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Otolaryngology manifestations of cocaine abuse.

Manifestaciones otorrinolaringológicas del abuso de cocaína

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Abstract

BACKGROUND: Cocaine abuse is a common practice with acute and chronic consequences in the upper airway, clinical suspicion by professionals is essential to identify cases.

OBJECTIVE: To describe the otorhinolaryngological manifestations related to cocaine abuse.

MATERIALS AND METHODS: A retrospective, descriptive, observational study was done with the medical records of patients who consulted for head and neck problems related to cocaine abuse from January 2016 to March 2019. Bibliographic research was conducted in MEDLINE, between 1990 and 2018, using the word "cocaine" and the following search terms: head and neck, nasal, oral cavity, pharynx, nasopharynx, oropharynx, hypopharynx, larynx, trachea, paranasal sinus, sinusitis, otitis, ear, hearing loss and vertigo.

RESULTS: During the study period, six patients with otorhinolaryngological manifestations related to cocaine abuse were treated. Four patients presented midline necrosis, one pyomyositis after intramuscular injection of cocaine in the neck and another subcutaneous emphysema after cocaine inhalation. The less frequent manifestations were mouth ulcers, pemphigus-like lesions, laryngeal edema or rhinitis.

CONCLUSIONS: The destructive lesions of the midline are the most common presentation of cocaine abuse, others can be observed, where a high index of suspicion of consumption can grant the etiological diagnosis.

KEYWORDS: Cocaine abuse; Mouth, Nose.

Resumen

ANTECEDENTES: El abuso de cocaína es una práctica común con consecuencias agudas y crónicas en la vía aérea superior, la sospecha clínica por parte de los profesionales es fundamental para identificar los casos.

OBJETIVO: Describir las manifestaciones otorrinolaringológicas relacionadas con el abuso de cocaína.

MATERIALES Y MÉTODOS: Estudio retrospectivo, descriptivo, observacional, en el que de enero de 2017 a marzo de 2019 se revisaron los expedientes de todos los pacientes que consultaron con problemas otorrinolaringológicos relacionados con el abuso de cocaína. Se realizó investigación bibliográfica en MEDLINE, entre 1990 y 2018, utilizando la palabra "cocaína" y los siguientes términos de búsqueda: cabeza y cuello, nasal, cavidad oral, faringe, nasofaringe, orofaringe, hipofaringe, laringe, tráquea, seno paranasal, sinusitis, otitis, oído, hipoacusia y vértigo.

RESULTADOS: Durante el periodo de estudio se atendieron seis pacientes con manifestaciones otorrinolaringológicas relacionadas con el abuso de cocaína. Cuatro pacientes tuvieron necrosis de la línea media, uno piomiositis después de la inyección intramuscular de cocaína en el cuello y otro enfisema subcutáneo después de la inhalación de cocaína. Las manifestaciones menos frecuentes fueron las úlceras bucales, lesiones tipo pénfigo, edema laríngeo o rinitis.

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CONCLUSIONES: Las lesiones destructivas de la línea media son la manifestación más común del abuso de cocaína, aunque pueden observarse otras, un alto índice de sospecha de consumo puede otorgar el diagnóstico etiológico.

PALABRAS CLAVE: Abuso de cocaína; boca; nariz.

INTRODUCTION

Cocaine is a powerful central nervous system stimulant mostly used as a recreational drug, and is most snorted, inhaled as smoke (crack cocaine) or injected intravenously. The drug is naturally found in low doses in the leaves of the coca plant and its extraction requires several chemical processes. Cocaine inhibits catecholamine reuptake, stimulates the central sympathetic nervous system, and increases the sensitivity of adrenergic nerve endings to norepinephrine, thus making the person feel euphoric, energetic and mentally alert.

After cannabis, cocaine is the most frequently consumed illegal drug in Europe, estimated to be used by 1% of the population (3,5 million people) in 2017. The European Monitoring Center for Drugs and Drug Addiction estimates that 5,2% (17,5 million people) has used cocaine at least once in their lifetime.

