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Acute Intoxication in Pregnant Women: Epidemiology and Management

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Abstract **Case Report**

Intoxication is a frequent reason for consultation in the emergency department, and may be voluntary, with the aim of self-medication, or accidental. However, intoxication in pregnant women is poorly described in the literature, with the majority of reports concerning case histories. This study analyses the epidemiological data on intoxication in pregnant women in the mother and child intensive care unit, describes the various clinical and toxic aspects, describes the management methods and assesses the maternal-fetal prognosis of intoxication in pregnant women.

Keywords: Intoxication, self-medication, child intensive care unit.

INTRODUCTION

Poisoning occurs when the body is exposed to a toxic agent through ingestion, inhalation, skin exposure, rectal or parenteral administration. Toxic agents may be chemicals, drugs or biological products of animal or plant origin [1].

However, intoxication in pregnant women is rarely described in the literature, with the majority of case reports.

Pregnancy is a veritable identity crisis, during which each woman mobilizes all her capacities to adapt to the changes she is confronted with; it is both a moment of physiological and psychological transformation. An unwanted pregnancy will be a source of stress for the mother [2].

Efforts to get rid of the unexpected can range from simple curettage to suicide attempts.

Description of the means used to study toxic substances and how they are managed in our department. Evaluation of the maternal-fetal prognosis of intoxication in pregnant women.

CLINICAL OBSERVATION

This is an 18-year-old, non-smoking patient primigravida at 16 SA. Admitted to emergency after being discovered unconscious in the shower (about 1 hour of exposure according to the family), the use of a non-compliant water heater led to the diagnosis of carbon monoxide intoxication. On admission to hospital, the patient was drowsy GCS: 13, apathetic, PEER to light, complaining of chest pain, tachycardic at 120 beats per minute, blood pressure 120/70mmhg, polypneic at 25 cycles per minute, conjunctivae normo-coloured; the gynecological-obstetrical examination was normal, notably normal reactive.

The electrocardiogram showed sinus tachycardia with no conduction or repolarization disturbances, and the cardiac Doppler echo was unremarkable, with normal systolic function and EF ejection fraction at 65%. The patient was started on high-concentration mask oxygen therapy at 12l/min on admission (HBOT hyperbaric oxygen therapy was not available).

The state of consciousness was normal after 1 hour of oxygen therapy, with good orientation in time and space and no notion of anterograde amnesia or other neuropsychic signs.

On admission to hospital, the patient was conscious GCS: 15; PEER, normocardic at 90 beats per minute, polypneic at 25 cycles per minute with BP at 13/07, conjunctivae normocolored; absence of bronchial hypersecretion; gynecological-obstetrical examination normal with no uterine contractions or metrorrhagia, with a normal, responsive FHR, and no other signs suggestive of fetal distress or abortion; the rest of the clinical examination was unremarkable.

After conditioning and monitoring: spo2:98% on room air, BP130/70 mmhg, HR:92bpm; the patient underwent gastric lavage at H2 of intoxication with 5l of saline; toxicological samples were taken and returned negative; the biological work-up revealed: Hb:9.9g/dl, PLQ:179000, correct renal function urea: 0.26, crea:7 without hepatic cytolysis; Troponin negative CPK: 82UI/l, CPKMB:26UI/l.

The patient was treated symptomatically with 2l/min oxygen therapy, RDB, antagonism and iron supplementation, with good clinical improvement.

Control laboratory tests remained normal; the patient was discharged from intensive care after 72 hours of hospitalization on iron supplementation.

Patient aged 24, single, with no notable pathological history, pregnant at 22 weeks' gestation, admitted to emergency after ingesting a large quantity of Harmel, considered a traditional remedy for abortion.

On admission to hospital, the patient was obnubilated with a GCS of 12/15, PEER to light, apyretic, hemodynamically stable: HR 60bpm, BP 110/70mmhg, polypneic at 30C/min, Sao2:95% on oxygen mask under 6l/min with concentrated urine.

