

Nasolacrimal Duct Obstruction and Orbital Cellulitis Associated With Chronic Intranasal Cocaine Abuse

George Alexandrakis, MD; David T. Tse, MD; Robert H. Rosa, Jr, MD; Thomas E. Johnson, MD

Objective: To report the association of acquired nasolacrimal duct obstruction and orbital cellulitis in patients with a history of chronic intranasal cocaine abuse.

Methods: Retrospective, consecutive case series. Results of imaging, histopathologic examinations, and clinical courses of these patients were studied.

Results: Five women and 2 men (mean age, 41 years) with a history of chronic intranasal cocaine abuse (mean, 11 years; range, 5-20 years) presented with epiphora and in some cases acute onset of periorbital pain, edema, and erythema associated with fever. The suspicion of intranasal cocaine abuse was made on anterior rhinoscopy with the detection of an absent nasal septum and inferior turbinate. Computed tomographic and magnetic resonance imaging findings in 4 patients included extensive bony destruction of the normal orbital wall architecture,

opacification of the sinuses, and the presence of an intraorbital tissue mass. Histopathologic examination of the nasolacrimal duct in 2 patients and of the orbital mass in a third patient revealed marked chronic inflammation with fibrosis causing secondary nasolacrimal duct obstruction. Six patients were treated with systemic antibiotics followed by dacryocystorhinostomy in 3 patients, and a pericranial flap to insulate the exposed orbit in 1 patient.

Conclusions: Chronic intranasal cocaine abuse can result in extensive bony destruction of the orbital walls with associated orbital cellulitis, and should be included in the differential diagnosis of acquired nasolacrimal duct obstruction. Anterior rhinoscopy is very helpful in establishing the correct diagnosis in these patients.

Arch Ophthalmol. 1999;117:1617-1622

COCAINE IS a naturally occurring alkaloid of the coca plant *Erythroxylon coca*. Its pharmacological properties are secondary to stimulation of the central and sympathetic nervous systems by augmenting the effects of norepinephrine. The use of cocaine as a topical anesthetic in ophthalmology was described by Koller¹ in 1884. Topically applied cocaine causes mydriasis by blocking the reuptake of norepinephrine and is used in evaluating anisocoria. In a higher concentration, cocaine may cause cycloplegia and upper eyelid retraction.²

Cocaine abuse is an ongoing public health concern and its prevalence and effects continue to draw attention. A US government report estimated that 28.7% of young adults aged 18 to 25 years have used cocaine at least once.³ When used illicitly, cocaine is usually taken intranasally, intravenously, or by inhalation. Depending on the route of administration, several medical complications can occur.⁴ Intravenous use can result in myocardial infarction, cardiac arrhythmia, rupture of the ascending aorta, cerebrovascular accident, subarachnoid hemorrhage, hypertensive crisis, seizure, pulmonary edema, and death.⁴ The intranasal route is associated with complications related to chronic irritation of

the delicate upper respiratory tract mucosa. Cocaine induces intense vasoconstriction and anesthesia of the respiratory mucosa; this may lead to mucoperichondrial ischemia, necrosis, and nasal septum perforation.^{2,5} Many intranasal cocaine abusers develop rebound nasal stuffiness, which is self-treated with over-the-counter nasal inhalants containing vasoconstrictors that contribute to further nasal mucoperichondrial necrosis. Repetitive cycles of vasoconstriction lead to marked destruction and result in osteolytic sinusitis with total bony and cartilage necrosis or saddle nose deformity.^{2,5,6}

We describe 7 consecutive cases of acquired nasolacrimal duct obstruction (NLDO) associated with chronic intranasal cocaine abuse. Three of the patients also presented with concomitant orbital cellulitis secondary to the chronic sinusitis and destruction of the orbital bony walls. Histopathological studies are presented and the management of these patients is discussed. Ophthalmologic complications associated with cocaine abuse are reviewed.

RESULTS

Seven consecutive patients (2 men and 5 women) with a mean age of 41 years (age range, 36-58 years) were identified. Pre-

From the Bascom Palmer Eye Institute, University of Miami School of Medicine, Miami, Fla.