The increasing intranasal abuse of cocaine has been associated with numerous medical problems. Among the most common complications are cocaine-induced midline destructive lesions, being septal perforation its most limited presentation.[1] But other disorders have been described related to cocaine abuse such sudden deafness,[2] oral pemphigus-like lesions[3] or pharyngo-laryngeal injuries.[4]

Levamisole is an immunomodulatory drug that was used to treat colon cancer before being withdrawn from the market in 2000 because of adverse effects such as neutropenia, agranulocytosis, purpura, and skin necrosis.[1] It is still being used in veterinary sciences as antihelminthic medication and as a cutting or bulking agent in cocaine due to its physical similarities.[1] Levamisole toxicity may result in purpura or necrosis of the ear helices, vasculitis, and neutropenia among other symptoms.[5]

We have not found a previous report about all the possible complications of cocaine abuse in the otolaryngology practice. Our goal was to describe all the head and neck manifestations of cocaine abuse with case presentations and a literature review.

This study aims to describe the otolaryngology manifestations related to cocaine abuse and determine the potential injuries of this toxic substance in the upper airway.

MATERIALS AND METHODS

A retrospective, descriptive, observational study was done with the charts of all the patients who consulted with head and neck problems related to cocaine abuse seen from January 2017 to March 2019. An informed consent was obtained from all the patients.

A literature review was performed using MEDLINE of all published cases of head and neck manifestations related to cocaine abuse using the

word "cocaine" and one of the following search terms: head and neck, nasal, oral cavity, pharynx, nasopharynx, oropharynx, hypopharynx, larynx, tracheal, paranasal sinus, sinusitis, otitis, ear, hearing loss and vertigo.

Results were limited to human subjects and articles written in English, French and Spanish between 1990 and 2018.

RESULTS

Case presentations

Six patients with otolaryngology manifestations related to cocaine abuse were seen during the study period (**Table 1**). Four patients presented with a cocaine-induced midline necrosis (**Figure 1**), one patient developed a pyomyositis after cocaine intramuscular injection in the neck, and another one suffered from subcutaneous emphysema after cocaine inhalation (**Figure 2**).

Literature review

Our review showed that cocaine-induced midline necrosis is the most reported presentation, followed by pinna or other facial necrosis. In **Table 2**, all the otolaryngologic manifestations related to cocaine abuse published in the medical literature and its frequency are summarised.

Cocaine consumption can have serious health problems. Although nasal septum perforation is familiar to most otolaryngologists, there are many other associated head and neck manifestations.

Cocaine-induced midline destructive lesions

Chronic inhalation of cocaine can cause necrosis and destruction of nasal and paranasal structures. These are called cocaine-induced midline destructive lesions. Cocaine progressively damages the mucosa and perichondrium of the nasal septum. This leads to ischemic necrosis of the septal cartilage and may result in perforation. Occasionally, destructive lesions may also be seen in the sinuses, the palate, and the pharyngeal walls.[1]

The mechanism by which cocaine induces necrosis is multifactorial and includes direct vasoconstrictive effects, local trauma from high-velocity inhalation of cocaine crystals, toxic effects of adulterants mixed with cocaine (levamisole, lidocaine), and damage from secondary infections.[1]

Patients with cocaine-induced midline destructive lesions suffer from chronic nasal obstruction, frequent nose bleeding, and/or severe facial pain. Physical examination reveals diffuse necrotizing ulcerative lesions, extensive crusting and septal perforation. In severe cases, necrosis may extend to the turbinates, the lateral wall of the nasal cavity, or the soft/hard palate, as it happened in 4 of our cases (**Figure 1**). These patients may present with nasal regurgitation, dysphagia, rhinolalia or nasal reflux. Inflammatory symptoms like fever, malaise, weight loss, arthralgia as well as inflammatory laboratory parameters are typically absent.[6]

These symptoms are also characteristic of granulomatosis with polyangiitis (Wegener granulomatosis). In one of our patients who didn't admit her drug abuse, it was difficult to make the differentiation between cocaine-induced midline destructive lesions and granulomatosis with polyangiitis. A correct diagnosis ultimately depends on the clinical history, the documentation of cocaine abuse, and exclusion of other possible etiologies such as inflammatory vascular diseases, tumours, or infections.[6]

In patients with cocaine-induced midline destructive lesions, MRI or CT shows abnormalities concentrated in the septum and turbinates, probably due to deposition of cocaine. Trimar-

