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Crack cocaine induced upper airway injury.

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CRACK COCAINE INDUCED UPPER AIRWAY INJURY

LÉSIONS RESPIRATOIRES SUPÉRIEURES LIÉES À L'USAGE DE CRACK

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SUMMARY. The paper describes the presentation and management of patients presenting with crack cocaine induced upper airway injury. The study involved a retrospective clinical series of six patients with crack cocaine induced upper airway injury. Demographics, symptoms, physical exam, flexible laryngoscopy findings, treatment and intervention were recorded. All patients with crack cocaine induced thermal injury presented with mouth or throat pain plus at least one other laryngeal symptom, such as globus sensation, dysphagia or throat tightness. On physical exam, the supraglottis was the most common subsite of endolaryngeal injury. The only statistically significant finding was the number of subsites on initial physical exam and flexible laryngoscopy and need for airway intervention (p = 0.001). Airway intervention was required in one patient, while the remaining patients were closely observed until resolution of symptoms. Upper airway injury should be suspected in patients who present with pain and laryngeal symptoms after smoking crack cocaine.

Keywords: crack cocaine, airway, burn, flexible laryngoscopy

RÉSUMÉ. *Nous décrivons la clinique et la prise en charge des patients souffrant de lésions respiratoires supérieures liées à l'usage de Crack, en nous basant sur une série rétrospective de 6 cas. Nous avons colligé la démographie, les signes et symptômes, les données cliniques et endoscopiques ainsi que le traitement. Tous souffraient de douleur bucco- pharyngées et au moins d'un signe laryngé parmi sensation de gonflement, dysphagie ou sensation d'étouffement. Á l'examen, la zone supra- glottique était la plus communément atteinte. Le nombre de zones atteintes corrélait positivement (p = 0,001) à la nécessité d'une intervention sur les voies aériennes, qui n'a cependant été nécessaire que pour 1 patient, les autres ayant été simplement surveillés jusqu'à disparition des symptômes. Une atteinte des voies aériennes supérieures doit être soupçonnée devant un patient se présentant avec des douleurs et des signes laryngés après avoir fumé du crack.*

Mots-clés*: crack, brûlure, voies aériennes, endoscopie*

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Introduction

By the 1980s, a new form of cocaine, called crack cocaine, started to gain popularity in the United States.[1,3] Through a chemical process, the combination of powdered cocaine hydrochloride with water and sodium bicarbonate produces a cocaine crystal.[1,3,4] As heat is applied, the crystals vaporize and can be inhaled into the lungs using various vaporizing methods.[5] Inhaling the vapor form of cocaine provides quick deliverance of the drug into the pulmonary vasculature and subsequently into the cerebral circulation.[4] Two major threats to the upper airway of smoking cocaine include thermal injury secondary to the high vapor temperature and complications of using a metal filter. The short glass pipe typically used does not allow the vapor to cool sufficiently before contacting the upper airway mucosa.[2] A filter, commonly fabricated out of Brillo screens, steel wool, brass or copper wire is required to keep the crystals in place within the pipe.[6] Pieces of superheated filters can be dislodged and subsequently inhaled, causing foreign body trauma and inflammation to the respiratory lining.[2,5-7]

Several case reports over the last decade have described the clinical presentation of patients inhaling cocaine vapor or filter particles; however, the immediate sequelae of this trauma are rarely reported and poorly understood. Additionally, literature delineating oral, oropharyngeal and endolaryngeal mucosal injury is very limited. We aim to describe the acute findings and management of patients with confirmed thermal injury secondary to crack cocaine use.

Methods

Approval for this clinical series was obtained through the Wayne State University Institutional Review Board. Charts from the Detroit Medical Center ranging from January 2011 to November 2015 with the ICD9 codes including burn of the epiglottis/larynx/trachea (947.1), inhalational burn (947.9), epiglottitis (464.30, 464.31) and supraglottitis (464.50, 464.51) were reviewed. Patient demographics, presenting symptoms, physical exam findings including flexible laryngoscopy, acute medical treatment, airway intervention, medication administered, and disposition were recorded. The physical exam, including the flexible laryngoscopy, was first completed by a resident, and the findings were subsequently verified by an attending otolaryngologist. This data was collected and then verified for accuracy by a second investigator. The two tailed t-test and Mann-Whitney U test were used to evaluate for statistical differences between patients. Results were considered significant when $p<0.05$.

Results

Six patients with thermal injury related to cocaine inhalation were identified (*Table I*). Due to airway complaints, otolaryngology evaluated all patients. The average age at presentation was 51.7 (SD 8.4, range 35-58) and the male to female ratio was 2:1. There was no significance found between age and need for airway intervention ($p=0.80$). All patients in this series were African American. No patient had a prior episode of upper airway burn secondary to crack cocaine. Five of the six patients were current cigarette smokers and all had a history of tobacco use. All patients were found to have cocaine detected on their urine drug screen.

