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The Pulmonary Complications of Crack Cocaine*

A Comprehensive Review

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ARDS=adult respiratory distress syndrome; BOOP=bronchiolitis obliterans and organizing pneumonia; MDR=multiple drug resistant

Key words: crack cocaine; pulmonary and complications

Since the development of "crack" cocaine in the latter part of 1980, it has become the most frequently abused controlled substance in the United States. It is estimated that almost 6% of high school seniors in the United States have used cocaine, and in 27% of these, crack was the abused substance. In one study, 38% of individuals in a drug rehabilitation program admitted to the use of crack.[1]

Cocaine hydrochloride is a fine white powder derived from the leaves of the plant Erythroxylon coca and is the end product of a complex chemical process. It is used mainly by nasal inhalation or injected intravenously occasionally mixed with heroin. Cocaine hydrochloride is not heat stable and therefore cannot be smoked. When bought in the street, it is often mixed with filler substances such as lactose, caffeine, talc, or lidocaine and therefore is not pure.[2]

Cocaine hydrochloride can be prepared as a free-base, which alters the chemical properties and the method by which the drug is used. Free-base is a cocaine alkaloid that melts at 98°C, vaporizes at higher temperatures, and is heat stable. These properties allow it to be smoked. Most users prepare free-base from a baking soda and cocaine hydrochloride mixture that is boiled in water and cooled. The precipitate is either filtered or extracted by adding a solvent such as ether or alcohol. Cocaine remains dissolved in the solvent. The solvent mixture can be evaporated leaving a residue of relatively pure cocaine crystals. This crystalline precipitate is called "rock" cocaine because of its appearance or "crack" cocaine because of the crackling sound made by the crystals when heated. However, in some cases, not all the ether is evaporated, which puts cocaine smokers at high risk for burns of the upper airways. Different methods exist for smoking cocaine. The most common involve glass or regular pipe, or mixing cocaine with tobacco or marijuana in cigarette form.[2-4]

Cocaine when smoked is readily absorbed through mucous membranes and reaches the cerebral circulation within 6 to 8 s. By comparison, the intravenous route requires approximately 16 to 20 s while nasal insufflation requires 3 to 5 min. The half-life of cocaine in the blood ranges between 60 to 90 min depending on plasma cholinesterase levels.[2,5] The euphoric effect of cocaine is almost instantaneous and the ease with which it is achieved has made crack cocaine the most desired and abused substance. Cocaine either in the form of crack or free-base is most frequently associated with pulmonary complications.[6]

Pharmacologic Action of Cocaine

Cocaine is a local anesthetic, has sympathomimetic properties, and is a strong stimulant of the central nervous system. The anesthetic effect is achieved by blocking conduction of the nerve impulses via inhibition of sodium channels in the neuronal cells. In contrast to the effects of cocaine on peripheral nerves, its systemic effects on the nervous system are probably mediated by blocking the presynaptic reuptake of the neurotransmitters norepinephrine and dopamine. This produces an accumulation of neurotransmitter at the postsynaptic receptor sites. Activation of the sympathetic nervous system by this mechanism produces tachycardia, hypertension, vasoconstriction, agitation, mydriasis, hyperthermia, and predisposes to arrhythmias. Tachyphylaxis often develops, requiring higher doses of cocaine to achieve the same euphoric effect. It is with these higher doses that medical complications usually arise. Crack cocaine use has become so prevalent that it is often overlooked by the patient in the process of obtaining a medical history. When asked about drug abuse, many patients neglect to mention the smoking of a drug, considering only intravenous or nasal use to represent abuse. This is analogous to the taking of a history from a patient with alcoholic cirrhosis who may consider beer as harmless. Table 1 outlines the various pulmonary complications of smoked cocaine.

Acute Respiratory Symptoms

The lungs are the principal organs exposed to the combustion products of crack cocaine. Acute respiratory symptoms usually develop within several

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Table 1—*Pulmonary Complications of Smoked Cocaine*

1. Acute respiratory symptoms
2. Exacerbation of asthma
3. Thermal airway injury
4. Deterioration in lung function
5. Pneumothorax and pneumomediastinum
6. Bronchiolitis obliterans with organizing pneumonia
7. Pulmonary hemorrhage
8. Noncardiogenic pulmonary edema
9. Pulmonary infiltrates with eosinophilia/interstitial pneumonitis
10. Pulmonary vascular disease/pulmonary infarction

hours of use but in some cases symptoms may develop within minutes.[7] The acute respiratory complaints include the following: (1) cough with carbonaceous sputum production; (2) chest pain with or without shortness of breath; (3) hemoptysis; and (4) exacerbation of asthma. These symptoms are more common with active cocaine abuse.

