Traditional Medicine with Cade Oil in Pediatric Emergency Care

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Abstract:

Despite the advancements in modern medicine, traditional medicine remains relevant in our country. This is exemplified by the practice of traditional therapy using cade oil, commonly referred to as "camel soap," "tar," or "القطران," which is increasingly observed in Tunisia. It is not uncommon to admit patients to the hospital who are victims of these practices.

We conducted a prospective study over a 6-year period from January 2018 to December 2023. We included newborns, infants, and children under 14 years of age who had undergone traditional therapy with cade oil. We excluded all other forms of traditional therapy.

The aim of our study was to raise awareness of the increasing frequency of cade oil traditional therapy in our region, the potential damages that can result, and the importance of taking appropriate medical and legislative measures to address this phenomenon.

Twenty cases were recorded in our department for this reason. All children had a low socioeconomic status. Thirteen were from rural areas and seven were from urban areas. The reasons for resorting to these practices included a lack of resources in one case, and in all other cases, parental beliefs in the effectiveness of these methods compared to modern treatments. The average interval between the application of cade oil and consultation at the hospital was 14 hours. The average age was 18 months, with a minimum age of 10 days and a maximum of 4 years. The primary reason for consultation was gastrointestinal symptoms, including diarrhea and vomiting, often in a febrile context.

Medical therapeutic management included symptomatic measures in all cases, with specific treatment provided in 2 cases. Legislative measures involved all living patients, represented by notification to the child protection delegate. The outcome was favorable for 14 cases, unfavorable with death in four cases, and neuro-sensory sequelae in two other cases.

Prevention plays a crucial role in addressing this phenomenon by raising awareness among both parents about the side effects of irrational use of this product and healthcare personnel about the need to consider cade oil poisoning in children presenting with unexplained multi-organ involvement to ensure early and appropriate management.

Keywords: Cade oil, Tar, Child, Poisoning, Traditional therapy

INTRODUCTION

Traditional therapy with cade oil, used for addressing physical, mental, or social health issues, is becoming increasingly embedded in Tunisian culture. The danger lies in exposing children to the harmful effects of cade oil through these popular practices.

Cade oil is distilled from the branches of *Juniperus oxycedrus*, which, despite its known toxicity due to its phenol content, continues to be used in traditional medicine.

Poisoning from this product manifests through cardiovascular disturbances (hypotension, tachycardia), neurological disorders (headaches, hypotonia, seizures, or even coma), respiratory issues (acute pulmonary edema and respiratory distress), and gastrointestinal symptoms (diarrhea, vomiting).

Diagnosis is based on a combination of anamnesis, clinical findings, laboratory results, and radiological examinations.

Management is multidisciplinary, focusing primarily on symptomatic measures.

METHODS

This is a retrospective study covering a 6-year period from December 2017 to December 31st, 2023, involving children hospitalized with a reported history of cade oil use.

The study was conducted at the Pediatric Intensive Care Unit of Hédi Chaker University Hospital in Sfax. We included all infants, children, and adolescents under 14 years of age who had undergone an application of tar. We excluded children who were poisoned by substances other than cade oil. During this study, we documented the epidemiological, clinical, and outcomes.

RESULTS

We compiled data on twenty cases with an average age of 18 months (ranging from 10 days to 4 years). There were 2 boys and 8 girls. Seven children were from urban areas, while the remaining 13 were from rural areas. Three modes of poisoning were observed: inhalation (1 case), cutaneous application (6 cases), and transcutaneous application via scarification (9 cases). The combination of cutaneous and respiratory routes was seen in 4 patients.

Neurological manifestations were present initially in 16 patients, including:

- Consciousness disturbances ranging from apathy and drowsiness to coma in 5 patients.
- Behavioral disturbances in 6 patients, including moaning in 3 patients and plaintive behavior in 3 patients.
- Axial and peripheral hypotonia in 4 patients.
- Seizures in 5 patients.

Thirteen of our patients showed signs of dehydration upon admission. Eight of these had severe dehydration (stage III), and seven patients have also presented associated hemodynamic disturbances. Respiratory manifestations were described in 12 patients: tachypnea (n=12), signs of respiratory distress (n=6), and abnormal pulmonary auscultation (n=5 patients).

Cade oil application was made in multiple locations on the same patient. Parents applied

the oil to the ear lobules, around the nostrils, on the forehead, chest, and extremities. Scarification lesions on the forehead, trunk, and all four limbs were noted in 12 patients (Figures 1 and 2).

