



Case Report

Bug off! Severe toxicity following inhalational exposure to *N, N*-diethyl-meta-toluamide (DEET)Erica M. Tavares, MD¹, Bryan S. Judge, MD¹, J.S. Jones, MD*

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ABSTRACT

N, N-diethyl-meta-toluamide (DEET) is an insect repellent currently used by millions of people since 1956. DEET has an excellent safety profile and has remarkable protection against mosquitoes, ticks and various other arthropods. Toxicity is unusual, and is generally associated with incorrect, or overuse of the product. We report a patient with severe toxicity following inhalational exposure to a “bug bomb” containing 98% DEET.

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1. Introduction

N, N-diethyl-*m*-toluamide (DEET) is an insect repellent typically formulated with aerosols, creams, liquids, lotions, pump sprays, or towellettes that can be applied to the skin [1]. It is also found in home insect foggers or “bug bombs” used to fumigate indoor living spaces. The concentration of DEET in these products ranges from 5 to 100%. In 2012, the Environmental Protection Agency required bug bombs to have improved instruction labels, with pictures to emphasize important actions users need to take, such as leaving the home for at least 2 h and ventilating the area for an additional 2 h [2]. However, exposure narratives from case reports suggested that many users did not follow or read label instructions. In a 10-state study of illnesses and injuries related to so-called total release foggers, researchers found more than 3200 incidents over roughly an eight-year period, for a rate of 27 per 10 million people per year [2]. The incidents included minor respiratory, gastrointestinal, cardiac, neurologic, and eye and skin problems. There have been very few cases of severe DEET toxicity reported. We report a case of accidental DEET toxicity by inhalation, after improper use of a “bug bomb” containing 98% DEET.

2. Case report

A 65-year-old male with a history of coronary artery disease was found unresponsive in his small mobile home. The patient was last seen normal by family members 24 h prior to presentation. In the

emergency department (ED), his vital signs were pulse 125 beats per minute, respirations 8 breaths per minute, blood pressure 102/57 mm HG, and temperature 37.2 °C. The patient demonstrated rigid, decorticate posturing, tremors, sonorous respirations, and dilated, fixed pupils. His physical examination was essentially unremarkable, and he demonstrated no stigmata of any toxidrome. He was intubated and initial pertinent laboratory studies included a serum lactate of 9.3 mmol/L and bicarbonate of 13 mmol/L. Head CT and chest radiograph were unremarkable. Initial EKG showed sinus tachycardia (heart rate, 141 beats/min) with a QTc of 434 ms, without acute ischemic changes. The patient was transferred to the ICU and within hours of admission, the patient became hypotensive, but remained tachycardic with a blood pressure of 70/41 mm Hg that was refractory to fluid resuscitation and required intravenous norepinephrine. An EEG was performed and indicated toxic-metabolic encephalopathy without seizure activity.

Initially the cause of his altered mental status was unknown, but comprehensive blood and urine drug screens returned showing only the presence of DEET. This finding correlated with further history obtained from family members, who reported that the patient had deployed a “bug bomb” in his mobile home. Found in the patient’s home was a 4-ounce can of home insect fogger containing 98% DEET. The mobile home’s windows and doors were all tightly closed when the patient was found by EMS. The first serum DEET concentration (Time 0 in Fig. 1) was obtained approximately 7.5 h after initial presentation to the hospital. Subsequent serum DEET concentrations were drawn daily until levels were undetectable which occurred by hospital day seven.

On hospital day three, an MRI of the brain showed no signs of cerebral edema or anoxic brain injury. By hospital day six, the patient was extubated, but he was still not following commands and demonstrated only minimally comprehensible speech. On hospital day eleven, he was

