

# Pulmonary manifestations arising from the use of crack

## *Manifestações pulmonares decorrentes do uso de crack*

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### ABSTRACT

Crack is considered the most powerful and addictive form of cocaine and lungs are affected immediately after inhalation. It is associated with various manifestations such as barotrauma, exacerbation of bronchial asthma, acute pulmonary edema, alveolar hemorrhage, obliterating bronchiolitis with organized pneumonia, and vasculitis. Its acute clinical manifestations characterize the “crack lung” syndrome with its own image manifestations.

**Key words:** Crack Cocaine; Cocaine-Related Disorders; Emergency Medical Services; Lung Diseases.

### RESUMO

*O crack é considerado a forma mais potente e viciante da cocaína, sendo o pulmão acometido logo após a sua inalação. Associa-se a diversas manifestações como barotrauma, exacerbação da asma brônquica, edema agudo pulmonar, hemorragia alveolar, bronquiolite obliterante com pneumonia organizada e vasculite. Suas manifestações clínicas agudas caracterizam a síndrome “pulmão do crack”, com manifestações imaginológicas próprias.*

*Palavras-chave:* Cocaína Crack; Transtornos Relacionados ao Uso de Cocaína; Medicina de Emergência; Pneumopatias.

### INTRODUCTION

The smoke from crack, after being inhaled, quickly reaches the mucous membranes and pulmonary alveolar epithelium due to the high volatility of its combustion's products. It quickly reaches the bloodstream due to the large pulmonary surface for absorption and diffusion of substances, producing an effect that is faster than the intravenous administration of cocaine.

Pulmonary manifestations associated with the use of crack are multiple and characterized mainly by barotrauma, exacerbation of bronchial asthma, acute pulmonary edema, alveolar hemorrhage, bronchiolitis obliterans with organizing pneumonia, and vasculitis.<sup>1-3</sup> Its acute clinical manifestations include the “crack lung” syndrome, which is the purpose of this article.

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## CLINICAL MANIFESTATIONS OF CRACK INHALATION

The term crack lung is used to describe an acute pulmonary syndrome that occurs after crack inhalation. The associated clinical manifestations derive from dry cough, hemoptysis or with dark sputum, chest pain, dyspnea, wheezing, fever, and diffuse alveolar infiltrate.<sup>2</sup> Lungs are the first organs to be exposed to crack combustion products; therefore, respiratory symptoms can occur from minutes to a few hours after use.<sup>3</sup>

It is probable that crack induces pulmonary vasoconstriction, causing cellular ischemia and likely direct toxic effect on the alveolar-capillary endothelium and hypersensitivity reaction to the inhaled components.<sup>1</sup> The coughing mechanism seems to be associated with irritation caused by the drug on subepithelial receptors. The dark sputum seems to result from inhalation of carbonaceous wastes. Chest pain, often present one hour after using the drug, and exacerbated by a deep inspiration, can represent a local sensory response to the irritation of airways. Other causes of chest pain that need to be investigated among crack smokers include acute myocardial ischemia, pneumothorax, and pneumomediastinum.<sup>2</sup> Hemoptysis is another frequent finding, present in 6 to 26% of crack users.<sup>3</sup> It can result from rupture of blood vessels present in the tracheal or bronchial submucosa, or originate in the alveolar-capillary membrane.

Radiological images of affected patients may show pulmonary interstitial and alveolar diffuse opacities. In addition, a small pleural effusion may be present.<sup>2</sup>

The anatomical histopathological analysis of lung fragments obtained from patients who are crack inhalants reveals alveolar lesion and hemorrhage injury with interstitial and alveolar cellular infiltrates rich in eosinophils with IgE deposition.<sup>4</sup>

## BAROTRAUMA

The description of barotrauma related to the use of crack is recent, however, it has been previously mentioned in marijuana users.<sup>1</sup> They are associated with pneumopericardium, subcutaneous emphysema, pneumothorax, and pneumomediastinum; these latter two being commonly associated.

