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Carbon monoxide and pregnancy: Cause of miscarriage in pregnant women in the first trimester?

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ABSTRACT: Carbon monoxide poisoning during pregnancy is a rare and potentially serious condition. Fetal complications are uncommon, related to anoxic lesions. The severity of these complications does not depend on the level of maternal COHb.We report the case of 25-year-old woman 2 months pregnant and half accidentally exposed to domestic gas who was received in a state of unconsciousness with medium abundant bleeding in gynecology and obstetrics emergencies of the maternity souissi CHU in Rabat where we have seen an abortion in progress with expulsion of the product of conception.

KEYWORDS: Carbon monoxide, first trimester of pregnancy, miscarriage.

1 INTRODUCTION

Acute carbon monoxide (CO) poisoning during pregnancy is a relatively rare condition with serious adverse effects to mother and fetus, and the resulting intrauterine hypoxia can lead to fetal death or severe neurological sequelae [1]. Its literature is old and little known [2]. It is the leading cause of toxic morbidity and mortality in the world. This potentially lethal intoxication can be voluntary as part of an attempt at autolysis. It is nevertheless, in Europe, more frequently accidental [3]. Its consequences are potentially serious for the fetus when it occurs in pregnant women because the fetus is particularly exposed to the risk of hypoxia due to the strong affinity of its hemoglobin for CO which easily crosses the placenta [4]. We report the case of a 25-year-old woman, 2.5 months pregnant, accidentally exposed to domestic gas who was received in a state of unconsciousness with moderate bleeding in the gynecology and obstetrics emergency department of the maternity souissi in Rabat. with a review of the literature to understand the physiopathological mechanism specific to pregnant women and the consequences on pregnancy as well as on the embryo or fetus.

2 OBSERVATION

25-year-old female primigravidae married for 04 months admitted in a context of disturbance of consciousness with amenorrhea of 02 months and a half. The questioning of the members of the family reveals a notion of domestic gas poisoning following a leak on the water heater during the day before the fortuitous discovery of the couple in a state of unconsciousness. That is to say approximately 10 hours after the occurrence of the incident. The examination found an unconscious patient with a Glasgow score of 12/15, polypneic with swaying of the wings of the nose, intercostal indrawing and cyanosis of the extremities. The pleuropulmonary examination finds a symmetrical, polypneic thorax with a respiratory rate of 30 cycles / min with an unaudible vesicular murmur and additional noises difficult to characterize; hemodynamically the blood pressure was 10 / 6cmHg with a heart rate of 157 beats / min. The gynecological examination found a pelvic uterus, globular, with minimal metrorrhagia speculum with beginning of expulsion of the product of conception and on vaginal examination a central cervix erased at 50% and permeable to the 2 openings. No obvious signs of the presence of a foreign body. Considering the dyspneic state of the patient, she was directly admitted to the emergency room to be under scope and under an oxygen mask with an initial saturation of 90% before being admitted to the intensive care unit for adequate treatment. She stayed in an intensive care unit for 72 hours where she received treatment based on non-invasive ventilation (NIV) with 100% FiO2. The initial

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paraclinical workup found on gasometry a complex disorder of metabolic acidosis and respiratory alkalosis, on the chest x-ray of the front an atelectasis, on the electrocardiogram (ECG) a sinus tachycardia, on the cardiac Doppler ultrasound no particularity and on the toxicological assay a positive return carboxy hemoglobin (HbCO). The course under initial treatment was complicated by organ failures: cardiovascular (hypotension, tachycardia, septal hypokinesia of LV with troponin positive), metabolic (hypoxia with persistence of metabolic acidosis). Faced with the worsening of the patient's clinical condition and the absence of hyperbaric oxygen in our health facility, invasive ventilation with 100% FiO2 was initiated and continued for 4 hours until the metabolic disorders were corrected. and improvement of clinical and laboratory signs. The patient was subsequently extubated and put on an oxygen mask with good tolerance. After a 48-hour stay in intensive care, she was declared discharged without sequelae with good ambient air saturation.