Three patients presented to the emergency department within four hours of experiencing the thermal injury, while one presented after 12 hours. The remaining two patients presented around 8 hours after

Table I - Demographics and presenting vital signs

	Mean ± SD (range) or n (%)
Age (years)	51.7 ± 8.5 (35-58)
Gender	
Male	4 (66.6)
Female	2 (33.3)
Race	
African American	6 (100)
Current tobacco user	
Yes	5 (83.3)
No	1 (16.7)
Systolic blood pressure (mmHg)	166 ± 39.4 (115-223)
Diastolic blood pressure (mmHg)	97.2 ± 14.6 (77-116)
Heart rate (bpm)	75.5 ± 10.8 (59-88)

thermal injury. There was no significance noted between the timing of presentation and airway intervention (p=0.76). The mean heart rate, systolic blood pressure and diastolic blood pressure on presentation was 75.5 bpm (SD 10.8, range 59-88), 166 mmHg (SD 39.3, range 115-223), and 97.2 mmHg (SD 14.6, 77-116), respectively. There was no significance between presenting heart rate, systolic and diastolic blood pressure, and need for airway intervention (p=0.09, 0.67, and 0.84).

All patients complained of pain in the mouth or throat with associated globus sensation, dysphagia or throat tightness. Two described dysphonia and one patient complained of shortness of breath and difficulty breathing. The mean number of presenting symptoms was 3.5 (SD 1.8, range 1-6). No patient complained of pruritis or urticaria. Statistical significance between number of presenting symptoms and airway intervention was not detected (p=0.16).

On initial evaluation one patient was in respiratory distress. No patients had identifiable injury to the face, lips or oral cavity, or were drooling. All patients underwent a complete physical exam and flexible laryngoscopy to visualize the upper airway (*Table II*). Supraglottic injury was identified in four patients,

Table II - Subsites involved on physical exam

	Patient 1	Patient 2	**Patient 3***	Patient 4	Patient 5	Patient 6
Face						
Oral cavity						
Supraglottis	Moderate		**Severe**	Moderate	Mild	
Oropharynx			**Severe**		Mild	Mild
Glottis						
Hypopharynx		Moderate				

* Bold indicates the patient who was intubated
Mild, moderate, and severe indicates degree of edema identified on exam

while there was injury to the oropharynx of two patients (*Fig. 1*), and injury to the hypopharynx in one patient. No injury to the glottis or subglottis was appreciated in any patient. A significant correlation was found between the number of subsites involved and the need for airway intervention (p=0.001).

All patients received intravenous steroid due to airway edema. Due to the severity of the edema and clinical presentation of respiratory distress, one patient also received an antihistamine and an H2 blocker. Three patients underwent a soft tissue neck x-ray and one patient underwent a chest x-ray, which were both non-diagnostic. After clinical evaluation

