Eyelash Formation Secondary to Latanoprost Treatment in a Patient With Alopecia

Eyelashes have a protective function against airborne particles. In addition, loss of eyelashes can be cosmetically unacceptable to some patients. For eyelash loss due to alopecia, treatments including oral steroids or other systemic medications have limited efficacy and can be potentially dangerous to the eye. We report growth of eyelashes after treatment with latanoprost in a patient with total loss of eyelashes for 5 years secondary to alopecia.

Report of a Case. A 53-year-old woman was referred to the glaucoma service of Devers Eye Institute, Portland, Ore, by an outside ophthalmologist. She was referred as a glaucoma suspect based on a positive family history of glaucoma, an enlarged cup-disc ratio without other signs of glaucomatous optic neuropathy, and intraocular pressures in the low 20s by Goldmann application tonometry. She had repeatable normal standard automated achromatic visual fields.

Five years prior to referral, she reported an allergic response presumed to be associated with ibuprofen that resulted in total loss of the eyelashes in both her eyes (Figure, left) and diffuse scalp hair thinning. Her eyebrows and hair and other parts of her body were not affected. She complained about an intermittent foreign-body sensation and her appearance. At the time of referral, examination revealed no evidence of an inflammatory process, eyelid tumor, or self-epilation. Findings from the remainder of her examination and history were unremarkable and included no past or present psychiatric disorders. Her intraocular pressure was 21 mm Hg and early glaucoma and optic neuropathy were noticed. Glaucoma and eyelash loss secondary to alopecia were diagnosed. Because of the hypotensive and hypertrichosis effect of latanoprost (Xalatan), it was offered as therapy. Three weeks following treatment, eyelashes were noticeable to the patient. Two months later full growth of her eyelashes occurred (Figure, right).

Comment. Diffuse shedding of hair is called alopecia or telogen effluvium. Drug-induced alopecia is usually confined to the scalp; however, the eyebrows, eyelashes, and body may be involved. This effect is thought to be due an immunologic mechanism against the hair follicle or melanocytes. The hair follicles are prematurely stimulated into apoptotic degeneration (catagen) and quiescence (telogen) resulting in shedding of the follicle. This immunologic process may inhibit the follicle from entering the anagen phase of growth. In most cases the hair follicle is not destroyed and after the disease activity abates, the hair follicle may spontaneously enter a normal cycle of anagen with resultant growth of the terminal hair. However, the chance of spontaneous growth decreases as the time period of the telogen phase increases.

Latanoprost, a phenyl-substituted analog of prostaglandin F2α, lowers intraocular pressure by increasing uveoscleral outflow. In addition to the ocular hypotensive effect, latanoprost causes hypertrichosis and hyperpigmentation of the iris and eyelashes. In a retrospective unmasked trial, Johnstone reported that in 43 patients receiving latanoprost treatment monocularly, hypertrichosis occurred in all of the eyes treated. Prostaglandins and specifically prostaglandin F2α, analogs bind to cell surface receptors activating phospholipase C. This enzyme orchestrates a variety of responses in cells such as stimulating gene expression and division. In our patient with eyelash loss of 5 years, it is possible that the dormant hair follicles were stimu-
lated to enter anagen and growth of eyelashes occurred.

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4. Camras CB, Alm A, Watson P, Stjernschantz J. Sarcoidosis. Respiratory or ocular symptoms. 2 In each case, it has been pos-

Sarcoid Inflammation and Obstruction of the Nasolacrimal System

We saw a patient with a medial canthal lesion and epiphora of the left eye of 18 months’ duration. Evaluation led to the diagnosis of sarcoidosis, but not before progressive cardiac involvement occurred, with a fatal outcome.

Report of a Case. A 58-year-old white woman was referred to our service for evaluation of a medial canthal lesion (Figure 1) and tearing of the left eye. Previous unsuccessful treatment by a community ophthalmologist included several months of oral antibiotics, antifungals, and topical corticosteroids, culminating in a left dacryocystorhinostomy. The incision included removal of the cutaneous lesion, which was found to be attached to the lacrimal sac. Biopsy specimens of lacrimal sac mucosa demonstrated noncaseating granulomas with epithelioid histiocytes (Figure 2). Three weeks postoperatively, the cutaneous lesion recurred and enlarged.

Her medical history was significant for an unspecified cardiac dysrhythmia treated with digoxin. Examination disclosed a 10 × 10-mm broadly elevated, erythematous mass with firm nodular components over the lacrimal sac. Nasal mucosa appeared normal.

Within 2 weeks of presentation, the patient was admitted to another hospital with atrial fibrillation. We recommended a serum angiotensin-converting enzyme determination and a gallium scan, which confirmed the diagnosis of sarcoidosis. During cardioversion, an embolus created a middle cerebral artery occlusion, which was fatal.

