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An unusual case of pediatric acute nicotine poisoning due to a dermal exposure

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ABSTRACT

Acute intoxication with nicotine is possible to cause nonspecific clinical signs and may be serious and lead to the death. We report a rare and severe form of acute nicotine poisoning secondary to dermal absorption of tobacco.

1. Introduction

The richness of nicotine characterizes not only all the specificity of tobacco plants but also its toxicity. Nicotine is most frequently encountered in snuff "Neffa", chewing tobacco, cigarettes, cigars and pipe tobaccos or in a limited number of pesticides[1]. Acute poisoning with nicotine may cause non specific clinical signs, and seriously lead to the death. We report the case of an infant admitted to pediatric intensive care for a rare and severe form of acute nicotine poisoning secondary to dermal absorption of Neffa. We review, through this article, the characteristics and mechanisms of nicotine intoxication as well as recommended therapeutic modalities.

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2. Case report

A 20-month-old boy was presented to pediatric emergency department for respiratory distress. He was immediately admitted into pediatric intensive care unit and diagnosed with an acute cutaneous intoxication with Neffa. His trunk was coated with Neffa during the two days preceding the hospitalization; This practice has been being widespread in the family as a traditional remedy of

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eczema.

The clinical examination on admission noted episodes of vomiting, tachycardia and tachypnea. The neurological examination found a consciousness disturbance, and his pupils were in miosis, so he was rapidly intubated and ventilated with a continuous sedation. After intubation, he presented recurrent generalized seizures. The clinical presentation was suggestive of a muscarinic syndrome with a nicotinic and encephalic syndromes related to a progression of toxicity. The results of chest x-ray, CT scan and baseline metabolic assessment were normal. Serum and globular cholinesterase activity were low.

The management of this infant was based on skin decontamination by abundant washing and symptomatic stabilization of vital failures. Hypotension responded to the administration of norepinephrine vasopressor in combination with volume repletion. Seizures were treated with benzodiazepines. Atropine was administered to treat muscarinic symptoms. The patient had a clinical improvement within 72 h. After being extubated, his was discharged at home without sequela.

3. Discussion

Nicotine is a natural water-soluble alkaloid obtained from the dried leaves and stems of tobacco plants (*Nicotiana tabacum* and *Nicotiana rustica*)[1]. Nicotine can be absorbed following inhalation, ingestion, or dermal contact and then rapidly absorbed through the skin, alveoli, oropharyngeal mucosa, and gastrointestinal mucosa[1]. After absorption, it moves quickly to brain and diffuses easily into tissues. The affinity is higher in the liver, kidneys, spleen and lungs[2]. Nicotine is extensively metabolized by the liver with the two main metabolites being cotinine and nicotine N-oxide[2]. Because of the long half-life of cotinine (20 h), and that of nicotine is approximately 2 h, it appears to be the best biomarker for tobacco use or exposure. It is possible to detect unchanged nicotine in the urine[2,3].

According to the statistics from US Poison Control Centers, the number of exposure to nicotine has increased since 2010, especially in children under 5 years old[4].

Acute nicotin poisoning has a biphasic profile. The early effects will initially be stimulants such as nausea, vomiting, pallor, abdominal pain, hyper salivation, bronchorrhea, tachypnea, hypertension, tachycardia, miosis, ataxia, tremors, fasciculations and convulsions. The delayed phase is depressive including the central nervous system and respiratory depression causing dyspnoea, bradycardia, hypotension, shock, mydriasis, weakness, muscle paralysis and coma[5]. Multiple toxidromes may be associated. As doses increase, loss of nicotinic receptor specificity may occur and result in signs of muscarinic cholinergic toxicity. The highest levels of poisoning can lead to death[3]. Serum nicotine measurement is generally of little use for purposes other than forensics. Serum nicotine concentrations of above 2 000 μg/L are often associated with serious toxicity as reported by Davis *et al*[6]. It was the first reported case of nicotine poisoning after topical application of a traditional remedy containing nicotine, as for our case. The authors realized the serum and urine nicotine measurement as it was difficult to obtain an accurate history. In our case, the diagnosis was confirmed by the clear medical history described by the parents. Several modes of voluntary or accidental poisoning with nicotine have also been reported : Oral self-administration or intravenous injection of nicotine solution or nicotine liquid used in electronic cigarettes; The application of transdermal patches containing nicotine; The ingestion of nicotine pesticides, as well as swallowing or accidental ingestion of tobacco or tobacco products by children; Ingestion of boiled tobacco water used enemas for intestinal parasites[7-9]. The dermal absorption of nicotine has also been reported after spraying or applying insecticides containing nicotine to skin or clothing and during contact with tobacco leaves in the workplace[10].

The management of nicotine poisoning is mainly symptomatic and generally favorable. In cases of dermal exposure to nicotine such as the reported case, contaminated clothing should be removed and affected areas should be washed. Alkaline solutions that could increase absorption should be avoided. In severe ingestion, administration of aqueous activated charcoal may be indicated. There is no specific antidote for nicotine. Atropine may be used to treat symptoms associated with muscarinic receptor stimulation[6]. Benzodiazepines are used for convulsions.

A high concentration of nicotine can cause serious poisoning and death even by topical application. Adequate and timely management ensures, in the vast majority of cases, a resolution of the toxicity without sequela. Emergency physicians should be aware of this mode of poisoning, and public health efforts should be made to minimize such exposure. This case highlights the importance of the accurate history as a corner stone in the early management of these patients.

Conflict of interest statement

The authors report no conflict of interest.

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