Respiratory Medicine Case Reports 25 (2018) 22–24

Contents lists available at ScienceDirect

Respiratory Medicine Case Reports

journal homepage: www.elsevier.com/locate/rmcr

Case report

Up in smoke: An unusual case of diffuse alveolar hemorrhage from marijuana

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A B S T R A C T

Diffuse alveolar hemorrhage (DAH) can be a serious and life threating condition. Illicit substance use has been associated with DAH, with cocaine being the most widely reported. Marijuana use has been associated with pulmonary complications in the form of pneumomediatsium, pneumothorax, bullous disease, and pulmonary aspergillosis. We present a case of diffuse alveolar hemorrhage (DAH) resulting from marijuana inhalation, a finding rarely described in the literature.

A 21-year-old male presented with several episodes of hemoptysis after drinking alcohol and smoking marijuana. He reported smoking 5–8 joints per day of marijuana (he denied use of bongs or other inhalant aids). His respiratory exam revealed bilateral fine rales. Laboratory evaluation included leukocytosis with left shift, normal platelets, coagulation profile, and a urine toxicology screen positive for tetrahydocanabinoid (THC). Chest CT revealed bilateral diffuse alveolar infiltrates suggestive of DAH. A bronchoscopy with BAL of bilateral upper lobes consistent with DAH with negative microbiologic studies, hemosiderin laden macrophages were present. Additional workup included a normal Echocardiogram, negative autoimmune serologies. His hemoptysis resolved with supportive care.

DAH is a potentially fatal disease that has been associated with illicit substance use, most commonly cocaine. Recently, reports have surfaced associating marijuana use with DAH, though these cases have all involved the use of bongs or other inhalant aids, leading to the hypothesis that combustibles and inhaled particles may be the etiologic factor. This is the second report of DAH developing after smoking only marijuana, though the etiology for the association between marijuana use and DAH remains uncertain.

1. Introduction

Diffuse alveolar hemorrhage (DAH) is a serious and life threating condition, characterized by the presence of new pulmonary infiltrates, hypoxemia and hemoptysis. It results from bleeding within the lung parenchyma from a variety of pathologies, including autoimmune process, infections, malignancy, cardiovascular disease and illicit drug use [1]. Illicit drug use can cause diverse pulmonary manifestations depending upon the specific substance used, mode of delivery, duration of use, and presence of additives [2]. Marijuana use in US has doubled in the past decade, and there are currently more than 22 million users [3]. DAH has been described as an immediate complication of inhalation drugs, particularly associated with cocaine and synthetic marijuana [4,5]. Here we describe a case of DAH following binge smoking of non-synthetic marijuana.

2. Case report

A 21-year-old male was admitted with the acute onset of hemoptysis. The night prior to admission he had smoked several (> 5) marijuana joints and subsequently developed blood streaked sputum that became bloodier over time. He presented to ER found to have in no distress but was coughing up blood. Vital signs: Blood pressure 122/84 mmHg, Pulse 92 bpm, respiratory rate 20/ minute, with an oxygen saturation of 98% in ambient air. He was alert and oriented without any evidence of oronasal bleeding. His respiratory exam revealed diffuse rhonchi, remainder of his exam was unremarkable.

Additional history was significant for 2 days of nasal congestion and headaches but no fever, chills, sweats, myalgia, sore throat, skin rashes, or joint pain/ swelling. He reported smoking 2–5 non-synthetic marijuana joints per day (though denied use of any inhalant aid, i.e. bongs), and had no significant occupational exposures or other illicit drug use.

Initial evaluation included a hemoglobin of 16.4 g/dl, leukocytosis of 30.1 k/μL (83% neutrophils, normalized within 24 hours) and

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https://doi.org/10.1016/j.rmcr.2018.05.028
Received 15 February 2018; Received in revised form 28 May 2018; Accepted 30 May 2018

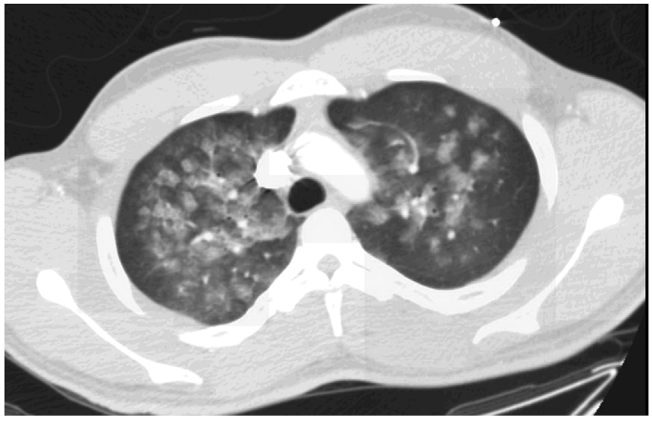

Fig. 1. CT scan of chest showing bilateral infiltrates.

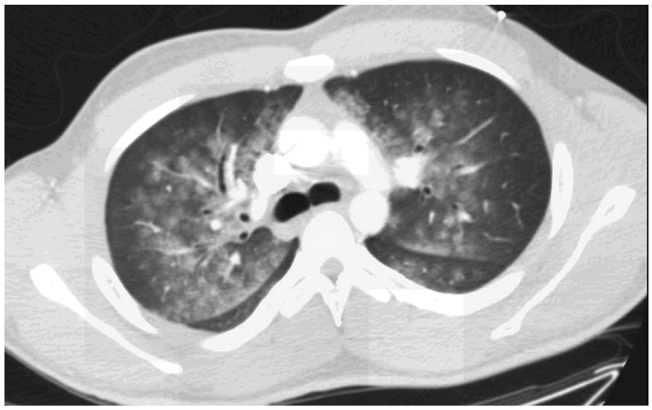

Fig. 2. CT scan of chest showing again diffuse infiltrates.