The patient was transferred to intensive care, and hemodialysis was performed secondarily, following a decrease in diuresis to 0.4ml/kg/k.

The following day, the patient presented with a deterioration in GCS to 10, associated with hypoventilation, stagnation of bronchial secretions and hypercapnia on gasometry, prompting intubation with artificial ventilation and sedation with 0.3mg/kg/h of midazolam; two days later, the fetus died, and expulsion was carried out without haemorrhage.

The evolution was marked by an improvement in renal function and diuresis to 1ml/kg/h under 80mg furosemide.

On the tenth day of hospitalization, the patient was extubated after assessment of her neurological condition; the laboratory work-up was normalized. Clinical examination revealed a mild distal sensory deficit, and a significant motor deficit rated at 2/5 involving the extremities. She benefited from intensive motor physiotherapy for 2 months while in hospital.

The patient was 22 years old, G2P1 (G1 miscarriage 4 months), currently estimated at 22 SA, with a history of multiple attempts at autolysis.

Admitted to the emergency department for attempted suicide after deliberately ingesting 2 tablets of rat poison (cypermethrin), causing dizziness, nausea, vomiting and abdominal pain.

Radiological investigations, including electrocardiography, chest X-ray and abdominal ultrasound, were normal.

Initial laboratory work-up was normal: Hb 12g/dl, urea 0.45g/l, creatinine 10mg/l without hepatic cytolysis; cardiac enzymes negative.

The patient was treated symptomatically with oxygen therapy at 2l/min, RDB, antagon with good clinical improvement.

The patient was discharged from the intensive care unit after 72 hours of hospitalization, with a psychiatric opinion, without sequelae or maternal and/or fetal complications.

A 19-year-old single woman with no medical history of pathology was admitted to the emergency department for status epilepticus in the context of poisoning during a pregnancy estimated at 21 weeks of amenorrhea.

linical examination on admission found an unconscious patient with GCS 9, pupils equal and reactive to light, generalized hypotonia of all four limbs and sharp, kinetic osteotendinous reflexes, with no signs of focalization; hemodynamically and respiratory stable: HR 95bpm, BP 110/70 mmhg, euphemistic 18c/min.

The clinical picture was marked by persistent neurological disorders and worsening hemodynamic instability that had become refractory to vasoactive drugs, leading to death 72 hours after intoxication.

This is a 26-year-old patient, G2P0 (G1 4-month miscarriage), currently 7 months pregnant, on bromocriptine (Parlodel 2.5 mg) n for a year for prolactin adenoma.

Admitted to emergency for attempted suicide after ingesting 20 tablets of Parlodel, causing dizziness, nausea, vomiting and abdominal pain. On admission to hospital, the patient was conscious, GCS: 15; well oriented in time and space, PEER, complaining of abdominal pain, tachycardic at 120 beats per minute, polypneic at 25 cycles per minute with hypotension at 9/6 without respiratory distress, the conjunctivae were

normo-coloured; the gynaeco-obstetrical examination was normal, notably normal reactive RCF and absence of other signs suggesting foetal distress or abortion, the rest of the clinical examination was unremarkable. With 2l of saline at H1 of intoxication; blood, urine and gastric toxicology samples were taken and returned negative.

Initial laboratory work-up was normal: Hb 12g/dl, urea 0.45g/l, creatinine 10mg/l with no hepatic cytolysis; cardiac enzymes negative.

The patient was treated symptomatically with oxygen therapy at 2l/min, RDB, antagon with good clinical improvement. The patient was discharged after 72 hours of hospitalization without sequelae or maternal and/or fetal complications.

Patient aged 19, married for 1 year, with no notable pathological history, pregnant at 22 weeks' gestation, admitted to the emergency department for attempted suicide due to potentially serious iron intoxication.

On admission to hospital, the patient presented with predominantly digestive symptoms, notably abdomino-pelvic pain with nausea, vomiting (in total 8 episodes of vomiting, including 2 at home and 6 in the emergency department), obnubilated with a GCS of 12/15, PEER to light, apyretic, hemodynamically stable: HR at 60bpm, BP at 110/70mmhg, polypneic at 30C/min, Sao2:95% on oxygen mask under 6l/min with concentrated urine.

