MED EMERGENCY / URGENCE

ISSN 2222-9442

MJEM

MEDITERRANEAN JOURNAL OF EMERGENCY MEDICINE

Original Articles

Case Reports

Endorsed by Lebanese Resuscitation Council

Indexed by Index Medicus

2018 - N°26

CANNABINOID HYPEREMESIS SYNDROME WITH HYPERLACTATEMIA: A CASE REPORT

HAMELIN PL, RENARD A, MICHOUD G, DEFUENTES G, AGOUSTY M, BRUYANT AC. Cannabinoid hyperemesis syndrome with hyperlactatemia: A case report. Med Emergency, MJEM 2018; 26:36-8.

Key words: cannabis, emergency department, hyperemesis syndrome, hyperlactatemia.

ABSTRACT

Introduction: The Cannabinoid Hyperemesis Syndrome (CHS) is a severe abdominal syndrome characterized by abdominal pain and intractable vomiting among long-term cannabis users. The normality of paraclinical examinations, outside of a potential hyperleukocytosis, is one of the diagnostic criteria and rules out other differential diagnosis. Actual diagnosis and therapeutics tests include showers and hot baths to provide relief to these patients.

Case report: We are reporting the case of a patient who presented at the emergency department for a CHS. He was also presenting an extremely high hyperlactatemia, known as a marker of severity in emergency medicine. The clinical and biological evolution was favorable after rehydration and a hot shower, which allowed patient discharge.

Conclusion: Many cases of severe metabolic manifestations, secondary to cannabis uses, have already been described, but an increase in lactate level after natural cannabis intoxication has never been described. Yet, an increase in lactatemia coupled with the CHS could perhaps enable us to detect patients in need for medical supervision.

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Senior involved in the patient care: Bruyant AC

Article history / info:

Category: Case report
Received: Sept. 04, 2017
Revised: Sept. 20, 2017
Accepted: Oct. 04, 2017

Conflict of interest statement:

There is no conflict of interest to declare

INTRODUCTION

The Cannabinoid Hyperemesis Syndrome (CHS) characterized by abdominal pain and intractable vomiting was first described in 2004 by Allen et al [1]. It occurs among patients with chronic and daily uses of cannabis. This syndrome's time of diagnosis is fairly long because of the persistent normality in paraclinical examinations. This normality has incidentally been recognized as one of the diagnostic criteriaby Simonetto et al. [2]. Besides, in 2017, 1% of adults in Europe between the age of 18 and 64 were admitted using cannabis on a regular basis [3]. According to the regional monitoring center for emergency departments in Provence-Alpes-Côte d'Azur, abdominal pain is the second presenting symptom among people aged between 15 and 54. Also, there is a real possibility that CHS is under-diagnosed. We are thus presenting a clinical case of a regular cannabis-user, in order to illustrate this syndrome that is rarely referred to in primary care situations.

CASE REPORT

A 33-years-old patient was admitted at the emergency room for abdominal pain, measured at 70/100 on the visual analog scale, associated with an abrupt onset of recurrent vomiting twelve hours before, without any signs of transit disorder. He reported no history of medical problems except a non-insulin dependent diabetes, treated with nutritional-hygienic measures. He also declared, at first, no use of medication, toxins or narcotics. Clinical examination showed a tachypnea (RR: 30.min^{-1}, SpO_2: 99% in ambient air) with significant sweating, but without any signs of respiratory distress. Patient was neurologically (GCS 15) and hemodynamically (CF: 60 bpm, BP: 150/90 mmHg) stable. However, he vomited several times during examination. His epigastrium was sensitive and inconstantly guarding. Moreover, the abdomen was very flexible and depressed without any organomegaly. Body temperature measurement was 35°C and hemoglucotest (with an empty stomach) was 8.8 mmol.L^{-1} with neither glycosuria nor ketonuria. ECG showed normal-oriented sinus rhythm and did not show any conduction or repolarization disorders. The biological assessment showed a hyperleukocytosis with polymorphonuclear neutrophils, without any associated increase in CRP, as well as a hyperlactatemia with respiratory alkalosis (**Table 1**). In the presence of a syndrome with abdominal pain and hyperlactatemia, the paraclinical examination is completed with an abdominopelvic scanner, both with and without contrast agent. It revealed no abnormalities.

