



Cannabinoid-induced acute pancreatitis

To the Editor,

While most cases of acute pancreatitis are caused by biliary tract pathologies, alcohol consumption and infections, cases of pancreatitis related to drugs are so rare, as they constitute approximately 2% of pancreatitis cases (1). In the literature, while there have been several reported cases of pancreatitis induced by drugs, such as metronidazole, tetracycline, azathioprine and diuretics, cases of pancreatitis related to an illicit drug, tetrahydrocannabinol (THC) are rarely reported (2,3). We present a case who had repetitive attacks of pancreatitis due to chronic THC abuse.

A 28-year-old boy was referred to the emergency unit with nausea and severe abdominal pain radiating to the back. The patient had no remarkable medical and family history and was not taking any prescription medications. He had smoked half a pack a day and had not consumed alcohol. He had a regular use of illicit drug, THC, for 3 months. Physical examination revealed no abnormal finding except for epigastric tenderness and diminished bowel sounds. His laboratory tests were remarkable for leukocytosis and increased level of amylase and lipase. Triglyceride and calcium levels were normal. Abdominal ultrasound showed features of edematous pancreatitis. Magnetic resonance cholangiopancreatography (MRCP) revealed no abnormal finding related to the gallbladder and biliary ducts. Conventional treatment palliated symptoms and normalizes the abnormal laboratory values. The patient was admitted to the emergency unit with the second attack of pancreatitis two weeks after discharge. The same work-up with the first pancreatitis attack was performed for the patient and no specific well-known etiology was found for recurrent pancreatitis. The questioning of the patient revealed intense THC intake in his usual life. The patient was diagnosed as recurrent pancreatitis due to THC and was treated symptomatically. The amylase and

lipase levels with his complaints regressed in a week. No attack of pancreatitis occurred after discontinuation of the drug at the 4-month follow-up.

The United Nations Office on Drugs and Crime estimates that approximately 166 million people used cannabis in 2006, equivalent to 3.9% of the global population aged 15-64 (4). There are clear ramifications associated with long-term cannabis use, including infertility, erectile dysfunction, visual disorders, and schizophrenia; however, pancreatitis is one of the least reported complications of this drug. This coexistence was first reported by Grant et al. in 2004 as a case report (3).

The mechanism of how THC causes pancreatitis is unclear, but it is thought to be via the cannabinoid receptors. These receptors have also been reported to activate the mitogen activated protein kinases p38 and cJun N-terminal kinase, which are involved in early acinar events leading to acute pancreatitis and induction of pro-inflammatory cytokines. Michler et al. (5) showed that the activation of cannabinoid receptor 2 reduces inflammation in acute experimental pancreatitis via intra-acinar activation of p38 and mapkinase 2 dependent mechanisms. The clear-cut mechanism of cannabinoid induced pancreatitis needs to be clarified. However, it is problematic due to difficulty in monitoring cannabinoids, illegality of cannabis and getting patients to admit to drug use.

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