The prevalence of these symptoms varies depending on the study (Table 2). One of the first studies reporting on the prevalence of the acute respiratory symptoms from cocaine smoking was that by Delbono et al.[8] Cough with sputum production was present in 61% of subjects who were smoking cocaine, while wheezing and dyspnea were present in 50% and 44%, respectively. Itkonen et al[9] described 12 of 19 subjects (63%), all of whom were habitual cocaine smokers (most of whom also smoked cigarettes) and complained of respiratory tract symptoms. Ten individuals (58%) complained of cough, and an additional ten people noted shortness of breath. The study by Tashkin et al[10] evaluated the prevalence of acute respiratory symptoms in a large sample of habitual smokers of crack cocaine with other smoked substances such as tobacco and marijuana. More than 40% of cocaine users reported having a cough productive of black sputum and 39% reported chest pain within 12 h of smoking cocaine. Although of high prevalence, these symptoms were experienced infrequently by most of the subjects. In the study by Suhl and Gorelick[11] of 23 subjects who were habitual cocaine smokers, about 50% reported one or more respiratory symptoms during the period of cocaine use. While the mechanism inducing the cough is not clear, a possible explanation is that noxious components within the inhaled smoke irritate the airway epithelium provoking cough. The black sputum is characteristic of crack cocaine smoking and is attributed to inhalation of carbonaceous residue from butane- or alcohol-soaked cotton torches used to ignite the cocaine.[12]

Chest pain was often reported by the subjects usually within 1 h of smoking. When experienced, it was usually worse on deep inspiration. The mechanism of the chest pain may represent a local sensory response to acute airway irritation from the high concentration of inhaled cocaine itself or from the products of combustion of crack.[10] It is conceivable that both symptoms of chest pain and carbonaceous sputum are related to the technique used to ignite crack cocaine. Butane is one of the methods used to ignite crack cocaine, and it is possible that the butane or its combustion products may contribute to the acute bronchial irritation when smoking cocaine. Other causes of chest pain that need to be excluded in subjects who smoke crack cocaine include acute myocardial ischemia and infarction, pneumothorax, and pneumomediastinum.[5]

Hemoptysis was reported in 6 to 26% of crack users.[10,11] Bleeding may be a result of rupture of bronchial or tracheal submucosal blood vessels or originate from the alveolar-capillary membrane. With regard to the effect of cocaine on chronic respiratory symptoms, the study by Tashkin et al[10] demonstrated that heavy use of cocaine is not associated with an increase in prevalence of symptoms such as chronic cough, chest pain, dyspnea, sputum production, or wheezing.

Effects of Crack Cocaine on Pulmonary Function

The data regarding the effect of free-base cocaine on pulmonary function as measured by standard pulmonary function tests are variable. The differences in the results of the various studies may be attributed to several factors: (1) sample size—in some of the studies, the sample size was very small; (2) potentially confounding influences of other smoked substances that often were not taken in consideration; (3) inadequate or absence of a control group; and (4) amount and purity of free-base cocaine smoked as well as the pattern of smoking, depth of inhalation, number of puffs taken, and the device used to smoke the cocaine are all difficult to control. Spirometry

Table 2—*Prevalence (%) of Acute Respiratory Symptoms in Free-base Cocaine Smokers*

Source	No. of Subjects	Cough, %	Sputum, %	Wheezing, %	Dyspnea, %	Hemoptysis, %	Other
Delbono et al[8]	36	61	61	50	44	—	—
Itkonen et al[9]	19	58	—	—	58	—	—
Tashkin et al[10]	202	43	13	<5	8	6	38% chest pain
Suhl and Gorelick[11]	23	26	21	32	21	26	—

seems not to be affected by cocaine smoking when results were controlled for tobacco and marijuana.

