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Case Series

Cade Oil Poisoning: A Report of Three Clinical Cases

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Abstract

Cade oil, extracted by distillation from the branches of *Juniperus oxycedrus*, continues to be used in traditional medicine, topically or orally despite its toxicity. This oil can be responsible for fatal poisoning, due to its high phenol content, which affects several systems.

Three cases of poisoning by this oil were reported: a 4-year-old child, admitted for a state of convulsive pain whose imaging was in favor of a reversible posterior encephalopathy syndrome (PRES syndrome), a 15-month-old infant admitted in a state of multiorgan failure and a 2-year-old infant, admitted for bradycardia. The three cases were put under resuscitation measures with good progress.

Keywords: *Cade Oil Poisoning; PRES Syndrome; Phenol Content*

Introduction

Cade oil is an oil widely used in traditional Moroccan medicine, it has different pharmacological properties: keratolytic, healing and antifungal. It is used in the preparation of some cosmetic products and is used in veterinary medicine [1]. Poisoning with this oil results from the ingestion of a large quantity, or from prolonged and extensive dermal application.

This oil can be responsible for fatal poisoning due to the high proportion of hydrocarbons and phenols, the latter being the most toxic component responsible for the majority of systemic symptoms [2].

According to data from the Moroccan Poison Control and Pharmacovigilance Center (CAPM), between 2008 and 2018, forty-six cases of poisoning were noted, including 7 deaths, i.e. a case fatality rate of 13% [3].

Case Reports

Observation N° 1

4-year-old child, female, admitted for a state of apyretic convulsive disease with application of cade oil on the forehead, eye contours and nose following a febrile episode.

The admission examination found a child obsessed with tachycardium at 100 bpm, polypneic at 40 cycles/min apyretic, SO_2 at 97% in ambient air, TRC<3s, ROT present, blood glucose at 0.9 g/l, BP 9/7 mmHg.

Paraclinical investigations: brain CT scan was normal, CSF normal, CRP 33 mg/l, WBC at 10000 e/mm, PNN 9780 e/mm, hepatic cytolysis: ASAT 9981 u/l, ALAT 3263 u/l, PAL 400 u/l, GT 120 u/l, normal hemostasis workup, blood glucose 1.1 g/l, CPK at 700 U/L, troponin 4 ng/l, renal failure; Urea 0.76 g/l, blood creatinine 12.1 mg/l, negative urine drug test.

The child benefited from conditioning and monitoring, rehydration, the use of N-acetylcysteine, anticonvulsant treatment, skin decontamination, filling with diuresis monitoring.

During the hospitalization, the child presented blindness, a brain MRI showed an aspect of a PRESS syndrome involving the posterior occipital and parietal region and to a lesser extent the frontal region. The fundus was normal.

The evolution was marked by the spontaneous recovery of vision with normalization of disturbed biological values.

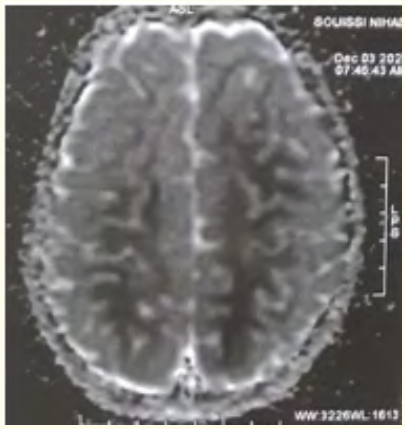

Figure 1: *Brain MRI diffusion sequence shows an occipito-parietal hypersignal.*

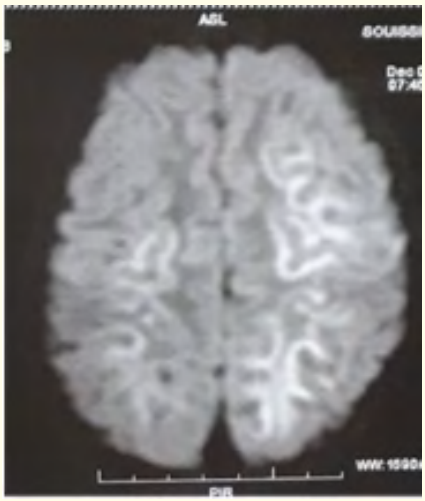

Figure 2: *Brain MRI flair sequence shows an occipito-parietal hypersignal.*

Observation No. 2

15-month-old female infant, notion of skin application of cade oil on the face to treat measles. A few hours later, she developed asthenia and hypotonia. The clinical examination found a patient who was obsessed with slightly reactive blood pressure, bradycardium at 60 beats/min, BP 9/6 mmHg, oxygen saturation was normal.