3 DISCUSSIONS

Carbon monoxide poisoning is the most well-known poisoning in France. The InVs (Institute of Health Surveillance) records 4000 cases per year and according to this institution its incidence is underestimated for several reasons: the non-mandatory declaration, smoke poisoning are excluded from the declared case, and finally, since this gas is odorless and colorless and symptoms do not always appear, some cases of CO poisoning are often misdiagnosed [5]. Pregnant women constitute a particularly fragile population vis-à-vis CO poisoning due to the physiological changes induced by pregnancy with consequences that can be extremely serious for the fetus, as long as there is no parallelism. strict between the clinical state of the mother and the severity of the poisoning of the child [3]. In terms of pathophysiology, CO passively crosses the placental barrier to enter the infant's circulation, the ability to diffuse CO increasing with gestational age in proportion to the weight of the fetus. Conversely, oxygen must cross the placental barrier for elimination of fetal CO to begin. There is therefore a delay in detoxification of the fetus compared to maternal detoxification. In addition, fetal hemoglobin has more affinity for CO than maternal hemoglobin. The hypoxia is much more marked, which increases the binding of CO to all hemoproteins. This explains the major risk of toxicity to the central nervous system of the fetus and the fact that the severity of fetal intoxication cannot be judged on the clinical status of the mother [6]. CO causes a decrease in the transport of oxygen (O2). It binds to hemoglobin with greater affinity than O2, thus forming carboxy hemoglobin (HbCO). The HbCO also induces a shift to the left of the oxyhemoglobin dissociation curve, reducing the release of O2 in the tissues. CO also binds to proteins such as myoglobin and cytochrome P450 and disrupts the use of O2 at the tissue level, especially by the myocardium. Finally, there is direct cellular toxicity. The fetus is particularly vulnerable to CO, which is often teratogenic, the passage of CO from the mother to the fetus is passively facilitated also by cytochrome P450 [2]. This passage would be all the more important as the pressure gradient in HbCO is high between the mother and the fetus. Its increase is also related to gestational age, fetal weight as well as increased placental blood flow and maternal hemoglobin concentration [1,7]. Fetal HbCO levels are 10-15% higher than maternal levels, fetal hemoglobin has a higher affinity for CO than adult hemoglobin. In addition, fetal elimination of CO takes longer, as it dissociates much more slowly from the fetus than adult hemoglobin. For this reason, the severity of fetal poisoning cannot be assessed solely by maternal condition [8]. In addition to the mechanisms described, it is reported by some authors that there are lesions of the ischemia-reperfusion type during tissue reoxygenation. Indeed, reoxygenation would lead to the production of oxygenated free radicals. This would be responsible for lipid peroxidation followed by the degradation of membrane fatty acids leading to the formation of unstable hydro peroxides in cell membranes, especially in the brain, with impaired functioning [1,9].

From the point of view of consequences, in the first trimester, various malformations deemed non-pathognomonic of CO poisoning have been reported including cleft lip [10] or limb malformations [11]. Some abnormalities in brain development have also been reported, including microcephaly and telencephalic dysgenesis [4,12]. From the second trimester of pregnancy, cerebral sensitivity to CO appears to be greater and lesions more frequent [13], mainly observed in the lenticular nuclei and more rarely in the brainstem, cerebellum and spinal cord [4]. Neonatal and long-term sequelae may result from these attacks [14]. In our review of the literature, we did not find cases of miscarriages as is the case in our observation. Could this miscarriage be linked to the amount of CO inhaled by our patient? However, she would have been exposed to CO for about 10 hours. We cannot explain the mechanism by which she expelled the product of conception. The mechanism that led to this miscarriage remains to be elucidated. The clinical manifestations of CO poisoning, dominated by neurological signs, are very polymorphic and variable from patient to patient and from time to time, making diagnosis sometimes difficult [3]. From a clinical point of view, the manifestations observed in pregnant women are identical to those in the general population, except that nonspecific clinical signs such as dizziness, nausea, vomiting, may be related to pregnancy. which can lead to a delay in the diagnosis of intoxication [15].

Francis Wattel et al [7]. reported in their study that there is no correlation between the degree of severity of maternal intoxication and the risk of fetal distress, the performance of an obstetric examination and an ultrasound would be necessary in the immediate aftermath of intoxication, the ideal being to have a prolonged follow-up of the fetus, the newborn and the

child. When it comes to management, the availability of oxygen is of great importance. The administration of oxygen is the basic treatment in the acute phase, allowing an increase in the rate of elimination of CO by O2 [16]. Treatment for acute CO poisoning during pregnancy begins with removing the victim from the environmental source of CO. Then 100% normobaric oxygen should be administered immediately and hyperbaric oxygen should be considered. The only possible non-teratogenic treatment for pregnant women with CO poisoning is hyperbaric oxygen therapy. Hyperbaric oxygen is mandatory for all pregnant women with impaired consciousness or COHb levels of 20% or more [17]. Despite the absence of hyperbaric oxygen, the management of our patient was successful because we managed to get her out of the respiratory distress and the fatal risk that she incurred without care.

4 CONCLUSION

Acute Carbon monoxide poisoning during pregnancy is relatively rare. Often accidental and in addition to potentially fatal consequences for the fetus, it can lead to spontaneous abortion in the first trimester as is the case in our observation

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