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RESEARCH ARTICLE

ACUTE RENAL FAILURE DURING POISONINGATTETRAHYDROCANNABIOL: ABOUT A CASE

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Abstract

Acute kidney failure (ARI) is a common condition affecting approximately26.9% of pediatric age patients. Its severity is variable, ranging from a simple disturbance of the biological balance to the need to resort to extra-renal purification and the commitment of the vital prognosis. Acute renal failure is defined as a sudden (occurring within 1 to 7 days) and sustained (>24 hours) decline in renal function, reflected by an increase in plasma creatinine; it is associated with the inability of the kidneys to excrete the waste products of nitrogen metabolism with an increase in blood urea.), other etiologies can be observed including the toxic origin. We report the case of a 14-year-old child from a non-consanguineous marriage, without particular pathological ATCDS, without notion of toxic or drug intake, admitted for AKI revealed by food vomiting and liquid diarrhea evolving for 5 days, with biological balance sheet urea at 1.69 and creatinine at 17.59. On examination the child was conscious stable normocardium at 92 bpm normotensive at120/80mmhg eupneic at 18 cpm diuresis at 1.1cc/kg/h; weight and height inaverage, no edematous syndrome or sign of overload, the urine dipstick was negative without glucosuria, the etiological assessment was negative, the patient was put on oral hyperhydration alone (3L/d) with a marked improvement in renal function urea to 0.38 and creatinine at 5.4 after 24 hours, the questioning was resumed which noted the notion of consumption of tetrahydrocannabiol. In conclusion, ARI is considered a diagnostic and therapeutic emergency whose toxic origin is always to be mentioned above all in adolescents.

Introduction:-

Acute renal failure in children has long been considered the side effect of failing hemodynamics in the context of severe disease. The diagnosis was made essentially following the sudden increase in creatinine. There was no consistent definition, which made it difficult to collect data on the frequency, etiology, and prognosis of acute kidney injury in childhood.

However, regardless of the severity, all patients who have presented with ARI are exposed to a risk of sequelae: the formation of irreversible lesions can progress to chronic renal failure (CRI) and increases the risk of recurrence. Due to the seriousness of its prognosis and the costs it generates, its prevention represents a major public health issue.

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Since 2012, the diagnosis of AKI has been based on clinical and biological criteria reflecting renal function, in particular diuresis and plasma creatinine according to the classification developed by the KDIGO working group (kidney disease: Improving Global Outcomes) (1). According to this classification, an ARF is defined by the presence of at least one of the following 3 diagnostic criteria:
- increase in plasma creatinine ≥ 26.5 µmol/L in 48 hours
- increase in plasma creatinine ≥ 1.5 times the baseline value in the last 7 days
- diuresis < 0.5 ml/kg/h for 6 hours

The presence of one of these criteria makes it possible to make the diagnosis of AKI at stage 1. The KDIGO classification makes it possible to establish 3 stages of severity according to the values of plasma creatinine and diuresis (Table 1).

stadium	Plasma creatinine	diuresis
1	≥ 26.5 µmol/l (0.3 mg/dl) in 48h OR 1.5 to 1.9 times baseline plasma creatinine in the last 7 days	< 0.5 ml/kg/h for 6h to 12h
2	2.0 to 2.9 times baseline plasma creatinine	< 0.5 ml/kg/h for at least 12h
3	3.0 times baseline plasma creatinine OR Plasma creatinine ≥ 353.6 µmol/L (4.0 mg/dL) ORInitiation of extra-renal purification	< 0.3 ml/kg/h for at least 24h OR Anuria for at least 12h

Table 1:- Classification of ARI according to the KDIGO criteria.

The most common classification is that according to the localization: pre-renal, renal or intrinsic and post-renal or obstructive.

Through the observation of a 14-year-old child and a review of the literature, we propose to take stock of a pre-renal origin of secondary ARFto a decrease in renal perfusion, the cause of which is intoxication with tetrahydrocannabiol.

Observation:-

This is a 14-year-old child from a non-consanguineous marriage, without ATCDSparticular pathological condition, with no notion of toxic or medicamentause intake, admitted to the service for ARI revealed by food vomiting and liquid diarrhea evolving for 5 days, with biological assessment of urea at 1.69 and creatinine at 17.59.

On clinical examination, the child was conscious, hemodynamically and respiratory stable: normocardium at 92 bpm, normotensive at 120/80 mmHg, eupneic at 18 cpm, diuresis at 1.1 cc/kg/h, average weight and height, no edematous syndrome or signs of overload, the urine dipstick was negative without glycosuria.