SUBJECTS AND METHODS

The study conformed to the policies outlined by the Human Subjects Subcommittee of the University of Miami School of Medicine, Miami, Fla. The described patients were identified from a review of the medical records of the Bascom Palmer Eye Institute for a 6-year interval (1993-1998). All patients underwent a complete history, physical examination, and ophthalmic evaluation. Diagnostic tests included evaluation of the lacrimal system with the dye disappearance test and Jones test, and evaluation of the sinuses and orbits with computed tomographic (CT) or magnetic resonance imaging.

senting signs and symptoms included epiphora or peri-orbital pain, edema, and erythema associated with fever. The suspicion of intranasal cocaine abuse was made on anterior rhinoscopy with the detection of an absent nasal septum and inferior turbinate. All patients had a history of chronic intranasal cocaine abuse with an average of 11 years (range, 5-20 years). Lacrimal irrigation confirmed complete blockage. Computed tomographic and magnetic resonance imaging studies in 4 patients disclosed extensive destruction of the orbital wall architecture, opacification of the sinuses, and the presence of an intraorbital tissue mass. Findings from histopathologic examination of the nasolacrimal duct (NLD) and orbital mass revealed marked chronic inflammation with fibrosis causing secondary nasolacrimal duct obstruction. Six patients were treated with systemic antibiotics, followed by dacryocystorhinostomy (DCR) in 3 patients and a pericranial flap in 1 patient. The cases are summarized in the **Table**.

REPORT OF CASES

CASE 1

A 39-year-old man with a 12-year history of intranasal cocaine abuse complained of severe left retrobulbar pain aggravated by nasal breathing, diplopia, and constant tearing of the left eye for several months. His medical and ocular histories were otherwise unremarkable. He denied intravenous cocaine use. Visual acuity was 20/20 OU. Motility showed decreased abduction and absent adduction of the left eye. External examination disclosed marked left peri-orbital swelling and erythema, inferior displacement of the left globe, and saddle nose deformity. The remainder of the ophthalmologic examination was unremarkable. Anterior rhinoscopy disclosed that the nasal septum and middle and inferior turbinates were absent. The terminal point of the NLD was occluded by scar tissue. Lacrimal irrigation confirmed complete blockage.

Computed tomographic scan revealed absent bony and cartilaginous nasal septum, and extensive loss of normal ethmoidal and antral bony architecture (**Figure 1**). The left medial orbital wall and floor were absent. A course of oral antibiotics was given without improvement. Bi-

opsy of a left medial orbital soft tissue mass disclosed dense fibrosis with chronic inflammation, partially involving the skeletal muscle. Occasional Russell bodies were present (**Figure 2**). Cultures were negative for organisms. A course of oral prednisone (1 mg/kg per day) was started. Because of persistent orbital pain and minimal response to steroids, a pericranial flap harvested from the forehead was used to insulate the exposed orbit from the external environment and air turbulence within the nasal cavity.⁷ The flap was secured into position within the orbit by wedging it between the posterior floor and periorbita.⁷ Postoperative anterior rhinoscopy revealed a smooth, mucosalized tissue layer lining the medial wall and orbital floor. Motility showed improved abduction of the left eye. The patient was able to breathe without orbital discomfort. Tearing persisted, but he declined surgery to relieve this symptom. He died 14 months later secondary to a myocardial infarction.

CASE 2

A 40-year-old woman with a 20-year history of intranasal cocaine inhalation had acutely decreased vision in the right eye and severe right periorbital pain with fever. Ocular history was notable for amblyopia of the left eye and chronic right dacryocystitis secondary to complete NLDO. She had undergone uneventful right DCR 6 weeks prior to presentation. Her medical history was otherwise unremarkable and she denied intravenous cocaine use. The visual acuity was 20/60 OD and 20/100 OS. Extraocular motility of the right eye disclosed restricted upgaze and downgaze. External examination showed moderate right periorbital swelling, erythema, and a saddle nose deformity. The remainder of the ophthalmologic examination was unremarkable. A large septal perforation was present on anterior rhinoscopy. Computed tomographic scan revealed opacification of the right ethmoid sinus, right maxillary sinus, and both frontal sinuses with destruction of the normal ethmoidal and antral bony architecture (**Figure 3**). Treatment with intravenous antibiotics was started with rapid resolution of symptoms. Blood cultures were negative for organisms. One month later, she denied periorbital pain and visual acuity was 20/30 OD. Motility was unchanged. She later committed suicide.

