

Emerging lethal electronic cigarette drug trends: acute respiratory distress syndrome and death from a “Zombie Apocalypse”

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Introduction:

Electronic cigarettes (e-cigs) or vaping devices were originally marketed as alternative methods for nicotine delivery instead of smoking traditional cigarettes. An estimated 1 in every 20 Americans use vaping devices, with the highest prevalence among 18–29-year-olds (20%). Injury from vaping, particularly lung injury, has gained recent national attention.¹ In addition, these devices are also used for inhalation of other substances, including flavored oils, marijuana, synthetic cannabinoids, and methamphetamines.² Recent reports describe abuse of insecticides, colloquially termed “wasping,” via e-cig inhalation resulting in a rise of vaping overdose cases. It is further reported that synthetic cannabinoids (“synthetic marijuana”, “spice”, “K2”) are sometimes supplemented with bug spray and loaded into e-cigs. Insecticides and synthetic cannabinoids are combined reportedly for a more rapid onset and an increase of the “high” from the drugs. Pyrethroids in certain insecticides induce potent sympathetic activation while strong agonism of cannabinoid receptors by synthetic cannabinoids lead to a host of physiologic, mood, and psychotic effects often producing a catatonic “zombie-like” state, with drooling and loss of motor function.³⁻⁷ Per U.S. News World Report, authorities in Indiana responded to nearly a dozen cases in a single day of “zombie-like” behavior due to the user’s inability to talk or respond.⁸ We describe the clinical course of a

patient affected by the lethal consequences of “wasping.”

Case Description:

A 29-year-old male developed severe shortness of breath, altered mental status, seizures, diaphoresis, and vomiting after inhaling “hits” of an e-cig which contained synthetic cannabinoids laced with heated and crystallized bug spray at a college party.⁹ The emergency responders recorded a fever of 104°F, pulse of 150/min, respiratory rate of 42/min, and oxygen saturation of 68% on room air. Physical exam was pertinent for diffuse rhonchi, agitation, hyperreflexia, and increased work of breathing. He was transported to an emergency department, where he was intubated, but immediately suffered a cardiac arrest with pulseless electrical activity. Spontaneous circulation returned after 13 minutes of resuscitation, but the patient remained non-responsive. His urine drug screen was positive for cannabinoid metabolites. Laboratory studies included a white blood cell count of 37/ μ L, bicarbonate of 14 mEq/L, lactate of 7 mmol/L, arterial pH of 7.05, pCO₂ of 70 mmHg, pO₂ of 103 mmHg. His imaging revealed significant right-sided and developing left-sided pulmonary infiltrate. Bronchoscopy was consistent with acute respiratory distress syndrome (ARDS) without evidence for infectious etiology. He remained unresponsive, and the brain MRI showed diffuse cerebral edema suggestive of severe anoxic brain injury. Despite

aggressive care, his clinical and mental status failed to improve, and he eventually expired.

As of February 2020, the CDC reported 2,807 cases of e-cig or vaping product-associated lung injury cases requiring hospitalization. While the presence of multiple chemicals, such as tetrahydrocannabinol, may damage the lung, vitamin E acetate is strongly related to e-cig-associated lung injury. Most patients who suffered lung injury after vaping tetrahydrocannabinol reported obtaining their vaping products from informal sources rather than commercial sources.¹ It is possible that pernicious compounds in e-cig aerosols may be more likely to be present in this setting.

In addition to the well-described characteristics of acute lung injury from vaping, synthetic cannabinoids can directly cause severe agitation, confusion, vomiting, hypertension, tachycardia, and death from cardiac dysrhythmia or ischemia, by stimulating the sympathetic nervous system via norepinephrine release and parasympathetic blockade. Additionally, myocardial oxygen demand and carboxyhemoglobin levels are increased with resultant decrease in oxygen saturation.^{4,5} Methamphetamines contribute as CNS stimulants that reverse vesicular monoamine uptake transporter-2, causing accumulation of serotonin, dopamine, and norepinephrine.¹⁰ Bug spray contains pyrethroids that prolong neuronal excitation and affect cardiac conduction via action on sodium, chloride, and calcium ion channels. GABA-gated chloride channels are also affected with higher concentrations of pyrethroids.^{6,7} Corticosteroid therapy and supportive care is often used for vaping lung injury. Therapy is aimed to decrease sympathomimetic output, reduce serotonergic tone, and treat end-organ toxicity. Medications given include GABA-A agonists, principally benzodiazepines, along with intravenous fluids.⁷ Recently, intravenous Lipid Emulsion Therapy has been successful in treating acute synthetic cannabinoid intoxication.¹¹

Conclusion:

Electronic cigarettes, originally intended to deliver vaporized nicotine, are reportedly utilized to abuse synthetic cannabinoids and insecticides. In this case report, we describe a 29-year-old man who presented with dyspnea, altered mental status and

seizures following use of an e-cigarette which was laced with synthetic cannabinoids and insecticides. The patient developed cerebral edema and ARDS and eventually expired indicating the necessity to further study patterns of abuse and pathophysiologic injury involving e-cigarettes.

Author Contributions:

C.S. and R.H. conceptualized the importance of the presented case, helped with writing the original draft, and supervised the project. P.D. helped identify valid resources and helped with writing the original draft. J.R. helped with writing the original draft. All authors helped with analysis, review, and editing.

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