsy, which tended to make the diagnosis of temporal arteritis equivocal, the internist instructed the patient to continue the prednisone therapy. At that visit the sedimentation rate was found to be 12 mm/h. Because of the gastrointestinal effects of prednisone, the internist prescribed nizatidine (Axid), a histamine H₂ blocker, to be taken with the prednisone. On January 28, 1992 the sedimentation rate was found to be 18 mm/h, and the internist instructed the patient to decrease her prednisone dosage to 30 mg/day for the following 3 weeks, and then to reduce it to 20 mg/day.

The patient's ocular health was reassessed after she had been on prednisone therapy for 6 months, at which time her pressures were found to be very little higher than before the prednisone therapy was begun: right eye 32 mm Hg and left eye 28 mm Hg. Results of fundus examination and Humphrey visual field examination were negative.

The prednisone dosage was tapered over a period of 11 months, during which none of the symptoms of temporal arteritis returned, with one exception: when the dosage was decreased to 15 mg/day there was a recurrence of temporal headaches and the sedimentation rate increased to 32 mm/h. At this point, the dosage was increased to 30 mg/day for a period of 2 weeks, after which the tapering was resumed. Adverse effects of the prednisone, such as swelling of the face, gastrointestinal disturbances, and muscular weakness gradually dissipated during the 11-month period.

Approximately 4 months after the discontinuation of the prednisone therapy, the patient complained of almost debilitating pains in the shoulders, hips, and trunk, with occasional mild (but persistent) head pains. Until the head pains occurred, she thought the pains were manifestations of prednisone withdrawal, but a complete blood work-up revealed an erythrocyte sedimentation rate of 69 mm/h. On the basis of her symptoms and the high sedimentation rate, her medical practitioner concluded that the polymyalgia rheumatica and temporal arteritis had returned, and immediately resumed prednisone therapy beginning with 60 mg/

day. The long-term prognosis, as this paper is submitted for publication, is for protracted prednisone therapy but, hopefully, at a relatively low dosage.

Comment. When this patient originally visited the Eye Care Center, she was concerned that her blurred vision, headaches, and feeling of pressure in her eyes could have been due to glaucoma: no symptoms or signs of temporal arteritis were evident at that time. However, when she came to the clinic 6 weeks later with severe temporal and ocular pain, but her IOP's were found to be within normal limits (for this patient), the tentative diagnosis of temporal arteritis was made. The tentative diagnosis was supported by the second sedimentation rate, performed 6 days after the tentative diagnosis was made. The episodes of transient loss of vision began to occur several days before the patient's primary care physician was able to see her, so it is fortunate that prednisone therapy was begun without waiting for the temporal artery biopsy to be performed: a delay of another week or more could have resulted in severe and permanent loss of vision.

It is worthy of emphasis that in spite of the apparently normal temporal artery biopsy (performed 10 days after the prednisone therapy was begun), the internist wisely elected to continue the prednisone therapy. The importance of treating the condition as if it were temporal arteritis in the absence of definite proof to the contrary was demonstrated by: (1) the fact that the prednisone dosage of 60 mg/day controlled the transient losses of vision, temporal headaches, and pains in the proximal extremities, and (2) the fact that these symptoms returned 3 months later when the dosage was lowered to 15 mg/day.

Finally, this patient demonstrates the importance of closely monitoring the prednisone therapy, on the basis of sedimentation rate and the patient's symptoms. At various times during the management of the disease, a high sedimentation rate together with temporal headaches and transient losses of vision gave evidence that the dosage was inadequate, whereas a low sedimentation rate together with pronounced adverse effects indicated that the dosage could be reduced.

CONCLUSIONS

Because patients often associate headaches with eyestrain, and because the pain of temporal arteritis is often in the vicinity of the eye, many patients will seek out an eye care practitioner in the relatively early stages of the disease process, before vision loss has occurred. When episodes of transient loss of vision begin to take place, the eye care practitioner is likely to be consulted. For these reasons, optometrists should be aware of the symptoms and signs of temporal arteritis and should waste no time in referring the patient to a neurologist or internist for a clinical diagnosis and appropriate treatment.

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