CASE 3

A 58-year-old woman with a 5-year history of chronic intranasal cocaine abuse had acute onset of severe right periorbital pain and fever. Her ocular history was unremarkable. Her medical history was notable for morbid obesity, non-insulin dependent diabetes mellitus, and chronic obstructive pulmonary disease. She denied intravenous cocaine use. External examination revealed right periorbital edema and erythema and a prominent saddle nose. Hertel exophthalmometry was 23 OD and 19 OS. On compression of the right lacrimal sac, purulent discharge was expressed through the canaliculi. The visual acuity was 20/30 OD and 20/20 OS. Aside from a mild right posterior subcapsular cataract, the remainder of the findings from the ophthalmologic examination were normal. Anterior rhinoscopy disclosed a perforated nasal sep-

Summary of Cases

| Patient No./ Age, y/Sex | Side | Findings | Anterior Rhinoscopy | Computed Tomographic or Magnetic Resonance Imaging Scan Findings | Treatment |
|----------------------------|-------|---|--|--|---|
| 1/39/M | Left | NLDO, orbital cellulitis | Absent nasal septum, middle and inferior turbinate | Extensive destruction of septum, ethmoid, and antral bony structures Absent left medial orbital wall and a soft tissue mass enveloping the medial rectus muscle | Oral steroids and antibiotics, pericranial flap |
| 2/40/F | Right | Status after DCR for NLDO, orbital cellulitis | Large nasal septal perforation | Destruction of the medial orbital wall with mucosal thickening and extensive loss of normal architecture in the nasal cavity and ethmoid sinuses Diffuse opacification of the ethmoidal and maxillary sinuses | Intravenous antibiotics |
| 3/58/F | Right | NLDO, orbital cellulitis | Perforated nasal septum, absent inferior turbinate | Opacification of the right maxillary and ethmoid sinuses with bony destruction of the right medial orbital floor | Intravenous antibiotics |
| 4/42/F | Left | NLDO | Nasal septal defects | Not done | Oral antibiotics, DCR |
| 5/37/F | Right | NLDO | Absent nasal septum and inferior turbinates | Not done | |
| 6/36/F | Left | NLDO | Large nasal septal perforation | Not done | Oral antibiotics, DCR |
| 7/36/M | Left | NLDO | Absent nasal septum, middle and inferior turbinate | Extensive destruction of septum, ethmoid, antral bony structures, left medial orbital wall and floor A left medial soft tissue mass was present | Lost to follow-up |

*NLDO indicates nasolacrimal duct obstruction; DCR, dacryocystorhinostomy.

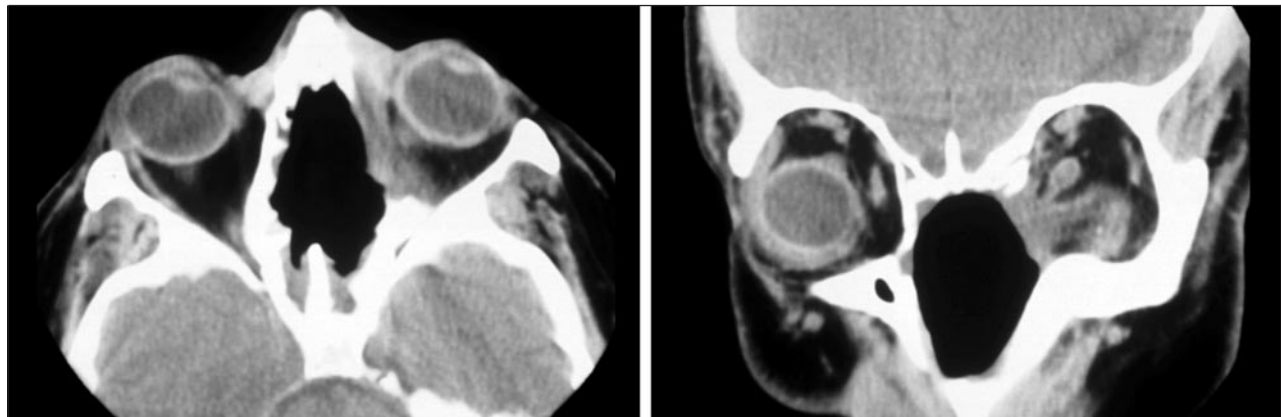


Figure 1. Case 1. Axial (left) and coronal (right) computed tomographic scans show extensive destruction of septum, ethmoid, and antral bony structures. The left medial orbital wall is absent and a soft tissue mass envelops the medial rectus muscle.