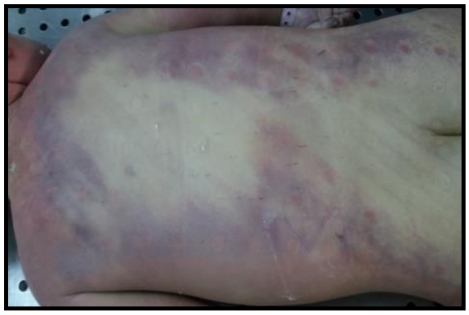

Figure 1: *Image showing scarification lesions on the back*

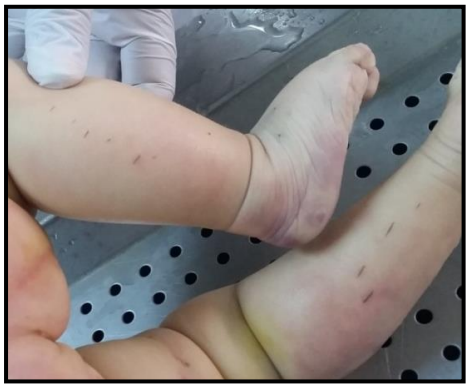

Figure 2: *Image showing scarification lesions on the feet*

The biological tests performed for all patients showed: leukocytosis (n=9), transient neutropenia (1 patient), thrombocytopenia (3 patients), thrombocytosis (3 patients), normochromic normocytic anemia (n=6), hyponatremia (n=11), hypokalemia (n=8), metabolic acidosis (n=12), hepatic cytolysis (n=7), and functional renal failure (n=3).

Toxicological screening blood and urine tests were performed in 4 patients; they were positive for organophosphates in 2 patients. Cholinesterase activity testing was performed for 2 patients, showing normal serum activity and low globular activity.

Chest X-rays were performed for all patients, with normal results in 16 patients and only 4 showing pulmonary radiological abnormalities.

Brain CT scans were performed in 8 patients, revealing toxic origin anomalies in 2 patients, old anterior abnormalities in 1 patient, and normal results in 5 cases.

Brain MRIs were performed for 3 patients, showing in one case a hyperintense signal in the thalamus, probably of toxic origin, and in the other two cases, bilateral and symmetrical signal abnormalities in the cortical-subcortical regions, basal ganglia, and corpus callosum, suggestive of toxic encephalopathy (Figure 2).

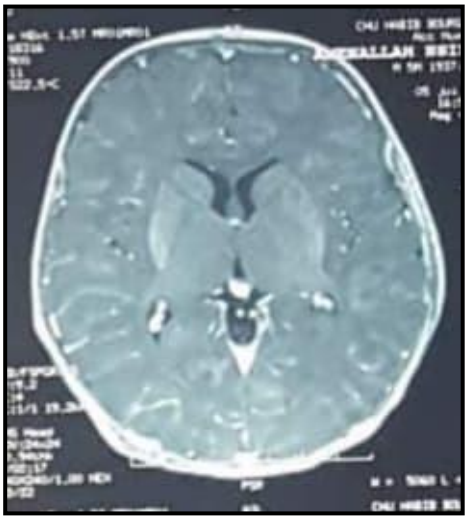

Figure 2: *Brain MRIs showing in one case a hyperintense signal in the cortical-subcortical regions, basal ganglia, and corpus callosum*

The electroencephalogram (EEG), performed for eight patients, was abnormal in 7 cases. It showed severe global anoxic-ischemic brain damage (see Figure 4) in three patients, slow and

polyspike waves in the temporal and bilateral temporal occipital regions with diffuse rapid rhythms in three patients, and poorly organized with the presence of multifocal anomalies in the remaining two cases.

Symptomatic treatment was based on rehydration in all cases. Antibiotics were administered to 18 patients. Anticonvulsant treatment was given to 8 patients. No specific treatment or antidote for tar was administered. However, Pralidoxime and Atropine were given to two patients whose enzymatic assays and toxicological evaluations showed low globular cholinesterase activity with the presence of organophosphates in the urine.

The outcome was favorable for 15 patients. Two cases resulted in severe neuro-sensory sequelae, and the remaining 5 cases had a fatal outcome.

