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

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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.


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 Koller 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.

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. When used illicitly, cocaine is usually taken intranasally, intravenously, or by inhalation. Depending on the route of administration, several medical complications can occur. 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. 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. 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.

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-
senting signs and symptoms included epiphora or 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. 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 periorbital 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. Biopsy 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. The flap was secured into position within the orbit by wedging it between the posterior floor and peri-orbita. 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 abuse 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-
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-
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. 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. They postulated that angle-closure was induced by the mydriasis caused by cocaine. Nemeth et al disclosed 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 al 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 al 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.

In 1988, Newman et al 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 al 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 Chiou 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. Recently, Bartley 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 peri orbital inflammatory response. However, this was minimally helpful and the patient finally underwent insulation of the exposed orbit with a pericranial flap. The peristeum and overlying subperiorbital 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. 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, otorhinolaryngologists, 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 cocaine 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.

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