Given hospital admission delay of over 6 h, treatment was symptomatic only, based on oxygen therapy, Oedes 20mg/dr, Clopram 1inj/8h, if vomiting, Deferox 500mg, which is Deferasirox cp, single dose (dosage 15mg /kg/pds; loading dose, then maintenance 5-10mg/kg over 4h, Lovenox 0.4UI/dr, saline and 5% glucose infusion No bicarbonate gastric lavage or activated charcoal was administered.

The patient was put on symptomatic treatment based on 2l/min oxygen therapy, with good clinical improvement. Biological monitoring remained normal, and

DISCUTION

Pregnancy is a physiological state during which two main periods of development can be distinguished [3]. Pregnancy induces a certain number of physiological changes in pregnant women, involving pharmacodynamic and pharmacokinetic modifications to the drugs they ingest. Notable physiological changes include [4].

Biological changes: hypercoagulation, with a 5-fold increase in thromboembolic risk during pregnancy and after delivery, as well as a greater risk of bleeding due to rapid consumption of coagulation factors.

Fetal albumin levels rise throughout development to equal or exceed maternal levels, resulting in fetal accumulation of drugs with greater affinity to fetal proteins than to maternal proteins [5].

Increased metabolism of certain drugs by pregnant women due to increased enzymatic activity (cytochrome P450, Uridine diphosphate gluronosyl transferase and N-acetyl transferase mainly), which reduces fetal exposure [6].

The majority of molecules are eliminated in the urine (in unchanged form or as degradation products). From the start of pregnancy, glomerular filtration is increased by 80%, due to an increase in renal blood flow induced by a decrease in renal vascular resistance and an increase in cardiac output. In addition, glomerular filtration is increased by the increase in the plasma free fraction of substances during pregnancy, except during the last 3 weeks, when the filtration rate slows down, necessitating dosage adjustment. Other routes of elimination (hepatic, salivary, cutaneous) are negligible [8].

The impact of drugs during pregnancy also depends on the placenta, a veritable hub of essential maternal-fetal exchange [9]. The amount of drug that crosses the placenta depends not only on physicochemical characteristics, but also on maternal pharmacokinetic parameters and placental factors, which vary according to the term of pregnancy. Drugs can be divided into three groups according to their transplacental passage (limited, high and excess). This degree of transfer governs the passage of drugs from mother to fetus. Therapeutic options and toxic risk assessment can be derived from these pharmacokinetic principles [10].

Pregnancy is an important moment in a woman's life, and represents the decisive phase in the elaboration of her identity, previously prepared by the earlier stages of development. In this sense, it represents a veritable identity crisis, during which each woman mobilizes all her capacities to adapt to the changes (bodily, biological and psychosocial) with which she is confronted. Pregnancy is a special time in a woman's life: a time of both physiological and psychological transformation.

As the pregnancy progresses, this imaginary child becomes increasingly real. The perception of fetal movements and bodily changes, ultrasound images of the child and knowledge of gender all contribute to this movement. The fusional experience of early pregnancy becomes more outward-looking, as the child becomes increasingly individualized.

All these psychic manifestations are more or less reinforced by the woman's psychosocial environment: the support of the partner, the presence of the mother, the couple's living conditions, whether or not the pregnancy was planned, and the woman's personal history, are all elements that can accentuate or attenuate them.

The same is true of some women, where the desire to be pregnant is the only thing at stake, and who have repeated pregnancies because they're just looking for a feeling of fulfillment. As the desire for pregnancy predominates, they don't achieve the desire for a child. (Jean-Michel Darves-Bornoz; women's issues in psychiatry).

During the first three months of pregnancy, the woman experiences a state of "being pregnant"; bodily modifications are minimal, and she has no representations of the child. Nevertheless, the first three months are rich in emotions. The mother-to-be goes through the joy of being pregnant, and the apprehension of a possible miscarriage.

