Oxycodone-Acetaminophen Abuse by Inhalation

Isca Hershkowitz MD[1], Avivit Cahn MD[2], Tal Keidar Haran MD[3], and Alon Y. Hershko MD PhD[1]

Departments of [1]Medicine C, [2]Endocrinology and Metabolism and [3]Pathology, Hadassah Medical Center, Faculty of Medicine, Hebrew University of Jerusalem, Jerusalem, Israel

KEY WORDS: acetaminophen, oxycodone, opioids, substance abuse

IMAJ 2024; 26: 54–55

Illicit drug abuse is a widespread medical problem with numerous sequelae. One of the major challenges in reaching a diagnosis is the difficulty in obtaining accurate details during the medical interview. We describe a patient who initially denied drug abuse while presenting a bizarre nasopharyngeal disease secondary to inhalation of oxycodone-acetaminophen powder.

PATIENT DESCRIPTION

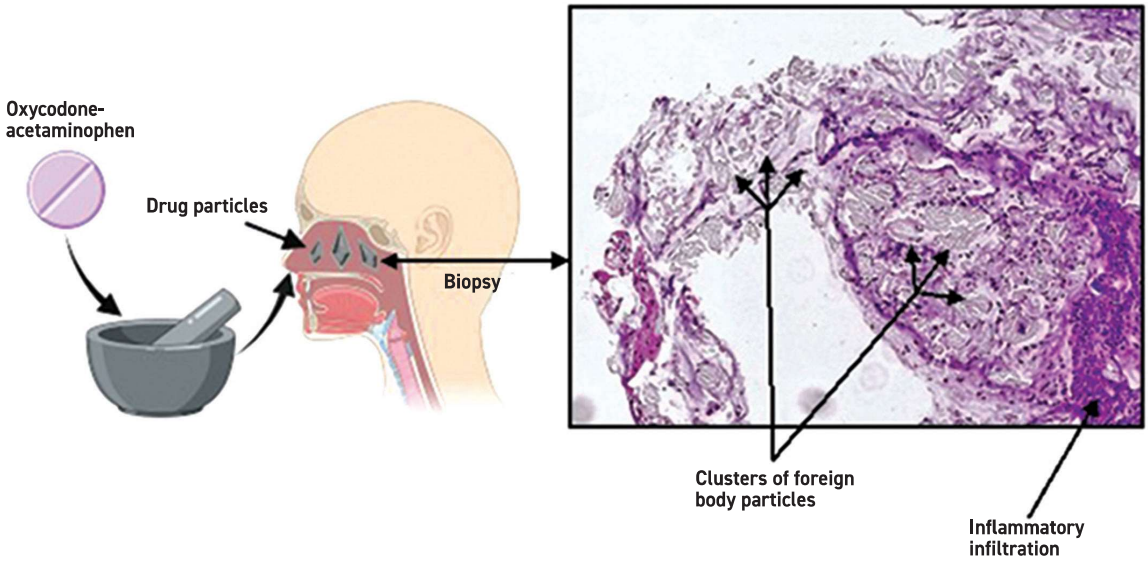
A 26-year-old male was admitted to the department of internal medicine with weakness, arthralgia, and a 10 kg weight loss over the past year. He presented with dysphonia and dysphagia as well as a persistent sore throat. He reported cigarette smoking but denied excessive alcohol drinking or illicit drug abuse.

During his stay in the internal medicine ward, his peripheral white blood cell count reached a peak value of 28.3 × 10^9/L (normal range 4.5–11) with 86.1% neutrophils. Platelets increased to a maximum of 811 × 10^9/L (150–450). An investigation for active infections was negative in the following tests: blood and throat cultures, serologic tests for cytomegalovirus, Epstein-Barr virus, human immunodeficiency virus, venereal disease research laboratory test (VDRL), and Q fever. Immunoglobulins subclasses were within normal limits.

Due to persistent symptoms the patient underwent an endoscopic evaluation of the nose and larynx. A white plaque was observed along the nasopharynx and the trachea. A tis-

Figure 1. A tissue biopsy from the right nostril stained with hematoxylin and eosin.

