



Case Report

A rare case of cade oil poisoning complicated by acute pancreatitis and acute tubular necrosis



Mohammed Azizi^{a,*}, Abderrahim El Kaouini^a, Mohammed Amine Lafkih^a,
Mohamed Zakaria Bouayed^a, Houssam Bkiyar^{a,b}, Brahim Housni^{a,b}

^a Anesthesia, Intensive Care and Resuscitation Department, MOHAMMED VI University Hospital Center, Oujda, Morocco

^b Simulation Center, Faculty of Medicine and Pharmacy, Oujda, Morocco

ARTICLE INFO

Keywords:

Cade oil
Acute intoxication
Traditional medicine
Acute pancreatitis
Acute kidney failure

ABSTRACT

Introduction: Cade oil is often used in traditional medicinal practices despite of its toxic effects, hence the occurrence of intoxication incidents often requiring intensive care.

Case presentation: We present the case of a young patient with no prior medical history who was exposed to significant doses of Cade oil both on skin and ingested, and who subsequently developed an apyretic consciousness disorder warranting an admission to our ICU department for specialized management.

Discussion: in this chapter we discuss the place of cade oil within Morocco's unsupervised medicinal practices. We also detail the spectrum of cade oil poisoning which is rarely reported in the literature, before discussing the therapeutic options.

Conclusion: The phenol derivatives of Cade oil, which is still used frequently and widely, are responsible of an acute intoxication, mainly impairing the cardiovascular, respiratory and renal functions. A pancreatic involvement is rarely reported.

1. Introduction

Cade oil, also known as Katran oil, contains hydrocarbons and phenols. Cade Oil intoxication often occurs in the context of traditional medical practice.

Often applied through the skin and rarely taken orally, Cade oil is believed to relieve pain, digestive and/or bronchial symptoms [1]. Following a passage to the bloodstream, phenol derivatives can induce a multi-organ failure, typically involving cardiovascular and/or respiratory complications.

We report the case of a young patient, with no significant medical history, hospitalized in our department for the management of a multi-organ failure following a Cade Oil poisoning.

2. Case presentation

We report the case of a 17 years old male, with no prior medical history whatsoever, who suffered from an acute diarrhea, for which he consulted a "traditional healer" within his village, who applied Cade Oil

on the patient's neck, torso and back without any immediate skin reaction reported. The patient was also instructed to ingest 3 large spoons of Cade oil daily.

24hours following Cade Oil usage, the patient presented a loss of consciousness for which he was rushed to the ER of provincial Hospital then transferred to the Anesthesia and Resuscitation Department of Mohammed VI University Hospital. Initial assessment found an unconscious patient with a Glasgow Coma Score of 10/15 with no obvious neurological deficit and equally reactive pupils, bradycardic at 45 bpm, polypneic at 30 cpm with shallow breathing. The rest of the clinical examination was normal.

Arterial Blood Gas measurement showed a pH at 6.8, Alkaline Reserves at 5 mmol/L (Base Excess = -25) pCO₂ at 25 mmHg PO₂ at 115 mmHg with lactate level at 2.79 mmol/L.

Biological assessment showed high serum creatinine levels of 1281 μmol/L and serum urea levels at 69.6 mmol/L.

The patient also had a hyperkalemia at 7 mmol/l, with a urinary ratio at Na/K < 1.

An EKG revealed Widened QRS complexes with peaked T waves.

* Corresponding author.

E-mail addresses: Azizimed264@gmail.com (M. Azizi), abderrahimfmpo19@gmail.com (A. El Kaouini), Aminelafkih23@gmail.com (M.A. Lafkih), medzakaria.95@gmail.com (M.Z. Bouayed), 7b.Houssam@gmail.com (H. Bkiyar), brahimhousni@yahoo.fr (B. Housni).

<https://doi.org/10.1016/j.amsu.2022.103562>

Received 2 January 2022; Received in revised form 29 March 2022; Accepted 31 March 2022

Available online 4 April 2022

2049-0801/© 2022 The Authors. Published by Elsevier Ltd on behalf of IJS Publishing Group Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Table 1

Laboratory finding during icu stay.

