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Cade Oil Poisoning: A Case Series

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Abstract

Objectives and Study: Cade oil is aromatic oil obtained from the branches and wood of *Juniperus oxycedrus*, common in the Mediterranean region and widely used in traditional medicine. This oil contains many chemical compounds with neurological, cardiac, renal, respiratory, hepatic, and gastrointestinal toxicity. Cade oil poisoning often requires intensive care admission due to the severity of the clinical picture. The objective of this study is to highlight the multiple manifestations found in the pediatric population due to cade oil exposure objectifying its significant toxicity. **Methods:** The authors report during this article a series of five cases of cade oil poisoning on pediatric patients hospitalized in the pediatric intensive care unit of El HARROUCHI hospital at the CHU Ibn Rochd in Casablanca during the period from 11/01/2022 to 12/07/2022. The patients have been exposed, a few hours before their admission, to an external cade oil application used by parents for therapeutic purposes. **Main Findings:** Our patients were aged from 1 month to 4 years, the average age was 1.5 years with a female predominance and a sex ratio of 1.5:1. The patients had no prior medical history, and the cade oil application was spontaneously declared by the parents of only 2 patients, 3 of them reported the use of it after the detection of the substance by the clinician. For all the cases, cade oil was applied to treat fever. All five (5) patients presented initial neurological signs. 3 of them were admitted to an acute consciousness disorder and the 2 other patients presented respectively a convulsive status epilepticus and generalized-onset seizure. We report respiratory symptoms in 4 cases ranging from a simple caught, rhinorrhea to severe respiratory distress. Three patients presented acute liver failure with very high transaminase levels associated with acute kidney failure. Two of them presented digestive symptoms such as abundant hematemesis, vomiting, and watery diarrhea. All patients received high doses of N acetylcysteine in their initial treatments. The evolution was unfavorable for 4 patients who developed a multiorgan failure, 3 of them died, with a good clinical improvement in the fifth patient

after supportive and symptomatic treatment. **Conclusion:** Cade oil poisoning remains a very frequent situation in our context. Its toxicity is widely described in the literature. The increasing number of cases admitted, and the seriousness of the clinical picture require mass awareness among the population and the scientific community toward the use of medicinal plants.

Keywords

Cade Oil Poisoning, Case Report, Pediatric Intensive Care Medicine

1. Introduction

Cade oil, also called "Katran", remains one of the most used products in Moroccan folk medicine. This substance is readily available to the general population. Therefore, cade oil poisoning is far from being an exceptional situation in our context, justifying the large number of pediatric cases observed in a short period of time.

Cade oil mainly contains phenols (derivatives of guaiacol and cresol), a sesquiterpenoid (cadinene), and an Alcohol (carbinol) known for their significant toxicity. Frequently applied topically, and rarely ingested, the systemic passage of its chemical compounds often involves multi-visceral failure. Cade oil poisoning cases are characterized by polymorphic and severe clinical pictures.

During this article, we report a retrospective study involving 5 cases of cade oil poisoning admitted to the pediatric intensive care unit. The objective is to underscore the seriousness of clinical manifestations found during cade oil poisoning in pediatric patients to raise awareness in the scientific community about this deadly situation.

2. Methods and Patients

This was a retrospective descriptive study of 5 patients admitted to the pediatric intensive care unit of a tertiary care hospital Abderrahim El Harrouchi from November first, 2022, to December first, 2022, that have been exposed, less than 10 hours before their admission, to external cade oil application.

Data collected included first the symptoms leading to the use of cade oil, then the clinical, biological, and radiologic presentations including the therapeutics used for each patient, and, finally, the clinical evolution of each case.

3. Results

3.1. Medical History

Patients were aged from 1 month to 4 years, with no prior medical history (Table 1).

The average age of our patients was 1.1 years with a female predominance and a sex ratio of 1.5:1. All of them had no prior medical history. In 3 cases, cade oil

Table 1. Medical history.

