



## Case report

## Pneumorachis after cocaine sniffing

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## ABSTRACT

Air in the epidural space is called pneumorachis. The usual mechanism of pneumorachis is air diffusion from the mediastinal tissue layers through the inter-vertebral foramen. Alternatively, air can diffuse directly after spine traumas (e.g., blunt deceleration with vertebral dislocation) or medical procedures. Several mechanisms could explain pneumomediastinum and pneumorachis after cocaine sniffing. Passive apnea and/or cough that occur after sniffing can cause intra alveolar hyper-pressure, which is responsible for alveolar rupture and air diffusion. Another mechanism is alveolar wall fragility and rupture induced by repeated cocaine sniffing, in turn causing air diffusion to the mediastinum, sub-cutaneous tissues and the epidural space. The diagnosis is usually made on Chest tomography scan. Management consists in close monitoring in the intensive care unit to detect aggravation of pneumomediastinum and pneumorachis, which would require surgical management. Supplemental nasal oxygen can be given to accelerate nitrogen washout. We present a case of a 28 years old male who presented to the emergency department for chest pain directly after sniffing cocaine. A computed tomography scan of the chest showed pneumomediastinum, pneumorachis and sub-cutaneous emphysema. The patient was admitted for 24 h: after that delay, surveillance chest tomodensitometry showed stability, and he could be discharged without further treatment.

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## 1. Introduction

Air can diffuse into the mediastinal and sub-cutaneous tissue layers after trachea-bronchial rupture, esophageal perforations, facial lesions, alveolar rupture secondary to bronchial hyper-pressure (e.g., asthma attacks, foreign body inhalation, intense cough, mechanical ventilation, vomiting, Valsalva maneuver, extreme effort) or diseases that cause alveolar wall fragility (e.g., pulmonary infections, emphysema, sarcoïdosis and silicosis, pulmonary fibrosis and drug toxicity) [1]. Air in the epidural space is called pneumorachis or aerorachia [2]. The usual mechanism is air diffusion from the mediastinal tissue layers through the inter-vertebral foramen. Alternatively, air can diffuse directly after spine traumas (e.g., blunt deceleration with

vertebral dislocation) or medical procedures (e.g., lumbar puncture, epidural injection) [3]. Cocaine sniffing can cause pneumomediastinum [4].

We report a case of pneumomediastinum, sub-cutaneous emphysema and pneumorachis following cocaine sniffing. This is the first reported case of pneumorachis secondary to cocaine sniffing.

## 2. Case

The patient was a 28 years old male non smoker, with no medical or surgical history. He had been sniffing cocaine daily for the last 3 years, and did not report any other illicit drug abuse. Immediately after sniffing cocaine in a party, he felt an intense retrosternal and neck pain, without any cough, trauma or vomiting.

On admission he did not complain of shortness of breath. His vital signs were normal, blood pressure 122/77 mmHg, pulse rate 77, respiratory rate 20 and temperature 37 °C. Pulmonary auscultation showed normal vesicular murmur and cardiac auscultation showed regular heart beats without murmurs. His physical exam was normal except for cervical crepitations on skin

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palpation. Neurological exam was normal. The patient did not show any symptom or sign of medullar compression or meningismus.

Electrocardiography showed sinus rhythm without signs of ischemia. Laboratory troponin test was less than 14 ng/L (normal range). Myocardial ischemia in a case of chest pain in a cocaine user was eliminated. Chest CT-Scanner showed diffusion of air into mediastinal, sub-cutaneous and epidural tissue layers (Fig. 1(a)–(b)), with no evidence of medullar compression. There was no air diffusion in deep cervical tissue layers. The patient was admitted to the hospital for 24 h and treated with pain-killers. A second chest CT-scan performed 24 h after admission did not show any aggravation of the pneumomediastinum or pneumorachis. The patient was then discharged home with a final diagnosis of pneumomediastinum and pneumorachis secondary to cocaine sniffing.

### 3. Discussion

Cocaine smoking or sniffing is a risk factor for diffusion of air in thoracic tissue layers [5]. Then air can reach skin tissues, epidural space (pneumorachis) and peritoneum. Cases of pneumorachis have been reported in the literature after thoracic trauma, blunt deceleration injury, oro-tracheal intubation, marijuana smoking with a special device [6] and severe asthma attack. We report here the first case of pneumomediastinum and pneumorachis secondary to cocaine sniffing.

Several mechanisms could explain pneumomediastinum and pneumorachis after cocaine sniffing. Passive apnea and/or cough that occur after sniffing can cause intra alveolar hyper-pressure, i.e.,

barotrauma, which is responsible for alveolar rupture and air diffusion [7]. Barotrauma is generated by increased intrapulmonary pressure and a subsequent high transmural gradient between the alveoli and the surrounding interstitial space. After the rupture, air diffuses to interstitial space, and then gets to the mediastinal soft tissue layers. The mediastinum communicates easily with deep cervical tissue layers and sub-cutaneous cervical space. Finally, mediastinal air migrates through the inter-vertebral foramens towards epidural space and the pneumorachis is formed [7]. An additional mechanism could be involved, i.e., alveolar wall fragility induced by repeated cocaine sniffing [8].