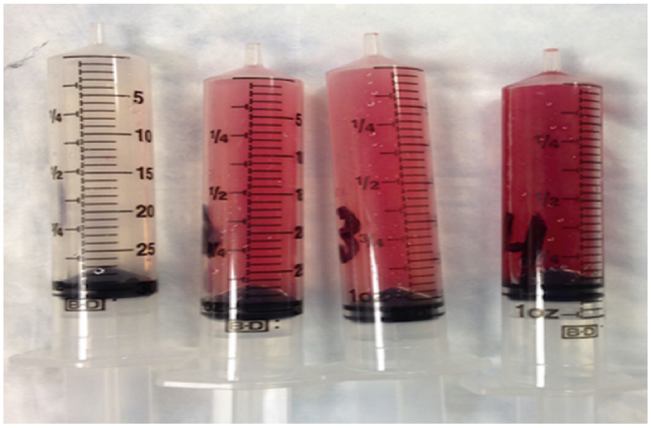

Fig. 3. Broncho-alveolar lavage of Right upper lobe (RUL) of lung.

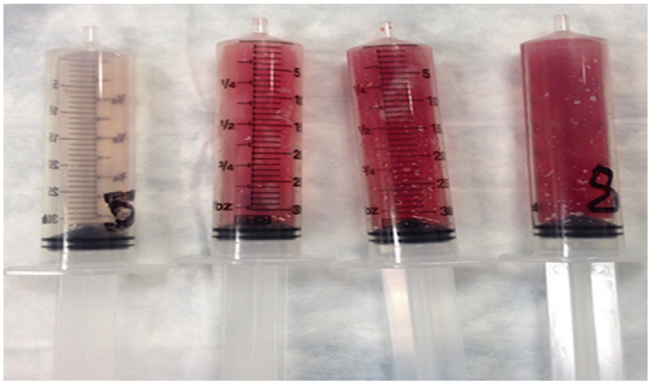

Fig. 4. Broncho-alveolar lavage of left upper lobe (LUL) of lung.

normal platelets. Serum chemistries, renal function, liver function, and coagulation studies were all normal. Urinalysis showed 0–2 RBC/ hpf and urine toxicology screen was positive only for tetrahydrocannabinol and negative for cocaine or any other illicit drugs. A Chest x-ray revealed diffuse patchy infiltrates, verified by a Chest CT showing extensive bilateral ground glass infiltrates with upper lobe predominance (Fig. 1) and (Fig. 2).

The patient was started on empiric antibiotics (piperacillin/tazobactum, vancomycin and ciprofloxacin). Infectious work up, including blood and sputum cultures, legionella urinary antigen, quantiferon TB test, and HIV antibodies were all negative. A respiratory viral panel was positive for rhinovirus. Echocardiogram was completely normal. Flexible bronchoscopy was performed and revealed blood-streaked secretions diffusely. Successive bronchoalveolar lavage (BAL) was performed in the right and left upper lobes (RUL, LUL), and both appeared consistent with DAH (Figs. 3 and 4). Cell counts were remarkable for a RBC of 44000 cells/μl with 380 white cells /μl (79% macrophages) from the RUL, and RBC of 37000 cells/μl with 363 white cells (74% macrophages) from the LUL. BAL stains and cultures were all negative, and cytology revealed only erythrophages and hemosiderinophages. An extensive rheumatological serologic evaluation including (ESR, CRP, C-ANCA, P-ANCA, Anti-GBM, rheumatoid factor, complement levels, and ENA panel) were negative. Urine cytology did not show any dysmorphic RBCs.

The patient's condition improved with resolution of the hemoptysis, and he was discharged in stable condition. Outpatient pulmonary function testing was normal and a repeat Chest imaging had normalized. He was counseled to avoid smoking marijuana and remained symptom-free at his follow up visit.

3. Discussion

DAH can present as a spectrum of nonspecific symptoms ranging from cough, dyspnea, and hemoptysis to severe hypoxemic respiratory failure requiring mechanical ventilation. Diagnosis is confirmed by bronchoscopy when successive BAL aliquots show progressively hemorrhagic return, and there are iron laden macrophages on cytology [6,7].

Inhalation injury as a cause of DAH has been associated with illicit drug use, especially cocaine, amphetamine and synthetic cannabinoids [8]. Repeated deep inhalation followed by Valsalva maneuver may increase intra alveolar pressure resulting in injury to the alveolar-capillary membrane leading to hemoptysis, pneumothorax, pneumomediastinum and non- cardiogenic pulmonary edema [9,10]. In addition, marijuana inhalation involves a 66% larger volume, 40% deeper inhalation, and 4 times longer breath hold when compared to smoking tobacco [8]. This technique could predispose to inhalation injury.

Only three cases of DAH associated with marijuana use have been described in the literature, two cases after inhalation of synthetic cannabinoid [11,12]. And one case after using a bong made of homemade plastic materials [13]. Our case is unique in that this patient was smoking non-synthetic marijuana and was doing so without the use of any inhalation aids or techniques. It's possible that the rhinovirus URI may have predisposed him to airway inflammation and then the significant marijuana inhalation resulted in inhalational injury. An extensive work-up for other causes of his DAH was unrevealing and his symptoms resolved without treatment. It's worth noting that rhinovirus has not been associated with DAH.

This case highlights a potential new complication from marijuana inhalation that should be considered in the differential diagnosis of patients presenting with DAH. The mechanism by which marijuana

might lead to DAH warrants further investigation.

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