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Table 1. Clinical cases

Age/gender	Main complaint	Physical exam	CT/MRI	Diagnosis	Administration route	Treatment	Sequels	Frequency/ duration
35/man	Neck pain and swelling	Neck swelling	SCM abscess	SCM pyomiositis	Intramuscular	Antibiotics	None	Weekly/5 years
58/woman	Nasal obstruction	Saddle nose deformity, nasal crusting and rhinorrhea, septum perforation	Extensive erosive process involving the palate, nasal cavity, and maxillary sinus	Cocaine-induced midline necrosis	Nasal route	Keep using nasal cocaine. Surgical endoscopic debridement	Lost for follow-up	Weekly/15 years
61/woman	Nasal crusting and facial disfiguration	Saddle nose deformity, nasal crusting and rhinorrhea, septum perforation	Crusting, mucosal necrosis, absent inferior turbinates, and maxillary sinus cavities	Cocaine-induced midline necrosis	Nasal route	Resume cocaine use. Surgical endoscopic debridement. Local flap reconstruction	Saddle nose deformity	Monthly/20 years
45/man	Nasal obstruction	Absence of most intranasal structures with palatal and nasal septal perforations	Total septum necrosis, absent of middle and inferior turbinates	Cocaine-induced midline necrosis		Keep using nasal cocaine. Local debridement	Bilateral midface flattening and a persistent saddle nose deformity	Weekly/> 20 years
47/man	Nasal crusting and obstruction	Nasal crusting and septum perforation	Septum and inferior turbinate necrosis	Cocaine-induced midline necrosis	Nasal route	Resume cocaine abuse	Severe nasal crusting	Weekly/> 20 years
42/man	Neck pain and swelling	Neck swelling	Extent neck emphysema	Neck and thoracic. Subcutaneous enphysema	Nasal route	Airway control. Prophylactic antibiotics	None	Sporadic/< years

SCM: sternocleidomastoid.

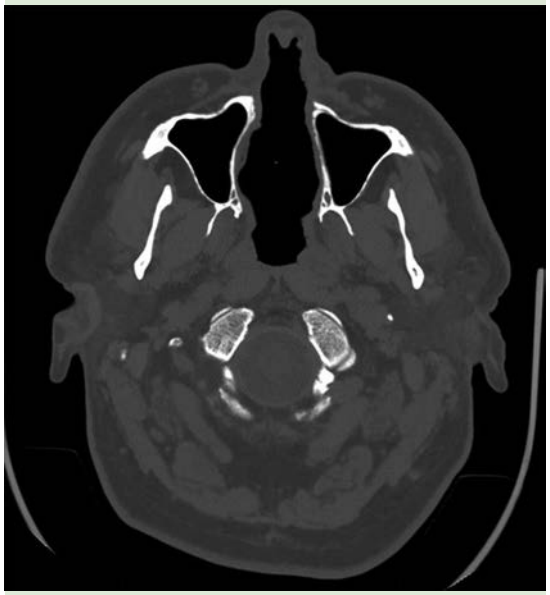

Figure 1. CT shows extent necrosis of central sino-nasal structures.

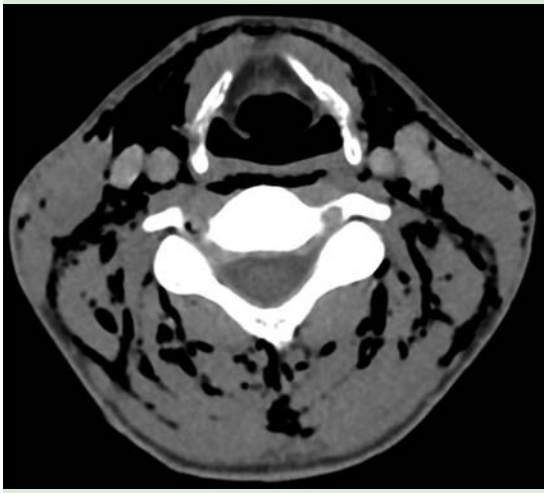

Figure 2. CT shows extent emphysema of the neck.

chi et al. even showed that a nasal perforation had a positive predictive value of 88.9% for