	AT ADMISSION (H0, DAY 1)	AFTER HYPOKALEMIC MEASURES (H8, DAY 1)	AFTER IHD SESSION (H24, DAY 1)	48H OF HOSPITALIZATION (H48, DAY 2)	AFTER CVVHDF SESSION (H80, DAY 4)	PRIOR TO ICU LEAVE (DAY 6)
WBC (10 [3]/ μ l)	9,51	–	12,09	16,22	18,01	12,33
CRP (mg/l)	18,8	–	39,0	153,4	160,3	102,9
Procalcitonin (ng/ ml)	0,02	–	0,04	1,3	0,9	0,12
serum creatinine (μ mol/l)	1281	1305	483	768	253	133
Serum UREA (mmol/l)	69,6	70,1	34,1	57,3	32,8	23
kaliemia (mmol/l)	7,0	6,5	4,5	5,1	4,3	4,0
blood pH	6,80	6,90	7,30	7,15	7,34	7,41
urinary ph	–	–	–	6,1	6,7	–
Alkaline Reserves (mmol/L)	5	7	17	13	21	21
Serum lipase (U/L)	57	–	61	976	891	543
CPK (UI/l)	125	–	265	3112	1983	703
ASAT (U/L)	43	–	47	107	100	44
ALAT (U/L)	38	–	41	99	81	37

WBC=Wight Blood Count, CRP=C Reactive Protein, CPK=Creatinin Phosphokinase, ASAT = Aspartate Transaminase, ALAT = Alanine Transaminase, H=Hours, IHD=Intermittent Hemodialysis, CVVHDF=Continuous Venovenous Hemodiafiltration.



Fig. 1. Axial C+ portal venous phase Swollen pancreas, enhanced uniformly, with discrete edema in favor of a CTSP's 2 grade acute acalculous pancreatitis.

The patient benefited from hypokalemic measures (given the electrical signs of hyperkalemia) made of a bolus injection of short-acting insulin (glucose serum was simultaneously administered to avoid a hypoglycemia) and alkalization using 2 boluses of 80mL (1mL/kg) of sodium bicarbonate 8.4% with a follow-up Kalemia measurement at 6.5 mmol/l.

Given the persistent hyperkalemia associated to acidosis, and the oliguria revealed during follow-up clinical assessment, the patient was placed on intermittent hemodialysis (Duration: 4h; Blood flow rate: 200mL/min gradually increasing up to 500mL/min; Dialysate flow rate: 500mL/min; LMWH bolus 4000 UI at the initiation; Ultra-filtrate: 2L total), which normalized the Kaliemia at 4.5 mmol/L and improved the pH level to 7.3 thus improving the patient's consciousness, with a GCS at 15/15 with resumption of diuresis.

48hours later the patient complained of an acute abdominal pain located to the upper abdomen. Physical examination revealed an epigastric tenderness, the biological assessment found a Lipasemia at 7 times the normal value (976 U/L), CPK levels at 10 times the normal value (3112 U/L) and a moderate liver cytolysis (ASAT at 107 U/L, ALAT at 99 U/L) (Table 1). The cytological and biochemical

examination of urine showed myoglobinuria as well as hematuria with a urinary pH of 6,1. The patient was first hydrated using 2L of Ringer Lactate with strict monitoring of fluid inputs/outputs. The follow-up urinary pH was measured at [3,6].

A decision was made, in concertation with the nephrology department, to first perform an injected abdominal CT to confirm the diagnosis of a pancreatitis, then to put the patient under CVVHDF (Continuous Venovenous Hemo-diafiltration).

The abdominal CT revealed a diffuse enlargement of the pancreas with a discrete infiltration of the peripancreatic fat with no pancreatic necrosis consistent with a CTSP's 2 grade acute acalculous pancreatitis (Stage C of Balthazar with 0% necrosis) (Fig. 1).

The patient was put under post-dilution CCVHDF (Duration: 24h; Blood flow: 170mL/min; Dialysate flow: 1300mL/h; substitution flow: 2000mL/h; weight loss: 2000mL/h). The management also consisted of restricting of enteral feeding as long as the abdominal pain persists, pain-controlled analgesia using IV morphine.

The patient recovered a diuresis with a gradual lightning of urine and a urinary pH measured at 6.7, he was gradually weaned off morphine without any residual pain, oral feeding was resumed gradually 3 days later, his blood work showed a blood pH at 7.34, a serum creatinine level at 253 μ mol/L, a serum lipase at 891 U/L, with a CPK at 1983 U/L (Table 1).

The patient was monitored during an additional 48 hours, then transferred to the nephrology department before being discharged.

3. Discussion

Cade oil or oxycedar tar oil also known as "guetran er-raguig" is one of the most widely used oils in Morocco's medicinal practices [2]. It is obtained by dry distillation of the Juniperus oxycedrus's branches [3].

Used for therapeutic purposes, this oil can be responsible for a significant number of poisonings, which are sometime fatal [4,5]. Phenol remains the most toxic component responsible for most systemic symptoms observed during intoxication. Its absorption is fast and its metabolism is essentially hepatic.

Systemic toxicity is multi-organic and may be explained by the formation of cytotoxic metabolites. The hydroxylation of phenols produces semi-quinone radicals whose oxidation leads to the formation of toxic free radicals when the amount ingested exceeds the liver conjugation capacity. This poisoning can also result in acute tubular necrosis due to both direct cytotoxicity, hemodynamic disorders and precipitation of hemoglobin and myoglobin in the renal tubules [5].