During this period, a number of symptoms can be observed, due to hormonal and emotional factors linked to the onset of pregnancy:

During the third trimester, the mother begins to recognize the different rhythms between herself and her baby. Gradually, over the course of the pregnancy, the mother evolves towards a specific state that the English psychoanalyst WINNICOT has called "primary maternal preoccupation". This is a psychic state that becomes more pronounced towards the end of pregnancy and the first months of the baby's life, preparing the mother-to-be to take care of her baby.

Consequences for both mother and fetus could be devastating. They highlighted common features among women at high risk of suicide during pregnancy: current or past history of psychiatric disorders, young, unemployed, single or socially isolated, unwanted or unplanned pregnancies, alcohol and/or drug intoxication, history of physical or sexual violence. They recommended that health and social authorities in different countries use these risk factors as a basis for prevention and monitoring of patients at high risk of suicide. These characteristics are also found in other studies:

A Bulgarian study [8], examined 224 cases of intoxication in pregnant women. The most common age group was 25-35, and drug intoxication was the most frequent (75.8% of all intoxications). Out of 14 women followed through to the end of pregnancy, 2 children carried malformations (rate 14.3%).

According to INSERM epidemiological data [8], among the 10,700 annual deaths by suicide in France between 2000 and 2010, a mental disorder associated with the death was present in 49.2% of women. The psychic disorders associated with suicide were overwhelmingly mood disorders (32.7%).

Intoxication may vary according to country, region, tradition and degree of development. Acute intoxication in pregnant women remains underestimated, given the small number of studies carried out. According to studies carried out in various regions of Turkey [2-9], the majority of acute poisoning cases were reported to be in women (71%), 7.5% of whom were pregnant [1, 2].

The study showed a predominance of intoxication during the second trimester of pregnancy (68%), followed by 28% during the third trimester, with 4% of cases occurring during the first trimester.

Psychiatric problems are frequently present in the histories of intoxicated patients, explaining the impulsiveness and fragility that led to the act of intoxication. In our series, 12% of patients were being followed for psychiatric pathologies, and 32% had a history of suicide attempts. Mauri C. reported a psychiatric history in 66% of patients, of whom 35% suffered from mood disorders, 15% from personality disorders and 9% from schizophrenia [10].

Physiologically, acetylcholine exerts two types of effect, muscarinic and nicotinic, via two different receptors. The muscarinic effect exerted on parasympathetic post-ganglionic fibers (and on a few rare sympathetic fibers) results in excitation of smooth muscle fibers. The effect is blocked by the para-sympathicolytic atropine.

Nicotinic syndrome: results from the accumulation of Ach in the motor plate and pre-ganglionic synapses of the sympathetic system, manifested by: muscular weakness which may affect the respiratory muscles and aggravate breathing difficulty, muscular fasciculation, tachycardia, which may mask the muscarinic bradycardia, and finally hypertension.

Central syndrome: manifested by confusion, anxiety, irritability, ataxia and sometimes convulsive coma.

The recently described intermediate syndrome, which may occur 1 to 4 days after the acute phase, is characterized by motor deficits in specific areas (proximal muscles, neck flexors, cranial pairs and respiratory muscles). The particular aspect of the intermediate syndrome lies in the severity of the respiratory impairment, requiring prolonged monitoring for at least four days of any organophosphorus intoxication [11]. Cardiac damage: The progressive onset of cardiogenic shock or atrio-ventricular block, with the possibility of sudden onset of asystole.

This includes anticholinergic treatment (atropine sulfate) and a cholinesterase regenerator (pralidoxime methyl sulfate). Atropine sulfate is a genuine antidote to organophosphate poisoning. It acts within minutes on muscarinic and central cholinergic receptors. The aim of using atropine is to improve respiratory function and reduce cholinergic syndrome.

In certain cases. The effect is rapid on muscular signs. Oxime does not pass the blood-meningeal barrier well, and so does not improve consciousness. There are other Oximes on the market, such as obidoxime. Trimedoxime and HI-6 are reserved for military use, and are available in syringes for self-injection. 100% of our patients with organophosphate poisoning received atropine sulfate.