The patient inhaled powder prepared from tablets of oxycodone-acetaminophen. The stained specimen shows a fragment of tissue with active inflammation and clusters of embedded foreign material representing inhaled drug particles (created with BioRender.com).



sue biopsy from the lesion revealed fragments of squamous epithelium with hyperkeratosis and additional ulcerated fragments with active inflammation and embedded foreign body particles [Figure 1].

Introduction of the histological findings to the patient prompted him to admit oxycodone-acetaminophen abuse by inhalation of crushed tablets. It was therefore concluded that intranasal exposure to this substance accounts for chronic inflammation, leukocytosis, and thrombocytosis. The patient was eventually referred to a rehabilitation program.

COMMENT

Intranasal abuse of prescription opioids is a well-recognized problem. Inhalation is considered to stimulate enhanced euphoric effects [1]. Complications of intranasal abuse of opioids alone or in combination with acetaminophen are attributed to an inflammatory response to the active drug or its excipients [2].

In combined opioid-acetaminophen formulations, talc is used as a binder. This additive is known for its ability to induce local inflammation. Previously reported symptoms caused by the drug powder include nasal pain and congestion, sinus pressure or pain, dysphagia, otalgia, and dysphonia. Endoscopic findings can vary from white plaque and edema to perforation of the nasal septum. Histological specimens can demonstrate necrosis, inflammation, and the presence of foreign polarized material [2-4].

Another potential adverse effect is pulmonary talcosis characterized by a foreign body giant cell reaction in the lungs surrounding polarizable birefringent crystals [5].

CONCLUSIONS

Intranasal abuse of medications should be included in the differential diagnoses of patients with recalcitrant upper respiratory and oropharyngeal symptoms. We highlight the significance of repeated medical interviews in the setting of an acute disease that is suspected to be due to substance abuse.

Correspondence

Dr. A.Y. Hershko
Dept. of Medicine C, Hadassah Medical Organization and Faculty of Medicine, Hebrew University of Jerusalem, Jerusalem 91120, Israel
Email: alonh@hadassah.org.il

References

1. Hernandez SH, Nelson LS. Prescription drug abuse: insight into the epidemic. *Clin Pharmacol Ther* 2010; 88: 307-17.
2. Vosler PS, Contreras JI, Wang EW, Schaitkin BM, Lee S. Clinical and pathologic characteristics of intranasal abuse of combined opioid-acetaminophen medications. *Int Forum Allergy Rhinol* 2014; 4: 839-44.
3. Yewell J, Haydon R, Archer S, Manaligod JM. Complications of intranasal prescription narcotic abuse. *Ann Otol Rhinol Laryngol* 2002; 111: 174-7.
4. Lin RJ, Smith LJ. Laryngeal manifestation of intranasal acetaminophen abuse and review of literature. *Ear Nose Throat J* 2019; 98 (4): 192-4.
5. Goncalves TA, Barreto MM, Marchiori E. Pulmonary talcosis related to cocaine inhalation. *J Bras Pneumol* 2021; 47: e20210146.

Capsule

Autoimmune amelogenesis imperfecta in patients with APS-1 and celiac disease

Ameloblasts are specialized epithelial cells in the jaw that have an indispensable role in tooth enamel formation (amelogenesis). Amelogenesis depends on multiple ameloblast-derived proteins that function as a scaffold for hydroxyapatite crystals. The loss of function of ameloblast-derived proteins results in a group of rare congenital disorders called amelogenesis imperfecta. Defects in enamel formation are also found in patients with autoimmune polyglandular syndrome type-1 (APS-1), caused by AIRE deficiency, and in patients diagnosed with celiac disease. However, the underlying mechanisms remain unclear. **Gruper** et al. showed that the vast majority of patients with APS-1 and celiac disease develop autoantibodies (mostly of the IgA isotype) against ameloblast-specific proteins, the expression of which is induced by AIRE in the thymus. This situation results in a breakdown of central tolerance and subsequent generation of corresponding autoantibodies that interfere with enamel formation. However, in celiac disease, the generation of such autoantibodies seems to be driven by a breakdown of peripheral tolerance to intestinal antigens that are also expressed in enamel tissue. Both conditions are examples of a previously unidentified type of IgA-dependent autoimmune disorder that we collectively name autoimmune amelogenesis imperfecta.

Nature 2023; 624: 653
Eitan Israeli