Table 2. Most frequent otolaryngologic manifestations of cocaine abuse

Clinical manifestations
Cocaine-induced midline destructive lesions[a]
• Septal perforation
• Turbinate necrosis
• Hard/soft palate necrosis
• Sinus destruction
Oral ulcers[b]
• Oral sores
• Pemphigus-like lesions
Hearing loss[c]
Pinna/nose/facial necrosis[b]
Subcutaneous emphysema[b]
Invasive fungal sinusitis[c]
Pott's puffy tumor[c]
Laryngeal injuries[b]
Oropharyngeal injuries[c]
Sudden Airway Obstruction[c]

[a] More than 20 cases reported in the medical literature.
[b] Between 5 and 20 cases reported in the medical literature.
[c] Less than 5 cases reported in the medical literature.

cocaine-induced midline destructive lesions. On the other hand, granulomatosis with polyangiitis lesions are less severe and are scattered, reflecting a diffuse inflammatory process. The absence of renal or pulmonary involvement does not exclude limited granulomatosis with polyangiitis.[6]

ANCA's directed against proteinase 3 (PR3) or myeloperoxidase have long thought to be specific markers for granulomatosis with polyangiitis. However, Trimarchi et al. reported positive ANCA staining in 14 out of 25 patients with sinus and nasal necrosis secondary to cocaine.[6]

Recently, Wiesner et al. studied human neutrophil elastase (HNE) ANCA's in patients with cocaine-induced midline destructive lesions. HNE-ANCA's were detectable in 84%. Blood samples from patients with Wegener granulomatosis, microscopic polyangiitis or from healthy controls were all HNE-ANCA negative. They concluded that HNE ANCA's might be a way to discriminate cocaine-induced midline

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destructive lesions from granulomatosis with polyangiitis, whereas testing for PR3 ANCA's may not.[7]

Micro-abscesses in the vascular wall and perivenulitis are common in both cocaine-induced midline destructive lesions and granulomatosis with polyangiitis. Stromal granulomas with giant cells, deeply located necrosis and extravascular micro-abscesses are found only in granulomatosis with polyangiitis.[6]

In acute lesions, thorough curettage should be performed to eliminate all active foci. Surgical correction of soft and hard tissue is delicate and doomed to fail if cocaine abuse continues. Therefore, reconstruction should only be initiated after a 12 to 18 months drug free period, confirmed by regular urine testing. Most often requested procedures are septal and palate reconstruction, rhinoplasty and closure of naso-cutaneous fistula.[6] There is no benefit in using immunosuppressive therapy. In only one of our cases reconstructive surgery was performed to improve nasal deformities. In two other cases the patients did not quit cocaine abuse, thus reconstructive surgery was not considered an option.

Reconstruction of cocaine-induced midline destructive lesions is challenging due to a myriad of reasons. First, anatomical structures are often left with a complex 3-dimensional defect. Secondly, the vascularity of the affected structures is often impaired and surgical flaps are therefore prone to failure. Thirdly, patients have to remain free of cocaine use in order to secure long-term surgical success.[8]

Most common surgical complications include localized septal collapse, delayed mucosal healing, and inadequate correction of septal deflection. One case study report on poor healing after endonasal surgery, even 10 years after cocaine abuse was ceased.[8]

Surgical correction will surely fail if the patient resumes the drug abuse. There is, however, no consensus on the acceptable abstinence period prior to surgery. Some authors consider an abstinence period of 6-18 months, confirmed by serial urine analyses, to be sufficient, while others demand the patient to be drug-free for several years.[8] At the very least, patients must be informed of the risks of continued cocaine abuse. They should confirm that they are no longer abusing cocaine and are committed to remain clean permanently.

Other sinonasal manifestations

In a retrospective questionnaire-based study, Schwartz et al. reported that nearly half of daily cocaine users experience symptoms such as frequent sniffing, diminished olfaction, nasal membrane irritation with nasal crusts or scabs and recurrent nosebleeds. Cocaine abuse should be considered as a cause of recalcitrant chronic rhinosinusitis, especially in teenagers and young adults.[9]

Chronic invasive fungal sinusitis is a rare but potentially aggressive form of invasive fungal disease that occurs in typically immunocompetent and healthy patients. Chronic invasive fungal sinusitis is characterized by a slow-onset yet progressive tissue destruction and angio-invasive non-granulomatous inflammation.[10] Symptoms are often non-specific, and can include nasal obstruction, disturbed smell, facial pain, rhinorrhea, headache and epistaxis. Left untreated, chronic invasive fungal sinusitis may even lead to severe neurological complications.[10]

Cocaine users are likely predisposed to bacterial and fungal infections due to soft tissue necrosis and local nasal immunosuppression.