Other studies point to impairment of fine motor skills, visual acuity and recent memory during child development. Finally, recent meta-analyses have shown a significant increase in the risk of leukemia and brain tumors.

Ingestion of aluminum phosphide is immediately followed by dry mouth, intense epigastric and retrosternal pain, and repeated vomiting; diarrhea, sometimes bloody, is secondary.

The main aim of treatment is to combat shock by taking appropriate measures and maintaining the patient's life until phosphine (PH3) has been completely excreted by the kidneys in the form of phosphatides and by the lungs in unchanged form.

The administration of activated charcoal can help reduce the toxic load and increase the likelihood of a positive outcome. However, its efficacy in cases of Phostoxin® intoxication has not been demonstrated in several studies, and its administration requires the addition of water, which could aggravate intoxication.

Oxygen therapy via nasal tube or mask, airway clearance and protection via endotracheal intubation in comatose patients, assisted ventilation if necessary, and blood gas monitoring are all used to combat metabolic acidosis Antiarrhythmics, cardioversion and temporary pacemakers can improve arrhythmias. Magnesium sulfate has been successfully used as a membrane stabilizer in the treatment of arrhythmias observed during the first 24 hours, particularly supraventricular arrhythmias. If bicarbonate levels fall below 15 mmol/l, metabolic acidosis is corrected by intravenous administration of bicarbonate solutions at a dose of 50 to 100 ml until HCO3- levels of 18 to 20 mmol/l are achieved.

The indication for activated charcoal must take into account the instantaneous or potential severity of the patient's intoxication, and the known contribution of activated charcoal to the treatment of a given intoxication among the other purifying, antidotic or symptomatic therapies that may be proposed.

The administration of repeated doses of activated charcoal is recommended if the patient has ingested a potentially lethal dose of Carbamazepine, phenobarbital, Dapsone, quinine or theophylline that may necessitate the use of invasive extra-renal purification techniques [12]. The optimal dose has not been determined, but an initial dose of 50 to 100g of activated charcoal followed by approximately 12.5 g/h or (50 g/4 h) is recommended in adults.

All situations leading to incomplete combustion of a carbon-containing substance due to a lack of oxygen result in the production of CO. The causes are therefore extremely numerous (fire, defective appliance, traditional "canoune" heating in rural Morocco) [5].

The clinical picture in our patients is dominated by neurological signs such as headache, vertigo and disturbed consciousness.

Cardiovascular sequelae of CO intoxication are frequent. Therefore, patients admitted to hospital with CO intoxication should have a baseline ECG and a cardiac biomarker assay.

In our study, troponin was positive in 10 patients with sinus tachycardia presenting with myocardial sideration.

Consciousness, and in pregnant women [4], as hyperbaric oxygen therapy is associated with less severe fetal damage on average. It accelerates the dissociation of CO from hemoglobin and mitochondrial respiratory chain proteins, shortens the half-life of HbCO, increases blood oxygen concentration and reduces the production of free radicals [8]. The half-life of maternal HbCO is estimated at four hours in room air, 1.5 hours with normobaric oxygen therapy and only 20 minutes with hyperbaric oxygen therapy at 3 ATA. Since the fetus takes longer to reach equilibrium and eliminate CO than the mother, emergency treatment can theoretically prevent the formation of fetal lesions, and reduce cerebral edema and long-term neurological sequelae [3]. Because of this slower elimination of fetal CO, the duration of hyperbaric oxygen therapy should theoretically be increased in pregnant women [46]. Some authors [46], have proposed the following indications: a maternal HbCO level above 20%, the presence of clinical signs of CO intoxication, and the existence of fetal heart rhythm abnormalities. These authors recommend continuing hyperbaric oxygen therapy if maternal or fetal signs persist 12 hours after the start of the first session. In France, the recommendations on what to do in the event of carbon monoxide poisoning date from 2005 [12, 11].

