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RESEARCH ARTICLE

PARAPHENYLENEDIAMINE POISONING : A CASE REPORT AND REVIEW OF LITERATURE

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Abstract

Paraphenylenediamine (PPD) intoxication is a medical emergency with a very poor prognosis in the absence of early and adequate treatment. Its pathophysiological mechanism is now known and results from the destruction of muscle cells by peroxidation phenomena leading to major rhabdomyolysis. The diagnosis is essentially clinical, guided by a rapid history. As there is no specific antidote, treatment is mainly symptomatic and aims at securing the VAS, reducing intestinal absorption of the toxicant, as well as managing cervico-facial oedema and complications of rhabdomyolysis. In hospital, treatment includes gastric lavage, activated charcoal, filling with alkalinisation, antioxidant treatments, corticosteroids, diuretics and even dialysis. In order to shed light on the danger of this practice, the authors have taken the liberty of reporting the clinical case of PPD intoxication in a young woman admitted to the maternity ward of the Harouchi Hospital in Casablanca.

Introduction:-

Paraphenylenediamine (PPD) is an aromatic amine derived from aniline and has been used since the end of the 19th century for cosmetic purposes, mainly as an additive to henna for dyeing hands, feet or hair, in several African and Middle Eastern countries. Its absorption causes systemic toxicity, which explains its misuse for suicidal purposes. In Morocco, PPD is available over the counter in the form of "Takaout" rock and its misuse for abortifacient purposes is very common. In order to shed light on the dangerousness of this practice, the authors have taken the liberty of reporting the clinical case of PPD intoxication in a young woman admitted to the maternity ward of the Abderrahim Harouchi Mother and Child Hospital in Casablanca.

Observation:-

A young woman (26 years old) with no previous history, presented to the maternity hospital emergency room for a suspected abortion at 3 months, with the notion of ingestion of toxic products. The little information we gathered from the history and the black marks on the fingers pointed to the intake of PPD (figure 1). On admission, the neurological examination revealed an obnoxious patient with symmetrical reactive pupils. She was apyretic and the blood glucose level taken at that time was 1.49. The patient was polypneic with abdominal breathing and SpO2 = 75% on free air. Pleuropulmonary auscultation revealed bilateral crackles. Hemodynamically, she was tachycardic at 120 bpm and hypotensive at 07/04. Diuresis was 400cc/12h. Physical examination revealed hypersialorrhea with cervico-facial edema. The gynecological examination revealed a violated cervix and the fingernail came back soiled with blood. Fetal heart activity was absent. The patient was rapidly intubated and put on mechanical ventilation

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(volume controlled mode, FiO2 at 100%, PEEP at 10, Vt at 380 mL). Saturation rose to 96%. Adrenergic support with noradrenaline was started and an echocardiographic probe showed good overall cardiac contractility, a preserved ejection fraction, a dilated right ventricle and a non-compliant IVC of 23. The emergency ECG did not reveal any particular abnormalities apart from a tachycardia. The arterial blood gas performed showed (ph: 7.03, Po2: 114 mmgh, PCo2: 40 mmgh , HCO3- :10.6mmol/l and lactatemia at 7.9). Biologically, an anaemia of 6.3 was found, as well as thrombocytopenia of 36,000, hyponatremia of 129, hyperkalaemia of 6.4, liver cytolysis increased to 3 times normal, renal function very impaired (urea >2.68, creatinine of 91), CPK increased to 77, PAL to 268, CRP to 311, LDH to 1430 and hypoalbuminaemia to 17. The therapeutic management was initially symptomatic, with abundant gastric lavage, aggressive filling associated with alkalization, diuretics and corticotherapy. As her condition worsened in the intensive care unit, she was transferred to the nephrology department for dialysis. The evolution was marked by the accentuation of her haemodynamic instability during dialysis despite the continuous administration of high doses of noradrenaline, then by her death on her return to the intensive care unit following an unrecovered cardiorespiratory arrest.

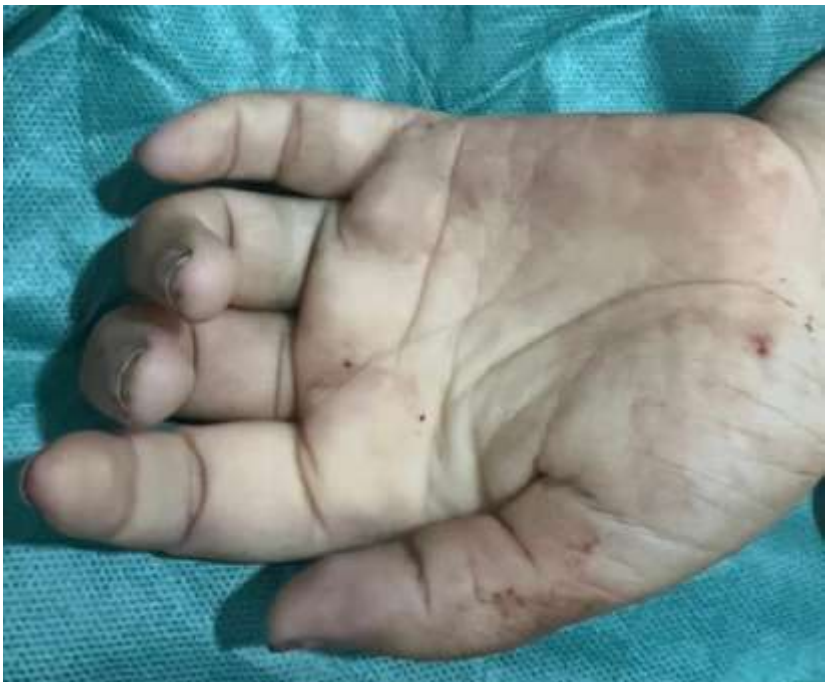

Figure 1:- Image showing the black marks on the fingers pointed to the intake of PPD.