The laboratory work-up was normal: CPK, troponin, renal and hepatic function, ionogram and PT.

In the case of bradycardia, cardiac exploration revealed a type I atrioventricular block.

The child benefited from conditioning with skin decontamination and strict supervision. The course was marked by the worsening of bradycardia up to 54 beat/min requiring the administration of atropine.

The evolution was good with the normalization of heart rate and neurological status.

Observation N° 3

2-year-old infant, male, good psychomotor development, admitted for a state of apyretic convulsive pain with the notion of application of cade oil to the mother's nipple to initiate weaning from breastfeeding.

On admission the obsessed infant, with cold extremities, BP at 80/60 mmHg, tachycardium at 150 bpm, polypneic at 50 cycles/min, SO_2 77% AA, TRC>3 s, hypoglycemia at 0.4 g/l.

The paraclinical assessment: cerebral CT showed vasogenic diffuse edema, normal CBC, CRP at 3 mg/l, ASAT 239 u/l, ALAT 178 u/l, normal hemostasis workup, LDH at 673 u/l, CPK at 2659 U/L, RA 9.2 meq/l, renal failure; urea 1.23g/l, blood creatinine 11.4 mg/l, NA+ 126 meq/l K+ 4 meq/l, troponin 6.2 ng/l, normal methemoglobinemia.

The child benefited from conditioning and monitoring, filling, correction of hydroelectrolyte disorders, administration of bicarbonate, and monitoring of diuresis.

The evolution was marked by the normalization of disturbed biological values.

Discussion

Cade oil is widely used in Morocco for therapeutic purposes. Phenol remains the most toxic component, its absorption is rapid and its metabolism is essentially hepatic. The renal and hepatic toxicity of phenol has been proven [4], in addition to the systemic symptoms due to the hydroxylation of phenols which produce semi-quinone radicals whose oxidation leads to the formation of toxic free radicals when the quantity ingested exceeds the liver's capacity for conjugation [5].

Systemic effects include alteration of the nervous system by seizures, hypotonia, mental confusion, hepatic cytolysis by centrilobular necrosis [6,7].

Methemoglobinemia, hemolytic anemia, haematological abnormalities or metabolic acidosis may also be found [8].

Cardiovascular disorders such as sinus bradycardia, myocardial excitability disorders, arterial hypotension, and cardiovascular collapse have been reported in the literature [5,9,10]. In the 2nd reported case, the infant presented with severe sinus bradycardia which necessitated the administration of atropine.

Studies have shown that toxic doses of phenol cause initial high blood pressure, followed by a marked drop in this pressure, this is due to a depression of the central vasomotor system leading to a loss of vasoconstrictor tone, and also a decrease in cardiac output secondary to a direct effect of phenol on the myocardium [11]. This explains the complication noted in our case, responsible for the press syndrome which resulted in blindness that was fortunately reversible.

Acute kidney injury is considered one of the main side effects of phenol [7]. This is a side effect with a poor prognosis, which has been documented in 2 of our patients, and which has progressed favorably under symptomatic treatment.

Respiratory and psychiatric disorders have also been reported in the literature [12].

Treatment of poisoning is based on rapid skin decontamination with soapy water. For systemic forms, therapeutic management is essentially symptomatic. Haemodialysis is indicated in cases of anuric renal insufficiency, the use of N-acetylcysteine is used to neutralise free radicals resulting from hepatic biotransformation [8], and the administration of methylene blue is indicated in cases of methemoglobinemia.

Due to its many beneficial effects, N-acetylcysteine is recommended by some poison control centers in the treatment of poisoning from essential oils such as penny mint oil, clove essential oil, phenols and hepatotoxic plants, due to the fact that it contains glutathione levels, antioxidant properties that act as free radical scavengers [13].

The prognosis of cade oil poisoning depends on the age of the patient, the route of exposure, the concentration of phenol and the duration of use.

In the literature, ingestion of 50 to 500 mg of phenol is fatal, and during dermal application, the prognosis depends on the amount applied and the duration of exposure [4].

Conclusion

Poisoning by cade oil remains a public health issue in areas where its traditional use is still common. However, its misuse or inappropriate use can cause severe toxicity.

Prevention is based on increased awareness and clear public information about the risks associated with its unregulated use. Management is essentially symptomatic, and the prognosis is generally favourable when treatment is started early.

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