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RESEARCH ARTICLE

INTEREST OF EARLY TOXICOLOGICAL SAMPLING DURING ALPHACHLORALOSE INTOXICATION , A CASE REPORT

Yasmine Aouam[1,2], Sabah Benhamza[1,2], Kenza Damaane[1,2], Mohamed Lazraq[1,2], Youssef Miloudi[1,2], GhizlaneBerdai[2,3], Imane Rahmoune[2,3], Ahlam Meftah[1,2,3], Houda Filali[2,3], Ghizlane Msik [1,2], Abdelali Bennaji[1,2] Abdelhak Bensaid[1,2]

1. Department of Anesthesia and Intensive Care, Hôpital 20 Août 1953.
2. Hassan II University, Faculty of Medicine and Pharmacy, Casablanca.
3. Pharmacology and ClinicalToxicologyLaboratory.

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Abstract

Alphachloralosis intoxication is very common and constitutes a real public health problem. Diagnosis is difficult because of its aspecific symptoms, which may erroneously point to other pathologies, hence the importance of good questioning. The clinical picture is suggestive of disturbed consciousness, myoclonus, sharp osteotendinous reflexes with changes in pupillary diameter, hypersialorrhoea and bronchial hypersecretion. A definitive diagnosis is based on the detection of alphachloralose in gastric fluid, urine and blood. Toxicological samples must be taken as soon as possible after ingestion, otherwise they will be negative. Progress is usually favourable, and the severity of intoxication depends on the dose ingested. Treatment is mainly symptomatic and is based on evacuation of the toxic substance, protection of the airways and control of myoclonus .

Introduction:-

Alphachloralose poisoning is most common in developing countries. Both a stimulant and a depressant of the central nervous system, alphachloralose was long used as a hypnotic and was withdrawn from the market in the 1980s. It is currently used as a rodenticide or taupicide in the form of powder or grains of varying concentration [1].

In Morocco, alphachloralose is a rodenticide available in 3 g or 7 g grain sachets, or in packs of 3 g or 9 g sachets, under several trade names (Raticidose®, Raticide 50®, Ratoxide®). It is on sale in grocery stores, pharmacies and rural souks at a modest price. Because of its easy availability and lack of regulation and control of sales, it is implicated in a large number of intoxications, both accidental and deliberate. All brands of alphachloralose rodenticide are for professional use only [2]. According to the CAPM annual report in 2014, alphachloralose occupies the second leading cause of pesticide poisoning in Morocco with a percentage of 18.8%. Thus, according to the number of pharmaco-toxicological analyses carried out at the laboratory of the Hassan II University Hospital in Fez in 2020, 43 positive chloralose cases out of a total of 201 samples were intended for pesticide testing [3]. The toxic dose is estimated at around 1 g in adults, 20 mg/kg in children and the lethal dose at 0.1 g/kg, with wide individual variations. The severity of clinical signs is proportional to the dose ingested.
We report the case of a young patient suffering from voluntary alphachloralose intoxication in order to highlight the importance of early toxicological sampling and the regulation of rodenticide sales in Morocco.

Corresponding Author:- Yasmine Aouam
Address:- Department of Anesthesia and Intensive Care, Hôpital 20 Août 1953.

Clinical observation :

We report the case of Mr. O.E, 30 years old, treated for schizophrenia for 2 years with Olanzapine and for depression with Amilzulpride, who presented to the emergency department at H2 after ingesting 5 packets of alphachloralose-based rat poison, as reported by his family.

Initial clinical examination revealed a comatose patient with an initial Glasgow score estimated at 6/15, pupils miosis, no sensitivomotor deficit, polypnea at 25 cycles per minute, pulsed O2 saturation at 90% in free air with no signs of respiratory struggle, hypersialorrhea and bronchial hypersecretion. Blood pressure was 110/60 mmHg, heart rate 65 beats/min, capillary glucose 0.9 mmol/L and temperature 35.6 C. Initial management consisted of intubation with mechanical ventilation, rewarming, administration of activated charcoal and copious gastric lavage. Neurological examination after stabilization revealed no hypertonia, osteotendinous reflexes were normal and the Babinski sign was negative. The patient also showed conjunctival hyperemia without skin rash.

Symptomatology was marked by the occurrence, on day 2 of ingestion, of two convulsive seizures lasting less than 2 min each and 20 min apart, predominating in the face , upper limbs and subsequently generalizing, despite sedation with midazolam at a dose of 5mg/hr. The patient received a loading dose of phenobarbital 10mg/Kg, after which the seizures ceased, and was then put on sodium valproate anticonvulsant therapy. Hemodynamically, the patient was hypotensive (08/46 mmHg), unresponsive to filling and requiring the introduction of vasoactive drugs, and had a plateau fever. Bacteriological samples were taken from the patient's lungs, urine and blood, and a lumbar puncture was performed, all of which were negative. The lactatemia level remained normal.

