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Case Report

Fatal Collective Intoxication with Glue Thistle *(Atractylis gummifera)* about 2 Cases

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ABSTRACT

Atractylis gummifera intoxication is a serious and often fatal intoxication if not treated early and effectively. It constitutes a real problem of Public health especially for the child population. We report the case of a collective intoxication following the accidental ingestion of *Atractylis gummifera* root by two children aged 5 and 10 years old. In order to take stock of this intoxication whose diagnosis is clinical, the treatment is symptomatic and prevention is paramount.

Keywords: *Atractylis gummifera*; Collective intoxication; Children

INTRODUCTION

Atractylis gummifera L is a plant of the Asteraceae family known as glue thistle, the Arabs call it Addad or chouk el-eulk (chewing glue thistle) [1]. It is a herbaceous, thorny and fragrant plant, perennial by its underground part (Figure 1), which is found in the Mediterranean region, in North Africa and Southern Europe [2,3]. It strongly resembles the wild artichoke and is responsible each year for many cases of poisoning, most often serious and fatal [3] caused mainly by the ingestion of the root, especially fresh [1]. In Morocco, this plant is most often available from herbalists, but it also grows in the wild, making it accessible especially to rural children, who are the most affected age group [4]. Through two clinical cases of collective and accidental intoxication of two children (brother and sister) collected in the emergency department of the Mohamed V hospital of Tangier, we make a point on this intoxication, whose diagnosis is clinical, the treatment is symptomatic and whose prevention remains the main remedy.

Figure 1: *Atractylis gummifera*: National Museum of Natural History.

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CASE REPORT

Observation no 1

A 5 years old girl, without any particular pathological antecedent living in a rural area, was admitted one evening in spring, to the emergency room of the hospital Mohamed V of Tangier for the treatment of a convulsive disorder. The interrogation of her parents revealed the acute onset of a generalized tonicoclonic seizure without regaining consciousness after an accidental ingestion 48 hours previously of an unknown quantity of the root of a wild plant, pulled up in the fields that the parents brought back with them. On admission the patient was comatose with a Glasgow score of 5 pupils in reactive bilateral mydriasis. Her blood pressure was 112/65 mm of Hg and her heart rate and respiratory rate were 120 beats/minute and 20 cycles/minute respectively. Cardiothoracic auscultation revealed diffuse bilateral snoring rales the child was also apyretic, with cutaneous and conjunctival jaundice, dark and sparse urine when catheterized. The abdominal examination was without abnormality with no particular smell of breath. Capillary blood glucose was 0.34 g/L. Five min after admission the child presented a generalized tonic-clonic seizure. The immediate treatment consisted of 30% glucose serum, intrarectal valium followed by IV phenobarbital in a loading dose, due to the persistence of the crisis the patient was put on mechanical ventilation, volume expansion with a physiological NaCl solution and deep sedation with benzodiazepines and morphinomimetics.

The poison control center was contacted and confirmed that it is *Atractylis gummifera*. So the girl received a loading dose and then maintenance doses of N acetyl cysteine. Blood gases were in favor of a deep metabolic acidosis with a high anion gap (pH=7.02, PCO_2=33.3 mm of Hg/HCO_3=8.5 mmol/l). The bioassay revealed a Hb at 12 g/dl, WBC at 18,000 mm^3, a collapsed prothrombin level of 20%, impaired renal function (uremia at 0.37 g/L, creatinine at 16 mg/L), intense cytolysis (Aspartate Amino-Transferase (ASAT) at 4163 IU/L, Alanine Amino-Transferase (ALAT) at 2390) and rhabdomyolysis (Creatine Phosphokinase (CPK) at 2900 IU/L). The serum electrolytes showed: Hypocalcemia at 60 mg/L, hyponatremia at 125 mmol/L and hyperkalemia at 6 mmol/L without electrical signs on the electrocardiogram, motivating the use of alkalinization with bicarbonate solution, and insulin in combination with a 10% glucose solution. Four hours later the girl presented a hemodynamic instability resistant to filling, stabilized by the introduction of vasoactive drugs. The evolution was fatal with the installation of multiple organ failure (Renal, Hemodynamic, Neurological and Hepatic).

