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Review Article

Diffuse Alveolar Hemorrhage Due to Synthetic Cannabinoid Inhalation

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Introduction

Synthetic Cannabinoid Receptor Agonists (SCRAs, aka "K2," "Spice," and other street names) are an increasingly popular drug of abuse and are difficult to regulate as manufacturers alter formulations in an attempt to stay one step ahead of law enforcement [1]. Due to variable chemical compositions and more potent agonism of endogenous cannabinoid receptors than naturally occurring Δ (9)-tetrahydrocannabinol, clinical presentation is unpredictable and often far more severe than that of cannabis intoxication [2]. We report the case of a 59-year-old man with diffuse, bilateral pulmonary infiltrates after SCRA inhalation.

Case Presentation

A 59-year-old man with a history of cocaine, alcohol, and K2 abuse, Hepatitis C, and schizophrenia was admitted to the Medical Intensive Care Unit (MICU) after a witnessed, generalized tonic-clonic seizure in the waiting room of a psychiatry clinic. The patient admitted to using K2 at least three times in the days prior.

Initial vital signs in the emergency department were notable for a pulse oximetry (SpO2) reading of 86% on a non-rebreather mask. High-Flow Nasal Cannula (HFNC) was applied at 40Lpm, FiO2 of 80% and the SpO2 increased to 94%. Initial venous blood gas revealed lactate of 21 mmol/L, pH 6.9, pCO2 57mmHg, and HCO3 of 10 mmol/L, which later normalized and was attributed to the seizure. The initial arterial pO2 was 50mmHg. Basic metabolic panel was unremarkable. A hepatic panel was notable for elevated alkaline phosphatase and elevated trasaminases. Complete blood count demonstrated white blood cell count of 17.4/μL with 63% lymphocytes, hemoglobin 15.3 g/dL and a normal platelet level. Coagulation panel was within normal limits. Serum ethanol level was undetectable and 5-panel urine drug screen was negative. A chest X-ray showed diffuse, bilateral pulmonary parenchymal opacities (Figure 1). Shortly after admission to the MICU the patient had a witnessed episode of small-volume hemoptysis.

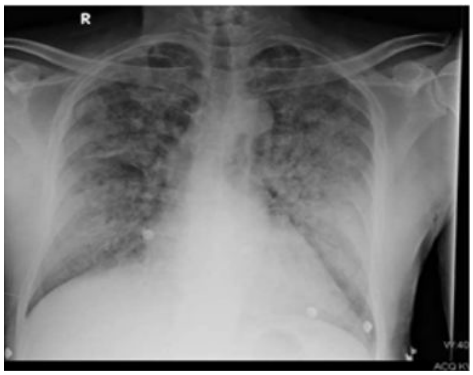

Figure 1: Chest X-ray on Admission with Diffuse Pulmonary Infiltrates.

Collateral information was obtained from an outside hospital where the patient had been admitted previously; the patient had had a seizure in the setting of K2 intoxication and then hemoptysis with diffuse airspace opacities on chest imaging (Figure 2). At the time of the prior seizure the patient had a cardiac arrest requiring advanced cardiac life support and endotracheal intubation. On the current presentation, the patient's hypoxia stabilized with the use of HFNC and he had no further episodes of hemoptysis so bronchoscopy was deferred.

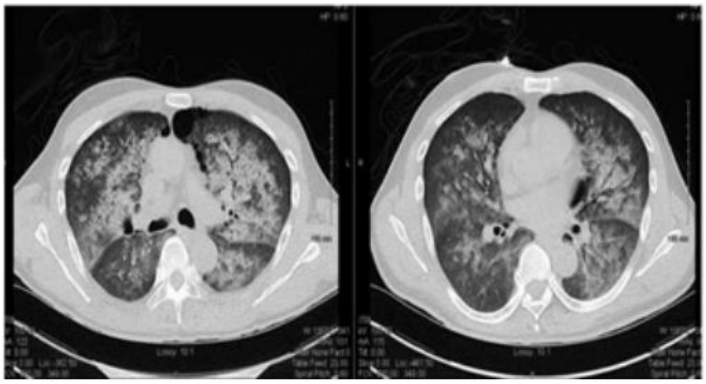

Figure 2: Chest CT from Outside Hospital Two Months Prior to Admission with Bilateral Airspace Consolidations and Ground Glass Opacities.