Patient	*Gender*	*Age* (*months*)	*Past medical history*	*Time of use* (*Before admission*)	*Symptoms found before application*
A	M	11	-	8 H	Fever + rhinorrhea + sneezing + cough
B	F	1	-	4 H	Fever + dyspnea + wheezing + diarrhea
C	M	2	-	6 H	Fever + cough + wheezing + atopic dermatosis
D	F	48	-	6 H	Fever + hemicorporal partial seizure
E	F	5	-	6 H	Fever + rhinorrhea + dyspnea + seborrheic dermatisis

Patient	*Chief complaint*	*Other symptoms reported*
A	Acute consciousness disorder	Abundant hematemesis + diarrhea
B	Respiratory distress	Vomiting + diarrhea
C	Respiratory distress associated with a generalized onset fever	-
D	Convulsive status epilepticus	-
E	Respiratory distress followed by acute consciousness disorder	-

was applied by parents, while for the 2 other patients, the application was done by grandparents. Two parents spontaneously declared the use of this substance, and the others recognized its use after its detection during the physical exam. In this series, all patients have been exposed to external application of cade oil which concerns the scalp, nostrils, and the sole of the foot.

Cade oil was used to treat a fever with neurological, respiratory symptoms (rhinorrhea, dyspnea, wheezing, cough, focal onset fever) (Table 1) or skin lesions. Fever is reported to be the first symptom motivating the use of cade oil, followed by respiratory symptoms in the second position. The average time between the application of the substance and the admission was 6 h. The skin surface covered by the substance was equal to all our patients and was estimated at 20%.

In our series, neurological signs remain the first complaint motivating parents to consult, 60% of our patients presenting an acute consciousness disorder, the others reporting seizures. 40% of our patients presented a digestive symptom with hematemesis, vomiting and diarrhea.

3.2. Clinical Examination

All the patients were initially admitted to pediatric emergency service where the initial physical examination was performed.

Table 2 summarizes the results of the initial examination.

Physical exam objected that all patients presented a pathological neurologic examination; 4 of them were unconscious, while one was found hypotonic and lethargic. On admission, acute respiratory distress was found in 60% of the cases. Respiratory examination was mainly abnormal. 60% of patients presented pathological auscultation and 80% of them had clinical respiratory distress signs. Only one patient was hemodynamically unstable on admission. Initially, fever was objectified in 3 patients, the 2 others were either apyretic or hypothermic.

Table 2. Initial examination.

Patient	*Vital signs*				*Neurological examination*	*Respiratory examination*	*General exam*
	BP	*HR*	*RR*	*PO*			
A	50/30	150	50	78%	Comatose patient, GCS 07/15, SRP	PA: diffused bilateral crackles	Hypothermic, Mottled skin
B	08/04	170	60	75%	obtunded, GCS 11/15, SRP	PA: diffused wheezing with fine crackles at right pulmonary apex. Intercostal and subcostal retraction, abdominal breathing Peripheral cyanosis	Apyretic
C	80/40	190	68	85%	lethargic, hypotonic, SRP	PA: diffused bilateral wheezing with crackles Nasal flaring, intercostal and subcostal retraction.	Febrile at 38.5˚C Sunken fontanel and eyes,
D	10/06	110	40	89%	Unconscious, GCS 08/15, SRP	PA: Normal	Febrile at 39.5˚C
E	09/04	130		70%	Unconscious, GCS 08/15, SRP	PA: Normal Intercostal and subcostal retraction	Febrile at 38.9˚C

BP = blood pressure cmHg, HR = heart rate ppm, RR = Respiratory rate cpm, PO = pulse oximetry (on room air), GCS = Glasgow coma scale, SRP = symmetric and reactive pupils, PA = pulmonary auscultation.

3.3. Biological Presentation

The kidney function was evaluated by serum urea, creatinine levels, and calculated creatinine clearance. Muscle enzymes measured included the creatine kinase (CK) enzyme and lactate dehydrogenase (LDH). We used transaminases (alanine transaminase and aspartate transaminase) with bilirubin levels to evaluate liver function. The procalcitonin (PCT) and C-Reactive protein (CRP) were measured as a part of the infectious and inflammatory workup. Coagulation profile included Prothrombin time (TP), activated cephalin time/activated partial thromboplastin time (APTT) and fibrinogen assay. The results are reported in Table 3.

60% of patients presented a hyperkaliemia with acute kidney failure and acute liver failure with high transaminases levels on admission. 80% of the cases presented an extensive rhabdomyolysis with very high muscles enzymes levels. Leukocytosis was reported in 4 cases while anemia was found in 2 patients. 2 patients had a disturbed coagulation profil with low prothrombin time and prolonged APTT. Gazometry (Table 4) was performed after an initial conditioning of the patients and objectified a respiratory acidosis for 2 patients, 3 cases had a low PaO_2/FiO_2 ratio.

