

## A Case of Cannabis-Induced Pancreatitis

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### ABSTRACT

**Context** There are no previous reports of acute pancreatitis associated with cannabis use in the general population. Drugs of all types are related to the aetiology of pancreatitis in approximately 1.4-2.0% of cases.

**Case report** We report the case of a 29 year old man who presented with acute pancreatitis after a period of heavy cannabis smoking. Other causes of the disease were ruled out. The pancreatitis resolved itself after the cannabis was stopped and this was confirmed by urinary cannabinoid metabolite monitoring in the community.

**Conclusion** To our knowledge this is the first description of a case of cannabis induced pancreatitis. However, the link is difficult to establish and further evidence is required to prove the association.

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### INTRODUCTION

The major causes of pancreatitis are well-known and, in the UK, gallstones and ethanol are established as the leading factors [1]. Many drugs are known to aggravate pancreatitis but there is little information in the literature as to whether or not cannabis can cause pancreatitis [2]. We are reporting a case of cannabis-induced pancreatitis and, to our knowledge, this has never been previously described.

### CASE REPORT

A 29 year old man presented to Accident & Emergency with a one day history of sudden onset, severe abdominal pain radiating to the back, plus nausea and vomiting. The patient had recently returned from a yachting holiday in The Netherlands. He had no previous medical or surgical history, no history of alcohol use, no history of trauma and took no medication.

On physical examination, the patient's abdomen was distended and diffusely tender. His bowel sounds were reduced. Rectal examination proved negative. Blood tests revealed an amylase of 1,997 IU/mL (reference values: 0-180 IU/mL), a raised white cell count ( $17.2 \times 10^9 \text{ L}^{-1}$ , reference values:  $4-11 \times 10^9 \text{ L}^{-1}$ ) and a moderately elevated C-reactive protein level (132 mg/L, reference values: 0-10 mg/L). Serum values of urea, creatinine, AST, ALT, alkaline phosphatase, triglycerides, cholesterol, calcium and bilirubin were within reference limits.

Ultrasound examination of the abdomen showed no abnormalities, the structure of the pancreas appeared normal and there was no evidence of gallstones or biliary tree dilatation/obstruction. An abdominal CT scan revealed an inflamed pancreas but no evidence of necrosis and an ERCP confirmed that there were no gallstones, biliary tree pathologies or structural abnormalities in the pancreas.

The only significant factor in our search for a cause was that the patient's consumption of

cannabis had increased greatly over the past few weeks during his journey around The Netherlands. This was noted but we were unsure of its significance at the time.

Initially, we managed the patient supportively. Despite the intervention of the acute pain team, his abdominal pain did not completely settle. Large doses of oral morphine did not help with the worst of the pain. Three days after admission, the patient had a second episode of severe, escalating pain. This attack of pancreatitis required sedation. His amylase rose to 3,200 IU/mL, the CRP to 523 mg/L and the liver function tests were elevated (AST, 212 IU/L; reference values: 3-35 IU/L. ALT, 101 IU/L; reference values: 3-35 IU/L). His attitude whilst on the ward alerted the nursing staff that he might have smoked cannabis. Believing there might be some connection between the above events, we queried whether it would be a good idea to measure the patient's serum cannabinoid levels. When questioned, the patient denied any drug use and there were no cannabinoids detected in his blood.

He then went on to suffer a third attack of acute pancreatitis pain ten days post admission. A rise in amylase to 2,905 IU/mL, CRP of 491 mg/L and, this time, there was a detectable amount of THC (tetrahydrocannabinol) - a cannabinoid metabolite - in his blood. A second abdominal CT scan revealed the initial signs of pancreatic necrosis in the tail of the pancreas and he was transferred to a High Dependency Unit bed for monitoring. The patient now admitted that he had been smoking cannabis throughout his stay in hospital, brought in for him by a friend. He believed that the soothing effects of cannabis would settle his abdominal pain. However, in this case, it only seemed to exacerbate his symptoms. The patient was unwilling to accept this possible causality and suffered a fourth episode of severe acute abdominal pain, 18 days post-admission and 4 days after an operation to debride a small necrotic area of his pancreas. Again, his serum THC levels were elevated.

The patient was strongly advised to cease his cannabis use and eventually his pain resolved

itself completely four weeks after admission. At follow-up he was doing very well, he had no signs or symptoms of pancreatitis and he had effectively given up his use of cannabis (this was confirmed by urinary THC analysis performed by the community drug and alcohol team).

## DISCUSSION

Cannabis is the most popular illicit drug in the world. Research on the impact of regular, long-term use shows its complications include erectile dysfunction, infertility, hearing and visual disorders [3], and schizophrenia [4]. To our knowledge, this is the first case of cannabis-induced pancreatitis in the literature; the reasons for this include the difficulty of excluding all other possible causes and problems with monitoring serum cannabinoid levels (the half-life of cannabinoids in the human body is known to be relatively short as it is rapidly taken up by the tissues and metabolised [5, 6], making it difficult to test for). It may also be possible that cannabis-induced pancreatitis is dose-related and, indeed, our patient only presented after several weeks of heavy usage. Our patient had been smoking cannabis for 3 months before the onset of acute pancreatitis.

In our case, all other possible causes of pancreatitis were ruled out. There was no history of alcohol use and no family history of pancreatitis. There was no evidence of gallstone disease, and the serum values of calcium and triglycerides were normal. The patient was not taking any other medication or narcotics.

No data exists regarding a possible mechanism for cannabis-induced pancreatitis. The current state of knowledge of the biochemistry of cannabinoids is increasing at an exponential rate and, with discoveries of cannabinoid receptors in unexpected areas of the body, new potential research/treatment avenues are appearing at an increasing rate. Cadas *et al.* [7] reported that cannabinoid receptors are present in the stomach and small intestine. Animal experimentation demonstrates that they are involved with

various intestinal peptides and smooth muscle motility. It may be feasible that sustained activation by exogenous cannabinoids interfere with pancreatic secretions in relation to enteric events or the smooth muscle component of pancreatic ducts and sphincters. The proportion of cases of pancreatitis caused by drugs of all types is estimated to be around 2% in the general population [8, 9]. Clear evidence of a definite association with pancreatitis, by means of rechallenge tests or consistent case reports supported by animal experiments or data on the acute incidence of pancreatitis in drug trials exists for didanosine, sodium valproate, aminosalicylates, oestrogen, and calcium [8]. An association with drug-induced pancreatitis is likely, but not proven, for thiazide diuretics, ACE inhibitors, some NSAIDs, clozapine and statins [9]. Many drugs have been reported to be associated with acute pancreatitis; however, lack of rechallenge evidence, consistent statistical data, evidence from experimental studies or evidence of a possible mechanism prohibit definitive proof for most of them. Despite the low incidence of drug-induced pancreatitis, all patients with acute pancreatitis of an unknown aetiology should be carefully questioned about drugs which could be possibly responsible for triggering the disease.

In conclusion, we have described a case of cannabis-induced pancreatitis. This is the first such case in the literature and it reinforces the fact that drug-induced or aggravated pancreatitis is a serious problem. Considering the frequent use of cannabis in the general population, this case should be kept in mind. The association needs to be explored but is problematic because of the difficulty in monitoring cannabinoids, the illegality of cannabis and getting patients to admit to drug use.

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**Keywords** Analgesia; Cannabis; Cannabinoids; Case Reports; Pancreatitis;

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**Abbreviations** THC: tetrahydrocannabinol

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