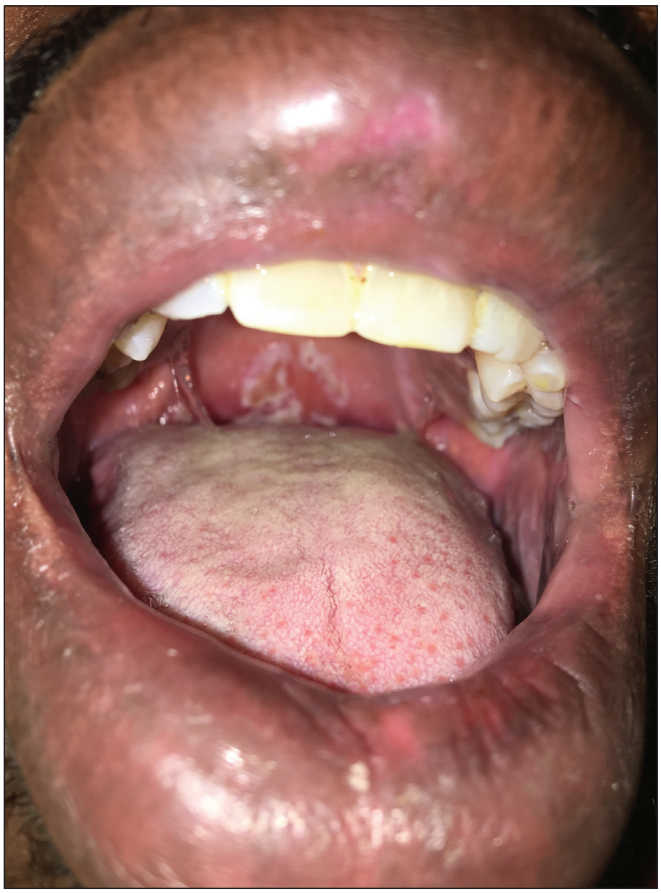

Fig. 1 - Soft palate thermal burn secondary to inhaled crack cocaine

and flexible laryngoscopy, one patient was urgently intubated awake in the operating room using a Miller blade. On evaluation, severe edema of the oropharynx and supraglottis was identified. Additionally, although not statistically significant, this was the only patient with a chief complaint of respiratory difficulty. The remaining five patients did not require airway intervention at any point. All patients were admitted for close observation. Four patients were placed in the emergency department overnight observation ward, which included continuous pulse oximeter and close nursing monitoring. The remaining two patients were observed in the intensive care unit. Two patients reported resolution of symptoms within 12 hours of the inhalation, while three patients experienced resolution within 24 hours. The patient who was intubated met weaning parameters with a cuff leak at around 36 hours and was subsequently extubated after repeat fiberoptic laryngoscopy confirmed resolution of the supraglottic edema. There was no significance noted between the time of symptom resolution and airway intervention (p=0.13).

Discussion

Several case reports have described serious thermal injury to the upper respiratory tract secondary to the use of crack cocaine. Osborne et al. and Zacharias et al. postulated that tissue trauma occurred secondary to extremely high vapor temperatures contacting respiratory mucosa.[2,8] In other case studies, patients found to have crack cocaine associated thermal injury presented to the emergency department with pain, respiratory distress, stridor, globus sensation, dysphonia, odynophagia and/or drooling.[2,5,7,9] In our study, all six patients presented with symptoms of pain in the throat or mouth with associated globus sensation, dysphagia, and/or throat tightness, which is similar to previous descriptions.

On flexible laryngoscopy, we found 67% (4/6) of our patients had a supraglottic injury, 33% (2/6) had trauma to the oropharynx, and 17% (1/6) of these patients had trauma to the hypopharynx. Our laryngoscopy findings were also similar to what others have described. Snyderman et al. reported epiglottic, left aryepiglottic fold, and false vocal fold edema, increased secretions in left pyriform sinus, and immobility of the left true vocal fold.[7] They hypothesized that the location of thermal injury may preferentially affect the supraglottic tissues due to the reflexive protective mechanism of the larynx.[7] Interestingly, very high deposition values on upper airway tissue have been found using cigarette smoke inhalation vapor as a model.[10] Our findings corroborate these findings as most patients experienced supraglottic versus oropharyngeal injury. Five patients underwent either a chest x-ray or soft tissue neck film with no metallic foreign body or significant edema visualized.

All of our patients received intravenous steroids due to airway edema. This treatment was similar to what others provided after initial presentation and exam findings.[2,5,7,9] None of our patients received antibiotics. Most patients (67%) experienced resolution within 24 hours with subsequent discharge. One patient, who presented with obvious respiratory distress and significant edema found on flexible bedside laryngoscopy, was urgently intubated. By 36 hours this patient met weaning parameters, had a cuff leak, and was subsequently extubated. Osborne et al. describes variability in the management of supraglottic edema.[11] At their institution, immediate intubation was described as the standard in the setting of supraglottic edema findings on fiberoptic laryngoscopy.[11] Our algorithm for airway intervention, such as intubation, is based on the combination of clinical severity and laryngoscopy findings. Close observation in a monitored bed can be considered in a clinically stable patient with mild or moderate airway edema secondary to thermal injury. Airway management must be tailored using all available information.

There are several limitations inherent to a retrospective clinical series. There are significant restraints in data collection due to variation in charting methods. There were limited patients as upper airway injury secondary to crack cocaine use is a rare condition; therefore, multivariate statistics could not be completed and correlations are limited due to underpowered data. The type of filter used within the crack pipe was not recorded; therefore, differentiation between injury secondary to superheated vapor versus molten filter (metal) inhalation was not possible. Due to the low incidence of cocaine associated thermal injury, amount of edema on presentation and other presenting findings could not be statistically correlated to need for airway intervention.

Conclusion

Upper airway thermal injury secondary to crack cocaine is an uncommon cause of upper airway trauma. All patients with crack cocaine induced thermal injury presented with pain with other laryngeal symptoms, such as globus sensation, dysphagia and/or throat tightness. The supraglottis was the most common subsite of injury. Airway intervention was uncommonly required in this clinical series. In patients who present with pain and laryngeal symptoms around the time of crack cocaine use, the airway should be evaluated with laryngoscopy. Appropriate management includes steroid, possible airway intervention and close observation.

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