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CASE REPORT

MESENTERIC ISCHEMIA AFTER MASSIVE CANNABIS AND COCAINE USE IN A YOUNG SUBJECT

El wassi Anas, El kinani Siham, Zalarh Fadoua, Abdelhak Ettaoussi, Khaija Kamal, Abdessamad Majd, Mounir Bouali, Abdelilah El bakouri and Khalid El Hattabi

Department of visceral surgery, University Hassan II, Faculty of Medicine and Pharmacy of Casablanca, CHU Ibn Rochd, University of Hassan II, Casablanca, Morocco

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Corresponding author:
El wassi Anas

ABSTRACT

Cannabis and cocaine use cause marked vasoconstriction, which can lead to a variety of systemic complications, particularly gastrointestinal. Among these, mesenteric ischemia is particularly difficult to diagnose, and can have serious consequences if not treated promptly. We report the case of a 19-year-old man presenting with mesenteric ischemia related to chronic cocaine and cannabis use. The diagnosis was established by computed tomography (CT). He required emergency surgery with segmental resection of the necrotic small intestine and placement of an ileostomy. The literature on cocaine-induced intestinal ischemia reveals great variability in clinical and therapeutic outcomes. Biological tests lack specificity, and are negative in most cases. We suggest that testing for cannabis and cocaine should be carried out systematically in all young patients admitted for a vascular accident, regardless of location.

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INTRODUCTION

Cannabis is the most widely used drug in the world, particularly among young people. Cocaine ranks third. A few cases in the literature report the association between cannabis and cocaine and mesenteric and renal infarction (1). Cannabis is considered a "soft" drug because, unlike "hard" drugs such as heroin, cocaine or crack, it does not lead to physical dependence or overdose. Tetrahydrocannabinol, the active ingredient in cannabis, diffuses rapidly through the body and interacts with cannabinoid receptors, particularly in the brain, heart and blood vessels (2). The most commonly recognized negative effects are the risk of psychological dependence and the possibility of decompensating an underlying psychiatric pathology. In recent years, cannabis has been identified in the literature as a significant vascular risk factor (2). Cocaine has also been clearly identified as a cardiovascular risk factor, with numerous cases of myocardial infarction described (3-4). Several observations of cerebral infarction (5,6), aortic dissection (7), mesenteric and renal ischemia (9,10) and distal arteriopathy (11,12) have been reported. We report a case of mesenteric ischemia secondary to massive consumption of cannabis and cocaine in a 19-year-old man who presented to the emergency department with a bowel obstruction.

CASE REPORT

The patient was 19 years old, a chronic 20 Pa unweaned smoker, an occasional cannabis and high-dose cocaine sniffer, and a chronic unweaned alcoholic. Admitted to our department with a bowel obstruction. Clinically, the patient presented for 6 days with a sub-occlusive syndrome consisting of cessation of matter without cessation of gas, with diffuse abdominal pain and bilio-food vomiting, associated with scanty rectal discharge. Clinical examination revealed generalized abdominal defensiveness, with a rectal examination in which the finger came back soiled with normal-coloured stools. A radiograph of the abdomen without preparation showed hydro-aeric levels in the groin (Figure 1). On admission, the white blood cell count was 27,760/mm3 with a CRP of 392g/l. The rest of the workup was normal. Abdominal and pelvic CT scans showed a bowel obstruction upstream of a sub-stenosing parietal thickening in the ascending colon measuring 27.4 mm in maximum thickness, with a normal appendix and a small peritoneal effusion (Figure 2). Intraoperative exploration revealed a medium-abundance effusion of squinty liquid with the presence of false membranes in the inter-vesicles, with a perforated ileal thickening (figure 3) located 20cm from the last ileal loop and