DISCUSSION

Cade oil is a tar obtained by pyrolysis of the wood of the cade tree (*Juniperus oxycedrus*), a Mediterranean shrub commonly known as juniper (1-8). It is a viscous, thick, and homogeneous liquid that is black or dark brown color, with a distinctive unpleasant empyreumatic odor and a consistency similar to tar, with a bitter and caustic taste (1-8). It is produced from the carbonization of the trunks and large branches of old juniper trees.

Juniperus oxycedrus (Figure 4) is a plant widely distributed in countries with a Mediterranean climate (around the Black Sea and the Middle East) (9). It is one of the species in the juniper genus, belonging to the Cupressaceae family, and is highly valued for its essential oil and secondary metabolites, which are extensively used in traditional medicine (10). All parts of the plant contain highly aromatic essential oils (11).

Figure 4: *Branch of* Juniperus oxycedrus *(5)*

1. Pharmacological Properties of Cade Oil

A variety of pharmacological properties are documented in the literature, some scientifically proven and others still requiring further investigation. This product exhibits antipruritic activity (12), mainly due to the presence of phenols such as cresols (1,2,5,13), as well as keratolytic action (1,2,5,13), anti-inflammatory effects, antiseptic properties (9,14-17), healing activity (9,14,17), toning and energizing effects (18-20), and antimicrobial and antifungal action (9,10). Cade oil also has parasiticidal properties (1,2,5,13). Additionally, it demonstrates analgesic, antispasmodic, and calming effects (9,17-19,20,21), an impact on blood pressure with hypotensive effects independent of the adrenergic system, likely due to the vasorelaxant effect of methanol (13,17,23), antidiabetic activity through anti-amylase effects (9,24,25), diuretic action (9,24), anticholinergic effects, and anti-Alzheimer effects stimulating memory (15,17,26).

2. Toxicity of Cade Oil

When used for therapeutic purposes, cade oil can cause a significant number of toxic reactions, which can even be fatal. According to data from the global poison center CAPM, this toxicity has a mortality rate of approximately 10.5% (28). In our study, five cases were fatal.

Toxicity often results from iatrogenic causes, primarily due to ingestion of a large amount or, particularly in infants and newborns, from prolonged and extensive cutaneous application. The surface-to-weight ratio, combined with the immaturity of metabolic and elimination processes in infants, explains the severity of the toxicity when the product is applied topically (5).

In our series, no cases of ingestion were noted; one case involved inhalation, six cases were through cutaneous exposure, nine cases involved transcutaneous absorption via scarification, and four cases had both cutaneous and respiratory exposure.

Cade oil contains phenol, which has life-threatening effects (corrosive, cardiac, hemolytic, pulmonary, and renal) (3). It is the most toxic component. Its absorption is rapid, and its metabolism is primarily hepatic. Systemic toxicity is multi-organ and is explained by the formation of cytotoxic metabolites (semi-quinone radicals) when the absorbed amount exceeds hepatic conjugation capacity (3,5,6,8). The severity of intoxication ranges from benign symptoms to life-threatening conditions.

2.1. Neurotoxicity

Alterations in the central and peripheral nervous systems have been reported following cade oil intoxication, including involuntary movements, headaches, hypotonia, mental confusion, and even myoclonic or convulsive coma (4) due to increased acetylcholine release at the neuromuscular junction, leading to heightened central nervous system activity (28). In our study, consciousness disturbances were reported in 16 patients.

2.2. Cardiovascular Toxicity

Cardiovascular disorders (such as sinus bradycardia, myocardial excitability disturbances, hypotension, and even cardiovascular collapse) have been documented in the literature (3,13,15,32). Toxic doses of phenol cause initial hypertension followed by a marked drop in blood pressure (4). In our study, seven patients exhibited hemodynamic disturbances. However, no electrical anomalies were observed.

2.3. Respiratory Toxicity

Pulmonary involvement can range from pneumonia to acute pulmonary edema (OAP) (3,33). In our study, respiratory symptoms were illustrated in 12 cases, with four having evident radiological findings.

2.4. Nephrotoxicity

Renal damage can be reversible or permanent, affecting the glomeruli, tubules, and interstitium. Hematuria and/or albuminuria (34,35), functional renal insufficiency (3,4,7,36), and organic renal failure (37) can be

observed. In our study, three cases of acute functional renal failure were noted.

2.5. Hepatic Toxicity

Liver damage ranges from hepatic cytolysis to hepatic cellular failure (4). In our study, seven cases of hepatic cytolysis were recorded.