The main controversy focuses on the effect of free-base cocaine on the diffusing capacity. Weiss et al[13] initially described a decrease in diffusing capacity in two subjects who were free-base cocaine users. Both had a history of intravenous drug abuse and one also had history of septic emboli to the lungs. Although the authors attributed the decrease in diffusing capacity to direct damage of crack cocaine on the pulmonary vasculature, it is not possible to estimate the contribution of the intravenous drug abuse to the decrease in diffusing capacity. Tashkin et al[14] found no significant change in the diffusing capacity when they evaluated the relationship between free-base cocaine use and chronic respiratory symptoms and lung dysfunction in a large sample of habitual smokers of marijuana with or without tobacco who denied intravenous drug abuse. Unlike previous studies, this population was controlled for the possible confounding influences of marijuana and tobacco on lung function. In a more recent investigation by Tashkin et al[10] of 202 habitual crack cocaine users and 99 nonusers, they found a mild but statistically significant impairment in diffusing capacity after controlling for the use of other substances. One-third of crack users had a reduction in diffusing capacity. The mean diffusing capacity in this group was 79% of predicted, compared with 90% of predicted in the nonusers.

The variable results obtained in the two studies published by Tashkin et al[10,14] were attributed to differences in frequency, intensity, and duration of cocaine smoking. The smaller size of the population in the first group may also have contributed to the inconsistencies in the results. Two other studies[11,15] on habitual cocaine smokers failed to show a decrease in diffusing capacity. Table 3 lists the studies that evaluated the effect of cocaine smoking on the diffusing capacity. A decrease in diffusing capacity usually implies damage to the alveolar-capillary membrane. The mechanism of cocaine-related decrease in diffusing capacity is not clear, but several theories have been proposed, including the following: (1) direct damage to the alveolar-capillary membrane; (2) damage to the pulmonary vascular bed; and (3) interstitial disease due to concurrent intravenous drug abuse. Two other confounding issues pertinent to the change in diffusing capacity include the presence of anemia, which is known to produce a decrease in the diffusing capacity, and alveolar hemorrhage, which causes an increase in diffusing capacity. Anemia and occult alveolar hemorrhage are common in subjects who abuse crack cocaine. None of the studies above took the patients' hemoglobin levels into consideration in the calculation of the diffusing capacity.

Table 3—*The Effect of Crack Cocaine on the Diffusing Capacity*

Source	Diffusing Capacity (No. of Patients)
Weiss et al[13]	Decrease (2/2)
Itkonen et al[9]	Decrease (10/19)
Tashkin et al[14]	Normal (84)
Dean et al[15]	Normal (26)
Suhl and Gorelick[11]	Normal (23)
Tashkin et al[10]	Minimal decrease (202)

The findings of acute noncardiogenic edema, alveolar hemorrhage, and pathologic evidence of acute lung injury in patients who died of acute crack cocaine overdose support the hypothesis that cocaine causes damage to the alveolar-capillary membrane. Additionally, Susskind et al[16] showed increased lung permeability following long-term use of crack cocaine in seven subjects with the use of labeled technetium-99m-diethylenetriamine pentaacetic acid.

Cocaine has been shown to injure the pulmonary vasculature. Autopsy studies of 20 subjects who died of cocaine overdose showed pulmonary artery medial hypertrophy in 4 of 20 subjects (20%).[17] The medial hypertrophy involved either small- or medium-sized pulmonary arteries. Other nonspecific findings included pulmonary edema, congestion, and hemorrhage. The major parenchymal abnormality was numerous hemosiderin-laden macrophages in 7 of 20 (35%) subjects. Direct noxious effect of crack cocaine leading to cell damage or the vasoconstrictor effect of cocaine on the pulmonary vasculature progressing to medial hypertrophy and ultimately to pulmonary hypertension appear to be responsible mechanisms.

Airway Injury

Thermal airway injury resulting in severe reactive airway disease and tracheal stenosis has been reported.[18] The thermal injury to the tracheobronchial tree may be attributed to either inhalation injury from chemical byproducts carried in the smoke or to intratracheal ignition of the highly volatile ether residue used in the processing of the free-base form of cocaine. A highly publicized case involving a show business personality who suffered severe facial and body burns has served as a warning as to the dangers of the free-base form of cocaine.