Discussion:-

PPD intoxication is a rapidly fatal emergency if not treated medically. It is secondary to oral intake of the toxicant, with considerable systemic toxicity even at very low doses. (1) The dose-effect relationship is not yet fully defined but the threshold of toxicity is estimated at 3g (2). Its mechanism is now known and begins with the formation of oxidised derivatives, including benzoquinone diimide, which is responsible for the destruction of muscle cells by peroxidation of membrane lipids, leading to significant rhabdomyolysis. Its oxidation by enzymes results in the formation of primary amines leading to the "Brandowski base", with an inflammatory component by stimulating the secretion of cytokines which increase capillary permeability and explain the appearance of cervico-facial oedema in the initial phase. (3)

The diagnosis is mainly clinical, guided first by an interrogation which is not always carried out in this type of situation because of the emergency context. The history should be brief and its purpose is to clarify the circumstances of the intoxication (history, reason, quantity and time of ingestion, association with other products, etc.). The visualisation of black marks on the fingertips is very suggestive of the use of this type of product and should attract the attention of the clinician. The diagnosis is then based on the stereotypical appearance of cervico-facial oedema and macroglossia, on average 2 to 3 hours after ingestion, which can be life-threatening due to asphyxia, and generally requires the insertion of an orotracheal intubation tube or a tracheotomy to secure the airway

(4) The oedema syndrome is the primary reason for hospital admission and the only clinical sign that immediately points to PPD intoxication (5).

The evolution is most often through the development of rhabdomyolysis with massive release of myoglobins resulting in renal damage firstly through mechanical obstruction, responsible for the cessation of glomerular filtration and tubular obstruction, and secondly through ischaemic damage secondary to the release of vasoactive substances and proteolytic enzymes by the necrotic cells, leading to acute renal failure. (6) Fluid leakage due to muscle lysis is responsible for a drop in GFR, which leads to hypovolaemia and thus worsens the renal damage. (7)

Cardiac involvement has also been described in the form of acute toxic myocarditis and complicates the prognosis (8). It should be suspected in the presence of arterial hypotension and ECG abnormalities. Troponin measurement allows early diagnosis and initial assessment of myocardial damage (9).

Toxicological analysis of gastric lavage contents, blood and urine can confirm the diagnosis, but is not essential. Clinical examination and history-taking are often sufficient to make the diagnosis, especially in the emergency setting.

In the absence of an antidote, therapeutic management is based on symptomatic treatment and well-conducted resuscitation measures. Gastric lavage must be carried out as a matter of urgency, even beyond the 2nd hour after ingestion, respecting safety conditions, using a gastric or nasogastric tube, 4 to 8 L of physiological serum until the liquid is clear. Activated charcoal is an attractive adjunct to gastric lavage by breaking the PPD enterohepatic cycle and providing gastrointestinal dialysis. (10) Securing the airways by orotracheal intubation or tracheotomy (11) is essential in the event of respiratory distress, the mechanism of which may be either early cervico-facial oedema which compresses the VAS, rhabdomyolysis of the respiratory muscles (especially the diaphragm) or methaemoglobinaemia which worsens the already pre-existing hypoxaemia. The haemodynamic resuscitation of PPD intoxication is called "target resuscitation" and aims to obtain a diuresis of 8 l/d and a urinary pH greater than 7 until the disappearance of myoglobinuria, which only occurs in an alkaline environment. (12) It is based on rapid and massive vascular filling using 0.9% physiological serum, associated with 5% glucose serum, and alkaline hyperdiuresis using 0.14% bicarbonate serum. If diuresis does not resume after restoration of effective blood volume and satisfactory fluid balance, diuretics are essential to convert oligoanuric ARF into diuresis-preserved ARF with a better prognosis. (13) Antioxidant treatment with vitamin C (4g/d) or methylene blue (1 to 3 mL/Kg) is justified (14) and some authors (15) recommend corticosteroid therapy with hydrocortisone hemisuccinate or methylprednisolone (100 to 120 mg every 6 hours).

The extraction of PPD by extra-renal purification is impossible in view of its very large diffusion space in the muscle parenchyma, its hydrophobic character and its strong binding to proteins, and therefore dialysis is ineffective. (16) Nevertheless, extra renal purification is indicated in the case of persistent oligoanuria despite adequate resuscitation, profound metabolic acidosis or threatening hyperkalaemia.

Conclusion:-

Paraphenylene diamine intoxication is a public health problem in Morocco. In our context, it most often concerns young women who ingest the product for the purpose of autolysis or abortion. The diagnosis is essentially clinical, based on the observation of an oedematous syndrome involving the face and neck and a rhabdomyolysis picture. Management must be rapid and is based essentially on symptomatic treatment, in the absence of an antidote. The prognosis is generally unfortunate despite good management, hence the need to introduce new preventive and educational measures to make the population aware of the seriousness of this dangerous disease.

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