Given the clinical and radiographic findings, the patient was started empirically on methylprednisolone for presumed drug-induced Diffuse Alveolar Hemorrhage (DAH). By hospital day two, the patient was weaned from HFNC to low-flow nasal cannula and was able to maintain appropriate oxygen saturation. Further work-up revealed negative antinuclear antibodies, normal erythrocyte sedimentation rate and complement levels, negative anti-glomerular basement membrane and anti-neutrophil cytoplasmic antibodies. He was transferred out of the MICU to the general medicine floor and subsequently to the psychiatric service for inpatient treatment of schizophrenia and substance abuse. On a presentation to the ER some months later for an unrelated chief complaint, the patient reported continued abstention from K2 and a chest X-ray showed resolution of the diffuse airspace disease (Figure 3).

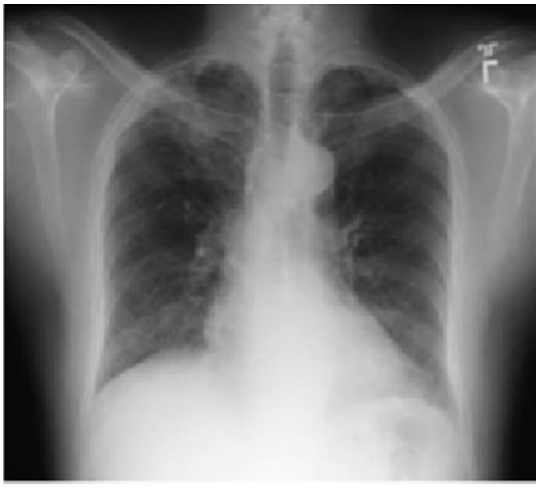

Figure 3: Chest X-ray Three Months after Admission with Resolution of Infiltrates.

Discussion

Our patient presented on two separate occasions with DAH and seizures after inhalation of a SCRA. Two prior cases of DAH associated with SCRA inhalation have been reported. The reported toxic effects of SCRAs include many central nervous system, cardiovascular, and pulmonary effects [1,2]. Some complications of SCRA abuse are thought to be due to vasospasm in the affected organs; cases of acute myocardial infarction as well as ischemic and hemorrhagic stroke have been reported in SCRA users, including younger patients without traditional cardiovascular risk factors [3,4]. Similarly, "Crack Lung" is a well-described complication of free-base cocaine inhalation with variable manifestations that include DAH; while the underlying cause is not fully understood, proposed mechanisms of alveolar damage include the high temperature of volatilized cocaine and cocaine-induced vasoconstriction in pulmonary capillary beds [5]. Notably, our patient's urine toxicology screen was negative for cocaine metabolites, thus "Crack Lung" is an unlikely cause of DAH in this case.

DAH secondary to SCRA use was first described in 2011 by Loschner, et al. in a 19-year-old man who presented with hemoptysis [6]. Their patient had bronchoscopic confirmation of DAH and reported daily K2 use. The patient improved with mechanical ventilation and empiric methylprednisolone. Similarly, Alhadi and colleagues presented a case of a previously healthy chronic K2 user with diffuse, bilateral pulmonary infiltrates [7]. Bronchoscopy with serial bronchoalveolar lavage revealed bloody, non-clearing secretions. A chronic, lymphocytic infiltrate was seen on transbronchial biopsy. The patient improved with mechanical ventilation and methylprednisolone; work-up of the alveolar hemorrhage was negative for infectious, malignant, rheumatologic or vasculitis-related etiologies. Furthermore, four SCRA compounds were detectable in the patient's blood, urine or saliva.

We were not able to confirm the presence of DAH bronchoscopically as our patient's hypoxic respiratory failure improved without the need for endotracheal intubation and bronchoscopy was deemed too high-risk. However, given the patient's clinical presentation and imaging findings, otherwise negative laboratory results, and dual temporal association with K2 use, it is most likely that this presentation represents another case of DAH secondary to inhaled SCRAs.

Conclusion

Our case highlights diffuse alveolar hemorrhage as a rare but important adverse effect of synthetic cannabinoid receptor agonist use. As in other forms of drug-induced DAH, withdrawal of the offending drug is likely the most important intervention. However, as the underlying mechanism remains unknown but may be inflammatory in nature, glucocorticoid therapy is a reasonable adjunct to supportive care for patients that present with hemoptysis and acute respiratory failure. The increasing prevalence of SCRA abuse underscores the urgent need to further characterize the cause of this potentially life-threatening complication.

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