Asthma

Acute exacerbations of asthma have been reported by several authors.[19,20] Rebhun[19] reported three cases in which crack cocaine seemed to cause exacerbation of asthma. One of the patients had history of childhood asthma and hayfever. He experienced cocaine-induced rhinitis with nasal itching when snorting

cocaine and developed cough and shortness of breath without wheezing, which were relieved with bronchodilator treatment. The other two patients developed bronchospasm, the first coincident with the inhalation of crack cocaine and the other 2 months after she had stopped smoking cocaine.

Rubin and Neugarten[20] described six patients who were hospitalized with acute exacerbations of asthma provoked by the inhalation of cocaine. All had a history of asthma and most were tobacco smokers. All the patients developed severe bronchospasm during or immediately after smoking cocaine. Three patients progressed to respiratory failure requiring ventilatory support. Wheezing was the predominant physical finding and all the patients responded to standard bronchodilator therapy and steroids. Bronchospasm may be a result of inflammation of the respiratory epithelium by either cocaine or adulterants.

Eosinophilic Lung Disease/Interstitial Pneumonitis

Pulmonary eosinophilia has been reported on several occasions after inhalation of cocaine.[21-24] Peripheral eosinophilia, when present, ranged from 13 to 20%. Symptoms of fever, cough, wheezing, hypoxemia, and peripheral lung infiltrates with significant blood eosinophilia and elevated IgE level on three separate occasions developed in one patient. Each episode occurred after the inhalation of cocaine. Bronchial lavage revealed an elevated eosinophil count. In another case with a similar clinical presentation, a transbronchial biopsy specimen revealed bronchial inflammation with eosinophilia. In all cases, there was prompt response to steroid therapy.

There are several isolated reports of an interstitial pneumonitis associated with smoking crack. In one case, a 33-year-old woman progressed to respiratory failure and death over 20 months.[25] Lung biopsy specimen revealed a histiocytic interstitial infiltrate with polarized foreign material later identified as crystalline silica. It is conceivable that the cocaine used by the subject was mixed with silica producing a pneumoconiosis. High-dose steroids and cyclophosphamide (Cytoxan) were unsuccessful in reversing the process. Celluose-induced granulomas in a cocaine sniffer have been reported to produce pulmonary damage in the crack user.[26]

BOOP: Bronchiolitis Obliterans and Organizing Pneumonia

Respiratory failure with bronchiolitis obliterans and organizing pneumonia (BOOP) documented by open lung biopsy specimen[27] was reported in a 32-year-old male crack user who presented with a 10-day history of nonproductive cough, fever, and dyspnea unresponsive to antibiotic and antitussive therapy. Symptoms improved rapidly after treatment with corticosteroids. The decrease in diffusing capacity and the widened alveolar-arterial oxygen pressure difference ($P[A\text{-}a]O_2$) gradient improved with treatment, but an obstructive ventilatory defect persisted. The absence of any identifiable infectious cause or known inhalational exposure makes this a diagnosis of exclusion. Another patient with BOOP due to smoking crack cocaine was seen at our institution. The patient was a 30-year-old woman who was brought to the emergency department because of agitation and dyspnea following the use of crack cocaine. She was 33 weeks pregnant and had no significant medical history. Approximately 1 week prior to hospital admission, she developed a dry cough and dyspnea. An arterial blood gas determination on room air revealed a PaO_2 of 41 mm Hg that