A paraclinical assessment was carried out: renal ultrasound having objectified kidneys of normal size with moderate bilateral ureterohydronephrosis; in the etiological assessment, hepatitis B/C/EBV/CMV/HIV serology were negatives; PCR covid 19 negative; rectal swab in search of negative E coli; ionogram without abnormality; NFS showed an HG level at 15.6 VGM 80.2 TCMH 29.8 PLQ 294000; the ECBU was sterile; normal 24-hour microalbuminuria at 21mg/24h; normal immunological assessment

The patient was placed on oral hyperhydration alone (3L/d) with marked improvement in renal function after 24 hours: urea at 0.38 and creatinine at 5.4. The questioning was resumed with the patient who this time mentioned taking tetrahydrocanabiol.

Discussion:-

Cannabis is the most widely used illegal drug in the world for its psychoactive effects, the latter being linked to its main phytocannabinoid, delta-9-tetrahydrocannabinol (THC) (1). Two US surveys report cannabinoid consumption by patients with kidney disease2,3, Another survey of 101 teenagers aged 13 to 19 with acute kidney disease reported that 23% of them used cannabis.

The pharmacokinetics of THC and its metabolites is known: their elimination is mainly intestinal and only 30% of a dose of THC is excreted by the kidneys (4). Consequently, the clinician does not expect to observe clinical particularities in chronic or acute renal failure who consume cannabinoids compared to a healthy subject. However, it seems important to assess the effect of cannabinoid consumption on the kidneys.

From a pharmacodynamic point of view, humans have two main receptors, CB1 and CB2, with their endogenous ligands, namely anandamide and 2-arachidonyl-glycerol. This collection of receptors and ligands is called the "endocannabinoid system". CB1 receptors are highly expressed at central and peripheral nerve endings, whose primary function is to inhibit the release of neurotransmitters. CB2 receptors are mainly present on inflammatory cells, where they exert anti-inflammatory effects, including inhibition of cytokine release (TNF-alpha, IL-1b). The system is mainly known for the binding of THC to central CB1 receptors during the consumption of so-called recreational cannabis.

CB1 and CB2 receptors would also be present in the kidney of healthy humans (5,6). However, their expression is very weak, except for the CB2 receptor expressed more in the glomeruli. The CB1 receptor is found in the proximal and distal tubules and in the intercalary cells of the collecting tubules (7). As for the CB2 receptor, it is expressed in podocytes (8). Little is known about the role of the endocannabinoid system in the healthy human kidney. It is difficult to extrapolate the results of animal experiments to humans, because their cannabinoid receptors are expressed differently.

No good quality clinical studies have been identified on the development of acute renal failure following the consumption of cannabinoids. Only cases and case series have been published in the scientific literature, which limits causal associations. Cannabis consumption may be responsible for acute pre-renal renal failure, due to functional acute renal failure linked to volume depletion during a cannabis hyperemesis syndrome9. The consumer of cannabis or synthetic cannabinoids then presents a syndrome which generally manifests itself by vomiting and abdominal pain usually relieved by taking hot showers (10). Diaphoresis and diarrhea can also occur and contribute to increased water loss (9,10).

Cannabis hyperemesis syndrome can give rise to a slightly different diagnosis from the uremia suffered by patients with end-stage renal failure and digestive disorders. Faced with a clinical picture evoking uraemia, health professionals must therefore examine whether cannabis consumption is the cause (11).

The nephrotoxicity of certain synthetic cannabinoids has been observed on several occasions. Acute tubular necrosis and, more rarely, acute interstitial nephritis have been reported after the consumption of these substances (9,12,13). In several cases, calcium oxalate crystals were found on kidney biopsy, probably related to plants used as cutting agents or to cannabinoid metabolites (9). The mechanism of synthetic cannabinoid nephrotoxicity is not yet understood. It is quite possible that this kidney dysfunction is due to contaminants present in these products (12,14). In some cases, the acute toxicity linked to the consumption of synthetic cannabinoids has been associated with a significant and permanent deterioration in renal function (15).

Conclusion:-

There are several kidney risks associated with the consumption of cannabinoids. The consumption of synthetic cannabinoids may be responsible for acute renal failure, mainly by acute tubular necrosis, but the physiopathological mechanisms explaining this lesion must be better defined9. The consumption of cannabis or synthetic cannabinoids can also lead to acute functional renal failure following a cannabis hyperemesis syndrome9. At present, there is insufficient evidence to support or refute a statistical association between cannabis use and permanent decline in kidney function, but several case reports and in vitro model studies urge us with caution

The main limitation of studies evaluating the effects of cannabinoid consumption on the kidney is, as other studies have already mentioned, the possible under-reporting of consumption by the subjects interviewed, by obeying the precautionary principle and pending studies providing more conclusive evidence on the subject, the health professional will benefit from making the ethical choice of advising against the consumption of cannabinoids in order to limit iatrogenic kidney damage.

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