Pergamum harmale is a widespread plant in the Mediterranean region. It is commonly used in traditional Moroccan medicine as a sedative and abortifacient, but exposes users to the risk of overdosing and poisoning. The pharmacologically active compounds of this plant include a number of ß-carboline and quinazoline alkaloids responsible for its pharmacological and toxicological effects.

Pharmacologically active compounds in P. Harmala include a number of ß-carboline and quinazoline alkaloids. Harmaline, harmine, harmalol, HARMOL and tetra hydroharmine have been identified and quantified as the main β-carboline alkaloids in P. harmala extracts. Seeds and roots contain the highest levels of alkaloids with low levels in stems and leaves and the absence of flowers. Harmine and harmaline accumulated in dry seeds ranged from 4.3% to 5.6%. Seed and root extracts are potent reversible and competitive inhibitors of Diagnosis of intoxication is based on knowledge of the plant and identification of alkaloids by high-performance liquid chromatography, or by the more sensitive gas chromatography/mass spectrometry method [1]. In the absence of adequate laboratory facilities to detect alkaloids. Emergency services and physicians should recognize and treat this specific intoxication according to the context and clinical presentation.

When administered orally to volunteers, whatever its chemical form, 2,4-D is rapidly absorbed from the gastrointestinal tract. The product appears in the bloodstream within an hour, reaching peak concentration after 4 hours. It may be distributed to certain tissues, including the liver and kidneys, but appears to accumulate very little there, as the residue concentrations measured are very low. Acid and salt forms of 2,4-D are excreted unchanged, while products in the ester form are hydrolyzed and excreted in the acid form. In humans, urinary elimination is rapid, with around 73% of ingested 2,4D excreted within 48 hours, only a small proportion being conjugated (IPCS 1997).

In our case, the evolution was marked by the spontaneous abortion of a stillborn baby, the onset of renal failure with urea at 1.36 g/l and creatinine at 19 mg/dl, as well as muscular and cardiac damage attested by CPK at 4000 µg/l and troponin IC at 3 ng/m, leading to death 72 hours after intoxication.

In an emergency, toxicological tests are only useful if they are specific and can be performed with the routine biological work-up. Moreover, quantitative methods are to be preferred to detection methods, because of the existence of dose-response relationships.

Blood tests are preferred, as the concentration of the toxicant is often better correlated with toxicity.

Negative results may be explained by the fact that sampling is carried out very early, when the toxicant has not yet been absorbed, or late, when the toxicant has already been metabolized or even eliminated. It is therefore essential to be familiar with the toxicodynamic and toxicokinetic characteristics of toxic products, and to specify the time between ingestion of the toxicant and sampling, in order to facilitate interpretation of the results of the toxicological analysis.

In this series, toxicological results were found in 4 cases (16%). The sampling media were gastric fluid in 10 patients, blood in 11 and urine in all pregnant women admitted to the department. Sample media number of samples positive rate negative rate.

In this study, iron was involved in 4% of cases of intoxication in pregnant women with suicidal intent. Symptoms begin with acute gastroenteritis, followed by a free interval, then shock and liver failure.

Iron is toxic to the digestive tract, cardiovascular system and central nervous system. The mechanisms involved in this toxicity are poorly understood, but excess free iron can disrupt enzymatic mechanisms such as oxidative phosphorylation, resulting in metabolic acidosis. Iron also catalyzes the formation of free radicals, behaving as an oxidizing agent and, as soon as protein binding.

When plasma protein binding is saturated, iron combines with water to form iron hydroxide and free H+ ions, further aggravating metabolic acidosis. Coagulopathy may occur in the early stages of intoxication, due to disruption of the coagulation cascade, and later, as a result of liver damage.

Free iron is a cellular poison through the formation of free radicals and lipid preoxidation. Its toxicity is triggered by saturation of transport and storage capacities, and it has a corrosive effect on direct and prolonged contact with the gastrointestinal mucosa, causing metabolic acidosis through uncoupling of alkaline phosphoryl and accumulation of metabolic products (lactic acid). In the event of an oral overdose, peak plasma levels are observed on average 4 to 6 hours after ingestion [7].