3.4. Radiological Presentation

The radiological examinations requested were guided by the initial symptomatology of each patient. In this series, all patients benefited from a chest X-ray and a computed tomography brain scan (without contrast).

Table 3. Biological tests on admission.

Patient	*Electrolytes*	*Transaminases*	*Total bilirubin*	*Kidney function*	*Troponins*	*Muscle enzymes*	*CRP/PCT*
A	Hyperkalemia Hyperphosphoremia	Normal	-	Altered	Normal	Elevated	Elevated 160/-
B	Hyperkalemia	Elevated 9N	Elevated	Altered	Elevated	Elevated	Elevated 120/7
C	Normal	Normal	Normal	Normal	-	Normal	Elevated 80/1.4
D	Normal	Elevated 3N	Elevated	Normal	Normal	Elevated	Negative
E	Hyperkalemia	Normal	Normal	Altered	Negative	Elevated	Negative

Patient	*CBC* (*Complete Blood Count*)	*Coagulation profile*
A	Normochromic normocytic anemia Leukocytosis with neutrophilia	disturbed
B	Leukocytosis with neutrophilia	Normal
C	Leukocytosis with lymphocytosis Thrombocytosis	Normal
D	Normal	normal
E	Hypochromic microcytic anemia Leukocytosis with neutrophilia Thrombocytopenia	disturbed

N = normal, CRP (mg/L), PCT (ng/ml).

Table 4. Gasometry results.

Patient	*pH*	PaO_2	PCO_2	HCO_3^-	*Ratio* PaO_2/FiO_2
A	7.20	138	73	28	138
B	-	-	-	-	-
C	-	-	-	-	-
D	7.28	88	67	24	88
E	7.32	51	62	20	51

PaO_2/PCO_2: mmHg, HCO_3^-: mmol/l.

2 patients had a pathological brain scan, the first objectified a diffused cerebral edema while the other report intra parenchymal hematoma. The initial chest X-ray was pathological for all patients (Table 5). 2 patients had initially a severe acute respiratory distress syndrome (ARDS).

For patient D a complementary imaging was performed (Brain MRI with angiography sequences) revealing bilateral isolated cortical venous thrombosis.

Patient B had an abdominal ultrasound revealing a normal size liver, with regular contours and a hypoechoic echo structure with multiple echogenic spots creating a "starry sky" appearance suggesting acute hepatitis.

Table 5. Radiological results.

Patient	*Chest X-ray*	*Brain scan*
A	Bilateral alveolar opacities	Diffused cerebral edema
B	Systematized right apical opacity with air bronchogram and a cardiomegaly	normal
C	Bilateral perihilar infiltrates	normal
D	Right basal alveolar opacity	Hyperdense parietal and frontal lesions (intra parenchymal hematoma)
E	Bilateral alveolar opacities	normal

3.5. Treatment

All patients received supportive and symptomatic treatment.

First, skin decontamination was performed using water and soap. Mechanical ventilation was required for 4 patients who presented a consciousness disorder with a Glasgow coma scale below 8 or severe respiratory distress.

Initially, 4 patients received deep sedation with anticonvulsant treatment (A, B, D, and E). Hemodynamic support was necessary for hemodynamically unstable patients using vasoactive drugs. We used high doses of N-acetyl cysteine for the first 3 days: 150 mg/kg as loading dose then 60 mg/kg/4hours. We then proceed to correction of hydro electrolytic and acid-basics disorders. None of our patients required hemodialysis considering the conservation of diuresis and the absence of dialysis emergencies. Probabilistic antibiotic therapy was initiated according to the suspected infectious site.

3.6. Clinical Evolution

During their hospitalization, patients A, B and E developed a multiorgan failure leading to death despite the supportive treatment established.

The evolution of patient C was favorable with good clinical improvement, biological tests were normalized within 4 days, and the patient was transferred a week later to the pediatric department.

Patient D is still, to date, hospitalized in the pediatric intensive care unit dependent on mechanical ventilation.

4. Discussion

Cade oil is aromatic oil obtained from the branches and wood of Juniperus oxycedrus.