It is related to the sharp increase in inspiratory effort and prolonged Valsalva maneuver, usually

performed by the user in an attempt to increase the drug effect. It also seems to be associated with intense cough, usually triggered by the direct effect of the drug on the tracheobronchial tree. These mechanisms determine the sudden surge of intrabronchial and alveolar pressure causing alveolar rupture and subsequent air penetration in the lung interstitium. The released open air can dissect the peribronchial connective tissue, within the mediastinum, pericardium, pleural cavity, or subcutaneous tissues, and determine pneumomediastinum, pneumopericardium, pneumothorax, or subcutaneous emphysema, respectively.<sup>2</sup>

The associated symptoms are dyspnea and chest pain. The diagnosis is suggested by the chest x-ray, which can show retrosternal gas, lateral displacement of the mediastinal pleura, and air around the diaphragm. It must be differentiated from esophagus perforation and tumors.<sup>4</sup>

## EXACERBATION OF BRONCHIAL ASTHMA

There is a strong association between the habit of smoking crack and exacerbation of bronchial asthma. It is possible that the mechanisms that intensify bronchospasm in crack users, who were previously asthmatic, result from bronchial hyper-reactivity similar to other non-specific causes such as: exposure to cold air, chemical products, stress, physical exercise, immunological reaction with formation of specific IgE, and direct release of bronchoconstricting substances on the bronchial tree.<sup>2</sup> The bronchospasm in users with bronchial asthma and constant exposure to crack can present itself as irreversibly and fatal.

## PULMONARY EDEMA

The pathogenesis of pulmonary edema after the use of crack is still unknown, however, it seems to be of a non-cardiogenic origin.<sup>1</sup> Its main pathogenesis stems from the increased pulmonary capillary permeability and high concentration of proteins in the bronchoalveolar lavage.

It is likely that the smoke from crack determines important adrenergic discharge that causes lesions in the microvascular structure, which increases the pressure in the venous return and determines pulmonary edema. It is also possible that cocaine increases

the systemic vascular resistance with left ventricular failure and consequent alveolar edema.<sup>2</sup> Cases of pulmonary edema by passive inhalation of crack have been described.

## ALVEOLAR HEMORRHAGE

The alveolar hemorrhage is often found in anatomopathological studies in crack users, whether acute or chronic, without hemoptysis, and evolving in asymptomatic form.<sup>1</sup>

Bronchoscopy may reveal blood from bronchi openings; hemosiderin in alveolar macrophages can be evidenced in bronchoalveolar lavages.

It is believed that the pulmonary vasoconstriction and cellular damage directly caused by the drug are the mechanisms that may trigger bleeding.<sup>2,5-7</sup>

## BRONCHIOLITIS OBLITERANS

Bronchiolitis obliterans is usually found in crack users with non-productive cough, fever, and dyspnea, and unresponsive to antibiotics and cough suppressants. Infectious causes and exposure to other known inhalational agents should be excluded. The administration of corticosteroids may be an effective therapy.<sup>1</sup>

Bronchiolitis obliterans commonly appears associated with pneumonia, presenting itself, in these cases, as diffuse alveolar infiltrate in thorax telerradiography and through the presence of micro-organisms in the bronchoalveolar lavage.

It is likely that these clinical manifestations arise from a direct drug toxic effect on the airways. Its association with pneumonia can occur as an idiopathic response to cocaine and substances used in its tampering or to contaminants found in objects used for its inhalation.<sup>2</sup>

## ALVEOLITIS, INTERSTITIAL PNEUMONITIS, AND FIBROSIS

The continued use of cocaine through inhalation can lead to pneumonitis and interstitial fibrosis. Its clinical manifestations are characterized by alveolar septal thickening and infiltration of neutrophils, lymphocytes, macrophages, and eosinophils, with

interstitial fibrosis evidenced by hyperplasia of pneumocytes type II.<sup>4,8,9</sup>

## VASCULITIS

The vasculitis associated with crack is necrotizing, with unknown mechanism, and associated with cerebral vasculitis.<sup>4</sup>

## COMPLEMENTARY PROPEDEUTICS

The availability of complementary tests for the evaluation of clinical manifestations resulting from crack inhalation is needed in many situations to determine the intensity, systematization, and associated complications.