tum and absent inferior turbinates. Computed tomographic scan revealed opacified right maxillary and ethmoid sinuses with bony destruction of the right medial orbital floor (**Figure 4**). She was treated with intravenous antibiotics with prompt resolution of symptoms. Hertel measurements after treatment were 21 OD and 19 OS. She declined surgical correction.

CASE 4

A 42-year-old woman with a 9-year history of intranasal cocaine abuse had a 2-year history of left-sided tearing and mucopurulent discharge. She denied ocular or medical problems or intravenous cocaine use. Lacrimal irrigation confirmed complete blockage. The visual acuity was 20/20 OU. Complete ophthalmologic examination

revealed no other abnormalities. Anterior rhinoscopy disclosed several nasal septal defects. She was initially treated with a course of oral antibiotics and subsequently underwent a left DCR procedure. She was last seen 3 months postoperatively with complete resolution of symptoms.

CASE 5

A 37-year-old woman with a 7-year history of intranasal cocaine abuse had a 1-year history of tearing and occasional discharge involving the right eye. Ocular and medical histories were otherwise unremarkable and she denied intravenous cocaine use. Visual acuity was 20/20 OU. Lacrimal irrigation confirmed complete blockage. A saddle nose deformity was present. Ophthalmologic examination was otherwise unremarkable. An absent nasal sep-

tum and inferior turbinates were noted on anterior rhinoscopy. She was initially treated with a course of oral antibiotics and subsequently underwent right DCR with en bloc removal of the NLD system. Histopathologic examination of the lacrimal sac/NLD disclosed a dense infiltrate of chronic inflammatory cells (mostly lymphocytes and plasma cells), periductal fibrosis, and a markedly narrowed duct (**Figure 5**). Special stains including Ziehl-Neelsen for acid-fast bacilli, Gomori methenamine-

silver, and Brown and Hopps disclosed no microorganisms. She was last seen 4 months postoperatively with complete resolution of symptoms.

CASE 6

A 36-year-old woman with a 12-year history of intranasal and inhalational cocaine abuse had left-sided tearing during the previous 6 months. She denied medical problems or intravenous cocaine use. Lacrimal irrigation confirmed complete blockage. Visual acuity was 20/25 OU and findings from a complete ophthalmologic examination were unremarkable. Anterior rhinoscopy revealed a large nasal septal defect. Histopathologic examination of the lacrimal sac disclosed a dense infiltrate of chronic inflammatory cells with fibrosis. She underwent left DCR and was last seen 2 months postoperatively with complete resolution of symptoms.

CASE 7

A 36-year-old man with a 15-year history of intranasal and inhalational cocaine abuse had left retrobulbar pain, diplopia, and left-sided tearing during the last 1 month. He denied medical problems or intravenous cocaine use. Anterior rhinoscopy revealed absent nasal septum and middle and inferior turbinates. Lacrimal irrigation confirmed complete blockage. Magnetic resonance imaging revealed ex-

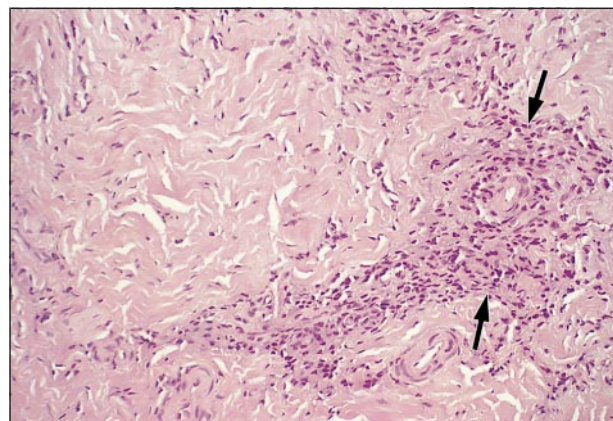


Figure 2. Case 1. Biopsy specimen of the medial orbital tissue mass showing dense fibrosis and chronic inflammation in a perivascular distribution (between arrows). Note the occasional Russell bodies (arrows) (hematoxylin-eosin, $\times 400$).

