A lethal danger in the home: turpentine poisoning

Ahmet Güzel, Mehmet Açıkgöz

Department of Pediatrics. Ondokuz Mayıs University Faculty of Medicine, Samsun, Turkey.
E-mail: dracikgozm@gmail.com
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Turpentine is an oleoresin obtained from various species of pine. In turpentine poisoning, various signs and symptoms of toxicity may develop, including hematuria, renal failure, loss of vision, chest pain, vomiting, severe coughing, gastroesophageal hemorrhage, hypotension, swelling of the throat and even death. We report a case of turpentine ingestion in a 9-year-old boy. The patient was admitted to our clinic with suspected intoxication after accidentally drinking from a glass that held a turpentine oil preparation used by his father for hair care. The patient displayed no significant signs and symptoms other than bradycardia and hypotension. Laboratory investigations revealed no abnormalities. The patient was hospitalized for close monitoring and observation. During a two-and-a-half-day observation period, hypotension was corrected with administration of dopamine and intravenous fluids. In this report, we wish to draw attention to the dangerous effects of plant-derived drugs.

Key words: turpentine, children, poisoning, emergency.

Herbal products play an important role in cases of fatal intoxication in childhood. Turpentine is an oily resin that is derived from a variety of pines; its main components are monoterpenes (α-pinene, β-pinene and 3–carene)[1,2]. Turpentine oil has a strong, bitter taste and is colorless and caustic[3]. A hydrocarbon derivative, it is found in paint solvents, shoe polish, floor polish, printer ink, adhesives, cosmetic products (e.g., shampoos and colognes) and in catarrh medications, carminatives and anthelmintic medications for therapeutic purposes, as it is a type of solvent[1,3-6].

As well as having many uses, turpentine has also been reported to cause fatal intoxication in children after accidental ingestion. The literature contains only a few pediatric cases associated with the ingestion of turpentine[5]. To our knowledge, the only such reported case in which the child survived is that of an 18-month male patient reported by Khan et al.[5], who experienced systemic complications due to oral ingestion of turpentine. An Australian study reports that the incidence of intoxication cases associated with turpentine in childhood is 0.79%. However, the authors did not report any clinical conditions or presence of complications in that study[7].

The intent of the present report is to draw attention to the phenomenon of turpentine intoxication, which is rare in children, but may be fatal.

Case Report

A 9-year-old male patient was brought to our pediatric emergency polyclinic 30 minutes after he accidently drank 50 ml of turpentine oil, which had been prepared by his father for hair care. On admission, the physical examination revealed the following: Glasgow Coma Score, 15/15; SpO_2, 97%; temperature (axillary), 36.8°C; pulse, 154 beat/min; respiratory rate, 21/min; and arterial blood pressure, 120/80 mmHg. Examination of other systems revealed no pathologic findings. The patient was monitored and followed up in the pediatric emergency department.

Laboratory studies at admission presented the following values: WBC, 6880/mm^3; Hgb, 12.9 g/dl; platelets, 178,000/mm^3. Biochemical tests included: Na^+, 136 mEq/L; K^+, 3.5 mEq/L;

Cl^-, 104mEq/L; Ca^{+2}, 9.9 mg/dl; BUN, 13 mg/dl; creatinine, 0.39 mg/dl; AST, 36U/L; ALT, 26U/L; CK-MB, 2.04 μg/L; CK, 110mU/ml; PT, 12 sec.; aPTT, 24 sec; INR, 1. Blood gas results were: pH, 7.38; pO_2, 72 mmHg; pCO_2, 40.8 mmHG; HCO_3, 24 mmol/L.

The patient developed hypotension at the 3rd hour of follow-up. Despite normal saline loading and a large dose of IV fluid (2500 ml/m^2), he had still hypotension and developed sinus bradycardia at the 14th hour of follow-up; intravenous infusion of dopamine (10 μg / kg/min) was therefore initiated. No additional treatment was delivered for prolonged aPTT, and his control values were normal. Following 8 hours of dopamine infusion, the patient had normal blood pressure values, and dopamine was discontinued. The patient was discharged after 3 days of follow-up, as he had no additional complaints.

Discussion

Intoxication by turpentine oil, a derivative of hydrocarbon, is very rare in childhood[3]. In one study, hydrocarbons were reported to rank 10th among agents causing intoxication through oral ingestion in children younger than six years old in the United States, and only 6 hydrocarbon intoxication-related deaths were reported among more than 100,000 pediatric patients between the ages of 18 and 24 months[8]. An Australian study investigated 1516 intoxication cases between the ages of 0 and 14, and reported a total of 12 pediatric cases associated with turpentine intoxication[7].

A review of the literature on turpentine intoxication turns up only a few cases where the complications were reported. Three Indian cases, which resulted in death, were reported in 1931, 1939 and 1964[3]. A fatal pediatric case involving a 16-year-old male patient who died of severe hypotension and status epilepticus after ingesting approximately 200 ml of turpentine oil was reported by Pande et al[3]. in 1994. Another case of fatal turpentine intoxication in childhood was reported by Harbeson[4]. The patient was an 11-month-old female infant who was given turpentine orally by her grandmother, because the latter suspected the child had worms. The infant was admitted to the emergency service due to coma, convulsions, arrhythmia and respiratory failure, and died after about 4 hours. The autopsy revealed symptoms of acute bronchitis, dilated thymus and lymphoid hyperplasia[4].

The average fatal oral dose of turpentine varies between 15 and 150 ml[6]. The 18-month-old male patient reported by Khan et al.[5] presented suffering from respiratory distress and fever 14 hours after accidental oral intake. Respiratory distress and fever became severe during follow-up, and he was later diagnosed with pleural effusion and pneumonia. Because the patient developed pleural effusion, necrotic lung injury and multiple abscess formation during follow-up, he underwent a decortication with thoracotomy and right center and lower lobe segmental resection[5].

Turpentine oil causes toxicity when absorbed by the lungs through inhalation or when it enters the gastrointestinal system through oral ingestion[3,5]. Clinical effects occur 2-3 hours after it enters the body. The most common effects of turpentine ingestion include a burning sensation in the mouth, pain in the lips, tongue, throat and esophagus, thirst, coughing, vomiting and diarrhea[3,5]. Other effects include pupillary contraction, dizziness, drowsiness, cold skin, hematuria, violet odor of the urine and difficulty in urinating[3-4]. Severe symptoms of toxicity that may result in death include aspiration pneumonitis, acute lung damage, pulmonary edema, resistant metabolic acidosis, serious hypotension, liver failure, renal failure, convulsions, arrhythmia and coma[3-5,9]. Effects in our case included hypotension, bradycardia and prolonged aPTT.

Death often occurs in such cases within the first 12-15 hours after oral intake[3]. Treatment of patients who are poisoned by turpentine oil generally involves support of respiration/circulation and use of a symptomatic approach[5]. American and European toxicology associations state that gastric lavage is contraindicated after intake of hydrocarbons because it increases the risk of aspiration[10]. Early use of steroids and prophylactic antibiotics is said to be ineffective in treating the lung injury secondary to turpentine aspiration[9]. This is because the concomitant fever and the presence of leukocytosis in such cases are associated with chemical pneumonitis due to turpentine aspiration[5]. We delivered to our patient a positive inotropic agent treatment through

IV replacement for apparent hypotension and bradycardia.

Given the widespread use of turpentine oil in cosmetic and household products as well as in industry, it must be kept in mind that turpentine can be a cause of fatal intoxication in childhood.

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