Numerous studies have reported the beneficial virtues of the latter, in particular its antimicrobial activity tested in vitro [1] its keratolytic and antipruritic action [2].

As previously mentioned, this oil contains phenols, trierpene and etheric oils [3].

Phenol is considered as the most toxic compound in this list, responsible for most systemic effects observed during cade oil poisoning [2].

The latter is easily absorbed through the skin, lungs, and gastrointestinal tract, widely distributed, and mainly metabolized in the liver via glucuronidation, sulfonation, and oxidation by CYP450 2E1 isoenzyme [4] with renal excretion.

Phenol toxicity can cause central nervous system excitation or depression responsible for neurological disorders ranging from coma to seizures [5] explaining the polymorphism of our patient's neurological state.

Severe phenol poisonings may also result in cardiovascular instability to include Cardiac dysrhythmias [6] [7] [8] such as sinus tachycardia, ventricular tachycardia, paroxysmal atrial fibrillation and severe hypotension [6] [9].

We report that phenol and cresol intoxication can be responsible for respiratory failure with acute respiratory distress syndrome (ARDS) [5] found in 3 patients included in our series. Cresol intoxication can also lead to bronchospasm with pulmonary edema in poisoned patients [10] which may explain the clinical presentation of patient C.

T. Hashimoto *et al.* [11] reported the increase of aminotransferase levels after cresol ingestion evoking probable hepato-cellular injury that can manifest even after a 24-hour asymptomatic period explaining the late onset of liver failure in patient D who had an initially normal hepatic function and presented 24 hours later a high transaminases level.

Acute kidney injury was found in most reported cases of cade oil poisoning [6] [9] [12], this one can be related to phenol's direct toxicity or result from extensive rhabdomyolysis or hemoglobinuria due to intravascular hemolysis reported during phenol poisoning [5].

Other abnormalities have been reported during cade oil poisoning cases including a decrease in prothrombin time with increased APTT [2] as found on patients A and E of our series.

Hemolytic anemia, platelet, bleeding and clotting disorders were described following exposure to phenol [2].

Exposed skin decontamination is an important step in the management of cade oil poisoning cases, skin should be copiously irrigated with soap and water, however 70% isopropanol (a common formulation of rubbing alcohol) was reported to be superior to water or soap and water for decontamination of phenol [13] [14].

N-Acetyl cysteine (NAC) is commonly used in acute hepatic failure, it provides cysteine, which is an essential precursor in glutathione production allowing to restitute glutathione reserve and neutralize free radicals, NAC by itself can binds to the toxic metabolites and eliminates free radicals [15] [16]. It also increases the blood flow and oxygen delivery to the liver and other vital organs [15] motivating its use for admitted cases.

5. Conclusion

Cade oil poisoning remains a very frequent situation in our context. The latter contains phenols known for their significant toxicity affecting multiple organs with life-threatening effects. Its high capacity of skin absorption in addition to

the reduced skin surface in the pediatric population can lead to severe poisoning cases evolving, days later, to a multiorgan failure. The increasing number of cases admitted and the seriousness of clinical presentations should lead to the regulation of the use of these substances.

Authors Contribution

The case series was written by Anass Erragh and Salma Bellaftouh. References were provided by Salma Bellaftouh, Anas Erragh, Amine Afif and Karima Amenzoui, first reviewed by Professor Kaoutar ElFakhr, approved by Professor Ouissal Aissaoui, Professor Samira Kalouch and Professor Abdelaziz Chlilek. All the authors have read and agreed to the final manuscript.

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No financial or non-financial benefits have been received or will be received from any party related directly or indirectly to the subject of this article.

Consent for Publication

Written informed consent was obtained from parents of the patients for publication of this case series and accompanying images.

Ethical Approval

Patients were diagnosed and treated according to national guidelines and agreements, our analysis looked retrospectively at outcomes of patients admitted, therefore, we did not seek ethical committee approval.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Karaman, I., Sahin, F., Güllüce, M., Ogütçü, H., Sengül, M. and Adigüzel, A. (2003) Antimicrobial Activity of Aqueous and Methanol Extracts of *Juniperus oxycedrus L.* *Journal of Ethnopharmacology*, **85**, 231-235. https://doi.org/10.1016/S0378-8741(03)00006-0

[2] Skalli, S., Chebat, A., Badrane, N. and Bencheikh, R.S. (2014) Side Effects of Cade Oil in Morocco: An Analysis of Reports in the Moroccan Herbal Products Database from 2004 to 2012. *Food and Chemical Toxicology*, **64**, 81-85. https://doi.org/10.1016/j.fct.2013.11.009

[3] Köse, H. (2000) Studies on the Germination of Some Woody Ornamental Plants Existing in Turkish Flora. III. *Juniperus oxycedrus L. Journal of Aegean Agricultural Research Institute*, **10**, 88-100.