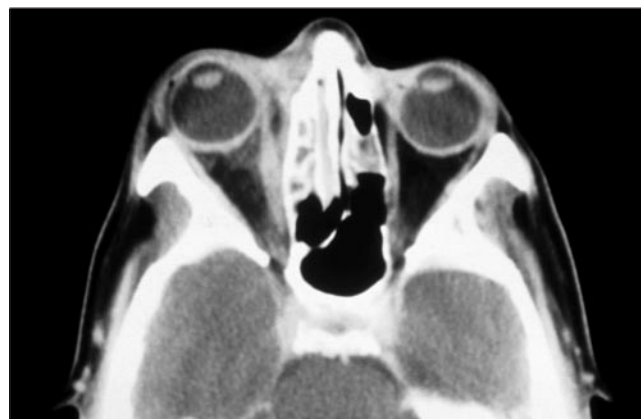


Figure 3. Case 2. Axial (left) and coronal (right) computed tomographic scans show mucosal thickening and extensive loss of normal bony architecture in the nasal cavity and ethmoid sinuses. Diffuse opacification of the right ethmoidal and maxillary sinuses is present.

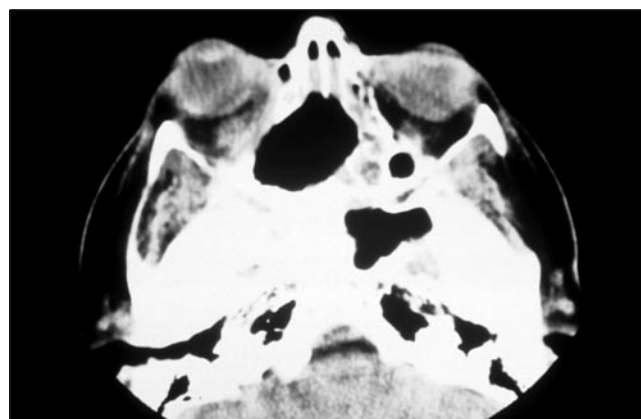


Figure 4. Case 3. Axial (left) and coronal (right) computed tomographic scan discloses opacification of the right maxillary and ethmoid sinuses with bony destruction of the right medial orbital floor.

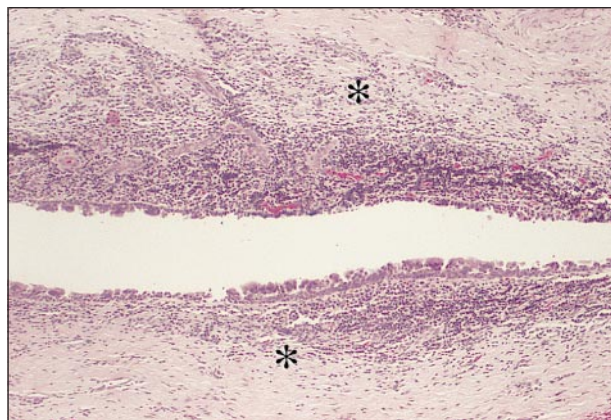


Figure 5. Case 5. Histopathologic examination of the lacrimal sac/nasolacrimal duct demonstrating a dense infiltrate of chronic inflammatory cells (mostly lymphocytes and plasma cells), periductal fibrosis (asterisks), and a markedly narrowed lumen (hematoxylin-eosin, $\times 200$).

tensive loss of normal ethmoidal and antral bony architecture. Destructive changes involving the medial orbital wall and floor were present. A left medial orbital soft tissue mass extending to the retrobulbar soft tissues and enveloping the medial and inferior rectus muscles was present (**Figure 6**). The patient was scheduled for a biopsy of the orbital mass but was lost to follow-up.