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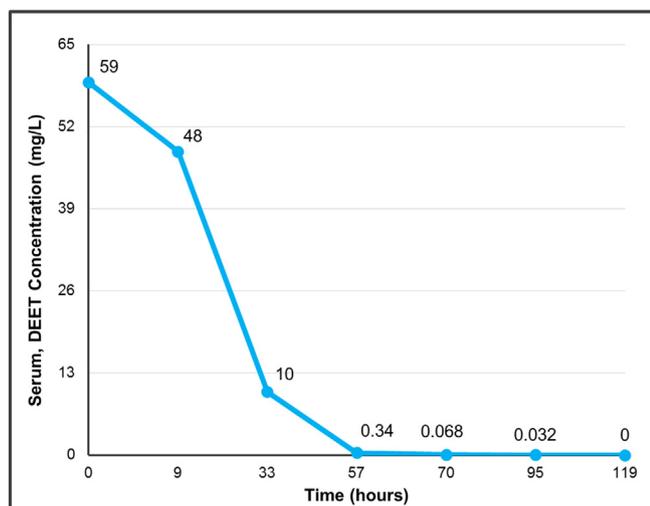


Fig. 1. Serial serum DEET concentrations in a patient with inhalation exposure to a product containing 98% DEET. Time 0 = 7.5 h after hospital arrival.

transferred out of the ICU, and his neurological exam had improved to following commands and speaking sentences, although he was still having trouble with muscle weakness and fine motor control. Upon questioning, the patient denied any suicidal intent from his exposure and reported that he was trying to fumigate his home thoroughly and didn't want any of the fumigant to escape through an open window. After fourteen days of hospitalization, he was discharged to home with outpatient physical therapy referral.

3. Discussion

Malaria, West Nile virus, Zika virus, Lyme disease, and Dengue fever are common causes of morbidity and mortality around the world. While arthropod bites may cause minor discomfort, a larger concern is the potential to develop a deadly systemic infection. The use of insect repellents to prevent these infections constitutes a fundamental public health effort [3]. Although DEET is one of the most commonly and widely utilized insect repellent, safety data are largely derived from animal studies and individual case reports [4]. The current literature includes limited studies evaluating pharmacokinetics and toxicity in humans. These reports of toxicity exist predominantly in the pediatric population and by way of dermal exposure or intentional ingestion [3–5]. There are no reports of severe DEET toxicity following inhalational exposure.

DEET is known to have neurotoxic and cardiotoxic effects, causing encephalopathy, respiratory depression, seizure and coma as well as profound hypotension [4–7]. Children seem to be particularly at risk for toxic encephalopathy [7]. The mechanism of action of DEET neurotoxicity is poorly understood. Animal studies have suggested it may disrupt the blood-brain barrier and induce neuronal apoptosis [3]. The neurologic symptoms seen in our patient are consistent with prior

toxic DEET exposures as is the hypotension though the tachycardia is an unexpected finding, especially in the presence of hypotension. However, a similar finding of persistent tachycardia was reported by Wiles et al. in a 37-year-old male who ingested 6 oz of a 40% DEET solution [4]. Treatment involves only supportive care.

The dose relationship between DEET exposure and symptoms reported in the medical literature is difficult to establish. Three case fatalities due to oral ingestion have had blood DEET concentrations reported [4]. These serum concentrations were 92.1 mg/L, 168 mg/L, and 240 mg/L. In comparison, our patient's initial serum DEET concentration (59 mg/L) was obtained approximately 7.5 h after presenting to the hospital. It took over 100 h for the DEET to be eliminated from our patient's serum (Fig. 1). Analysis of DEET in clinical and forensic cases usually involves specimens collected at one time point after exposure. One other study measured serial DEET concentrations after ingestion of 15–25 mL of a DEET-containing solution. In that case serum DEET concentrations were non-detectable by 48 h post-ingestion. DEET is oxidized by hepatic microsomes and metabolites can persist in fatty tissues for 1–2 months [8]. Since DEET undergoes hepatic oxidation, elimination of DEET in our patient may have been prolonged at the beginning of the elimination curve secondary to saturation [9]. A considerable amount of interindividual variability in activity of drug metabolizing enzymes relevant to DEET metabolism has also been reported [10].

4. Conclusion

Although DEET products are widely used and safe when used as directed, severe toxicity can result from inhalation and improper use of “bug bombs”. This case provides new information regarding serial DEET concentrations, and toxic effects after an inhalational exposure.

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