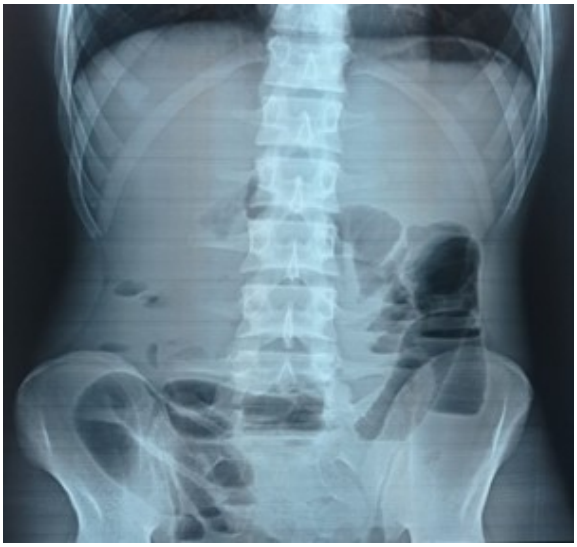

Figure 1. Hydroaeric levels in the hare

2, 4m from the duodenojejunal angle, with 130cm of upstream small bowel with signs of venous infarction scattered in some of the ileal loops (figure 4), revived with warm saline. The rest of the exploration was unremarkable. Segmental bowel resection was performed without incident, with placement of a double-barrel ileostomy. The post-operative course was marked at 6 days post-op by the appearance of generalized abdominal pain, with no other associated signs, and generalized abdominal tenderness. An abdomino-pelvic CT scan was performed, revealing: an occlusion of the mesenteric artery superior to 59mm from its origin, with signs of gastrointestinal distress. With medium-abundance pneumoperitoneum. And left anterior Nutcracker syndrome (figure5). Intraoperative exploration revealed a medium-sized peritoneal effusion with pus, and a medium-sized hemoperitoneum with 1 m of necrotic and sphacelic small intestine perforated at 40 cm from the ADJ and 10 cm from the JIC. A bowel resection was performed, with placement of a double-barrel ileostomy.

NB: after resection, the patient was left with a 50 cm small bowel. The evolution was marked by the onset of secondary short small bowel syndrome with worsening of the patient's condition and death

DISCUSSION

Acute mesenteric ischemia (AMI) is a serious medical emergency characterized by mesenteric vascular insufficiency, leading to intestinal ischemic injury. Diagnosis is based primarily on a multiphasic injected CT scan, which not only makes the diagnosis, but also assesses its severity. If not treated promptly, IMA progresses to irreversible intestinal necrosis, organ failure and death. The main indicators differentiating early from advanced stages are organ failure, increased serum lactates and the presence of signs of necrosis on CT images. Major CT signs include absent or reduced bowel wall enhancement, loop dilatation, parietal pneumatosis, as well as the presence of aeroportia or extradigestive gas bubbles, the latter being associated with increased severity. (13). Non-occlusive mesenteric ischemia is more frequent in elderly patients with reduced cardiac output, thus reducing irrigation of the mesenteric vessels(14) . In younger patients without vascular risk factors, the use of substances such as cocaine may be involved (15). Mesenteric vessels contain a large number of alpha-adrenergic receptors (16). Cocaine acts by blocking the reuptake of presynaptic monoamine neurotransmitters, enhancing the normal adrenergic response and leading to vasoconstriction (17). It also promotes calcium influx across the vascular endothelium, exacerbating further vasoconstriction (18). These mechanisms can reduce intestinal blood flow, leading to ischemia and even intestinal necrosis, with an increased risk of perforation. Although most cases of cocaine-related mesenteric ischemia are non-occlusive, this substance can also increase thrombus formation and platelet aggregation, while reducing fibrinolytic activity (19). Thus, anticoagulation may be considered until thrombus exclusion is achieved. We report a case of mesenteric ischemia secondary to massive cannabis and cocaine consumption in a 19-year-old man. This patient's only risk factor was chronic smoking and occasional cannabis and drug use. He snorted cocaine in high doses and had increased his consumption in the hours preceding the mesenteric ischemia. The exhaustive etiological work-up did not identify any cause for this infarction other than the concomitant use of cannabis and cocaine. The arterial work-up showed no signs of aortic or peripheral atherosclerosis. Biological tests routinely performed to check for thrombophilia or cardiovascular risk factors were normal. No cardiac rhythm disorders were detected.

