ORIGINAL RESEARCH PAPER

Emergency Medicine

A RARE CASE OF MOSQUITO COIL POISONING FOLLOWING ORAL INGESTION

KEY WORDS: Mosquito coil, pyrethroid poisoning, poisoning, toxicology, supportive treatment.

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ABSTRACT

Mosquito coils are commonly used household insect repellent products and generally contain synthetic pyrethroid compounds. Although pyrethroids have comparatively low mammalian toxicity, significant exposure, particularly following ingestion, can produce gastrointestinal, neurological and systemic manifestations. This is a case report of intentional ingestion of a crushed mosquito coil mixed with water in a 24 year old female who presented approximately 15 minutes after ingestion with abdominal pain, chest pain, nausea, vomiting, sweating, lacrimation, coughing and altered/confused mental status. On examination, she was afebrile, conscious and oriented, with a pulse rate of 90/min, blood pressure of 100/70 mmHg, respiratory rate of 18/min and oxygen saturation of 98% on room air. Cardiovascular, respiratory and abdominal examinations were unremarkable. Laboratory investigations were largely within the reported ranges, electrocardiography showed normal sinus rhythm, and toxicological analysis of the gastric aspirate was positive while the blood sample was negative. The patient was managed with gastric decontamination and supportive treatment and subsequently showed symptomatic improvement. This case highlights the uncommon possibility of clinically significant toxicity following ingestion of a household mosquito coil and emphasizes the importance of early recognition, appropriate toxicological assessment and supportive management.

INTRODUCTION

Mosquito coils are widely used household insect-repellent products, particularly in tropical and subtropical regions. The active insecticidal components of mosquito coils commonly belong to the synthetic pyrethroid group. Pyrethroids are synthetic derivatives of naturally occurring pyrethrins and are widely used because of their potent insecticidal activity and comparatively low toxicity in mammals. Their principal toxicological action involves modification of voltage-sensitive sodium channels, resulting in delayed channel closure, prolonged sodium influx and repetitive neuronal firing. At higher concentrations, pyrethroids can additionally affect chloride channels, including GABA-gated chloride channels, which may contribute to neurological manifestations such as seizures in severe poisoning.[1,2]

Human pyrethroid poisoning is relatively uncommon, despite the extensive use of these compounds. Following ingestion, symptoms may develop within minutes and commonly include sore throat, nausea, vomiting and abdominal pain, while increased secretions, dysphagia, dizziness, headache, fatigue, palpitations and chest tightness may occur in more significant exposures. Severe poisoning may result in altered consciousness, convulsions and coma, although most patients recover with appropriate supportive management.[1]

Mosquito coil poisoning is rarely reported compared with ingestion of liquid pyrethroid formulations. In a recent series, mosquito coils accounted for only 2 of 379 identified pyrethroid exposures with a known formulation, representing approximately 0.5% of cases.[3] This relative rarity makes the present case clinically relevant, particularly because mosquito coils are easily accessible household products and may not always be perceived by the public as potentially toxic.

Mosquito coil ingestion represents an uncommon form of pyrethroid exposure. While pyrethroid poisoning is well documented, reports specifically describing ingestion of mosquito coils are scarce. We report a case of acute symptomatic poisoning following ingestion of a crushed mosquito coil mixed with water, representing a rare mode of pyrethroid exposure.

Case Report

A 24-year-old female was brought to the emergency department by her husband with complaints of abdominal pain, chest pain, nausea, vomiting, sweating, lacrimation, coughing and a confused mental state. On further questioning, a history of ingestion of a crushed mosquito coil mixed with water was obtained. The patient reportedly developed symptoms approximately 15 minutes following ingestion and was subsequently brought to the hospital for evaluation and management. There was no history of loss of consciousness, seizures, blood in vomitus, fever or loose stools.

On presentation, the patient was conscious, cooperative and oriented to time, place and person. Her GCS was 15/15 and there was no focal neurological deficit. Her pulse rate was 90 beats per minute, blood pressure was 100/70 mmHg, temperature was 98.8 F and respiratory rate was 18 breaths per minute. Examination of the cardiovascular system revealed normal S1 and S2 without any murmur. Respiratory examination showed bilateral equal vesicular air entry without added sounds. Abdominal examination revealed a soft, non-tender abdomen. There was no evidence of respiratory distress, seizures or hemodynamic instability at the time of presentation.

Investigations

Laboratory investigations revealed a hemoglobin concentration of 12.5 g/dL, white blood cell count of 8,800/mm^3 and platelet count of 2.39 lakh/mm^3. Serum electrolytes showed sodium of 136 mEq/L, potassium of 4.2 mEq/L and chloride of 110 mEq/L. Renal function testing showed creatinine of 0.8 mg/dL and uric acid of 9.2 mg/dL. Liver function testing showed total bilirubin of 0.7 mg/dL, SGOT of 18 IU/L, SGPT of 14 IU/L and alkaline phosphatase of 58 IU/L. Random blood sugar was 108 mg/dL. Electrocardiography demonstrated sinus rhythm. Toxicological examination of the gastric aspirate was positive, whereas the blood sample was negative.