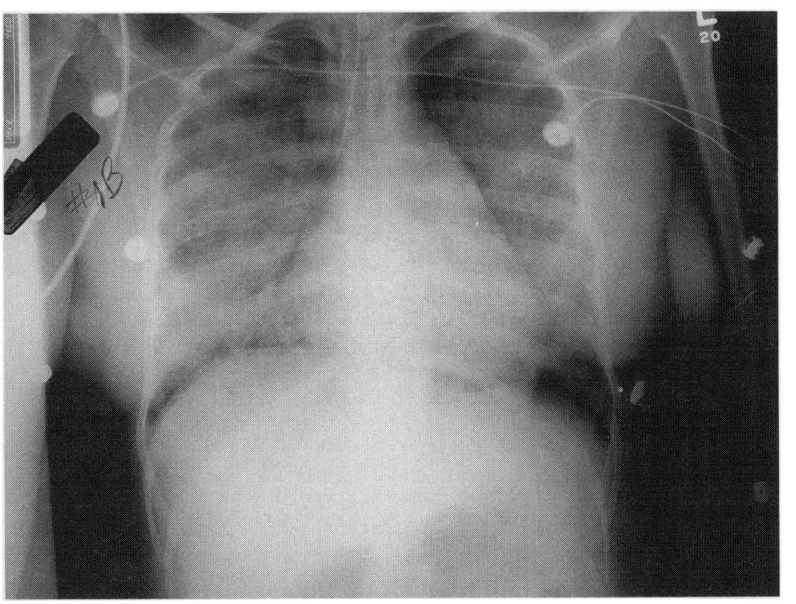

Figure 1. Hospital admission chest radiograph showing diffuse alveolar infiltrates.

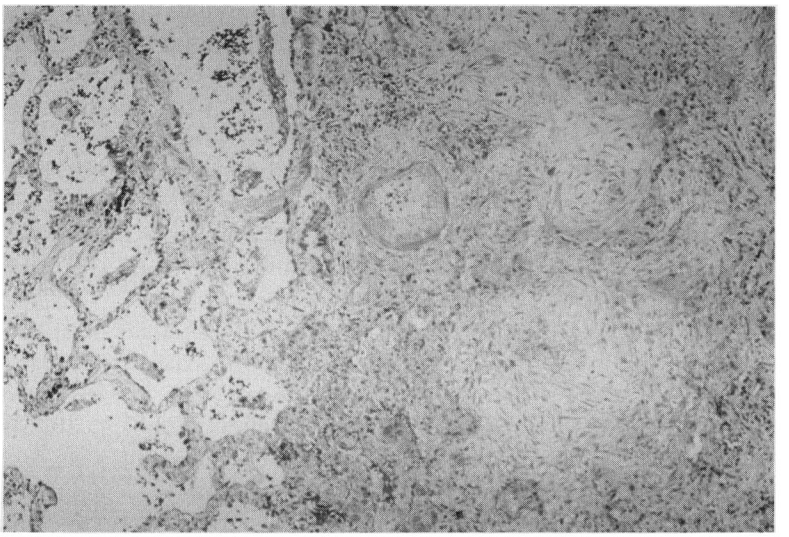

FIGURE 2. Open lung biopsy specimen showing features of BOOP (see text for details).

increased to 141 mm Hg on 100% oxygen. The chest radiograph showed diffuse alveolar infiltrates (Fig 1). Labor was induced with delivery of a normal infant. The patient was intubated for hypoxic respiratory failure and a bronchoalveolar lavage was performed that was negative for *Pneumocystis carinii* pneumonia, acid-fast bacilli, bacteria, fungi, and viral inclusion bodies. The HIV test was negative. An open lung biopsy specimen revealed areas with serpiginous fibroblastic plugs occupying the terminal bronchioles and alveolar spaces. The interstitium was thickened and infiltrated with mononuclear cells. Foci with collections of cholesterol clefts, foamy macrophages, and multinucleated giant cells were noted (Fig 2). These histopathologic findings were consistent with the diagnosis of BOOP. Despite high-dose corticosteroid therapy adult respiratory distress syndrome (ARDS) ensued and the patient died on the 48th hospital day. Postmortem findings included diffuse bilateral consolidation and fibrosis. We believe that crack cocaine was the cause of BOOP in our patient. Unlike the case reported by Patel et al,[27] our patient progressed to develop severe ARDS with no response to systemic corticosteroid therapy.

PNEUMOTHORAX, PNEUMOMEDIASTINUM, AND PNEUMOPERICARDIUM

Pneumothorax, pneumomediastinum, and pneumopericardium have been reported in subjects smoking crack cocaine.[28-35] Subjects usually present with chest pain or dyspnea. The radiographic diagnosis is enhanced by observing a retrosternal gas collection on the lateral radiograph, lateral displacement of the mediastinal pleura, and the presence of the continuous diaphragm sign (air outlining the diaphragm).[28] Of 71 crack smokers who presented to the emergency department with chest pain, 2 had a pneumomediastinum, 1 had a pneumothorax, and 1 had a hemopneumothorax.[36] The mechanism for this barotrauma seems to be related to the increase in the intra-alveolar pressure caused either by (1) deep inhalation followed by a Valsalva maneuver (often performed to increase the effect of the smoked cocaine), or (2) severe cough triggered by the cocaine.[29] This leads to alveolar rupture with air dissecting through the peribronchiolar connective tissue into the mediastinum, pericardium, pleural space, or subcutaneous soft tissues.

