Retinopathy as the Initial Presentation of Human Immunodeficiency Virus 2 Infection

Human immunodeficiency virus (HIV) 2 is a human lentivirus, which shares 40% to 50% genetic homology with HIV-1 but has distinct epidemiologic, biological, and clinical features. First described in Senegal in 1985, HIV-2 is endemic in many west African countries, but is uncommonly found outside of that region. Human immunodeficiency virus 2 has been reported in European countries and in North America. In the United States, less than 100 cases have been reported. In this small study, to our knowledge, patients with HIV-2 infection are limited. In HIV-1 infection, the pattern of symptoms and opportunistic illnesses are more similar for HIV-1 and HIV-2.

On review of risk factors for HIV, after the diagnosis of HIV-2, the patient admitted to having multiple heterosexual partners in Burkina Faso before moving to the United States.

Comment. The clinical course of HIV-2 is characterized by progressive CD4 lymphocyte depletion, which occurs at a slower rate compared with HIV-1. With advancing immunosuppression, the pattern of symptoms and opportunistic illnesses are quite similar for HIV-1 and HIV-2.

Risk factors for HIV-2 infection are analogous to those for HIV-1, with sexual contact as the predominant means of transmission (Table). Since 1992, US blood banks have screened all blood products with a combination HIV-1/HIV-2 enzyme immunoassay. No transfusion-acquired cases of HIV-2 have been reported. Coinfection with HIV-1 and HIV-2 has been described; however, there is evidence that HIV-2 infection may decrease the risk of subsequent HIV-1 infection.

Serologic testing for HIV-2 is readily available through most HIV testing centers and state and local health departments. Many commercially available HIV test kits combine assays for HIV-1 and HIV-2. The combined assay is reactive, HIV-1 Western blot testing is performed. If the combined assay is reactive, HIV-1 Western blot testing is performed. If the combined assay is reactive, HIV-1 Western blot testing is performed. If the combined assay is reactive, HIV-1 Western blot testing is performed. If the combined assay is reactive, HIV-1 Western blot testing is performed. If the combined assay is reactive, HIV-1 Western blot testing is performed. If the combined assay is reactive, HIV-1 Western blot testing is performed. If the combined assay is reactive, HIV-1 Western blot testing is performed. If the combined assay is reactive, HIV-1 Western blot testing is performed. If the combined assay is reactive, HIV-1 Western blot testing is performed. If the combined assay is reactive, HIV-1 Western blot testing is performed. If the combined assay is reactive, HIV-1 Western blot testing is performed. If the combined assay is reactive, HIV-1 Western
for vascular retinopathy. Human immunodeficiency virus 2 remains a rare disease in the United States but must be considered in at-risk individuals. Our case serves to increase awareness of HIV-2 infection, and indicates the epidemiologic features that should prompt HIV-2 testing (Table).

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Optic Neuropathy After Burns

We examined 2 new cases of postburn optic neuropathy, and 10 additional cases were identified in the medical literature. The onset was either immediate or delayed for weeks. Patients did not necessarily manifest symptoms of an encephalopathy before the onset of visual loss. Loss of vision was severe and bilateral in all cases, with disc edema present in most. Retinal hemorrhages and edema were observed in some cases. There was a capacity for spontaneous recovery.

We conclude that patients with cutaneous scalds or thermal burns may develop bilateral optic neuropathy, the pathogenesis of which has yet to be established. Visual loss following burns usually results from direct injury to the ocular surface. However, in some instances the visual loss is neurogenic. Our goal in presenting the following cases is to document the rarely reported occurrence of optic neuropathy following cutaneous thermal burns.

Report of Cases. Case 1. A 25-year-old man was burned on May 11, 1994, when the furnace that he was cleaning exploded, pinning him in a pit of smoldering ash. He did not lose consciousness and was alert but incoherent on admission to a local hospital, with partial- and full-thickness burns covering 90% of his body. His entire face was burned, and his eyelids were absent. Morphine and 10 L of intravenous fluid were administered. The patient was intubated, given a central line, paralyzed with drugs, and flown to a burn unit at another hospital. At the time of transfer, the size and reactivity of his pupils were described as normal.