COMMENT

The destructive effects of intranasal cocaine in midline nasal structures have been well documented.^{2,5,8-10} The pathogenesis of cocaine-induced destruction probably results from vasoconstriction of the small vessels, chemical irritation by the adulterants in cocaine, and repeated nasal trauma. In this report, we describe the first series, to our knowledge, of patients with acquired NLDO and acute orbital cellulitis associated with chronic intranasal cocaine abuse. They presented with either long-standing epiphora or an acute onset of periorbital pain, edema, and erythema associated with fever. All patients initially denied cocaine abuse; however, they later confessed after the destructive changes within the nasal cavity were revealed to them. All patients also denied intravenous cocaine use or other illicit drug use. Computed tomographic imaging disclosed opacification of the sinuses with extensive destruction of normal bony architecture of the orbit and adjacent structures. The differential diagnosis based on these radiographic findings includes nasal midline granuloma caused by Wegener granulomatosis, polymorphic reticulosis, or non-Hodgkin lymphoma; however, in our cases findings from histopathologic examination excluded these considerations.

A number of ophthalmologic complications related to cocaine abuse have been described and ophthalmologists should be aware of the ocular manifestations of cocaine abuse. These include acute angle-closure glaucoma, motility disturbances, corneal complications, preseptal cellulitis, orbital inflammation, orbital wall destruction, and optic neuritis.^{2,5,6,11-17} Mitchell and Schwartz¹¹ described a patient who developed primary angle-closure glaucoma shortly after intranasal cocaine

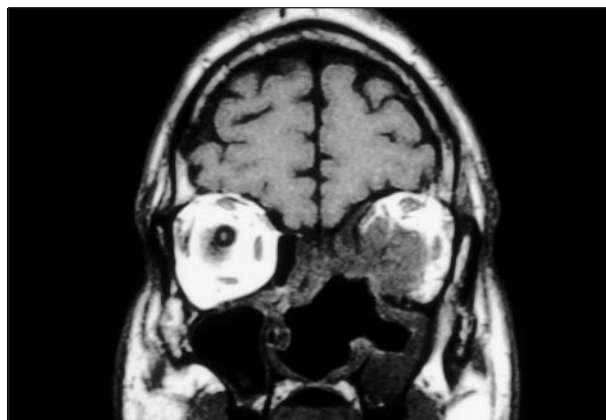


Figure 6. Case 7. Magnetic resonance image reveals extensive loss of normal ethmoidal and antral bony architecture with destruction of the left medial orbital wall and floor. A left medial orbital soft tissue mass extending to the retrobulbar soft tissues and enveloping the medial and inferior rectus muscle is present.

abuse. They postulated that angle-closure was induced by the mydriasis caused by cocaine. Nemeth et al¹² described 2 patients with internuclear ophthalmoplegia and another with trochlear nerve palsy who were using crack cocaine. These abnormalities were thought to be secondary to the hypertension and vasospasm induced by cocaine, leading to vascular occlusion. Sachs et al¹³ used the term "crack eye syndrome" to describe the corneal findings of superficial punctate epithelial keratopathy, corneal epithelial defects, and infectious keratitis associated with smoking crack cocaine. Corneal epithelial defects were first described by McHenry et al¹⁴ in 1989. The exact mechanism is not completely understood, but it was postulated that cocaine disrupts plasma membranes and cytoplasm resulting in alteration of corneal epithelial cell function. The corneal surface becomes anesthetized and can then desquamate with rubbing. The resultant corneal anesthesia may lead to neurotrophic keratopathy and secondary infectious keratitis.^{15,16}

In 1988, Newman et al² described a patient with bilateral optic neuropathy and osteolytic sinusitis associated with intranasal cocaine abuse. The authors postulated that the optic nerve involvement was due to the extensive ischemic necrosis of the mucosal and bony structures in close proximity to the optic nerves. In 1989, Goldberg et al⁵ described 3 additional cases of orbital inflammation and optic neuropathy associated with chronic sinusitis secondary to inhalation cocaine abuse. An inflammatory medial orbital wall mass contiguous with an ethmoid sinusitis and partial loss of the lamina papyracea was present in 2 of the 3 patients. Histopathology of the orbital mass disclosed nonspecific inflammation with variable fibrosis that was similar to the biopsy of our first patient. The authors supported that the orbital involvement is secondary to chronic inflammation in the adjacent sinuses. Recently, Underdahl and Chiou⁶ reported a patient with a 7-year history of nasal cocaine abuse with preseptal cellulitis. Extensive destruction of the medial orbital wall and nasal septum was present on CT imaging. A nasal biopsy specimen disclosed an inflammatory exudate with neutrophils similar to the NLD biopsy specimens of our patients.