The differential diagnosis includes esophageal perforation that can be ruled out by a barium swallow, infection, and tumor. Rarely is there any evidence of tension. The management is strictly supportive with spontaneous resolution occurring in most patients within 48 to 72 h. The administration of high oxygen concentrations may enhance resolution. Hospitalization is recommended in all patients for observation to exclude the development of a pneumothorax. In those in whom the pneumothorax is enlarging or in whom tension develops, a chest tube is indicated. Patients should be duly warned that repeated use of crack cocaine may produce a recurrence.

PULMONARY EDEMA

Pulmonary edema as a complication of intravenous use of cocaine was first reported by Allred and Ewer.[37] The patient presented with hypoxemia, respiratory failure, and hypotension and later died. since then, there have been several reports of patients presenting with pulmonary edema after smoking cocaine.[24,38-41] Chest radiographs usually show bilateral perihilar, interstitial, and alveolar infiltrates that are often symmetrical. Symptoms resolve within 24 to 72 h. The pathogenesis of free-base cocaine-induced pulmonary edema is unclear. In part this is due to the lack of hemodynamic data that would distinguish between cardiogenic and noncardiogenic mechanisms. Cucco et al[39] suggested damage to the pulmonary capillary endothelium based on a four-

fold elevation in protein concentration seen in the bronchoalveolar lavage fluid recovered from their patient. Another possible mechanism includes transient left ventricular dysfunction, either on the basis of ischemia or severe peripheral vasoconstriction due to the intense activation of the sympathetic nervous system. This hypothesis is supported by the study published by Lang and Maron[42] that showed that mongrel dogs given cocaine intravenously developed sustained hypertension. The left ventricular end-diastolic, left atrial, pulmonary artery, and central venous pressures were all elevated during the hypertensive phase. Significant decreases in cardiac output and stroke volume were observed. Increased lung water was measured in all animals in which pulmonary artery and left atrial pressures exceeded 30 mm Hg. By administering a β-blocker such as propranolol to mice either before or after intravenous cocaine, Robin et al[43] were able to reduce mortality to 18%. Interestingly, there was no decrease in the amount of lung water present. The exact mechanism by which β-blockade may enhance survival is not clear.[43] It is clear, however, that isotopic assessment of lung water in crack users shows a permeability defect, suggesting damage to the alveolar epithelium.[16]

The management of pulmonary edema, which can be severe enough to satisfy criteria for the definition of ARDS, is supportive. Diuretics, oxygen administration, and intubation with mechanical ventilation with or without positive end-expiratory pressure have all been used effectively. Improvement is usually rapid, with most patients demonstrating radiographic resolution in 3 to 5 days. In almost all cases there is a strong temporal relationship between the amount of crack inhaled (usually 1 g or more) and the onset of symptoms (usually within 1 to 2 h of smoking). Dyspnea was a universal feature. Often bronchoscopy with bronchoalveolar lavage, and occasionally transbronchial biopsy, was performed to exclude HIV-related infections such as *Pneumocystis carinii*.

Pulmonary Hemorrhage/Infarction

Diffuse alveolar hemorrhage associated with dyspnea and hemoptysis is a common manifestation of cocaine abuse, but not emphasized in the literature.[44,45] In the case presented by Godwin et al,[45] the patient had several episodes of hemoptysis, all of which were temporally related to smoking free-base cocaine. On one occasion, massive hemoptysis was noted. Lobectomy was performed, revealing intrapulmonary hemorrhage and hemosiderin-laden macrophages, but immunologic studies were negative. There was no concomitant renal disease. The chest radiograph returned to normal at 1 week.[45] Occult lung hemorrhage from cocaine use is more common and was found at autopsy in about 30% of subjects who died suddenly from cocaine overdose.[17]

