REPAIR OF COCAINE-INDUCED NASOPHARYNGEAL AND SINONASAL STENOSIS

Austin DeHart MD, Nicholas Deebel BS, Jonathan Young MD, Thomas Lee MD, Rajanya Petersson MD

1. Department of Otolaryngology – Head and Neck Surgery, Virginia Commonwealth University, Richmond VA
2. Virginia Commonwealth University School of Medicine, Richmond VA
3. Department of Otolaryngology – Head and Neck Surgery, Children’s Hospital of Richmond at Virginia Commonwealth University, Richmond VA

Introduction

Cocaine abuse is estimated to affect 4.2 million US citizens a year and is frequently used intranasally (Resulis 2013). This causes sympathetically-mediated vasodilatation, which can lead to ischemia, inflammation, and ulceration in the nasal, palatal, and pharyngeal tissue (Silvestri 2010). Occasionally this can progress to full thickness formation and has been reported in severe cases to cause extensive destruction, even to the clivos (Mollen 2016). A common cocaine addiction, leukosia, has been associated with especially severe tissue destruction through the induction of rapid-onset vasculitis (Larocque 2012). Leukosia is initially used in humans as an anesthetic and immunomodulatory agent, but it was associated with angioanaphylactose and cutaneous necrotizing vasculitis, especially affecting the earlobes. It was removed from the market in 2000, but is still used in veterinary medicine. Alternative causes of midline destructive lesions include malignancy, trauma, fungal infection, Wegener’s granulomatosis, and ulcerating midline lymphoma. Treatment for cocaine-induced abnormalities can be difficult. Cessation of cocaine use is imperative. Due to the drug’s effects on blood vessels, a vascularized flap is often considered due to poor local tissue perfusion (Collett 2014). While necrosis and tissue destruction have been previously described, to our knowledge this is the first reported case of complete sinonasal and nasopharyngeal stenosis associated with cocaine use.

Methods

An otherwise healthy 34-year-old woman presented for evaluation after a large piece of tissue fell out of her nose. She reported chronic, gradually worsening, bilateral nasal congestion, drainage, and the inability to blow her nose. On exam, she had a saddle nose deformity, a 2.5 cm caudal septal perforation, and complete bilateral vestibular stenosis with scarring between the lateral nasal wall and the anterior septal remnant with 100% nasal cavity obstruction. There was no nasal patency. The soft palate was retracted and adherent to the posterior oropharyngeal wall. Her history was significant for prior cocaine abuse, which she reported to have ceased using 4 years prior.

She underwent two stage operative repair. Her first procedure addressed her nasal cavity and the vestibular stenosis. Using image guidance, a combination of sinus surgery instruments and microdebrider were used to resect the bilateral nasal cavities. There was extensive scarring along the anterior 2/3 of the nasal cavities. Once the bilateral nasal cavities were re-established to the nasopharynx, bilateral Doy-cell nasal flaps were placed for 4 weeks postoperatively to prevent restenosis. The inferior turbinates bilaterally and the septal mucosa were preserved to be used for a staged septal perforation repair.

In the nasopharynx, the patient was noted to have a near total nasopharyngeal stenosis from scarring of the soft palate to the posterior pharyngeal wall. A staged septal perforation repair using bilateral rotational lateral nasal wall flaps was planned for a later time. Biopsies from the nasal cavity and posterior pharyngeal wall showed reactive respiratory epithelium and squamous mucosa with chronic inflammation.