The patient was alert and responding to commands on his arrival at the burn unit. His blood pressure was 170/110 mm Hg, and his pulse varied from 120 to 140 beats per minute. Arterial blood gas analysis showed a PO$_2$ of 66 mm Hg, oxygen saturation 88.7%, pH 7.24, and total carbon dioxide 22 mm/L. The hemoglobin level was 215 g/L, and the hematocrit was 65%. During escharotomies, a fall in his oxygen saturation necessitated a criocryothiroidotomy. He remained in the burn unit for 7 months, the first 4 of which he was maintained in a drug coma. His course was complicated by transfusions required for anemia, as well as transient renal failure, pneumonia, sepsis, and urinary tract and skin infections for which he received a variety of antibiotics. While in coma, he was examined several times by an ophthalmologist, who reconstructed his eyelids and treated him for bilateral exposure keratopathy complicated by an infection of the right cornea. There was no report of his pupillary reactions or of the fundus appearance.

When he was extubated 6 months after the accident, the patient reported poor vision in both eyes. This was initially ascribed to his corneal lesions. One month later, the patient complained specifically of a blind spot in the center of the visual field of both eyes. An ophthalmologist performed perimetry and concluded that he had bilateral optic neuropathy. Twenty-one months after sustaining his burns, the patient’s vision was unchanged, and he was referred for neuroophthalmic evaluation.

His best-corrected visual acuities were 20/20 OD and 20/200 OS. Color vision (Ishihara) was normal in both eyes. There were bilateral central scotomas on testing with the Goldmann perimeter. Both corneas were scarred below the visual axis, but the anterior segments were otherwise normal. His pupillary reactions and eye movements were unremarkable. Applanation intraocular pressures were 15 mm Hg OD and 14 mm Hg OS. Both optic discs were atrophic with sharp margins and 0.6 cup-disc ratios. The fundi were otherwise normal. No abnormalities were found on the general neurological examination or on magnetic resonance images of the orbits. The patient was unchanged when reexamined 1 and 2 years later.

Case 2. A 13-year-old girl suffered a flame burn in a house fire, which covered 85% of her body, including her face. Although further details are not available, it is known that the patient lost consciousness and then complained of poor vision when she first became sentient several days later. There had been no previous eye problems.

The patient was referred 3 months later because of impaired vision. At that time, she was alert and cooperative. Vision in her right eye was limited to counting fingers at 1 ft, and she could not see light with her left. Her color vision was nonexistent, and she could not cooperate for perimetry. Both pupils were dilated. The right had a faint response to light stimulation, whereas the left had none. Her ocular motility was unimpaired. The skin of all 4 eyelids was scarred. There was a slight central stromal haze of both corneas, but the conjunctivae, anterior chambers, irides, and lenses appeared normal. Her intraocular pressure seemed normal by palpation. The optic nerves were extremely pale with sharp margins. Both fundi were otherwise unremarkable. We were informed of subsequent improvement in vision.

Comment. Some patients with thermal burns suffer severe loss of vi-
sion even without direct injury to the eyelids or globes. Visual impairment in such cases is caused either by intracranial lesions or by optic neuropathy.

Intracranial cases are often lumped as burn encephalopathy, a term that specifies neither a characteristic neuropathological lesion nor a particular set of symptoms. Infection, metabolic disturbances, and hypoxia are undoubtedly contributing factors in some cases. Although the exact pathogenesis of burn encephalopathy is uncertain, it may develop from scalds, which makes it difficult to blame hypoxia or carbon monoxide inhalation. Direct heating of the brain cannot be a contributing factor, because the burns did not necessarily involve the head. In several reported cases, encephalopathy even developed from burns that were not particularly extensive.

Burn encephalopathy, which is typically heralded by seizures and diminished consciousness, may develop immediately or be delayed for more than a month. Profound and bilateral visual impairment has been reported in some patients. These patients were presumably blind on a cortical basis, but in several instances hydrocephalus may also have been a factor. We have identified only 10 published cases of visual loss from the intracranial complications of burns that were not associated with such confounding conditions as meningitis. Unfortunately, most of the case histories were incomplete or contained confusing information. For example, it is unclear how the diagnosis of cortical blindness could have been established in patients described as being in coma. In a few cases, there might also have been mechanical injury, which further confuses the issue of pathogenesis.