A 24-year old male cocaine user presented with nasal and palate pain, a history of nasal congestion

and rhinorrhea, and nonhealing nasal septal and palatal perforations. Histopathology revealed the fungus *Aspergillus flavus*. Surgical debridement was pursued and voriconazole was administered. Evaluation after five months showed a healthy mucosa without crusting or necrotic tissue.[11]

In a 28-year old immunocompetent female patient, clinical examination showed paranasal erythema and extensive damage of the columella, septum and nasal floor. Mycological culture analysis of a yellowish substance from the nasal floor revealed the growth of *Aspergillus flavus*. Complete clinical remission was achieved after surgical debridement and oral itraconazole treatment. Other therapeutic approaches have been proposed such as high doses of i.v. amphotericin B, followed by long-term p.o. itraconazole therapy. Surgical debridement is necessary since *Aspergillus* causes necrosis by infarction and direct angio-invasion.[10]

Tissue necrosis and ulceration related to intranasal drug use should be recognized as a potential risk factor for invasive fungal sinusitis. Early biopsy and culture analysis of septal or palatal perforation may lead to earlier diagnosis and prompt surgical and medical management of chronic invasive fungal sinusitis.[11]

Pott's puffy tumor is a subperiosteal abscess of the frontal bone associated with frontal osteomyelitis. It is a rare life-threatening complication of infectious sinusitis. Noskin and Kalish described a case of Pott's puffy tumor in a patient who chronically abused cocaine. The authors propose that recurrent mucosal insults provided an excellent environment for the growth of anaerobic bacteria and ultimately the growth of a Pott's puffy tumor.[12]

Auricular and facial necrosis

It is estimated that approximately 70%-88% of cocaine is contaminated with levamisole. The most common visible adverse effect reported in patients exposed to levamisole, peroral or by inhalation by nose, is a tender skin eruption that manifests as retiform purpura.[13] These lesions may occasionally evolve into bullae, which are often accompanied by necrosis of the skin. Most commonly affected areas include the earlobe and helix, the malar region and the nose tip.[13] Interestingly, the literature describes more female than male patients with a ratio of 2/1.[3] In our series we did not find any case of skin lesions related to cocaine abuse.

Histopathologic examination of patients with purpura of the ears, malar region or nose either shows a thrombotic vasculopathy, a leukocytoclastic vasculitis or a combination of both, with a subsequent necrosis of the epidermis and dermis.[13] A review by Poon et al. reports that the origin of purpura is in 64% solely a thrombotic vasculopathy, in 32% a thrombotic vasculopathy with leukocytoclastic vasculitis, and in 4% solely a leukocytoclastic vasculitis in a total of 22 patients with suspicion of levamisole adulterated cocaine-induced purpura of the face and ears.[5]

The pathogenesis of levamisole-induced vasculopathic purpura is unclear. However, antineutrophilic cytoplasmic antibodies (ANCA's) are frequently elevated in patients with purpura induced by levamisole-adulterated cocaine. The majority of patients (87-100%) test positive for perinuclear antibodies (p-ANCA's),[14] although antibodies against cytoplasmic ANCA's (c-ANCA's), cardiolipin and lupus coagulant are also regularly elevated.[5]

Whether the antibodies are pathogenic by inducing vasculitis or are merely bystanders produced by a general immune reaction is unclear. However, the more frequent histopathologic finding of thrombotic vasculopathy[5] might also suggest direct toxicity of levamisole on the endothelium.[13] Cocaine itself may also play a role in producing

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cutaneous lesions by vasoconstriction and endothelial activation. Older literature describes a 'pseudo-vasculitis' induced by cocaine alone, but this was before research on levamisole-adulterated cocaine was widely spread.[5]

