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Death following intentional ingestion of e-liquid

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Context: Electronic cigarette (e-cigarette) use is growing within the United States, resulting in both intentional and unintentional exposures to concentrated liquid nicotine or “e-liquid.” Nicotine has been culpable for severe poisoning and deaths in the past. However, sources of nicotine have traditionally been from cigarettes, cigars, or pesticides. Fatalities due to liquid nicotine are rare, and fatalities following ingestion of e-liquid are even scarcer. Case: We present a case of a 24-year-old woman who intentionally ingested up to 3000 mg of liquid nicotine intended for e-cigarette use. She was found in pulseless electrical activity and had return of spontaneous circulation (ROSC) after undergoing approximately 10 min of cardiopulmonary resuscitation with a blood pressure of 74/53 mmHg and a pulse rate of 106 beats/min. Despite aggressive supportive care, she ultimately died after she was found to have multiple acute infarcts, consistent with severe anoxic brain injury, on magnetic resonance imaging. The patient’s toxicologic testing, obtained shortly after ROSC, was notable for plasma nicotine and cotinine levels each >1000 ng/mL. Discussion: This fatality highlights the potential toxicity associated with suicidal ingestion of liquid nicotine.

Keywords Liquid nicotine; E-liquid; E-cigarettes; Public health

Introduction

Since their introduction in 2004, electronic cigarette (e-cigarette) use has increased in part due to their purported safety over conventional nicotine delivery systems such as cigarettes, cigars, and tobacco pipes. E-cigarettes vaporize liquid nicotine (“e-liquid”) instead of combusting tobacco leaves. The vaporized e-liquid is then inhaled (“vaping”). E-liquid is available in replacement vials with nicotine concentrations up to 36 mg/mL for immediate use, and base concentrations up to 100 mg/mL, which must be diluted before use. Ingestion or dermal absorption of these replacement liquid nicotine products can potentially cause toxicity.

Due to the increased prevalence of e-cigarettes, there is a growing concern for potentially toxic exposures to liquid nicotine. The American Association of Poison Control Centers’ National Poison Data System (NPDS) revealed that liquid nicotine for e-cigarettes was responsible for 1495 exposures in 2013. Of those exposures, 49 led to moderate or severe outcomes. In 2012, one death was reported to the NPDS after an intentional intravenous injection of e-liquid. Increasing unintentional exposures to e-liquid have prompted the United States (US) Food and Drug Administration (FDA) to propose authoritative oversight of e-cigarette and e-liquid sales. If such changes are instituted, product approval, addition of warning labels, and packaging requirements would be required and monitored by the FDA.

We report a fatality resulting from a suicidal ingestion of concentrated liquid nicotine intended for e-cigarette use.

Case report

A 24-year-old woman called 911 after an intentional suicidal ingestion. The time of ingestion is unknown. Upon first responder arrival, she was unresponsive and pulseless beside a suicide note, a partially ingested bottle of whiskey, and two empty 15 mL vials of concentrated liquid nicotine (100 mg/mL) (Fig. 1). Her home medications (trazodone, fluoxetine, and olanzapine) were present at the scene, but pill counts were appropriate to the dates prescribed, suggesting they were not co-ingested. First responders applied an automated external defibrillator (AED) and completed three rounds of CPR. The AED instructed, “No shock advised” between each round of CPR. Upon subsequent paramedic arrival, the patient was found to be in pulseless electrical activity (PEA). After a total of 10 min of CPR, she had return of spontaneous circulation with
a systolic blood pressure of 100 mmHg and a pulse rate of 120 beats/min. No medications were administered during CPR due to inability to obtain intravenous access. After placing an intraosseous line and administering 150 mg of succinylcholine and 5 mg of midazolam, medics performed endotracheal intubation. Prehospital glucose was not measured. During transport to the emergency department (ED), she received 200 mL of crystalloid through the intraosseous catheter.

Upon arrival to the ED 11 min later, vital signs were as follows: blood pressure, 74/53 mmHg; pulse rate, 106 beats/min; respiratory rate, 14 breaths/min via mechanical ventilation; temperature, 36.5°C; and oxygen saturation on 100% FiO₂, 97%. Her physical examination was notable for pupils fixed and dilated at 5 mm, pale and clammy skin, and incontinence of urine and stool. The only abnormality on pulmonary examination was decreased breath sounds in the left lung fields due to right mainstem intubation. This improved after retraction of the endotracheal tube to the appropriate position. No wheezes or rales were detected. Myoclonic jerks of the neck progressed to whole body myoclonus. She demonstrated slight extensor posturing to painful stimuli and was otherwise unresponsive.

Electrocardiogram demonstrated sinus tachycardia with a rate of 106 beats/min, slightly rightward axis, QRS 96 ms, and QTc of 483 ms.