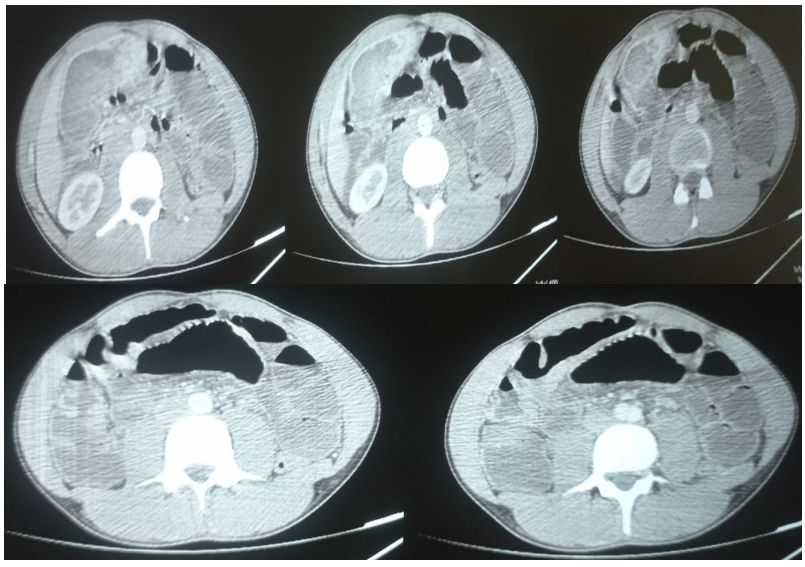

Figure 2. Gravesic distension with thickening of the ascending colon

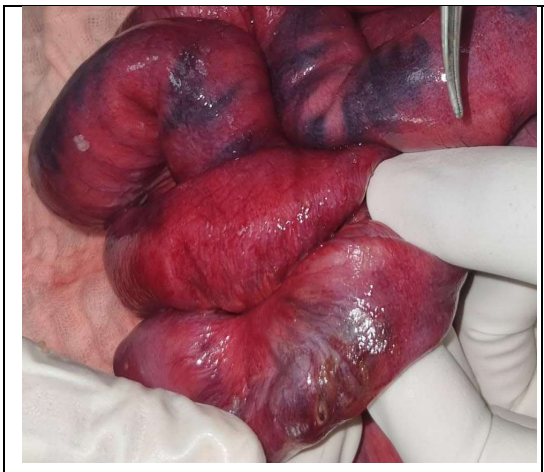

Figure 3. Perforated greaves

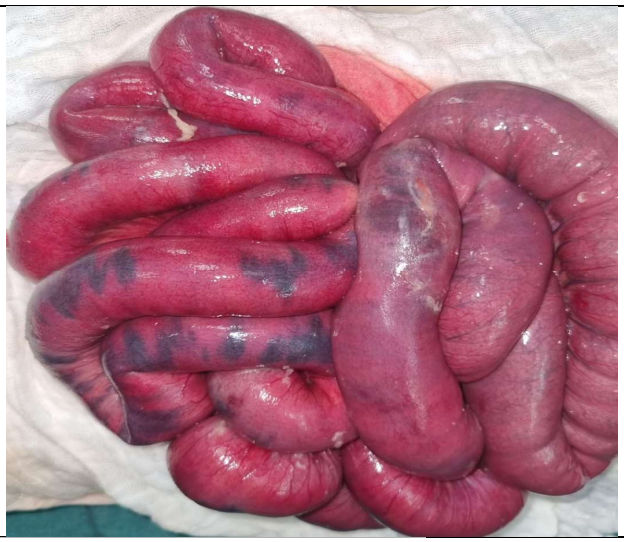

Figure 4. Infarcted greaves

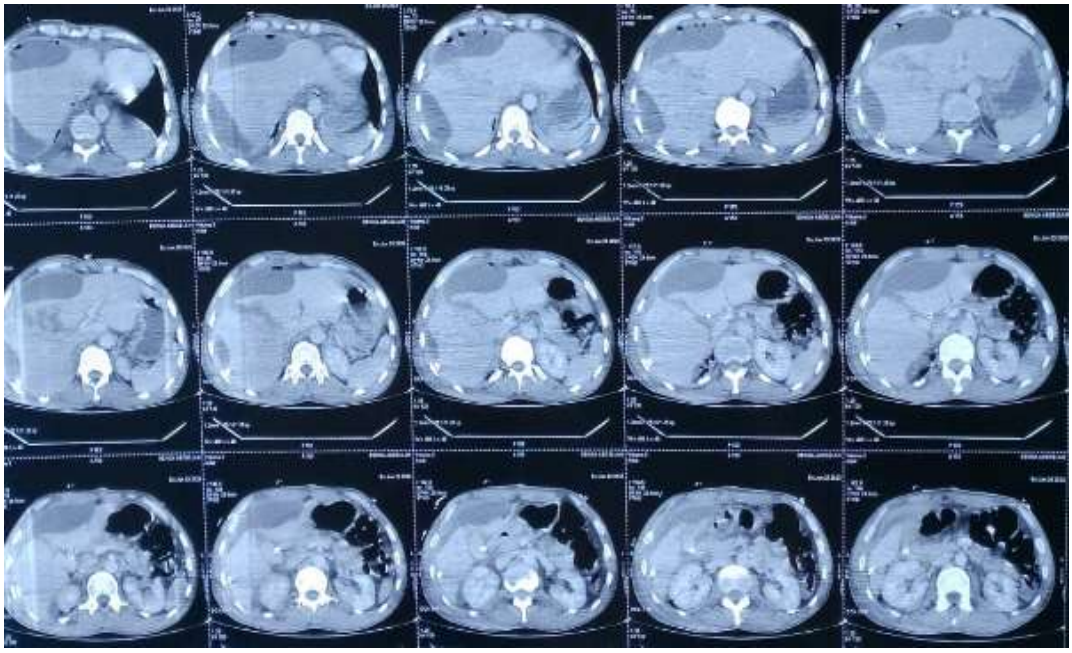

Figure 5. Abdominal-pelvic CT scan showing occlusion of the mesenteric artery superior and pneumoperitoneum

Arterial occlusions of the mesenteric or renal vascular system have already been reported in the literature in patients with a normal cardiovascular work-up. The processes by which cannabis and cocaine lead to vascular accidents are multiple. Cannabis reduces blood pressure, causes vasoconstriction and increases platelet adhesion and aggregation (20,21). Cocaine causes arterial hypertension, vasoconstriction and platelet proaggregation (22). The simultaneous absorption of cannabis and cocaine can potentiate the cardiovascular toxic effects of each substance (23), as can the concomitant absorption of alcohol. Another factor to be taken into account with cannabis is the mode of consumption: a "bong" (equivalent to 4-5 joints in a single breath) is thought to be associated with a higher vascular risk than a single joint, but data are lacking.

CONCLUSION

Cannabis and cocaine are implicated in the occurrence of vascular events through three main mechanisms: cannabis-related hypotension, cocaine-induced arterial hypertension, and arterial vasoconstriction and thrombosis formation. Despite the high prevalence of these substances, associated vascular events are rarely reported, suggesting that they may be underestimated as a toxic cause. It is essential to systematically investigate the use of cannabis and cocaine, as well as their mode of administration, in young patients admitted for a vascular accident, whatever the location. Such an approach would enable us to better understand and document the effect of these substances in these pathologies.

Conflict of interest: The authors declare no conflict of interest in relation to this article.

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