In summary, the mechanism by which levamisole-adulterated cocaine induces retiform purpura in its specific distribution pattern, remains poorly understood. Thrombotic vasculopathy, vasculitis, auto-antibody production and the direct toxic effects of both levamisole and cocaine all appear to play a role in the pathophysiology and symptomatology of levamisole-adulterated cocaine -induced auricular and facial necrosis.[5,13,14]

Cocaine-mediated hearing loss

Several cases of sudden hearing loss after cocaine consumption have been reported in young patients, most of them with improvement after corticosteroid treatment.[15] Stenner et al. described a case of a 26-year old man who presented to the outpatient department with severe bilateral hearing loss after intravenous injection of cocaine. Audiogram showed bilateral normoacusia after three days of treatment with intravenous sodium chloride, prednisolone and pentoxifylline.[16]

The exact pathophysiology of cocaine-mediated hearing loss remains unclear, but several hypotheses have been put forth. First, hearing loss may be caused by intralabyrinthine hemorrhage or cochlear ischemia due to the inherent vasoconstrictive properties of cocaine, although bilateral hearing deficits are unlikely caused by two independent vascular events. Some authors theorize that cocaine damages the hair cell's metabolism by blocking K^+-channels, such as seen in myocytes.[16]

Thirdly, although cocaine abuse has not been linked with autoimmune-induced hearing loss, it has been linked to autoimmune-induced brain pathology and could therefore theoretically affect the cochlea.[16] Effects of additional substances in cocaine could not be excluded.

Oral manifestations

Crack cocaine is a free base heat-stable form of cocaine that can be smoked. The usual way of consumption is with a glass straight pipe. The heated smoke is inhaled and comes into immediate contact with the buccal mucosa and palate before passing into the trachea and lungs.[17]

Crack users frequently have blisters, burns, sores and cuts on the lips and in the oral cavity. These may be the result of direct contact with the hot glass pipe, direct contact with hot smoke, or due to glass pipes breaking.[17]

A cross-sectional study found a 25% prevalence of oral mucosal lesions in crack cocaine-addicted participants, traumatic ulcers and actinic cheilitis being the most prevalent.[17]

Crack cocaine consumption has also been associated with a decayed teeth index, dental erosions, hyposalivation, periodontitis and gingival lesions, and with chromosome breaks and oral mucosal cell death.[18]

Intranasal cocaine use has been associated with pemphigus family disorders, mainly pemphigus vulgaris.[19] Cocaine contains an aromatic ring and hydrolysis could produce a phenolic intermediate. Cocaine can thus be considered a masked phenol drug. Phenol drugs induce pemphigus by disrupting cell adhesion by stimulating keratinocytes to release proinflammatory cytokines. Unidentified diluents in cocaine could also be the source of drug-induced pemphigus.[19] One of our patients who had a cocaine-induced midline necrosis developed pemphigus-like lesions which disappeared with cessation of cocaine consumption.

Laguna et al. described a case of a 37-year-old man with a 2-week history of painful erosive lesions on his oral and genital mucosa, along with bullae and erosions on his back. Histopathological analysis confirmed the diagnosis of pemphigus vulgaris. Urinary toxicology was positive for cocaine and the patient admitted the use of cocaine. The erosions rapidly improved after cocaine cessation. There were two outbreaks of pemphigus, despite a maintenance therapy with prednisone and mycophenolate mofetil. Both outbreaks coincided with a relapse in cocaine use. It is unclear whether the pemphigus was caused by cocaine itself or by a diluent.[19]

Blaise et al. reported a case of pemphigus-like cutaneous and mucosal ulcers, similar to our first case (**Table 1**). Biopsy showed infiltrates of neutrophils and plasma cells in the dermis or submucosa. The ulcerations significantly improved after cessation of cocaine use. At one year after discharge, no recurrent skin lesions were noted.[20] In our case, no histopathological signs for pemphigus were found, but the oral lesions disappeared after cocaine discontinuation.

