

Short reports

Cocaine induced eosinophilic lung disease

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Abstract

A patient developed fever, bronchoconstriction, hypoxaemia, pulmonary infiltrates, and serum and bronchoalveolar lavage fluid eosinophilia on two occasions after inhaling crack cocaine. Transbronchial biopsy specimens showed normal lung parenchyma but a dense eosinophilic infiltrate within the bronchial wall. Both episodes resolved promptly after treatment with corticosteroids. Eosinophilic lung disease may be a steroid responsive complication of crack cocaine abuse.

Recreational abuse of cocaine has increased dramatically over the past few years, largely because of the popularity of smoking the alkaloid base of cocaine known as crack or freebase. Pleuropulmonary complications of cocaine abuse have included pneumothorax, pneumomediastinum, pulmonary oedema, pulmonary haemorrhage, bronchiolitis obliterans, hypersensitivity pneumonitis, pulmonary artery medial hypertrophy, thermal injuries to the airways, and asthma.¹⁻³ We describe a patient who twice developed acute pulmonary infiltrates associated with serum and bronchoalveolar lavage fluid eosinophilia and histological evidence of eosinophilic airway disease after using freebase cocaine.

Case report

A 27 year old white man was well until November 1989, when he presented with cough, purulent sputum, fever, wheezing, and pronounced dyspnoea, all of sudden onset. He had no previous history of asthma, allergies or other respiratory problems. He had smoked 30 cigarettes a day for 14 years and had used cocaine for the preceding four years. His initial cocaine use was limited to intranasal "snorting," but he had started to use freebase cocaine frequently during the preceding month. He was treated with antibiotics in a community hospital but his condition improved only minimally over the next week before he was discharged.

Later the same day he presented to the Toronto Hospital because of persistent cough, yellow sputum, wheezing, sweats, chills, pleuritic chest pain, and malaise. Physical examination showed an ill looking man in moderate respiratory distress, with a respiratory rate of 26/min and an oral temperature of 37.6°C. There were diffuse inspiratory crackles and expiratory wheezes. The remainder of the examination showed nothing abnormal. The white blood cell count was $16.2 \times 10^9/l$ with 20% eosinophils. Arterial blood gas measurements performed with the patient breathing room air were pH 7.48; carbon dioxide tension (P_{aCO_2}) 4.3 kPa, oxygen tension (P_{aO_2}) 7.7 kPa. A chest radiograph showed a diffuse increase in interstitial markings, most pronounced in the perihilar regions. Lung function tests showed a mixed obstructive and restrictive picture with FEV₁ 1.58 (38% predicted); forced vital capacity (FVC) 2.90 (59% pred); FEV₁/FVC 54%; total lung capacity (TLC) 4.67 (75% pred); carbon monoxide transfer factor (TLC) 21.68 ml/min/mm Hg (72% pred). Bronchoscopy and bronchoalveolar lavage showed that many thin mucus plugs were obstructing the peripheral airways, and these contained a large number of eosinophils and degenerative debris. Transbronchial biopsy specimens showed normal lung parenchyma but the bronchial wall showed a prominent inflammatory cell infiltrate, composed mostly of eosinophils with some thickening of the basal lamina. Cultures and stains of bronchoalveolar lavage fluid for bacteria, fungi, mycobacteria, and *Pneumocystis carinii* were negative. The patient was treated with inhaled bronchodilators and supplemental oxygen but remained severely dyspnoeic and hypoxaemic with a persistent productive cough over the next two weeks. His eosinophil count rose to $3.7 \times 10^9/l$ and his serum IgE was over 2200 (normal <450) $\mu g/l$. A skin test for aspergillus gave no reaction and aspergillus serum precipitins were absent. Testing for the human immunodeficiency virus gave a negative result.

Treatment with prednisone, 30 mg daily, was started, producing rapid improvement in symptoms and dramatic radiographic clearing. After two and a half weeks of treatment the eosinophil count had fallen to $0.64 \times 10^9/l$ and serum IgE to 1437 $\mu g/l$, and the chest radiograph returned to normal. The patient failed to keep his follow up appointment for repeat lung function tests. One month later, shortly after resuming the use of freebase cocaine, he was readmitted to hospital with dyspnoea, productive cough, fever, and chills. His chest radiograph again showed a diffuse interstitial infiltrate. Bronchoalveolar lavage showed a high eosinophil count. He responded quickly to corticosteroid treatment and was discharged on a tapering dose of prednisone. He has not required corticosteroids for

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over a year and, despite the occasional use of small amounts of freebase cocaine, he remains well with a normal chest radiograph and normal lung function. A recent methacholine challenge test showed no bronchial hyper-reactivity.

Discussion

Our patient developed fever, dyspnoea, wheezing, pulmonary infiltrates, peripheral and bronchoalveolar eosinophilia, a high serum IgE concentration, and severe hypoxaemia on two occasions after using large amounts of freebase cocaine. These features had components of eosinophilic bronchitis,⁴ hypersensitivity pneumonitis, and acute eosinophilic pneumonia. The latter has been described as an acute idiopathic cause of respiratory failure that is responsive to steroids.^{5,6} One previous case of hypersensitivity pneumonitis induced by freebasing cocaine has been reported.⁷ In that case the bronchial wall eosinophil accumulation was not as striking as in the present patient, and recovery occurred on withdrawal of the offending drug. Our patient failed to improve appreciably after three weeks of abstinence from cocaine but recovered in dramatic fashion after starting corticosteroid treatment. Cocaine precipitated bronchoconstriction has been described in asthmatic patients,³ but our patient had no history of atopy or asthma and had a normal response to a methacholine challenge test.

The present case best fits a diagnosis of eosinophilic lung disease induced by a hypersensitivity reaction to crack cocaine. We assume that some component of inhaled

cocaine leads to IgE production in the upper respiratory tract, followed by eosinophil chemotaxis to the airways with subsequent release of mediators and tissue injury. Corticosteroid presumably reverses the injury by inhibiting eosinophil chemotaxis and adherence and mediator release.

Our patient has since used lower doses of freebase cocaine without further harmful effects. Perhaps tolerance develops with continued exposure to low doses of the offending drug.

The development of sudden hypoxaemia and bronchoconstriction, with interstitial infiltrates on the chest radiograph, should raise the possibility of eosinophilic lung disease induced by inhaled crack cocaine. Bronchoalveolar lavage may be indicated to exclude an infection and to give differential cell counts. The presence of serum or bronchoalveolar lavage fluid eosinophilia in a patient with persistent symptoms should lead to consideration of a short course of corticosteroid treatment.

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