Observation no 2

A 10-year-old boy with no notable history was admitted to the emergency room of Mohamed V Hospital, Four hours after his sister's admission for treatment of a seizure disorder. On admission the patient was in the midst of a generalized tonic-clonic seizure, pupil in reactive bilateral mydriasis, hemodynamically unstable; His blood pressure was 70/45 mm of Hg, heart rate was 120 beats per minute and respiratory rate was and 27 cycles/min. In addition, the child was apyretic; Capillary blood glucose was 0.46 g/L. Immediate management consisted of administration of glucose serum at 30%, intrarectal valium with lateral safety position followed by IV phenobarbital in loading dose with volume expansion by a crystalloid, the patient was put on mechanical ventilation due to the persistence of the convulsive crisis with deep sedation with benzodiazepines and morphinomimetics. With the same symptomatology as his sister, the child also received N acetylcysteine. The rest of the examination showed the existence of bilateral diffuse snore rales on cardiothoracic auscultation, cutaneous and conjunctival jaundice, and dark and sparse urine on bladder catheterization. The abdominal examination was without abnormality with no particular odor of breath. The blood gases showed a deep metabolic acidosis with a high anion hole' (pH=7.17/ PCO_2=30/HCO_3=7). The bioassay revealed Hb at 12 g/dl, WBC: 10,000/mm^3, prothrombin level collapsed at 21%, impaired renal function (uremia 0.52 g/L, creatinine at 18 mg/L, intense cytolysis ASAT at 3500 IU/L, ALAT at 2700 and rhabdomyolysis with CPK at 3700 IU/L). The serum electrolytes showed: hypocalcemia at 70 mg/L, hyponatremia at 129 mmol/L and hyperkalemia at 6.9 mmol/L with no electrical signs on the electrocardiogram, motivating the use of alkalinization with bicarbonate solution, and insulin in combination with a 10% glucose solution. The child is recovered from a first cardiac arrest by cardiac massage and injection of 0.5 mg of adrenaline but died after a second cardiac arrest five minutes later due to a multiple organ failure (hepatic, Renal, Hemodynamic and Respiratory).

DISCUSSION

Plant poisoning is still a worrying phenomenon due to its seriousness and the number of victims declared, thus presenting a real public health problem. According to recent statistics from the Moroccan Poison Control and Pharmacovigilance Center, plants and traditional pharmacopoeia products rank tenth among the causes of poisoning in Morocco [5]. Glue thistle is classified as a highly toxic plant [6] and is in fact one of the plants most often involved in fatal poisonings in Morocco and most Mediterranean countries [7] either by ignorance of its toxicity with abusive use for therapeutic purposes or by confusion with another plant, *Scolymus Hispanicus*, better known under the name of "*kouk lakhla*" or *Guernina* [8]. This confusion is more pronounced, especially among children, whose curiosity leads them to examine all parts of this plant with their hands and especially to taste it with their mouths. Even chewing the rhizome like a gum [9]. Between 1981 and 2008, 344 cases of glue thistle intoxication were declared by the Moroccan Poison Control Center (MPCC) [10]. These are mainly accidental poisonings involving mainly rural children under the age of 15 years with 64.65% of the cases whose collective component represents 54.15% of the cases, with 68 cases of death by this plant that were reported among the 344 cases, the majority were from rural areas. All parts of the plant contain the toxic

principles of the glue thistle. Which are, classified in order of decreasing concentration from the root, through the stem, the bracts, the flower, the seed and finally the leaf. The aerial parts of the plant are the least toxic [8-11]. The toxicity of the root is very high, only 50 g of root pulp can kill a 6 kg dog in less than 24 hours [10], while about 480 mg of root can be fatal for a 60 kg individual [1].

