Intranasal Hydrocodone-Acetaminophen Abuse Induced Necrosis of the Nasal Cavity and Pharynx

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Introduction

Abuse of prescription pain medication continues to be a large problem in today’s society. Intranasal abuse of drugs such as cocaine and heroin is well described. In our central Kentucky practices, we have seen the emergence of intranasal abuse of orally formulated hydrocodone/acetaminophen (HA) combinations and its toxic sequelae on the upper aerodigestive tract’s soft and hard tissues.

A small number of case reports have begun to detail the effects of intranasal hydrocodone-acetaminophen abuse (INHAA).\(^4\) These studies point to a more fulminant destructive disease process than that described in other types of nasal drug abuse.\(^5,6\) \(^1\) This behavior is reported with an acute and rapid destruction of the septum and palate, as well as cases that led to invasive fungal rhinosinusitis.\(^7\)

We present the first large series of patients presenting with upper aerodigestive tract pathology secondary to INHAA. From analysis of this group of patients, we hope to better characterize the presentation and manifestations of this condition.

Methods

Thirty-five patients were identified across three Otolaryngology practices in central Kentucky from 2004 to 2011. Patients documented to have nasal manifestations of INHAA were retrospectively identified through a variety of mechanisms including physician memory, searches of inpatient logs, and searches of diagnostic billing codes.

We then abstracted general demographic information, presenting symptoms, substance abused, duration of the abuse, prior substance abuse history and past medical history. Pathology reports, microbiology reports, operative reports, and archived endoscopic images were also abstracted from the inpatient and outpatient medical records. Patients were excluded if it was determined a different substance was abused, or if definitive evidence of abuse could not be established.

This study was approved by the University of Kentucky’s IRB (protocol #11-0345-p2H).

<table>
<thead>
<tr>
<th>Signs of INHAA</th>
<th>N (%)</th>
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<tbody>
<tr>
<td>Mucosal necrosis and crusting</td>
<td>27 (77%)</td>
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<tr>
<td>Septal perforation</td>
<td>18 (51%)</td>
</tr>
<tr>
<td>Oropharyngeal ulceration</td>
<td>11 (31%)</td>
</tr>
<tr>
<td>Palatal perforation</td>
<td>9 (26%)</td>
</tr>
<tr>
<td>Fevers/chills</td>
<td>3 (9%)</td>
</tr>
<tr>
<td>Facial swelling</td>
<td>3 (9%)</td>
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</tbody>
</table>

Discussion

- Our data reaffirms the destructive nature of INHAA. The majority of patients presented with necrosis of the oronasal mucosa, consistent with prior case reports.\(^6,7\)
- Septum and palate perforation rates are five times higher than those reported for intranasal cocaine abuse.\(^8,9\)
- Oronasal pain with crusty, white exudate overlying necrotic mucosa in the oronasal cavities is highly characteristic of INHAA.
- The pathological mechanism of tissue destruction induced by intranasal H-A is not well defined.
- Abstaining from INHAA in combination with local debridement reverses progression of the disease. Introducing alternative nasal analgesics, such as topical lidocaine is helpful.
- A majority of patients were abusing their own prescription pain medications at the time of presentation.
- Concurrent invasive fungal rhinosinusitis must be considered especially in the immune compromised patient with INHAA mucosal disease.

References

5. Greene D. Total necrosis of the intranasal structures and soft palate as a result of nasal inhalation of crushed OxyContin. Ear Nose Throat J 2006; 85(3) – 139
12. Greene D. Total necrosis of the intranasal structures and soft palate as a result of nasal inhalation of crushed OxyContin. Ear Nose Throat J 2006; 85(3) – 139