Comment. Since a review of cases of histologically verified sarcoidosis of the lacrimal sac in 1981,1 similar descriptions have appeared in the literature.2 In each case, it has been postulated that granulomatous inflammation of lacrimal tract mucosa, contiguous with similar infiltration of nasal mucosa, led to obstruction of outflow. Clinically significant blockage occurs in approximately 1.8% of patients with sarcoidosis.3

The differential diagnosis for granulomatous inflammation of the nasolacrimal system includes atypical mycobacterial infection, fungal infection, and Wegener granulomatosis. Several features of this case suggested a diagnosis other than sarcoidosis. Respiratory or ocular symptoms were absent. The medial canthal lesion was solitary; cutaneous involvement in sarcoidosis, while occurring in 9% to 37% of patients,4 more typically consists of erythema nodosum or clusters of small subcutaneous nodules. Nasal mucosal examination did not disclose the diffusely grayish appearance characteristic of sarcoid infiltration.

Patients with sarcoid-related obstruction of lacrimal outflow are at increased risk for failure after dacryocystorhinostomy. Of the 13 patients with histologically verified nasolacrimal system sarcoidosis reported in the literature, 9 had a total of 13 primary dacryocystorhinostomies. Four of the 13 surgeries resulted in failure. Although follow-up intervals and surgical technique (including placement of lacrimal stents) varied, the high failure rate suggests the presence of ongoing inflammation at the site of

Figure 1. A diffusely elevated, erythematous lesion is located over the left lacrimal sac.

Figure 2. Photomicrograph demonstrates the presence of noncaseating granulomas with epithelioid histiocytes and multinucleated giant cells within lacrimal sac mucosa (hematoxylin-eosin, original magnification × 100).

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the 7- to 11-o’clock positions and an RD bisected the fovea. Two large outer wall holes (OWHs) measuring 16 × 9 mm extended from the 7:30- to the 10:30-o’clock positions 8 mm anterior to the fovea. No inner wall hole (IWH) was visible with indirect ophthalmoscopy. Goldmann lens identified 3 pinpoint IWHs at the 10-o’clock position. There were no full-thickness breaks.

Inner wall holes were treated with transcleral cryotherapy and 0.6 mL of filtered air was injected through the temporal pars plana. The next day, both the RD and schisis cavity were flat. Laser photoagulation was applied around the OWHs (Figure). Ten months later, visual acuity was 20/30 OD. The retina and schisis cavity remained flat.

Comment. Pneumatic retinopexy is not a popular procedure for the repair of symptomatic retinoschisis-related RDs. These detachments, in which subretinal fluid extends well beyond the schisis cavity are rare and progressive. Surgical repair with pars plana vitrectomy, retinotomy, and simultaneous subretinal fluid drainage and intravitreal gas injection with cryopexy has been advocated.1,2 This case suggests surgical intervention with attendant risk need not be considered the standard or first option in all cases.

Histopathologic study findings concluded retinoschisis-related RDs may be caused by OWHs alone.3 However, RDs in these studies were limited to the area within or slightly beyond the schisis cavity and by definition they were not progressive rhegmatogenous RDs. Clinical studies are divided with respect to the observation of IWHs in symptomatic progressive RDs.1,2,4 Because contact lens examination was not always performed, it is possible that tiny IWHs below the resolution of indirect ophthalmoscopy may have been present but unrecognized in some cases. The importance of the IWH for the formation of progressive rhegmatogenous RDs may be underestimated.

Because OWHs are considered the important breaks, the goal has been to close OWHs with vitreoretinal surgical techniques. While OWHs alone may cause limited “schisis detachments,” IWHs may be more important in progressive rhegmatogenous RDs. They should be sought especially in patients with symptoms of acute vitreoretinal traction. The IWHs not visible with indirect ophthalmoscopy may be identified with Goldmann lens examination. Pneumatic retinopexy should be considered before more aggressive surgical techniques when full-thickness breaks or IWHs are identified superiorly. The initial goal of treatment should be to close these IWHs.

Fundus photograph of retina following pneumatic retinopexy. Laser scars demarcate the superior outer wall hole. Cryotherapy scar indicates location of treated inner wall holes. Schisis cavity and retina have flattened.

Pneumatic Retinopexy in a Progressive Rhegmatogenous Retinoschisis Retinal Detachment

Pneumatic retinopexy (PR) is effective for selected retinal detachments (RDs). Pneumatic retinopexy has failed in a previously reported case of retinoschisis-related RD.1 The successful management of a patient with progressive rhegmatogenous retinoschisis RD with pneumatic retinopexy is described.

Report of a Case. On August 27, 1998, a 52-year-old woman referred for retinal evaluation described photopsias, floaters, and a “curtain” in the right eye of 3 weeks’ duration. Best-corrected visual acuity was 20/40 OD. Bullous retinoschisis extended from nasolacrimal anastomosis. In the absence of long-term steroid administration, stents should probably be maintained for prolonged intervals.