## PULMONARY FUNCTION TEST

The pulmonary function of crack users may be altered,<sup>4</sup> however, when it is evaluated by spirometry, altered FEV1 and FVC values are not observed most of the times.

The diffusion capacity of CO can be decreased or normal. This decrease appears to be related to the direct damage to the alveolocapilar membrane, in the vascular bed, and interstitial disease due to concomitant use of intravenous drugs.<sup>4</sup>

## THORAX TELERADIOGRAPHY

The thorax telerradiography can show several alterations in crack users. The main findings include pneumothorax, pneumomediastinum, pneumopericardium, hemopneumothorax, bullous emphysema, and interstitial and alveolar diffuse opacities.<sup>1</sup> Figure 1 shows heterogeneous consolidations predominating in the top and middle lung fields, on a bilateral basis, one of them projected on the right hilum simulating a lymph node mass.

## THORAX COMPUTED TOMOGRAPHY (CT)

Edema (cardiogenic or not) and alveolar hemorrhage can manifest in CT by diffuse or multifocal

pulmonary impairment, with opacities in frosted glass, consolidations, interlobular septa thickening and pleural effusion. Excavated lesions may also appear as a result of pulmonary infarcts.<sup>4</sup> Figure 2 shows sparse focal consolidations, with air bronchogram in between, observing opacities in frosted glass on the periphery of the consolidations with a small nodule in the right lung.



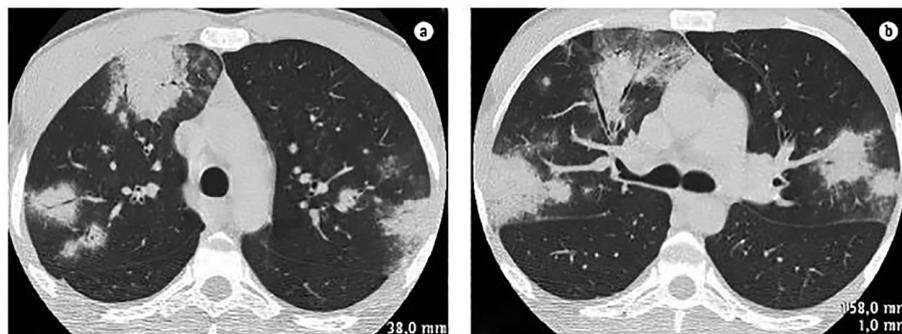
**Figure 1** - Thorax x-ray.  
Source: Mançano A, Marchiori E, Zanetti G, Escuissato DL, Duarte BC, Apolinário LA. Pulmonary complications after crack use: findings on high-resolution computed tomography of the chest. *J Bras Pneumol.* 2008; 34 (5): 323-7. Reprinted with the authors' permission.

## SCINTIGRAPHY

There have been reported cases in which disruptions in the alveolocapilar membrane integrity determines alterations in scintigraphy.<sup>4,7</sup> The pulmonary epithelium lesion seems to be consequent to increase in pulmonary permeability by the direct action of crack vapors and release of inflammatory mediators from effector cells or structures exposed to the smoke.

## BRONCHOALVEOLAR LAVAGE AND SPUTUM

The analysis of bronchoalveolar lavage among cocaine smokers revealed large numbers of eosinophils, Charcot-Leyden crystals, large populations of alveolar macrophages often containing hemosiderin, and elevated protein concentrations. These results reveal the increased permeability of the alveolar epithelium and the likely interstitial alveolar inflammatory response triggered by the pulmonary aggression associated with drug use.<sup>4, 5, 7,9</sup>



**Figure 2** - Pulmonary complications after crack use.  
Source: Mançano A, Marchiori E, Zanetti G, Escuissato DL, Duarte BC, Apolinário LA. Pulmonary complications after crack use: findings on high-resolution computed tomography of the chest. *J Bras Pneumol.* 2008; 34 (5): 323-7. Reprinted with the authors' permission.

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