2.6. Digestive Toxicity

Phenol's corrosive effect leads to diarrhea and vomiting of varying severity, sometimes resulting in severe dehydration (13). Digestive symptoms were predominant in our study, affecting all of our patients.

2.7. Hematological Toxicity

Methemoglobinemia, deep vein thrombosis, and hemolytic anemia (38) have been reported after exposure to cade oil, with effects attributed to phenol (13,26). Neutropenia, thrombocytopenia, and consumption coagulopathy are also observed (13).

2.8. Adverse Effects on Skin and Mucous Membranes

Short-term, this substance has an irritant effect (8). Long-term, cade oil contains polycyclic aromatic hydrocarbons such as benzopyrene, which is known to be carcinogenic (8,39).

3. Management of a Child with Cade Oil Poisoning

A thorough interview with parents, their surroundings, or witnesses often allows for the precise determination of the cause of poisoning, the circumstances, the time of exposure, initial symptoms, and any complications, thus avoiding costly and sometimes unnecessary tests, especially toxicological analyses.

Apart from cyanosis associated with methemoglobinemia, the presence or absence of a blackish discoloration due to the toxic product can easily guide the diagnosis. A brown-black discoloration may be due to the presence of hemoglobin or myoglobin, but the blackish color of cade oil itself can also cause this coloration. The distinctive odor of cade oil is another important diagnostic clue.

Symptoms are very varied and can involve all organs, including central neurological disturbances, cardiovascular issues, respiratory, and digestive disorders. Certain biological abnormalities have significant diagnostic value and can even suggest the toxin (41). It is recommended to perform blood glucose testing to check for hypoglycemia (9,24,25), an electrolyte panel to detect ionic disturbances, a gas analysis to assess metabolic acidosis, and a coagulation profile to identify hemorrhagic syndromes secondary to hepatic failure or disseminated intravascular coagulopathy within the context of multi-organ failure.

Measuring transaminases helps assess the extent of cytolysis and toxic aggression (3). Radiological exams have non-specific indications for determining the presence of lesions or complications (e.g., chest X-ray for pulmonary edema, atelectasis, pneumonia). EEG remains valuable in cases of convulsive or myoclonic states and in monitoring post-anoxic comas (40). Digestive endoscopy is indicated in case of significant cade oil ingestion since some of its constituents are considered corrosive (42).

Toxicological analysis is certainly diagnostically useful. However, if the diagnosis is evident (history, symptomatology), its main relevance becomes medico-legal (41). Methemoglobinemia should be investigated in the presence of slate-gray cyanosis, unexplained by hypoxemia, and unresponsive to oxygen, when arterial blood shows a chocolate-brown hue unaltered by exposure to air or oxygen bubbling.

Regarding cholinesterase levels, a decrease in plasma and especially erythrocyte cholinesterase activity is usually a direct indicator of the severity of organophosphate and carbamate insecticide poisonings.

4. Severity Diagnosis

This is a fundamental step as it determines the therapeutic strategy and monitoring. The progression of poisoning is a dynamic process dependent on the kinetics and toxicodynamics of cade oil.

5. Therapeutic Management

Therapeutic management should begin as early as possible to prevent systemic effects. For systemic intoxication, therapeutic management is primarily symptomatic, based on resuscitation measures (ventilatory and hemodynamic support if indicated, anticonvulsants for seizures, correction of ionic and acid-base disturbances if biological anomalies are present, and administration of methylene blue in cases of methemoglobinemia (43)) to maintain vital signs and simultaneously decontamination to prevent systemic effects. Depending on the route of exposure, cutaneous and ocular decontamination or digestive decontamination is implemented.

No specific antidote for cade oil is currently available due to limited knowledge of the product. Emergency antidotes include Atropine for cholinergic syndrome and pralidoxime when toxicological tests are positive for organophosphates (41). Administration of N-acetylcysteine (Fluimucil®) may be considered to attempt to neutralize reactive metabolites from hepatic biotransformation.

Medico-legal management with a report to child protection is mandatory.

CONCLUSION

In Tunisia, cade oil is available to consumers without prescription or market authorization. Its irrational use leads to severe, sometimes fatal adverse events. Public awareness is essential. Likewise, informing healthcare professionals about these side effects is crucial to ensure early and effective management in a resuscitation unit in cases of poisoning.

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