In the case of more severe intoxication (over 60mg/kg), digestive hemorrhage, arterial hypotension, unconsciousness, convulsions, coagulopathy (DIC), myocardial infarction, metabolic acidosis, hyperglycemia and hyperleukocytosis may occur in addition to digestive disorders. Necrotizing hepatitis may occur 24 to 48 hours later.

Massive accidental overdosing with intravenous iron administration can lead to life-threatening multi-visceral failure, requiring intensive care.

Serum iron levels can be used to assess the severity of intoxication when clinical evidence is insufficient; levels above 500 ug/dl (90 umol/L) require hospitalization in an intensive care unit capable of managing multivisceral failure.

On the other hand, deferoxamine does not bind iron from transferrin, hemoglobin or cytochromes. The complex formed is ferroxamine, a soluble, pink-colored pigment which is eliminated within 24 to 48 hours, mainly in the urine and to some extent in the bile. Hemodialysis may be required to eliminate the complex in cases of renal failure or anuria. Chelation is discontinued once symptoms have disappeared, acidosis has normalized and serum iron levels are below 100ug/dL. Chelation usually lasts no longer than 24 hours.

Three commonly used iron removers. One (deferoxamine) is injected and two (deferiprone and deferasirox) are taken orally, which have been shown to be equally good at removing excess iron. Several trials have found that the combination of deferoxamine and deferiprone was more effective than using a single chelator.

We reported a high rate of gastric lavage practices that were not necessary, as long as it becomes ineffective an hour after ingestion of toxic. This therapeutic attitude has also been observed in Spain [12], which requires more effort to train emergency physicians in central and peripheral hospitals to save patients and hospitals the untimely costs of an unnecessary and probably dangerous act.

Prevention of suicidal intoxications, limiting risk factors (major depression, schizophrenia, alcoholism or life difficulties encountered by vulnerable people such as adolescents and women) But especially on the promotion of protective factors: marriage, religion, the role of parents, social support, family and friend ties, self-esteem, the importance of which seems to outweigh the limitation of risk factors

Impose strict standards for imported toxic products; no product should be introduced into Morocco if not allowed in the country of origin. Eliminate highly toxic products from the market and replace them with such effective and less toxic products. Impose as a standard the manufacture or import of diluted pesticides, marketed in small bottles to prevent death even if the amount ingested is significant. Promote strict legislation regarding the labelling of toxic products. Decentralization of health facilities to address the problem of delayed treatment and promote the availability of fans and necessary treatments. Installation of CO detectors near potential sources in the workplace and at home.

Toxicological concerns should not detract from the need for psychiatric counselling (medicolegal value) following the acute phase in cases of voluntary poisoning. Agitation, aggressive behaviour or anxiety must be managed (anxiolytic, sedation, [12, 11]. Or even restraint if necessary) while awaiting psychiatric evaluation. In the case of a request for medical discharge, it is recommended to contact the family and/or the person of trust and to record any action in the medical record. Particular attention is paid to the criteria of suicidal intentionality. If the risk of short-term recurrence is detectable, a specialized hospitalization should be set up, in particular in the absence of a critique of the act, in case of persistence of active suicidal ideation, major anxiety or manifest psychiatric disorder.

CONCLUSION

Pregnancy is a special period during which physical, psychological and physiological changes occur in women. Premature and unplanned pregnancy can be an additional source of stress for women. The diagnosis of acute poisoning, has evolved a lot, so next to the clinic, which is sometimes evocative but not always attractive, the contribution of analytical toxicology remains very interesting at this level to determine the toxic in question.

This study is the first in our country that analyzes the epidemiology of acute poisoning during pregnancy. Nevertheless, our study is limited by being retrospective and including the cases of a single center. In order to obtain more.

Conflict of Interest: The authors declare no conflicts of interest.

Others' Contributions:

All authors have contributed to the development of the work and endorse the document. The have also read and approved the final version of this manuscript.

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