Central visual impairment following burns is undoubtedly more common than one would infer from the paucity of published cases, because deficits such as coma would mask visual loss. In addition, victims might be too young to report the symptom. Based on our review of published cases, it appears that central visual loss following burns is a disorder of childhood, can be severe, can be caused by scalds or flame burns, can appear after a delay of several weeks, is accompanied by other neurological symptoms, and in some cases provides a remarkable opportunity for complete recovery. The apparent paucity of adult cases defies explanation. Fortunately, the incidence of burn encephalopathy seems to be decreasing.

Mooren was the first to report an optic neuropathy after cutaneous burns, a complication that he considered by no means rare. He also commented that neuropathy sometimes followed minor burns. Peripapillary hemorrhages were a common feature. His patients exhibited a great capacity for recovery; however, he did mention 1 case of severe bilateral loss of vision following superficial leg burns in which recovery did not occur. We found 10 published cases of optic neuropathy or neuroretinopathy ascribable to burns. Like the central cases described previously,...
Visual loss followed scalds or flame burns, was severe and bilateral, could appear after a delay of weeks (average delay, 3 weeks), and sometimes recovered. Unlike the central cases, optic neuropathy occurred in young adults and adolescents as well as in children. Seven of the patients were men between the ages of 17 and 25 years. In most cases the burns were deep, extensive, and involved the face. Half of the patients were otherwise neurologically normal at the time their visual impairment became evident. Seven patients had disc edema and either a hemorrhagic retinopathy or retinal edema. In 2 cases the optic discs initially appeared normal, and in 1 case they were described as pale. We found optic atrophy in our 2 patients, but these examinations were not performed until months after their loss of vision. The preservation of our first patient’s color vision despite optic neuropathy remains unexplained.

Many burned patients have respiratory, infectious, and metabolic complications and receive multiple medications. In light of the multiplicity of factors, it is impossible to identify with certainty the exact cause of the optic neuropathy that follows thermal burns, either in our cases or in those from the literature. However, the occurrence of optic neuropathy after scalds makes it unlikely that hypoxia or carbon monoxide inhalation could be responsible. The delayed onset and the remarkable capacity for recovery observed in some cases do not support an anoxic neuropathy. One possible mechanism is a toxic optic neuropathy from one of the medications. For example, hexachlorophene, which can cause edema of myelin, was once widely used to treat burns.25,26 It or another drug might have played a role in certain cases. Hypotension, especially if potentiated by anemia, could cause an ischemic optic neuropathy. Although hypotension was not documented in these cases, it might well have occurred. Because the face was burned in both of our patients and in at least 8 of the 10 published cases, it is possible that there was a direct thermal effect on the optic nerve or retina.

Although it would be difficult to reconcile with the retinal findings in some cases, another possibility is that visual loss resulted from demyelination mediated by a toxin released from burned skin. Demyelination would allow for restitution of vision. Williams23 described the autopsy findings in a patient who lost vision 15 days after being burned and who died 25 days later. In addition to subacute necrotizing lesions in the brain proper, there was extensive demyelination in the anterior visual pathways. Demyelinating lesions have been found in the brain at autopsy in several other cases, which raises the possibility that cutaneous burns are capable of inducing a demyelinating optic neuropathy.1,27,28 Some authors have theorized that the lesions might be caused by the release of a neurotoxin from burned skin.29-30 A circulating neurotoxin could explain the case reported by Resch and Sullivan,21 in which a patient, despite being blind, had no lesions of the eye, brain, or optic nerve when autopsied.

Patients who have suffered flame burns or scalding of the skin may develop bilateral optic neuropathy, usually with severe visual loss but a capacity for spontaneous recovery. Retinal hemorrhages and edema may be present. Visual loss may develop immediately or after a delay and need not be accompanied by neurological symptoms. The pathogenesis of the neuropathy has yet to be established, but in some cases it may result from demyelination.