The toxic principles of *Atractylis gummifera L. Atractyloside and carboxyatractyloside or gummiferin*, are mitochondrial poisons that block the oxidation-reduction phenomena involved in the formation of ATP. Their mode of action is at the origin of the increase in glucose consumption, the depletion of the hepatic and muscular glycogen stores and the inhibition of glycogen genesis, which explain the hypoglycemia encountered in severe cases [12,13].

The clinical picture of intoxication has several phases [1].

The latency phase from 6 to 24 hours or even 36 hours before the clinical picture is established

The symptomatic phase, which includes several clinical disorders:

- The digestive disorders: represented by nausea, vomiting, liquid diarrhea, blackish or rectorrhea, abdominal pain and bloating [2].
- Neurological disorders: once installed, they have a poor prognosis and can go as far as coma, which is accompanied by contractures, decerebration rigidity, trismus, collapse, congestion, hypersalivation with apnea in the final stage [13].
- Neurovegetative disorders: Often noted, they are represented by hypothermia.

The complications phase very poor prognosis. It includes respiratory disorders marked by progressive dyspnea and even asphyxia, cardiovascular disorders with tachycardia, rhythm disorder and low blood pressure, acute renal failure and fulminant hepatitis with very important cytolysis. Our two reported cases were already admitted in the complications phase with neurological disorders represented by convulsions and coma, cardiovascular disorders: tachycardia and hypotension, very severe cytolysis and rhabdomyolysis associated with acute renal failure. The detection of atractyloside, by thin layer chromatography, can be done either in the urine, in gastric lavage fluid or in the blood on silica gel [14]. The diagnosis of glue thistle intoxication is essentially based on the clinical history, the symptomatology and the detection of toxins of the plant in the gastric aspiration liquid or in the urine [1]. The diagnosis of glue thistle intoxication in our two reported cases was made in front of the onset of a state of apyretic convulsive disorder of a child living in rural areas after ingestion of an indeterminate quantity of the root of a plant torn from nature that the parents bring back with them, which helped confirmed the diagnosis. It is therefore advisable to think of intoxication with this plant in rural areas around the Mediterranean basin, in front of any acute convulsive coma associated with hypoglycemia, cytolysis with a collapse of the prothrombin level. These abnormalities are constant and should evoke he diagnosis [15]. At present, therapeutic management is essentially based on symptomatic and evacuating treatment, in the absence of a specific antidote. Digestive evacuation should be carried out as soon as possible by gastric lavage, administration of activated charcoal to reduce intestinal absorption of toxins and acceleration of intestinal transit.

Symptomatic treatment, always to be instituted as a priority, includes volume expansion, glucose loading, alkalinization in case of severe acidosis, coagulation factor intake, control of respiratory function, and administration of vasoactive drugs when necessary [16]. Nevertheless, subjects who have absorbed theoretically lethal amounts of the poison, Symptomatic treatment is always insufficient. Other therapeutic means have been described in the literature such as verapamil, dithiothreitol and N-acetylcysteine, compounds which may protect against toxic effects but provided they are administered before exposure [7]. Current research is exploring the route of immunotherapy [17]. Others are currently discussing the possibility of producing and using specific antibodies against the toxic components of *Atractylis gummifera L* [7,16], in the most severe situations liver transplantation may be considered [17].

Finally, prevention remains the most effective tool for dealing with this intoxication, which is still frequent and responsible for several deaths each year, especially in the child population, and is based essentially on raising awareness and informing families about the various dangers that their children can run. Thus health professionals are brought to know this intoxication to undertake early and effective management and recourse to the Poison Control Center in case of doubt. This remedy has contributed to reducing the number of deaths due to poisoning by this plant for several years in Morocco.

CONCLUSION

Glue thistle poisoning is still common in Morocco, most often accidental and sometimes fatal, especially for children in rural areas. The treatment is essentially symptomatic but unfortunately remains insufficient in serious intoxications, hence the interest of primary prevention, a remedy based on sensitization and information of the population in order to minimize or even avoid the occurrence of this dreadful intoxication.

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