Management And Clinical Course

The patient was kept nil per oral for 24 hours and a nasogastric tube was inserted. Gastric lavage with activated charcoal was performed, following which she received injection atropine 2 mL, injection pralidoxime 500 mg diluted in 100 mL normal saline, injection pantoprazole 40 mg and injection ondansetron

4 mg. The patient showed symptomatic improvement following the initial management and was subsequently continued on supportive treatment and clinical observation.

DISCUSSION

This case is unusual because of the ingestion of a mosquito coil after crushing and mixing it with water, followed by the rapid development of multisystem symptoms. Pyrethroid containing household insecticides are generally regarded as having relatively low toxicity in humans because mammals have lower sensitivity to their target sodium channels and possess efficient metabolic pathways for their detoxification. Nevertheless, substantial exposure can produce clinically significant toxicity.[1]

The symptoms observed in our patient occurred approximately 15 minutes after ingestion and included abdominal pain, nausea, vomiting, sweating, lacrimation, cough, chest pain and altered or confused mental status. These findings are compatible with the recognized clinical spectrum of acute pyrethroid exposure. Bradberry et al. reported that ingestion of pyrethroids can produce symptoms within minutes, particularly sore throat, nausea, vomiting and abdominal pain, while neurological and systemic manifestations may occur with more significant exposure.[1] A review of pyrethroid poisoning similarly describes vomiting, drowsiness, salivation and seizures among reported clinical manifestations, although most exposed patients have relatively mild toxicity.[3]

The mechanism of toxicity is primarily related to effects on neuronal ion channels. Pyrethroids delay the closure of voltage-sensitive sodium channels, producing prolonged sodium influx and repetitive neuronal firing. At higher concentrations, effects on chloride channels and GABA-gated chloride channels may contribute to neurological manifestations, including seizures.[1,4] This mechanism explains the broad range of neurological symptoms that may be encountered in pyrethroid poisoning.

The toxicological findings in our patient are also noteworthy. The gastric aspirate was positive, whereas the blood sample was negative. This finding supports exposure through ingestion but does not necessarily exclude systemic absorption, particularly because pyrethroids are rapidly metabolized. The diagnosis of pyrethroid poisoning is predominantly clinical and is supported by identification of the offending product or its active ingredient. Advanced toxicological assays may be available in specialized laboratories, but their routine clinical role remains limited.[2]

The management of pyrethroid poisoning is predominantly supportive because there is no established specific antidote for isolated pyrethroid toxicity.[2] Treatment should focus on maintaining the airway, breathing and circulation, correcting metabolic abnormalities and managing neurological or cardiovascular complications when present.[2] In patients with seizures, appropriate anticonvulsant therapy and airway support may be required.

A particularly important aspect of this case is differentiation of pyrethroid poisoning from organophosphate poisoning. Clinical features such as vomiting, excessive secretions, sweating and altered sensorium can create diagnostic uncertainty. However, pyrethroid poisoning has a different toxicological mechanism and does not have an established specific antidote. The majority of patients can be managed successfully with supportive and symptomatic therapy.[2]

In this case gastric lavage with activated charcoal was performed soon after ingestion, with atropine and pralidoxime administered during initial management. These interventions reflect the treatment provided in this case rather than specific therapy for pyrethroid poisoning, which is primarily supportive and symptomatic.[2] Pralidoxime has an established role in organophosphate poisoning but is not an antidote for pyrethroid toxicity. The role of gastric decontamination also requires careful consideration. Because gastric lavage can result in aspiration and other complications, it should only be considered in carefully selected circumstances after assessment of the potential benefits and risks.[2]

Previous case reports have also demonstrated that household mosquito-repellent formulations containing pyrethroids can produce significant clinical manifestations following ingestion. A published case of prallethrin poisoning described neurological and systemic manifestations and emphasized the importance of recognizing pyrethroid exposure in patients presenting with unexplained poisoning symptoms.[5] Another report described severe neurological toxicity, including status epilepticus, following pyrethroid ingestion, demonstrating that although most cases are mild, serious complications can occur.[6]

The overall prognosis of pyrethroid poisoning is generally favorable. Bradberry et al. reported that most patients recover within several days, although severe poisoning can occasionally result in convulsions, coma and death.[1] In the present case, the absence of seizures, respiratory compromise, significant cardiovascular abnormalities and persistent altered consciousness, together with improvement following initial management, was associated with a favorable clinical course.

CONCLUSION

Mosquito coil ingestion is an uncommon but clinically important form of pyrethroid exposure. Although pyrethroids generally have relatively low mammalian toxicity, ingestion can result in rapidonset gastrointestinal, neurological and systemic symptoms. A careful history of the type and formulation of the insecticide, early clinical assessment, appropriate toxicological evaluation and close monitoring are essential. Management is predominantly supportive be-cause no specific antidote is available for isolated pyrethroid poisoning.[1,2] This case emphasizes that commonly available household mosquito coils should not be considered harmless and highlights the need for appropriate storage, public awareness and prompt medical evaluation following ingestion.

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