During a second stage procedure, she underwent revision of her sinonasal stenosis and repair the nasopharyngeal stenosis. Membranous soft tissue scarring had recurred in the nasal cavity and was removed with microdebrider. A bilateral pan-sinus FESS was performed. To address the nasopharyngeal stenosis, a left laterally-based pharyngeal flap was planned, and a combined transoral and transnasal endoscopic approach was used (Figure 1). The soft palate was nearly completely scarring to the posterior pharyngeal wall. The thinnest area of stenosis was identified with transillumination near the uvula, this was bluntly entered transorally with a ball-tipped monopolar cautery (Figure 2). Endoscopic confirmation of the probe’s location was obtained (Figure 3). The opening was dilated with a right angled foroscope (Figure 4), and a 7200 Beaver blade was used to extend the opening laterally to the right (Figure 5). A red rubber catheter was placed to retract (Figure 6). Subsequently, the soft palate was sharply divided from the posterior pharyngeal wall along the posterior tonsillar pillar, from the uvula superiorly to the level of the mid-opharynx inferiorly, using a 7200 Beaver blade (Figure 7, 8). Excess, fibrotic scar tissue was removed using scissors and a microdebrider (Figure 9). The nasopharynx was re-explored endoscopically to evaluate the opening created (Figure 10). A 9/0 Prolene was used to line the nasopharynx with the posterior nasopharyngeal flap was fashioned from the tissue that was released, and a right laterally-based pharyngeal flap was design (Fig 11). The pharyngeal flap was elevated and rotated superiorly to line the mucosal defect along the posterior oropharyngeal wall to prevent circumferential scarring and re-stenosis (Figure 12, 13). A 28 French nasal trumpet and contralateral Doy-cell splint were inserted and left in place for 2 weeks.

At last follow-up, six months after surgery, her nasal obstructive symptoms were significantly improved, and she had no evidence of velopharyngeal insufficiency. Her nasal cavity was patient with no restenosis (Figure 14). Her velopharyngeal opening was approximately 1 cm and her pharyngeal flap was well healed (Figure 15, 16).

Discussion

Operative repair in this patient was challenging for several reasons. In patients with a history of drug abuse, relapse is often a concern, which would likely worsen her pathology and impair healing. Tissues often had poor vascular supply, impeding proper wound healing. Patient counseling to manage expectations and to confirm the cessation of drug use are critical. A staged approach focusing on the most significant subsite of anatomic pathology was chosen. The nasal cavity was addressed first since it was the most severe and improving this allowed endoscopic access to the nasopharyngeal stenosis. Image guidance was used as no appreciable landmarks were easily visualized. Care was taken to preserve inferior turbinates mucosa as donor tissue in case the patient wishes to eventually pursue a delayed septal perforation and saddle nose deformity repair.

Restenosis is also a prominent concern. To combat this, prolonged stenting was used after each stage of her procedures. Simply releasing the palate from the posterior oropharyngeal wall would have left a defect in the mucosa that would be prone to circumferential scarring. To prevent this, a laterally-based pharyngeal flap was chosen to introduce healthier vascularized tissue into the posterior nasopharyngeal defect.

Velopharyngeal insufficiency was also of concern, given the scared palate with probability of very limited motion. As such, it was decided to open one side of the nasopharynx first, and evaluate symptoms to ensure relief of obstructive symptoms without presence of velopharyngeal insufficiency.

Conclusions

Intra-nasal cocaine abuse can cause extensive soft tissue damage. We present an unusual case of cocaine induced sinonasal and nasopharyngeal stenosis and its operative management with a successful staged repair.

References


Contact

Austin N. DeHart, MD
Department of Otolaryngology – Head & Neck Surgery
Virginia Commonwealth University
Email: austin.dehart@vcuhealth.org

Case Report

A 55-year-old woman presented with progressive speech and swallowing difficulties since a head and neck radiation treatment for nasopharyngeal lymphoma 10 years prior. She had undergone radiation therapy to the nasopharynx and paranasal sinuses for nasopharyngeal lymphoma. At last follow-up, six months after surgery, her nasal obstructive symptoms were significantly improved, and she had no evidence of velopharyngeal insufficiency. Her nasal cavity was patient with no restenosis (Figure 14). Her velopharyngeal opening was approximately 1 cm and her pharyngeal flap was well healed (Figure 15, 16).