Chronic cardiac dysrhythmia led to the patient’s death 18 months after the onset of her medial canthal lesion: 5% of patients with sarcoidosis have life-threatening cardiac dysfunction secondary to granulomatous inflammation, including dysrhythmias. Prompt diagnosis of sarcoid inflammation and obstruction of the nasolacrimal system may lead to earlier detection and management of the vision- and life-threatening complications of sarcoidosis.

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Whether or not additional laser demarcation of OWHs is necessary to prevent redetachment is uncertain. Advantages of PR include lower risk of surgical complication and lower cost. Pneumatic retinopexy has failed in a similar case. However, it is uncertain whether IHWs were treated specifically. Further investigation will confirm whether PR is an effective, less invasive way to manage progressive rhegmatogenous retinoschisis RDs.

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Diffuse Unilateral Subacute Neuroretinitis in China

We report on the first known case of diffuse unilateral subacute neuroretinitis (DUSN) found in Asia. It is important for physicians in those areas in which DUSN has been described to remain vigilant in looking for its occurrence as well as being aware of its possibility in regions in which it has not yet been identified as endemic.

Report of a Case. A 24-year-old Chinese woman was first seen at the Department of Ophthalmology, Shanghai ChangZheng Hospital, September 15, 1998, with the complaint of a 2-week history of painless progressive loss of vision in her left eye. Her ocular history was unremarkable. On examination she was found to have a best-corrected visual acuity of 20/20 OD and 20/200 OS (her affected eye). There was no pallor of the left optic disc and no apparent arteriolar attenuation; however, a left relative afferent pupillary defect was present. Diffuse, small patchy retinal pigment epithelium changes with 2+ cells in the vitreous and a decreased macular reflex were present. Intraocular pressure was normal. Absence of a pattern visual evoked potential was noted and the flash visual evoked potential response was positive in the left eye. The right eye was normal.

On September 22, 1998, while the patient was being examined, a motile white worm was seen about 1.5 disc diameters inferior to the fovea. It was approximately 1500 µm long and 80 µm wide. The following day the worm was found inferonasal to the optic disc with its head and tail embedded more deeply in the retina. The nonsegmented cylindrical nematode just overlay the vein with an S-shaped configuration (Figure 1) and accompanying hemorrhage. One week later, the hemorrhage was absorbed and the nematode changed to a V shape (Figure 2).

The patient was born and raised in an urban area (Shanghai, China) with no experience of close contact with cats, dogs, or raccoons. She had never ingested raw fish nor had she drunk fresh water. No skin lesions or changes could be found. Results of a complete blood cell count with differential cell count were within normal ranges, with no eosinophilia. No worm or egg was found in her stool. Findings from a magnetic resonance imaging scan of the orbit and brain were normal. Fluorescein angiography of the left eye demonstrated scattered small areas of irregular hyperfluorescence with leakage near the nematode.

Laser photocoagulation (focal Argon green) was applied on October 15, 1998. The patient was followed up for more than 11 months without recurrence of the inflammation. Her best-corrected visual acuity in the affected left eye has remained 20/200. No other parasites were noted. The area of laser photocoagulation demonstrated evidence of chorioretinal scarring.

Comment. Diffuse unilateral subacute neuroretinitis is typically characterized by progressive visual loss secondary to inflammatory changes in the retinal pigment epithelium, retinal vessels, and optic nerve. It usually affects young healthy individuals. Evidence exists that DUSN is caused by infestation of a solitary intraretinal or subretinal nematode whose exact identity remains uncertain. Several species of nematodes, including Toxocara ca-
nis, Baylisascaris procyonis, and An
cylostoma caninum have been sug-
gested as the potential etiologic agent
of DUSN.

The nematodes have been clas-
sified into 2 different sizes. The
smaller nematode, measuring 400 to
1000 µm in length, is endemic to the
southeastern United States, the Ca-
ribbean islands, and Brazil. The
larger nematode, measuring 1500 to
2000 µm in length, has been de-
scribed in the northern midwest-
er United States.\(^3\)

**Toxocara canis** and **B procy-**
nis are the most common causes of
internal larva migrans in animals and
humans, and either parasite could be
involved in human ocular larva mi-
gans and DUSN. Morphologically,
the nematode in this case appears to
be a large one and is most likely
**B procyonis**. To our knowledge, this
is the first reported case of DUSN
occurring in Asia.

Accurate diagnosis of the dis-
eease is important because destruc-
tion of the worm in early stages will
halt the progression of visual loss.
Observation and destruction of the
nematode are the methods of choice
for accurately reaching a diagnosis
and treating patients with DUSN.
Medical treatment is generally be-
lieved to be ineffective because of
the relative impermeability of the
blood-retinal barrier. A recent re-
port confirmed this.\(^4\) Laser treat-
ment remains an effective means of
treatment.

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**Figure 2.** One week later from Figure 1, the nematode’s appearance has changed to a V shape, with its head and tail more deeply embedded in the retina. The hemorrhage has cleared.