On a therapeutic level, the patient was rehydrated with 750 mL of 0.9% sodium chloride. He also got analgesic treatment with a combination of paracetamol with morphine-titration, phloroglucinol and anti-emetic metoclopramide. Even though the patient was relieved on a pain level, the vomiting did not stop.

We finally diagnosed CHS when the patient admitted the use cannabis on a regular basis (three or four joints per day), and the normality of his lipase allowing us to move away from an acute pancreatitis diagnosis. Thereupon we offered him to take a hot shower to confirm our diagnosis and offer therapeutics relief. The shower relieved the patient immediately, thus confirming our hypothesis. He was discharged after normalization of his lactatemia, with the strong advice to stop all cannabis consumption in order to prevent relapse.

Parameter	Result	Norms
Sodium (mmol.L^{-1})	138	136 – 145
Potassium (mmol.L^{-1})	3.9	3.5 – 5.1
Chlorine (mmol.L^{-1})	**94**	98 – 107
Phosphorus (mmol.L^{-1})	**0.32**	0.87 – 1.45
Anionic gap	**26.8**	8.0 – 16.0
Glycemia (mmol.L^{-1})	**8.5**	3.9 – 6.1
Hemoglobin (g.dL^{-1})	**16.1**	12.0 – 16.0
Creatinine (μmol.L^{-1})	**90**	44 - 80
Urea (mmol.L^{-1})	6.2	2.5 – 8.3
Lipase (UI.L^{-1})	30	13 – 60
AST (UI.L^{-1})	**31**	10 – 30
ALT (UI.L^{-1})	29	7 – 35
Leukocytes (,μL^{-1})	**21810**	4000 – 10000
Neutrophil count (,μL^{-1})	**16227**	2000 – 10000
Platelets (,μL^{-1})	331000	150000 – 400000
Myoglobin (μg.L^{-1})	39	28 – 72
Troponin (hS)	< 3	< 14
CRP (mg.L^{-1})	**9.5**	< 5.0
D-dimers (mg.L^{-1})	< 0.27	< 050
Prothrombin time (%)	91	70 – 100
Fibrinogen (g.L^{-1})	**4.19**	2.00 – 4.00
pH	**7.529**	7.350 – 7.450
$PaCO_2$ (mmHg)	**22.5**	35.0 – 45.0
PaO_2 (mmHg)	**118.0**	83.0 – 108.0
Bicarbonates (mmol.L^{-1})	**18.7**	21.0 – 28.0
Carboxyhemoglobin A (%)	1.5	0.5 – 1.5
Methemoglobin A (%)	1.1	< 1.5
Lactates (mmol.L^{-1})	**6.4**	0.5 – 1.6

Table 1: Blood tests results

DISCUSSION

Iterative and intractable vomiting among young patients should at first indicate meningeal affection, and, if there is fever, a lumbar puncture should be performed right away after eliminating signs of neurological focalization and the presence of purpura. The etiological discussion would be different though, without a fever, and opens up many different possibilities: food or medicine intolerance, intracranial hypertension, vestibular syndrome, esophageal pathology, etc.

Outside of a functional or surgical context, abdominal pain among young adults should indicate adrenal insufficiency, vasculitis,

nocturnal porphyria crisis, lead poisoning, angio-neurotic edema, nocturnal paroxystic hemoglobinuria and auto-inflammatory diseases (starting with familial Mediterranean fever).

CHS's diagnostic criteria were first described by Simonetto et al. from a series of 98 patients [2]. The case studied in this article initially had a strong suspicion about CHS, but it raises the question of a high increase in lactatemia, which to our knowledge was never described as such in specialized literature. Indeed, some cases of acute renal insufficiency with metabolic alkalosis, secondary to vomiting, were described, while also showing an expansion of the anion gap that is very similar to our own observations but without, however, any dosage of serum lactate [4;5]. Lactatemia in emergency services is known to be a reliable marker of severity and an efficient screening tool [6]. This case report suggests that cannabis intoxication could induce acute and severe clinical diagnoses. As a matter of fact, cannabis-induced acute pancreatitis [7], myocardial infarction [8] or even cerebral infarction by multifocal vasoconstriction [9] are some of the conditions that can be witnessed in emergency services.

