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Selected Topics: Toxicology

ACETYLFENTANYL: AN EMERGING DRUG OF ABUSE

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□ **Abstract—Background: Opioid analgesics are widely used in health care, yet have significant potential for abuse. High doses are associated with potentially fatal respiratory depression, which caused 21,314 deaths in the United States in 2011. Acetylfentanyl, a synthetic opioid agonist closely related to fentanyl, recently emerged as a drug of abuse linked to numerous deaths in North America. Case Report: A 36-year-old male developed the habit of using a propylene glycol electronic cigarette filled with acetylfentanyl to aid relaxation. He purchased the drug online in a manner that appeared legal to him, which compromised his insight about the danger of the substance. He had been using the e-cigarette with increasing frequency while on medical leave, and his wife reported finding him weakly responsive on more than one occasion. At approximately 3 AM, the family activated 911 for altered mental status. His presentation included respiratory depression, pinpoint pupils, hypoxemia, and a Glasgow Coma Scale score of 6. He responded to serial doses of intravenous naloxone with improvement in his mental status and respiratory condition. Due to the need for repeated dosing, he was placed on a naloxone infusion and recovered uneventfully in intensive care. Why Should an Emergency Physician Be Aware of This?: Complications from emerging drugs of abuse, like acetylfentanyl, frequently present first to emergency departments. Prompt recognition and treatment can help avoid morbidity and mortality. Acetylfentanyl can be managed effectively with naloxone, although higher than conventional dosing may be required to achieve therapeutic effect.** © **2016 Elsevier Inc.**

□ **Keywords—opioid analgesics; drug overdose; street drugs; naloxone**

INTRODUCTION

Opium and its various preparations have been used medicinally for centuries for their analgesic properties (1). The term *opiates* refers to alkaloid chemicals that are derived from opium, such as morphine and codeine. The term *opioids* refers more broadly to drugs with agonist activity at opioid receptors, including synthetic chemicals, such as methadone and fentanyl. These drugs are widely used in health care for their analgesic effects, although they are likewise known for high abuse potential due to euphoric properties. Emergency physicians are trained to recognize and manage the opioid toxidrome, which should be considered core knowledge in emergency medicine curricula (2). Intentional or unintentional opioid overdoses commonly manifest with respiratory suppression, which can be lethal. In 2011, an estimated 678,522 emergency department (ED) visits occurred in the United States due to misuse of prescription and illicit opioids (3). The yearly death rate attributed to prescription opioid overdoses rose from 4,030 in 1999 to 16,917 in 2011; for heroin, these figures increased from 1,960 to 4,397 in 1999 and 2011, respectively (4). Fortunately, opioid overdoses can be managed effectively with naloxone, a competitive antagonist of the mu-opioid receptor. We present the case of a young man with an opioid overdose caused by acetylfentanyl, a recently emerged opioid drug of abuse.

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CASE REPORT

A 36-year-old male presented to the ED via ambulance with altered mental status. He was last seen normal at midnight that night. At approximately 3 AM, the patient's wife found that he could not be roused to stimulation and appeared to have difficulty breathing. She called the patient's brother, who activated 911. On arrival, paramedics found the patient with a Glasgow Coma Scale (GCS) score of 6 and oxygen saturation of 85%. During transport he was given 2 mg of intravenous naloxone, after which he was able to converse appropriately. Upon arrival to the ED, he had an intact airway and adequate spontaneous respirations, but his mental status had declined such that he was minimally aroused by painful stimuli. He was placed immediately into a resuscitation room, where he was found to be afebrile with a pulse of 97 beats/min, blood pressure of 173/97 mm Hg, respiratory rate of 17 breaths/min, and oxygen saturation of 97% on 2 L by nasal cannula. His blood glucose was 249 mg/dL, and his pupils were constricted to pinpoint. He was given another dose of 2 mg naloxone, with rapid improvement in his mental status. The patient then described a recent habit of smoking a propylene glycol electronic cigarette to aid relaxation. He stated that he purchased what he called "synthetic opium" legally online, which he used to fill the e-cigarette and inhale vapors—a process known as "vaping." The patient later admitted to adding the "synthetic opium" to alcoholic beverages as well. He had been on medical leave for the previous 3 days for the planned closure of a patent foramen ovale, which had been found after a transient ischemic attack 3 months earlier. With the free time during his medical leave, the patient had been using his e-cigarette frequently, and his wife found him difficult to rouse on more than one occasion. The patient denied recent trauma, fevers, anorexia, headaches, or any other symptoms. The history was obtained from the patient and his brother, and initially neither could identify the specific contents of the "synthetic opium."

The patient had no other medical history, except for the patent foramen ovale and transient ischemic attack. He took no prescription medications and denied known allergies. He smoked tobacco cigarettes daily, reported social alcohol consumption, and used an e-cigarette as noted. His physical examination showed pinpoint pupils initially, which changed to 3 mm bilaterally after naloxone administration. There was no nystagmus. His oropharynx, heart, lungs, and abdomen were normal on examination. His GCS was 15 after receiving naloxone. He had normal motor strength and sensation to light touch. Cranial nerves II to XII were intact, and no clonus was apparent. Examination of his skin showed no track marks or bruising. Laboratory evaluation showed a basic metabolic profile that was normal except for mild hyperglycemia and creatinine increased to 1.7 mg/dL from his baseline of 0.8 mg/dL. His creatinine kinase (CK) was elevated at 11,715 U/L. Venous blood gas analysis showed a pH of 7.12 and pCO_2 of 89 mm Hg. Other studies, including a complete blood count, liver function tests, urinalysis, and chest x-ray study were unremarkable. Computed tomography of the head was considered but not obtained, because the working diagnosis of an opioid toxidrome seemed clear based on his reported history and normal mental status after naloxone. Similarly, urine screening for drugs of abuse was considered but not obtained, because this was felt unlikely to alter management for the patient (5).

During his ED course, the patient's mental status began to decline again, so a third dose of 2 mg of naloxone was administered. He was aggressively hydrated with intravenous crystalloid due his elevated CK. Continuous waveform capnography was used to monitor his respiratory status, and a continuous infusion of naloxone was started at 1.5 mg/h. With these interventions, his mental status remained adequate, and results of arterial blood gas sampling improved to a pH of 7.32, pCO_2 of 55 mm Hg, and pO_2 of 84 mm Hg. He was admitted to the medical intensive care unit (ICU) in improved condition for further management of an opioid toxidrome, complicated by acute hypercarbic respiratory insufficiency and rhabdomyolysis with acute kidney injury. Upon request, the patient navigated to the website where he purchased the "synthetic opium," which showed chemical abbreviations and structures. Using this information, providers from the ICU and the regional Poison Control Center identified the substance as acetylfentanyl, an analogue of fentanyl with similar clinical effects.

DISCUSSION

Acetylfentanyl is a synthetic analogue of fentanyl that is similar to heroin in color, consistency, and pharmacologic activity (Figure 1). It acts in the human body by agonism at the mu-opioid receptor, where its activity is 15.7 times more potent than morphine and 3 times less potent than fentanyl (6,7). It was discovered in the late 1960s, about the same time that fentanyl was discovered. It remains unused in health care and, aside from rare use during the 1980s, did not establish a significant presence on the streets for decades. In 2013, the drug re-emerged after 3 kg and 12,400 pills were seized during a drug bust in Canada (8). In early 2013, fourteen deaths in Rhode Island and 50 deaths in Pennsylvania were linked to acetylfentanyl, prompting the Centers for

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