[4] Vearrier, D., Jacobs, D. and Greenberg, M.I. (2015) Phenol Toxicity Following Cutaneous Exposure to Creolin®: A Case Report. *Journal of Medical Toxicology*, **11**, 227-231. https://doi.org/10.1007/s13181-014-0440-1

[5] Downs, J.W. and Wills, B.K. (2023) Phenol Toxicity. StatPearls Publishing, Treasure Island, FL.

[6] Haddad, L.M., Dimond, K.A. and Schweistris, J.E. (1979) Phenol Poisoning. *Journal of the American College of Emergency Physicians*, **8**, 267-269. https://doi.org/10.1016/S0361-1124(79)80222-1

[7] Gross, B.G. (1984) Cardiac Arrhythmias during Phenol Face Peeling. *Plastic and Reconstructive Surgery*, **73**, 590-594. https://doi.org/10.1097/00006534-198404000-00012

[8] Truppman, E.S. and Ellenby, J.D. (1979) Major Electrocardiographic Changes during Chemical Face Peeling. *Plastic and Reconstructive Surgery*, **63**, 44-48. https://doi.org/10.1097/00006534-197901000-00008

[9] Koruk, S.T., Ozyilkan, E., Kaya, P., Colak, D., Donderici, O. and Cesaretli, Y. (2005) Juniper Tar Poisoning. *The Journal of Clinical Toxicology*, **43**, 47-49. https://doi.org/10.1081/CLT-45072

[10] Doughmi, D., Bennis, L., Berrada, A., Derkaoui, A., Shimi, A. and Khatouf, M. (2019) Severe ARDS Complicating an Acute Intentional Cresol Poisoning. *Case Reports in Critical Care*, **2019**, Article ID: 6756352. https://doi.org/10.1155/2019/6756352

[11] Hashimoto, T., Iida, H. and Dohi, S. (1998) Marked Increases of Aminotransferase Levels after Cresol Ingestion. *The American Journal of Emergency Medicine*, **16**, 667-668. https://doi.org/10.1016/S0735-6757(98)90171-1

[12] Achour, S., Abourazzak, S., Mokhtari, A., Soulaymani, A., Soulaymani, R. and Hida, M. (2011) Juniper Tar (Cade Oil) Poisoning in New Born after a Cutaneous Application. *BMJ Case Reports*, **2011**, bcr0720114427. https://doi.org/10.1136/bcr.07.2011.4427

[13] Monteiro-Riviere, N.A., Inman, A.O., Jackson, H., Dunn. B. and Dimond, S. (2001) Efficacy of Topical Phenol Decontamination Strategies on Severity of Acute Phenol Chemical Burns and Dermal Absorption: *In Vitro* and *in Vivo* Studies in Pig Skin. *Toxicology and Industrial Health*, **17**, 95-104. https://doi.org/10.1191/0748233701th095oa

[14] Hunter, D.M., Timerding, B.L., Leonard, R.B., McCalmont, T.H. and Schwartz, E. (1992) Effects of Isopropyl Alcohol, Ethanol, and Polyethylene Glycol/Industrial Methylated Spirits in the Treatment of Acute Phenol Burns. *Annals of Emergency Medicine*, **21**, 1303-1307. https://doi.org/10.1016/S0196-0644(05)81891-8

[15] Ershad, M., Naji, A. and Vearrier, D. (2023) N Acetylcysteine. StatPearls Publishing, Treasure Island, FL.

[16] Janes, S.E.J., Price, C.S.G. and Thomas, D. (2005) Essential Oil Poisoning: N-Acetylcysteine for Eugenol-Induced Hepatic Failure and Analysis of a National Database. *European Journal of Pediatrics*, **164**, 520-522. https://doi.org/10.1007/s00431-005-1692-1