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Nasopalpebral Lipoma–Coloboma Syndrome

Nasopalpebral lipoma–coloboma syndrome is an autosomal dominant syndrome characterized by congenital upper eyelid and nasopalpebral lipomas, upper and lower eyelid colobomas, telecanthus, and maxillary hypoplasia. Ours is the third report of this dysplasia-malformation syndrome and the first report in the ophthalmic literature.

This rare syndrome was first described in a Venezuelan family\(^1\) and later in a Turkish family.\(^2\) Our patient is the proband for the third family reported with this dysplasia-malformation syndrome and the first with reported computed tomographic findings.

**Report of a Case.** The patient was a full-term male newborn, born at 39 weeks gestation to a 36-year-old, gravida 2, para 2 mother. The patient’s mother had received good prenatal care and denied tobacco, alcohol, or other drug use during her pregnancy. The patient was delivered via spontaneous vaginal delivery with Apgar scores of 8 and 9 at 1 and 5 minutes, respectively. Oxygen was administered for 2 minutes following birth, after which the patient did well on room air. Birth weight was 3.3 kg; birth length was 42.3 cm.

At delivery, the patient was noted to have anomalous facial features. The parents denied any family history of similar findings. An older sibling at home was noted to be healthy.

On physical examination, the patient was alert, crying, and moving all extremities spontaneously. There were no signs of acute distress or neurologic impairment. The nares were patent bilaterally. The patient’s palate was intact and he had a single, midline uvula.

Ophthalmologic consultation was obtained at 1 day of age. Marked telecanthus and large, bilateral upper eyelid colobomas at the junction of the medial one third and lateral two thirds of the eyelids were noted (Figure 1). Lacrimal puncta and canaliculi were absent from the upper eyelids bilaterally. Both lower eyelid puncta were patent and in the normal position. Pupils were equal, round, and reactive; no afferent pupillary defect was present. Penlight examination of the anterior segment and dilated fundus examination results were within normal limits in both eyes. No corneal stain or opacity was present. The patient was noted to have a flat, wide nasal bridge with a soft, raised mass located between the eyes and involving the medial aspect of both upper eyelids. The rest of the physical examination results were unremarkable.

Computed axial tomography with 3-dimensional reconstruction revealed a normal brain structure. Evaluation of the bony anatomy disclosed normal intraorbital distance, prominent sutures in the anterior fontanelle, and mild maxillary hypoplasia. Lipomatous tissue involving the forehead and nasal bridge was noted (Figure 2).

A hearing test including otocoustic emissions and auditory brainstem response had normal results. Cytogenetic studies showed no evidence of a chromosome abnormality. It was felt that the patient’s craniofacial dysmorphism had resulted from a spontaneous mutation.

At 10 days of age, the patient underwent exploration of the lacrimal drainage systems and repair of the bilateral upper eyelid colobomas. Inspection of both upper lids failed to reveal any remnants of the lacrimal puncta or canaliculi. Probing of the lower puncta revealed normal anatomy on the right side, while a soft tissue density was noted at the level of the common canalicus on the left. Full-thickness tissue was then excised along all aspects of the coloboma to create fresh edges and a pentagonal-shaped defect. There was no tarsal remnant medially in either eyelid. The lateral tarsal remnant was normal. The defects were closed primarily, with several sutures placed between the tarsal remnants and the remnants of the medial canthal ligament to support the posterior lamella. The coloboma on the left was more lateral than that on the right, with greater deformity of the medial canthal region, resulting in a slightly abnormal contour of the reconstructed upper eyelid. The contour on the right side was more natural.