Initial laboratory studies drawn 6 min after ED arrival were notable for the following values: sodium, 133 mEq/L; potassium, 4.3 mEq/L; chloride, 96 mEq/L; bicarbonate, 17 mEq/L; BUN, 11 mg/dL; creatinine, 1.02 mg/dL; and glucose, 360 mg/dL. The initial serum lactate was 13.0 mmol/L. After infusion of 2 L of crystalloid and continuous infusion of norepinephrine at 0.2 mcg/kg/min, the serum lactate drawn 50 min later improved to 4.7 mmol/L. Urine drug screening performed by immunoassay was positive for amphetamines and benzodiazepines. Comprehensive urine drug screen by gas chromatography and mass spectrometry (GMCS) was positive only for nicotine, cotinine, and the patient’s home medications. Serum ethanol, acetaminophen, and salicylate concentrations were undetectable. The patient’s plasma nicotine and cotinine concentrations, measured upon arrival to the ED, were each >1000 ng/mL (measured by liquid chromatography/tandem mass spectrometry; LC/MS/MS). Absolute quantitative concentration was unavailable as the laboratory test was performed at another institution, and further dilution was not performed.

After admission to the intensive care unit, the patient demonstrated ongoing full body myoclonic jerking. The ICU team noted an absence of corneal, gag, and cough reflexes. Despite receiving 6 mg of intravenous lorazepam, 1800 mg of phenytoin, and 1000 mg of levetiracetam, she continued to have uncontrollable limb myoclonus. A electroencephalogram (EEG) performed during continuous myoclonus showed generalized suppression, consistent with profound generalized cerebral dysfunction. No epileptiform discharges or evidence of seizure activity was noted. The intensive care team started a cisatracurium infusion in an attempt to stop myoclonus. Norepinephrine was titrated off approximately 3.5 h after admission.

Magnetic resonance imaging of the brain was performed on hospital day 2 and showed multiple acute infarcts consistent with anoxic brain injury. The patient continued to be unresponsive. Given her poor prognosis, she was transitioned to comfort care and died 3 d post-ingestion.

**Discussion**

We present a case of cardiac arrest following intentional nicotine ingestion. Other medications were present in the home and detected on a qualitative drug screen, and the nicotine level was not reported in an exact level as it exceeded the maximum cutoff for the quantitative assay. Despite these limitations, we believe that nicotine to be the causative agent in the patient’s death for the following reasons: empty e-liquid vials at the scene previously containing a lethal amount of nicotine; clinical features of the case including cardiac dysrhythmias, muscle fasciculations, and cardiovascular collapse, which are all consistent with nicotine overdose; and markedly elevated (>1000 ng/mL) quantitative nicotine and cotinine levels drawn at the time of presentation. Intentional overdose of her home psychiatric medications is a possible contributing factor in her death, although we believe her clinical
presentation to be more consistent with nicotine toxicity than poisoning from these agents.

Nicotine toxicity is not a new clinical phenomenon. The addictive stimulant has been used worldwide for hundreds of years. It is available in many different forms, including cigarettes, dried tobacco leaves, patches, and gums. It is also used outside of the U.S. as a pesticide. The recent explosion of e-cigarette popularity, however, introduces a highly concentrated, widely available source of unintentional and intentional poisoning. A recent survey showed a 132.5% increase in e-cigarette sales across all markets between 2012 and 2013.6 Concurrently, US Poison Centers reported an increase of e-cigarette exposures by 9.60 per month in 2013.4 E-cigarettes and replacement nicotine solution are responsible for over 40% of nicotine-related calls to poison centers, and they are not currently regulated by the US FDA.7 Consequently, emergency providers and poison control centers should anticipate a continued increase in cases of e-liquid exposures and poisonings.

Published fatalities from e-liquid poisoning are exceedingly rare. A recent survey of 1700 cases of e-liquid exposure reported to poison centers found only one death involving parenteral injection of nicotine solution.3,4 Two fatal ingestions of a nicotine-containing solution were reported in 2010, but these cases involved extraction of nicotine from tobacco plants.8 A recently published case describes a 34-year-old man found in cardiac arrest at home with elevated post-mortem nicotine concentrations detected in gastric contents and blood. Empty vials of e-liquid were later found in his home.9

While inhalation of a single conventional tobacco cigarette results in absorption of 0.5–3 mg of nicotine, each cigarette actually contains anywhere between 10 and 30 mg of nicotine. Cigarette smokers usually maintain serum nicotine concentrations of 30–50 ng/mL.10,11 In contrast, e-liquid can contain nicotine concentrations up to 100 mg/mL.

This case highlights the potential for lethal overdose following liquid nicotine ingestion, which is concerning due to its wide availability and increasing popularity.

Declaration of interest

The authors report no declarations of interest. The authors alone are responsible for the content and writing of the paper.

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