Occasionally cocaine-induced rhabdomyolysis can be associated with chest pain and hemoptysis mimicking a pulmonary embolus.[46] The differential diagnosis also includes myocardial infarction and acute aortic dissection. The measurement of the serum creatine phosphokinase level as well as the presence of urinary myoglobin will establish the diagnosis of rhabdomyolysis, now considered one of the most common manifestations of cocaine abuse.[47,48] A lung scan and noninvasive studies of the venous system can exclude venous thromboembolic disease. In one series by Forrester et al,[24] 75% of the patients had evidence of hemoptysis clinically as well as pathologically. It is clear that a true crack lung syndrome exists that may include any of a combination of features: pulmonary hemorrhage, chest pain, pulmonary edema, and an interstitial lung process.[21,24] Possible mechanisms include the following: (1) vasoconstriction of the pulmonary vascular bed that may result in anoxic epithelial or endothelial cell damage producing alveolar hemorrhage and edema; (2) direct toxic effect of the inhaled substances on the alveolar epithelium leading to injury; and (3) possibly cocaine-induced thrombocytopenia.[49] One of the more common causes of hemoptysis in the north Philadelphia drug scene appears to be the use of crack cocaine. Almost all patients have unremarkable chest radiographs with diffuse blood staining of the airways demonstrated at the time of bronchoscopy. There appear to be no serious sequelae.

Management is supportive, with bronchoscopic evaluation, supplemental oxygen, and observation being the mainstays of therapy. Since HIV-related diseases are closely associated with cocaine abuse, we recommend fiberoptic bronchoscopy and lavage in all HIV-positive patients presenting with hemoptysis if the infiltrates do not clear in 4 to 5 days. Table 4 summarizes the results of bronchoalveolar lavage in crack smokers. There is no evidence to support the use of corticosteroids in the treatment of these patients. Rarely pulmonary infarction can occur as the result of crack cocaine abuse. In the only reported case (to our knowledge), Delaney and Hoffman[50] described a 23-year-old with hemoptysis, an abnormal angiogram, and a bloody effusion culminating in thoracotomy confirming a significant pulmonary in-

Table 4—*Bronchoalveolar Lavage Findings in Crack Cocaine Users*

1. Eosinophils[21]
2. Hemosiderin-laden macrophages[17]
3. Increased protein concentration[39]
4. Carbonaceous material[12]

farct. Cocaine may induce a hypercoagulable state by enhancing platelet aggregation as well as causing a transient deficiency of protein C and S.

HIV-Related Infections

Although it is the intravenous cocaine user who will be at significant risk for HIV-related infections and therefore AIDS, it must be kept in mind that a significant number of crack users have been intravenous drug addicts as well. In addition, the exchange of sex for drugs, so common in the crack user, is another major risk factor for the development of AIDS.[51,52] Therefore, all crack abusers seeking medical attention should be offered HIV testing. It is already apparent that tuberculosis is prevelant in almost epidemic proportions in crack houses located in the major US cities.[53] Given the emerging problems with multiple drug resistant (MDR) organisms in these cities, it will be only a matter of time before we begin to see MDR tuberculosis as a major health problem in the crack user. The close crowding, poor nutrition, erratic drug compliance, and shared inhalational paraphernalia found in crack houses all serve to spread this disease that we once almost conquered.[54] The current recommendations for the treatment of MDR tuberculosis is a five-drug regimen.[55]

In the treatment of any patient who is admitted to the hospital with a pulmonary problem related to the abuse of crack cocaine, it is well to remember that HIV-related problems may coexist. Bronchoalveolar lavage has proved to be a useful technique both in diagnosing the most common infections observed in HIV-infected patients as well as evaluating the pulmonary toxicity of crack cocaine.

Conclusions

Crack cocaine has become a major agent of drug abuse in the United States. Along with its use is the recognition of significant pulmonary toxic reactions. The pharmacologic action of the drug enhanced by rapid absorption via the smoking route is responsible for many of the reported complications. Often the history of abuse may be overlooked or neglected by the patient in view of the ubiquitous nature and availability of the drug. It is crucial to recognize that although the intravenous and nasal forms of the drug have usually been associated with most toxic reactions, crack cocaine is becoming the major offender, replacing the other forms of the drug in many communities. Treatment in most cases is supportive. There is need to educate users and potential users about the serious and potentially fatal effects of crack cocaine.

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