Pharyngolaryngeal manifestations

The use of "crack" and "freebase" cocaine has resulted in an increased number of patients presenting to emergency rooms with cocaine-related injuries of the upper aerodigestive tract. Crack cocaine is commonly smoked through a pipe or mixed with tobacco. When it is smoked, the rock is converted to hot vapor that could potentially burn the mucosa of upper aerodigestive tract.[21] In addition, smoked through a pipe, the metal screen becomes hot and may partially disintegrate, resulting in aspiration or ingestion of burning metal debris or smoldering crack particles. Due to the anaesthetic effect of the cocaine when it gets in contact with the mucosa, the patient is at increased risk for thermal burns to the oral cavity, oropharynx, larynx, esophagus and lower airway.[21] Because symptoms are nonspecific and histories often unreliable, the emergency room physician must keep a high index of suspicion for cocaine use. So, a complete examination of the upper aerodigestive tract is essential to make an accurate diagnosis and recommend appropriate therapy.[21]

Crack cocaine inhalation is a known cause of burn injuries of the upper airways and it has become a significant cause of adult supraglottitis between others. Pharyngeal and laryngeal burns may present with various and sometimes puzzling clinical manifestations, but the most characteristic symptoms are hoarseness, dyspnea with stridor, odynophagia, and dysphagia.[21] The differential diagnosis includes other inflammatory conditions of the upper airway, such as diphtheria, acute epiglottitis, caustic ingestions, foreign bodies, and neoplasms.

Pharyngeal injuries could vary from superficial to extensive necrotizing burns that involved the base of the tongue, epiglottis, and pyriform sinuses that are basically the result of inhaling hot vapors of free-base cocaine.

Supraglottic injuries may result from both thermal and chemical mechanisms and is highly more frequent, while injury in lower respiratory tract is often solely due to chemical mechanisms. It is a rare manifestation due to the glottic closure reflex, activated by heat or irritative stimulus.[22] It is important to remember that the risks of burns in pharyngeal and laryngeal tissues are further enhanced by the anesthetic effect of cocaine when it comes in contact with mucous membrane.[22]

Sometimes, acute inflammation of the upper airway as a result of cocaine inhalation can develop into a sudden airway obstruction which requires intubation or tracheostomy.[21] Polyposis or hyperplasia, as well as bilateral saccular cysts in the respiratory mucosa have been reported as a result

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of crack-related burns. Episodes of coughing after vigorous inhalation could increase intralaryngeal pressure that, combined with the irritative effects of crack on the mucosa, contribute to the formation of laryngoceles or saccular cysts.[23]

Thus, the abuse of crack cocaine is a growing problem, and the incidence of thermal and cocaine-induced injuries at the level of the upper aerodigestive tract will likely increase, risking life-threatening complications such as airway compromise. The increasingly reported cases indicate that clinicians should have a high suspicion for illicit drug use when adults present with symptoms suggestive of upper airway injury.

Other otolaryngologic manifestations

One of our patients developed a subcutaneous emphysema secondary to pneumomediastinum associated with drug abuse. In these cases, it is important to consider other serious associated conditions. Mechanisms of injury are related to rupture of marginal pulmonary alveoli caused by increased alveolar pressure due to barotrauma or in cases of inhalation of "free-based" or "crack" cocaine secondary to necrosis and perforation of the posterior pharyngeal wall from the toxic vasoconstrictive properties of the inhaled vapors.[24] Flexible nasofibroscopy is recommended to look for pharyngeal perforation or injury, followed by gastrografin swallow in the first 24 hours to assess for esophageal injury.[24] Subcutaneous emphysema secondary to alveoli rupture usually takes a benign course as in our case and should be managed conservatively with 24-48hours of observation with prophylactic antibiotics and oxygen therapy.[24]

Our last patient presented with a pyomyositis (pyogenic myositis), a bacterial infection of striated muscle tissue, at the level of the sternocleidomastoid muscle after attempting to inject cocaine intravenously. Pyomyositis of the sternocleidomastoid muscle is very rare condition and most described cases are associated with immunosuppressive conditions and are usually secondary to *Staphylococcus aureus*.[25] In our case, the responsible bacterium was not identified. No previous cases have been found in the medical literature related to drug abuse.

CONCLUSIONS

While cocaine-induced midline destructive lesions are the most common presentation of cocaine abuse, other manifestations may be seen, and the clinician must maintain a high index of suspicion for underlying cocaine consume.

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