The treatment is mainly based on rapid skin decontamination, it is done with soapy water, and should include all contaminated regions. For systemic intoxication, therapeutic management is mainly symptomatic and supports vital functions, based mainly on: the correction of hemodynamic and acid-base disorders by intravenous hydration with crystalloids and alkalization of urine, renal replacement therapies in case of severe metabolic acidosis or acute kidney failure, or even the resort to mechanical ventilation in cases of severe respiratory distress. *N*-acetylcysteine may be proposed to prevent free radical accumulation by hepatic biotransformation. Administration of methylene blue in case of methemoglobinemia.

Cases of acute renal failure have been reported in the literature [6,7] with acute tubular necrosis, which is mainly due to hemodynamic disturbances and precipitation of hemoglobin and/or myoglobin in the tubules. Hepatic cytolysis was also reported.

Acute pancreatitis is a rare complication of cade oil poisoning, which has been attributed to cade oil in our case given the absence of abdominal pain prior to ingestion of this product as well as the absence of any other somatic signs other than apyretic diarrhea.

The very significant rise in serum lipase levels with moderate cytolysis in the context of acute renal failure by acute tubular necrosis and toxic rhabdomyolysis and the likely absence of other probable etiologies (young age, correct calcium level, no notion of alcohol intake, serum triglyceride levels <1g) are in favor of our hypothesis.

The patient benefited from renal replacement therapies. Diuresis was gradually resumed. As for the acute pancreatitis, it also evolved positively. Overall, the patient's outcome was favorable.

4. Conclusion

Cade oil continues to be used in traditional medicinal practices, which sometimes leads to frequent intoxications mainly due to the phenol derivatives of the plant. The consequences are mainly cardiovascular, respiratory as well as renal. We have reported a rare case of pancreatitis due to Cade Oil skin application and ingestion, associated

with acute renal failure and rhabdomyolysis, The evolution was favorable, which we mainly attribute to the use of renal replacement therapies.

This article has been reported in line with 2020 SCARE guidelines [8].

Provenance and peer review

Not commissioned externally peer reviewed.

References

- [1] S. Benomar, A. Habzi, A. Tahri, M.S. Lahbabi, Connaissances et pratiques traditionnelles des mères en matière de soins des nouveau-nés, Arch pédiatrie 169 (7) (2000) 713–789. <http://www.sciencedirect.com/science/article/pii/S0929693X00801633>.
- [2] I. Teixidor-Toneu, G.J. Martin, R.K. Puri, A. Ouhammou, J.A. Hawkins, Treating infants with frigg: linking disease aetiologies, medicinal plant use and care-seeking behaviour in southern Morocco, J. Ethnobiol. Ethnomed. 13 (1) (2017) 4, <https://doi.org/10.1186/s13002-016-0129-4>.
- [3] G. Mazars, Pharmacopée traditionnelle du Maroc : Jamal Bellakhdar, La pharmacopée marocaine traditionnelle, in: Médecine arabe ancienne et savoirs populaires, N 320, Rev d'histoire la Pharm 86e année, 1998. Published online 1998, https://www.persee.fr/doc/pharm_0035-2349_1998_num_86_320_4719_t1_0465_0000_3.
- [4] S.T. Koruk, E. Ozyilkan, P. Kaya, D. Colak, O. Donderici, Y. Cesaretli, Juniper tar poisoning, Clin Toxicol (Phila). 43 (1) (2005) 47–49, <https://doi.org/10.1081/CLT-45072>.
- [5] A. Sanae, N. Rhalem, S.S.S. Abourazzak, R. Bencheikh, Intoxication mortelle à l'huile de cade à propos de deux cas, Urgence Prat (2008). Published online January 1.
- [6] A. Nejmeddine, INTOXICATIONS DU NOUVEAU-NE CAUSEES PAR LA « FERRAGA » : EXPERIENCE DU SERVICE DE REANIMATION NEONATALE, Published online, <http://wd.fmpm.uca.ma/biblio/theses/annee-htm/FT/2019/these50-19.pdf>, 1993.
- [7] K. Hafidi, Intoxications par les plantes et les produits de la pharmacopée traditionnelle chez l'enfant, Published online, <http://www.chu-fes.ma/intoxications-par-les-plantes-et-les-produits-de-la-pharmacopée-traditionnelle-chez-lenfant/>, 2014.
- [8] R.A. Agha, T. Franchi, C. Sohrabi, G. Mathew, A. Kerwan, The SCARE 2020 guideline: updating consensus surgical CAse REport (SCARE) guidelines, Int. J. Surg. 84 (2020) 226–230, <https://doi.org/10.1016/j.ijsu.2020.10.034>.