Given this notion of convulsions, a cerebral CT scan was performed, revealing no abnormalities, The biological work-up revealed an isolated hyperleukocytosis with no other abnormalities on the haemogram. Biological markers of inflammation (Procalcitonin and C-reactiveProtein) were negative, liver, kidney functions and blood ionograms were also normal. Toxicological tests carried out 24 h post ingestion on urine samples diluted by LC-MS-MS and blood samples were negative. The patient improved neurologically, hemodynamically and respiratorily, and was extubated after 6 days of mechanical ventilation. The patient was transferred to the psychiatric ward for further treatment.

Discussion:-

Alpha-chloralose acts primarily on the central nervous system. The hypnotic action results from a decrease in the activity of the ascending activator system. The excitatory action, on the other hand, is explained by an action on the two parameters of the cortical excitability cycle (shortening of neuronal recovery time and increase in neuronal synchronization) [4]. Neurological manifestations can range from a disturbance of consciousness to a calm chlorasic coma, often followed by a state of hyperexcitability with the appearance of myoclonus, predominantly of the face and limbs. Sometimes these myoclonus may be generalized and asynchronous, simulating a convulsive seizure [4], but it is difficult to distinguish between the two clinically. Hemodynamically, alphachloralose has a negative inotropic effect, while hemodynamic function remains clinically preserved. We also find sinus tachycardia, moderate hypotension and, in rare cases, shock in the event of massive intoxication.This was the case in our patient. Tracheobronchial hypersecretion is one of the most striking signs of alphachloralose intoxication, its association with the evocative context and the rest of the symptomatology, namely coma and myoclonus. In some cases, this hypersecretion is responsible for pulmonary congestion, the main complication of this intoxication [5], which was the case in our patient. Other clinical signs include hypothermia, mucocutaneous signs such as erythematous skin eruptions and conjunctival hyperemia, although these are relatively rare. Increased muscle tone, the presence of vivid, diffuse and polykinetic osteotendinous reflexes and a positive Babinski sign are very favourable, the latter not having been reported in our case. [6]

Being rapidly absorbed, the time to onset of signs varies from a few minutes to a few hours, depending on the dose ingested, the presentation of the product, associated toxic substances, the state of gastric repletion and inter-individual factors. [2]

On the paraclinical level, there are no biological abnormalities specific to this type of intoxication reported, although hyperleukocytosis may be found out side any infectious context as well as rhabdomyolysis [4]. Blood gases show hypoxemia in relation to bronchial congestion and metabolic lactic acidosis in relation to myoclonus [4]. Chloralose

has a large volume of distribution and accumulates in the kidneys, liver and central nervous system. It is 90% eliminated in the urine in conjugated form. There is no cumulative action. [3], [4]. In our patient, a toxicological work-up was carried out on principle, given the notion of psychiatric illness and the suspicion of intoxication with other substances, after 24 hours of ingestion. The results were negative, as 45% of the ingested dose was eliminated within 24 hours, with a short half-life of 5 hours, which explains the drop in alphachloralose concentrations in the urine, making it undetectable. This underlines the importance of the delay between ingestion and toxicological sampling.The electroencephalogram (EEG) reveals as lowed trace with delta waves of 2-3 cycles/s of high voltage, predominantly frontal and inconstantly symmetrical and synchronous spike-waves [5]. This typical trace may be replaced by zero activity in the form of a flat trace during massive chloralose intoxication [4].

Treatment is mainly symptomatic, aimed at maintaining vital functions and preventing myoclonus and convulsions, so intubation and mechanical ventilation are often essential in the first 24 hours. For myoclonus, benzodiazepines are the first molecules used, and are often effective. In some cases, other anticonvulsants are required, notably barbiturates and propofol [6],

Evacuatory treatment is also essential in the first few hours, activated charcoal is recommended for the first two hours after ingestion [5]. Gastric lavage can be performed, but must be abundant (10 to 15l). However, there is no formal consensus on the value of gastric lavage in cases of acute intoxication by Alphachloralose .

In most cases, the disease is cured without sequelae. Mortality following rat poisonings is low [6]. Coma is generally short-lived, lasting 8 to 12 hours, although in some cases it may be prolonged. The course of the disease depends above all on the dose ingested, but also on how early the patient is treated. Our patient's delayed neurological recovery and cardiovascular signs suggested a high dose, but fortunately we observed full recovery.

Conclusion:-

Alphachloralose intoxication is frequent and can be serious. A national strategy should be put in place to inform the population about the risks of alphachloralose, to inform health professionals about the properties of this toxic substance, and to stress the importance of early diagnosis and treatment. Finally, we need to regulate and control the marketing of this product.

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