At 5 months of age (Figure 3), the patient underwent transcoronal excision of the forehead, nasal, and bilateral upper eyelid lipomas as well as transnasal bilateral medial canthopexy. The lipomatous tissue was subcutaneous in the forehead and extended into the eyelids and medial canthus in a preseptal plane; it was discrete from the orbital fat. The punctae were again probed bilaterally at the time of this surgery and the lower canaliculi were found to be intact. However, no nasolacrimal duct remnants could be found. No discrete medial canthal tendons were visible, but there was dense tissue in the medial aspect of the lower eyelids which was felt to be of adequate strength to undergo a medial canthopexy. Two 1.0-mm holes were drilled...
Figure 3. The patient at age 5 months after bilateral upper eyelid coloboma repair, before undergoing excision of the bilateral nasopalpebral lipomas.

Figure 4. The patient at age 12 months, 7 months after transcoronal excision of frontal, nasal, and bilateral palpebral lipomas as well as transnasal bilateral medial canthopexy.

Future management of this patient will include further surgical repair of his telecanthus, nasolacrimal duct reconstruction, and continued follow-up for strabismus and amblyopia.

Comment. The similarities between the examination results of this patient and those of the previously reported cases of nasopalpebral lipoma–coloboma syndrome are striking. Penchaszadeh et al described 8 affected individuals from 1 family in Venezuela, while Akarsu and Sayılı later reported 7 similarly affected patients in 3 generations of a Turkish family. The syndrome was described as an autosomal dominant condition with congenital lipomas symmetrically present under the upper eyelid and nasopalpebral skin, giving rise to marked telecanthus. Eyelid colobomas were bilateral and symmetric, involving both upper and lower eyelids in the majority of cases. However, 1 patient in the Penchaszadeh et al series had only unilateral upper eyelid colobomas, similar to our patient. All patients described by Penchaszadeh et al had marked exotropia and a few patients had corneal or anterior subcapsular lens opacities. Fundus examination results were normal. Visual acuity tested in 2 adult patients was reported to be within normal limits or consistent with other ocular findings (cataracts and exotropia). Skull x-ray films showed normal intraorbital distances and midface hypoplasia, which our patient also exhibited.

Other syndromes which are associated with eyelid colobomas include Goldenhar syndrome, Treacher-Collins syndrome, frontonasal dysplasia, and Dellemann syndrome, and a new, unnamed syndrome described by Balci et al consisting of upper eyelid coloboma, hypertelorism, hypospadias, and mixed-type hearing loss. Defining features of these syndromes include preauricular appendages, epibulbar dermoid, and vertebral abnormalities (Goldenhar syndrome); orbital hypertelorism, nasal tip bifidity, and median facial cleft (frontonasal dysplasia); orbital cysts, agenesis of the corpus callosum, and punctate defects of the lip, philtrum, and nose (Delleman syndrome). Our patient did not exhibit any of these other key findings.

The pathogenesis of eyelid colobomas is not well understood since normal eyelid development does not produce clefting. It is possible that interference in the epithelial adhesion of the eyelid folds during gestation may cause maldevelopment of the eyelid and associated structures. Alternatively, it has previously been postulated that central defects such as disturbances in the migration of neural crest cells may produce symmetric eyelid colobomas such as those seen in Treacher-Collins syndrome.

The findings in this report were thought to be consistent with nasopalpebral lipoma–coloboma syndrome, an autosomal dominant dysplasia-malformation syndrome previously reported in 2 families in the genetics literature. We believe this patient is the proband for the third reported family of this rare dysplasia-malformation syndrome and the first case of nasopalpebral lipoma–coloboma syndrome reported in the ophthalmic literature thus far.

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Penetrating Orbital Injury by Automobile Wiper-Control Stalk

A restrained driver fell asleep at the wheel and crashed his truck into a tree. He was found walking near the crash site with the windshield wiper stalk impaled in his right cheek. It had penetrated the right maxillary sinus, both ethmoid sinuses, and the left orbit, contusing his left optic nerve and rendering him blind in that eye. To our knowledge, no such injury has ever been documented. Using data obtained from the crash site, we present a hypothetical reconstruction of the accident.

Report of a Case. A 21-year-old man fell asleep at the wheel and crashed his truck into a tree. He was found fully alert and walking near the scene, with the windshield wiper stalk impaled into his right cheek (Figure 1 and Figure 2). It had pierced the gingiva and penetrated the right maxillary, both ethmoid sinuses, and the left orbit, deviating and contusing the left optic nerve (Figure 3). The injury rendered him blind in his left eye and partially ophthalmoplegic.