Furthermore, cannabis is known to be the cause of arteritis, especially at the cerebral, myocardial and lower limbs levels, because of its vasoconstrictor effect – by its actions on CB-1 receivers – if not thrombotic on the smaller arteries and arterioles. This vasoconstriction reduces downstream flow and thus the tissue supply in oxygen. Splanchnic hypovascularization occurs through this mechanism, as well as the resulting hyperlactatemia. Thus we propose to qualify Simonetto work, by underlining the possibility of hyperlactatemia during CHS. On the same note, the description of recurrent and regressive acute renal failure by Abodunde et al. [5] allows us to discuss its functional determinism (secondary to dehydration induced by vomiting) and/or microangiopathic ischemia.

However, an elevation of serum lactate in a context of recurrent vomiting should evoke an intestinal beriberi by thiamine deficiency (Vitamin B1), often found among under-nourished and/or chronic alcoholics patients [10], which was not the case in our patient. Also, hyperlactatemia can be a sign of: hypovolemia by depletion, a decrease in arterial oxygen content, intoxication responsible for an intracellular oxygen impaired utilization (intoxication to hydrocyanics for instance), or an increase in lactates' hepatic synthesis (alcoholic consumption, long-term biguanide consumption, etc.). None of which were obvious in our observation.

CONCLUSION

The CHS remains very unknown among emergency doctors, despite being increasingly described in the specialized literature. It still is a diagnosis of exclusion against an acute abdominal syndrome and should not prevent doing examinations to discard a surgical pathology. However, a cannabis consumer presenting a painful abdominal presentation with hyperemesis and meeting the diagnostic criteria we mentioned above, should benefit from a hot shower, the only real diagnostic and therapeutic test. We also suggest, after this observation, to systematically perform a severity diagnosis by dosing serum lactate. Indeed, even if extremely severe metabolic forms were only reported for synthetic cannabinoids, because of anaffinity for CB1/2 receptors up to 200 times superior compared to natural cannabinoids [11], the physiopathology of tetrahydrocannabinol's action (cannabis' active substance) on target organs remains poorly-known. Thus, anytime hyperlactatemia (a severity marker in emergency medicine) occurs during an "uncompensated" chronical cannabis intoxication, there is a risk of single or multiple organ failure.

REFERENCES

1. Allen JH, Moore GM de, Heddle R, Twartz JC. Cannabinoid hyperemesis: cyclical hyperemesis in association with chronic cannabis abuse. Gut 2004; 53:1566-70.
2. Simonetto DA, Oxentenko AS, Herman ML, Szostek JH. Cannabinoid hyperemesis: A case series of 98 patients. Mayo Clin Proc 2012; 87:114-9.
3. European Monitoring Centre for Drugs and Drug Addiction. European Drug Report 2017: Trends and developments. Luxembourg: Publications Office of the European Union; 2017.
4. Price SL, Fisher C, Kumar R, Hilgerson A. Cannabinoid hyperemesis syndrome as the underlying cause of intractable nausea and vomiting. J Am Osteopath Assoc 2011; 111:166.
5. Abodunde OA, Nakda J, Nweke N, Veera RL. Cannabinoid hyperemesis syndrome presenting with recurrent acute renal failure. J Med Cases 2012; 4:173-5.
6. Levraut J, Lemoël F, Leplatois T. Intérêt du dosage du lactate en médecine d'urgence. Ann Fr Med Urg 2011; 3:185-91.
7. Lorvellec A, Thiriet L, Andrianjafy C, Gervaise A, Seigne AL, Rey P. Recurrent cannabis-induced acute pancreatitis. Presse Med 2015; 44:468-71.
8. Hodcroft CJ, Rossiter MC, Buch AN. Cannabis-associated myocardial infarction in a young man with normal coronary arteries. J Emerg Med 2014; 47:277-81.
9. Wolff V, Lauer V, Rouyer O, Sellal F, Meyer N, Raul JS, et al. Cannabis use, ischemic stroke, and multifocal intracranial vasoconstriction: a prospective study in 48 consecutive young patients. Stroke J Cereb Circ 2011; 42:1778-80.
10. Duca J, Lum CJ, Lo AM. Elevated lactate secondary to gastrointestinal beriberi. J Gen Intern Med 2015; 31:133-6.
11. Sherpa D, Paudel BM, Subedi BH, Chow RD. Synthetic cannabinoids: The multi-organ failure and metabolic derangements associated with getting high. J Community Hosp Intern Med Perspect 2015; 5:27540.