Nasolacrimal duct obstruction is the most common abnormality of the lacrimal system, and may be congenital or acquired. Presenting signs commonly include epiphora and acute or chronic dacryocystitis. Acquired obstruction can be primary or secondary. While primary acquired NLDO is idiopathic, secondary acquired lacrimal drainage obstruction may result from various infectious, neoplastic, inflammatory, traumatic, or mechanical causes.¹⁸ Recently, Bartley¹⁸⁻²⁰ described an etiological classification system and reviewed the literature on acquired NLDO; however, there was no mention of cocaine abuse as a cause. The mechanism of acquired NLDO in our patients is secondary to the chronic inflammation and scarring of the delicate respiratory mucosa from the chronic intranasal cocaine use. The large negative pressure generated by "snorting" cocaine permits retrograde delivery of the substance around the nasal mucosa of the NLD ostium. The mechanism of NLDO is due to either destruction or scarring of the NLD. Histopathologic examination of the NLD removed during DCR in 2 of our patients revealed a non-specific pattern of chronic inflammation with dense fibrosis. In case 5, the NLD was removed en bloc for histopathologic examination to provide information on the mechanism of NLDO. Destruction of the bony barrier insulating the orbit from the bacterial flora of the nasal cavity and ischemic necrosis of the orbital tissues lead to inflammation and impaired immune surveillance or ability to respond to infection. We believe the mechanism of orbital cellulitis in patients with chronic intranasal cocaine abuse is related to the combination of severe pansinusitis and destruction of the bony barrier between the sinuses and orbit. The bony wall destruction was radiographically documented in all of our patients with signs of orbital cellulitis.

Treatment of NLDO secondary to intranasal cocaine abuse is the same as in any other cause of NLDO. The acute dacryocystitis should initially be treated with a course of oral antibiotics. When the acute infection resolves, DCR should be considered. In recalcitrant cases of acute orbital cellulitis, intravenous antibiotics may be necessary. All our patients with orbital cellulitis responded promptly to this therapy. A course of oral steroids was given in case 1 to decrease the periorbital inflammatory response. However, this was minimally helpful and the patient finally underwent insulation of the exposed orbit with a pericranial flap. The periosteum and overlying subaponeurotic loose connective tissue enveloping the skull are collectively referred to as the "pericranium." This well-vascularized tissue has proved to be a versatile local flap in craniofacial surgery and in our patient successfully sequestered the orbit from the nasal cavity.⁷ Oral steroid therapy could be considered to reduce the intense orbital inflammation after assuring an absence of bacterial cellulitis and cessation of cocaine abuse. Management of these patients often involves the expertise of several specialists, including oculoplastic surgeons, otolaryngologists, and craniofacial surgeons. Referral to the internist may be necessary to evaluate for other potential systemic complications secondary to the cocaine abuse.

In summary, we report what is to our knowledge the first series of patients with clinical and histopathological evidence showing that chronic intranasal co-

caine abuse is associated with acquired NLDO. We recommend that any patient with epiphora should have a thorough anterior rhinoscopy as part of evaluating the lacrimal drainage system. Although anterior rhinoscopy is not routinely performed by ophthalmologists, this examination can provide valuable clues in establishing inhalation cocaine abuse as a potential cause of epiphora. Chronic intranasal cocaine abuse can result in extensive bony destruction of the orbital walls with associated orbital cellulitis, and should be included in the differential diagnosis of acquired NLDO.

Accepted for publication July 16, 1999.

This study was supported in part by the Fight for Sight Research Division of the National Society to Prevent Blindness, New York, NY (Dr Tse), and the Florida Lions Eye Bank, Miami, Fla (Dr Rosa).

