Intranasal drug use has long been recognized as a significant cause of sinonasal pathology. Historically, cocaine has been the most commonly cited offender and is associated with mucosal ulceration and necrosis, septal perforation, palatal defects, and saddle-nose deformity. Secondary to the increasing prevalence of opioid abuse, intranasal use of crushed medications remains an important consideration when evaluating patients presenting with intranasal debris and evidence of mucosal necrosis and/or bone destruction. Patients typically present with symptoms refractory to standard medical therapy (eg, intranasal steroid sprays, oral antibiotics).1,2 History may be difficult to elucidate whether patients are reluctant to divulge medication misuse and a high index of suspicion is key to diagnosis. Immune status is an important consideration in the development of a treatment plan as secondary infectious agents tend to play a more indolent role in immunocompetent individuals and a more invasive part in immunocompromised hosts.3 The clinical presentation and recommended treatment of isolated intranasal acetaminophen use, without an accompanying opioid, is presented.

**Patient #1**

A 38-year-old immunocompetent female presented with a 2-month history of severe right-sided facial pain, nasal congestion, and throat pain for which she admitted to frequent intranasal use of crushed acetaminophen tablets. Endoscopic examination revealed erythematous mucosa with scattered crusts and white debris. Computed tomography demonstrated nonspecific mucosal thickening within the right ostiomeatal complex and anterior ethmoid air cells. Operative debridement was performed and initial cultures revealed *Aspergillus* species. She was started on oral fluconazole and later switched to itraconazole secondary to gastrointestinal upset. She required 2 additional operative debridements, with cultures resulting in *Candida glabrata* and Methicillin-sensitive *Staphylococcus aureus*. Subsequent antimicrobials consisted of voriconazole, doxycycline, and amoxicillin, secondary to patient tolerance and culture sensitivities, as directed by our Infectious Disease consultants. Total antimicrobial duration lasted approximately 8 months. At treatment conclusion, sinonasal symptoms had resolved.

**Patient #2**

A 54-year-old immunocompetent male who initially developed right-sided nasal pain after removing an insect from his nose presented with a 3-month history of worsening pain radiating to his midface. He endorsed daily intranasal use of crushed acetaminophen tablets to ease the pain. Computed tomography demonstrated bony dehiscence along the right hard palate with abutting mucosal thickening (Figure 1). Endoscopic examination revealed pale mucosa with white debris throughout the...
right nasal cavity (Figure 2). Cultures were obtained and revealed *Aspergillus* species. Daily oral fluconazole was initiated and it was recommended the patient to discontinue intranasal acetaminophen use. His symptoms failed to improve, thus operative debridement and biopsy was performed. Intraoperative cultures were notable for concurrent *Streptococcus pneumoniae* and Methicillin-sensitive *Staphylococcus aureus*. He was advised to follow-up with Otolaryngology as well as Infectious Disease but failed to do either. His clinical progression is thus unknown.

Although chronic fungal rhinosinusitis is uncommon in immunocompetent patients, such cases have been reported. This report details 2 cases of intranasal acetaminophen use with tissue necrosis and intranasal colonization with the saprophytic opportunistic fungus *Aspergillus*. When evaluating patients with rhinosinusitis recalcitrant to standard medical therapy or in patients presenting with mucosal necrosis and/or bony destruction, intranasal medication misuse should be considered, particularly if findings are unilateral, pain is out of proportion with examination findings, or there is evidence of visible pill debris. Computed tomography and magnetic resonance imaging findings are often nonspecific.\(^1\) Biopsy of ulcerated nasal mucosa, areas of persistent crusting, and nasal septal or palatal perforations is are often warranted. Tissue culture, particularly fungal culture, is often revealing. Interestingly, preoperative pain that is often out of proportion to examination findings seems to be improved following operative debridement. Acetaminophen has no known vasoconstrictive properties, unlike its historic predecessor cocaine, which also leads to mucosal necrosis and local tissue destruction. Perhaps acetaminophen or its additives contribute to a mechanical and/or chemical irritation that result in tissue necrosis and the development of opportunistic infections.\(^4\) Therapeutic goals consist of drug abstinence, evacuation of intranasal debris, and treatment of secondary infections with oral antimicrobials. Recognizing that secondary infections are primarily fungal is paramount. Challenges include pain management and treatment compliance, particularly in this patient population, and collaboration with infectious disease and addiction specialists may prove helpful.

**Declaration of Conflicting Interests**

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

**Funding**

The author(s) received no financial support for the research, authorship, and/or publication of this article.

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