An injury of this nature has not been recorded in the forensic records of automobile companies in the United States. We present a hypothetical reconstruction by automotive crash analysts (L.S., J.M.).

Medical Aspects. The wiper stalk pierced the skin approximately 4 cm above the right upper lip margin. Apart from minor skin abrasions elsewhere and a rib fracture, there

Figure 1. Preoperative view of the windshield wiper stalk impaled in patient’s right cheek.

Figure 2. Computed tomography scout film shows penetration of stalk through paranasal sinuses and left orbit.

Figure 3. Coronal computed tomography film shows stalk deviating left medial orbital wall.
were no other injuries. The patient was neurologically intact. Visual acuity was 20/20 OD and no light perception (NLP) OS. There were no abnormalities of the right eye and orbit. The left eye was 4 mm proptotic, had complete ptosis, and a pupil dilated to 8 mm that did not react to direct light. A left afferent pupillary was present. The left eye was located in midorbital position, and ductions were absent in all directions. Biomicroscopy images showed no abnormalities, and intraocular pressures were 14 mm Hg in the right eye and 20 mm Hg in the left eye. Ophthalmoscopy results were normal in the right eye and revealed retinal hemorrhages that obscured the optic disc of the left eye.

A bifrontal craniotomy gave access to the anterior cranial fossa, nasal structures, and left orbit. After orbital bone fragments surrounding the wiper stalk were removed, its protruding portion was grasped with a wrench and pulled out through the entry site. It measured 25 mm in diameter at its leading edge, and was 150 mm long (Figure 4).

Postoperatively, the patient's vision in the left eye remained NLP despite his being administered high doses of intravenous methylprednisolone. Several months after the procedure was performed, the left ptosis and proptosis had resolved (Figure 5), and the eye had regained nearly full motion, but it remained blind. Ophthalmoscopy images disclosed a pale optic disc.

Crash Analysis. The crash involved the high-speed, off-road, frontal impact of a 1994 Chevy S-10 (General Motors, Detroit, Mich) pickup truck. The driver, who had reportedly been drinking, had fallen asleep. His truck crossed the road's center line, drifted off of the left side of the road, and struck a large tree with the right front bumper, grille, and hood (Figure 6). Based on crush measurements and the WinSmash\textsuperscript{1} crash-reconstruction program, the truck was traveling at a speed of 45 mph to 50 mph. The impact caused the vehicle to rotate clockwise so that it then struck a second tree near the front of the driver's door.

The driver was wearing the 3-point restraint, as evidenced by a clear imprint of the belt-webbing pattern on the plastic D-ring. There were no airbags. Although this impact was to the right of the vehicle's center line, it produced 20 cm of rearward intrusion of the toepan/footwell in the driver area and 5 cm
of rearward intrusion of the driver instrument panel. The steering column's shear capsules separated during the impact (Figure 7), freeing the steering column to rotate laterally and vertically.

**Comment.** How could a wiper-control stalk located on the left side of the steering column have pierced the driver's right cheek? We believe that as he fell asleep, he slumped forward and leftward, pulling a length of shoulder belt webbing out of the retractor (Figure 8 A). Physical evidence of clothing transfers on the interior door panel supports this hypothesis. On first impact with the large tree (Figure 8B), his head moved farther forward, locking the shoulder belt. The clockwise rotation of the truck moved it into the second tree to its left, causing a counterclockwise deceleration (Figure 8D), which stopped the clockwise rotation and leftward movement of the vehicle and driver. By this time, however, the steering wheel and column were rotating to the left. With the driver's face well forward of the steering-wheel rim (Figure 8C), the leftward-moving steering column acted like a heavy pendulum and pushed the leftward-pointing wiper stalk through the driver's right maxilla.

This is a sobering tale. Shoulder restraints could not protect the driver from extreme forward and leftward deviation of the head, which placed the driver in a position to be impaled by the wiper stalk. He is fortunate to have escaped with the isolated obliteration of 1 optic nerve.

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