Presented at the Association for Research in Vision and Ophthalmology annual meeting, Fort Lauderdale, Fla, May 9, 1999.

Corresponding author: David T. Tse, MD, Bascom Palmer Eye Institute, 900 NW 17th St, Miami, FL 33136 (e-mail: dtse@bpei.med.miami.edu).

REFERENCES

1. Koller C. Preliminary report on local anesthesia of the eye (September 15, 1884) [in German]. *Arch Ophthalmol*. 1934;12:473-474.
2. Newman NM, DiLoreto DA, Ho JT, Klein JC, Birnbaum NS. Bilateral optic neuropathy and osteolytic sinusitis: complications of cocaine abuse. *JAMA*. 1988; 259:72-74.
3. Johnston LD, Bachman JG, O'Malley PM. *Student Drug Use, Attitudes and Beliefs: National Trends 1975-1982*. Rockville, Md: US Dept of Health and Human Services; 1983. ADM publication 83-1260.
4. Michelson JB, Robin HS, Nozik RA. Nonocular manifestations of parenteral drug abuse. *Surv Ophthalmol*. 1986;30:314-20.
5. Goldberg RA, Weisman JS, McFarland JE, Kraus HR, Hepler RS, Shorr N. Orbital inflammation and optic neuropathies associated with chronic sinusitis of intranasal cocaine abuse. *Arch Ophthalmol*. 1989;107:831-835.
6. Underdahl JP, Chiou AGY. Preseptal cellulitis and orbital wall destruction secondary to nasal cocaine abuse. *Am J Ophthalmol*. 1998;125:266-268.
7. Tse DT, Goodwin WJ, Johnson T, Gilberg S, Meldrum M. Use of galeal or pericranial flaps for reconstruction of orbital and eyelid defects. *Arch Ophthalmol*. 1997;115:932-937.
8. Sercarz JA, Strasnick B, Newman A, Dodd LG. Midline nasal destruction in cocaine abusers. *Otolaryngol Head Neck Surg*. 1991;105:694-701.
9. Deutsch HL, Millard R Jr. A new cocaine abuse complex: involvement of nose, septum, palate, and pharynx. *Arch Otolaryngol Head Neck Surg*. 1989;115:235-237.
10. Schweitzer VG. Osteolytic sinusitis and pneumomediastinum: deceptive otolaryngologic complications of cocaine abuse. *Laryngoscope*. 1986;96:206-210.
11. Mitchell JD, Schwartz AL. Acute angle-closure glaucoma associated with intranasal cocaine abuse. *Am J Ophthalmol*. 1996;122:425-426.
12. Nemeth G, McHenry JG, Zeiter JH, et al. Oculomotor abnormalities secondary to crack cocaine. *Ophthalmology*. 1993;100(suppl):137.
13. Sachs R, Zagelbaum BM, Hersh PS. Corneal complications associated with the use of crack cocaine. *Ophthalmology*. 1993;100:187-191.
14. McHenry JG, Zeiter JH, Madion MP, Cowden JW. Corneal epithelial defects after smoking crack cocaine. *Am J Ophthalmol*. 1989;108:732.
15. Strominger MB, Sachs R, Hersh PS. Microbial keratitis with crack cocaine. *Arch Ophthalmol*. 1990;108:1672.
16. Zagelbaum BM, Tannenbaum MH, Hersh PS. *Candida albicans* corneal ulcer associated with crack cocaine. *Am J Ophthalmol*. 1991;111:248-249.
17. Ravin JG, Ravin LC. Blindness due to illicit use of topical cocaine. *Ann Ophthalmol*. 1979;11:863-864.
18. Bartley GB. Acquired lacrimal drainage obstruction: an etiologic classification system, case reports, and a review of the literature. *Ophthalmic Plast Reconstr Surg*. 1992;8(pt 1):237-242.
19. Bartley GB. Acquired lacrimal drainage obstruction: an etiologic classification system, case reports, and a review of the literature. *Ophthalmic Plast Reconstr Surg*. 1992;8(pt 2):243-249.
20. Bartley GB. Acquired lacrimal drainage obstruction: an etiologic classification system, case reports, and a review of the literature. *Ophthalmic Plast Reconstr Surg*. 1993;9(pt 3):11-26.