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CASE REPORT

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Amphetamine use associated with acute pancreatitis

Bayman Kamiran and Macaigne Gilles*

Department of Gastroenterology, Hospital Center of Marne la Vallee 77600 JOSSIGNY, France

Introduction

The MDMA (3,4-methylenedioxy-methamphetamine) is a synthetic psychotropic molecule of the amphetamine class. It is a stimulant of the central nervous system which possesses particular psychotropic characteristics, of a powerful serotoninergic which provides it in high doses or in case of regular use a neurotoxic power [1]. The MDMA is a consumable drug in the form of crystals or pills, more commonly known under the name of ecstasy.

The MDMA can cause short-term complications such as, among others, hyperthermia with dehydration, neurological diseases and acute hepatitis and long-term toxicities such as psychiatric, neurological, heart and liver. Acute pancreatitis induced by the consumption of MDMA has not previously been reported in the literature. We report, to our knowledge, the first observation of acute pancreatitis associated with recent use of the MDMA.

Case report

A 24-year-old young woman was hospitalized in May 2017 for intense and persistent epigastralgia since a few hours. She had not any previous history except smoking about 10 packs of cigarettes/year. There was no family history of pancreatic disease. At the interrogation, the patient reported a consumption of MDMA in crystals diluted in water during the previous evenings, the appearance of the acute abdominal pain; she consumed it for the first time 5 mg of MDMA diluted in water. She did not consume alcohol nor other drugs. On arrival, serum lipase levels were 8 times in the normal, serum ALAT levels were normal, and blood alcohol levels were negative. There was not any signs of clinical and biological gravity and the abdominal computerized Tomography realized with the injection of contrast agent after 72 hours of development, showed a global increase in pancreas size with homogeneous and discrete enhancement of peri pancreatic fat without necrosis (Stage C of Balthazar with severity score 2). The progress under medical treatment was beneficial with a resumption of the diet without pain.

The abdominal ultrasound was normal and the echo-endoscopy and the bilio-pancreatic MRI showed no vesicular lithiasis, the intra- and extrahepatic biliary ducts were normal and there was no increase for a pancreas divisum. The hepatic, lipid, and phospho-calcium balances were normal at admission to the emergency room. The serological survey for mumps virus, the cytomegalovirus, the Epstein Barr virusand the human immunodeficiency virus was negative. The search for anti-nuclear antibodies, neutrophil anti-cytoplasmic antibody (ANCA) was negative. Serum IgG4 level was normal. The absence of hepatic cytolysis on arrival was not in favour of Oddi sphincter dyskinesia. The toxic cause of acute pancreatitis related to the consumption of MDMA was retained. The patient no longer used drugs after discharge and was still asymptomatic more than 3 months after the initial episode. According to the French method of imputability of the medicinal acute pancreatitis [2]. The intrinsic imputability of MDMA was considered very likely: a) acute epigastralgias appeared a few hours after MDMA consumption, b) complete etiological balance of negative acute pancreatitis, c) rapid improvement of pancreatitis at cessation of consumption MDMA, d) absence of recurrence during follow-up.

Discussion and conclusion

In our case, the occurrence of acute pancreatitis following an increase of the amount of MDMA consumed could result in an accumulated dose effect causing pancreatic toxicity. The normality of the hepatic biological balance at her arrival to the emergency room, the absence of vesicular lithiasis in the echo-endoscopy results and the slow and the progressive decrease of the serum level of pancreatic enzymes despite a rapidly favourable clinical evolution were not in favour of the biliary origin of the acute pancreatitis. The rest of the etiological balance was negative.

There are two types of toxic effects of the MDMA. The first one concerns all acute poisoning which are not dose-dependent, and the second concerns the poisonings occurring in the medium or long-term dose-dependent. The MDMA, as all the amphetamine derivatives, increases the release of noradrenaline and dopamine engendering a sympathomimetic activity and, in particular, a positive chronotropic effect which causes many cardiac effects such as tachycardia, one hyper or an arterial low blood pressure. Cases of myocardial necrosis at some young subjects without a risk factor of vascular lesions have been reported [3], the physiopathological mechanism which can be the same as in case of use of cocaine. A combination of secondary small-caliber vasospasm of arteries to the massive release of catecholamines and the formation of thrombus by platelet aggregation mediated by catecholamines could be one of the mechanisms involved. Thus, as has been reported with the consumption of cocaine [4], the mechanism of acute pancreatitis induced by u-MDMA, even though uncertain, may be ischemic, linked to a peripheral vasoconstrictor effect [5].

The effects of chronic administration of methamphetamine on pancreatic tissues were histopathologically studied in experimental models. Ito et al .[6] demonstrated that histological toxic lesions in

exposed rats were severe regional hemorrhage, partial acinar cell necrosis, destruction of the acinar cells, neutrophile infiltration, interstitial vessel dilatation, interstitial edema and fatty cell invasion, fibrosis and cirrhosis-like lesions with destruction and degeneration of the acinal cells. The autopsies demonstrated a severe acute necrotic hemorrhagic pancreatitis, with only scattered slight hemorrhaging in the brain and lungs. These findings indicate that chronic administration of methamphetamine to rats evoked significant changes in pancreatic tissues including some degeneration of the endothelial cells of the small vessels in this hypoxia-vulnerable organ.

Contrary to the pancreatic toxicity of DMDA, the hepatic toxicity has been reported more frequently in the literature through different mechanisms such as vascular involvement and direct toxicity of ecstasy [7], which can be the cause of acute hepatitis sometimes severe [8]or chronic liver disease [9].

Ecstasy is the major synthetic substance of the past 15 years.

This aetiology of pancreatitis may appear limited in frequency due to the increasing consumption of this drug. This cause is undoubtedly underestimated, which must to incite to be vigilant and wary about it.

The diagnosis of acute pancreatitis MDMA establishes certainly, a diagnosis of exclusion, but given the increasing consumption of this substance, it is quite possible that this diagnosis is in use more frequently. Its frequency is maybe underestimated due to a non-systematic etiological research. It is necessary to consider it, once we meet a young patient, hospitalized for acute pancreatitis non-A non-B. Although in our case we cannot demonstrate a definite causal link between MDMA consumption and the occurrence of acute pancreatitis, it is probably a new association. Thus, the consumption of MDMA could be added to the list of possible aetiologies of acute pancreatitis.

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Correspondence: Macaigne Gilles, department of Gastroenterology, Hospital Center of Marne la Vallee 77600 JOSSIGNY, France, Tel: 606508686; E-mail: gmacaigne@ghef.fr

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