Another physiopathological process can be responsible for air diffusion. In fact air diffusion could have started in a facial or cervical structure, than air can migrate to epidural space. In fact, Cocaine sniffing is responsible of destruction of nasopharyngeal structures. The pathophysiology of cocaine induced destructive lesions is multifactorial. It includes ischemia, infection, impaired mucociliary transport and decreased immunity. Destruction of these structures can cause air leak and diffusion. A case of pneumocephalus secondary to cocaine abuse was reported in 2011, the authors concluded that air diffused from nasal midline structures [9]. In our case, this hypothesis is unlikely because nasopharyngeal physical examination did not show structural destruction and on chest tomodensitometry there was no air diffusion in deep cervical tissue layers.

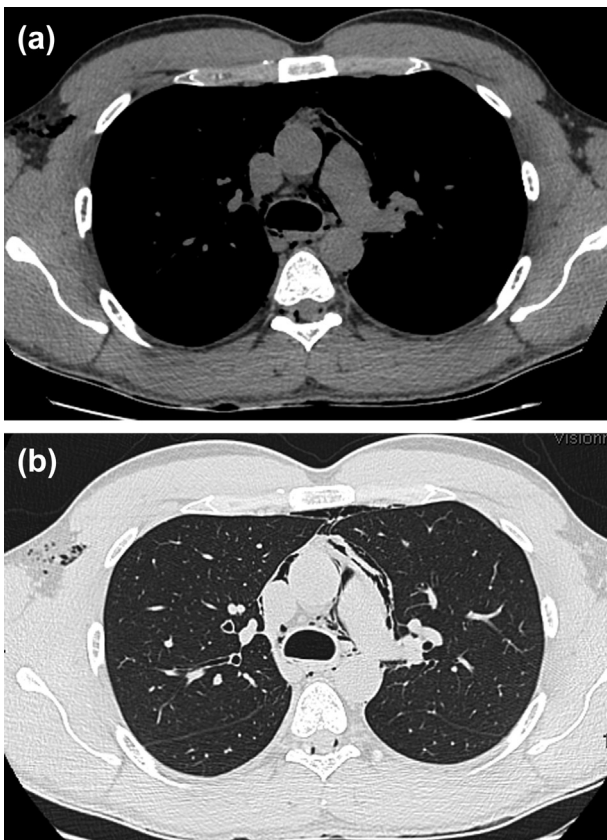
Several cases of pneumomediastinum secondary to cocaine sniffing or smoking have been reported. In 2004, James S.M. et al. reported a case of “crack inhalation-induced pneumomediastinum”. The CT-images showed free gas in the mediastinum. The patient recovered rapidly without intervention other than oxygen delivery, he was discharged after three days when his chest X-Ray returned to normal [10]. In 2003, Micha Maeder et al. had reported a pneumomediastinum and bilateral pneumothorax as a complication of cocaine smoking. No information was given about oxygen use or length of stay in the hospital. The air collection resolved spontaneously [11].

The lack of previously reported cases of pneumorachis associated with cocaine sniffing might be at least in part explained by under-diagnosis. In fact, cocaine and illicit drug users likely tend to present less to the ER when symptoms occur after sniffing; it might thus well be that many cases of pneumorachis are not diagnosed and heal spontaneously without any medical care.

As in similar cases of non traumatic pneumorachis, the patient recovered completely without any sequela or complication [5]. He stayed in the hospital less than 24 h, i.e., much less than other pneumorachis cases reported in the literature. He was actually discharged immediately after the CT-scan performed at 24 h had shown stability, while previously reported cases admitted for pneumomediastinum or pneumorachis were monitored for much longer. No oxygen was delivered, which is atypical since many patients with pneumothorax or pneumomediastinum are treated with oxygen to achieve nitrogen washout [5]. This observation thus suggests that patients admitted for pneumorachis after cocaine sniff-induced intra-bronchial hyper-pressure can be monitored for a short period of time and do not require oxygen therapy. Additional observations are required to confirm these findings.

### 4. Conclusion

Pneumomediastinum has been recognized several decades ago as a non life-threatening complication of cocaine sniffing. Association with a pneumorachis in the same patient had not been reported so far. Treatment consists in a simple monitoring during a short period of time.



**Fig. 1.** Unenhanced axial transverse CT image, (a) mediastinal window (b) lung parenchyma window, showing Pneumomediastinum together with air in peridural space and right axillary sub-cutaneous emphysema.

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