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Case Report



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Opioid-induced pancreatitis: A case study

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ABSTRACT

Rationale: Opioids, the class of drugs, frequently used for pain relief in acute pancreatitis are known to act as a dual-edged sword. They are capable of causing acute pancreatitis though very rare.

Patient's Concern: A 45-year-old male presented with complaints of acute onset, severe pain in the abdomen, radiating to the back. The symptoms were preceded by regular oral intake of opium.

Diagnosis: Opioid-induced pancreatitis.

Interventions: He was managed conservatively with non-opioid analgesic-acetaminophen 15 mg/kg every 6 hours along with intravenous Ringer's lactate for 5 days.

Outcomes: The patient was discharged in stable vitals.

Lessons: Drug-induced pancreatitis, though a rare entity, must be dealt with a high degree of suspicion once the other common factors are ruled out. Thus, this case highlights the importance that opioids can act as a double-edged sword in the way that it is given for pain relief in patients suffering from pancreatitis and can also be the culprit in causing it.

KEYWORDS: Acute pancreatitis; Drug-induced pancreatitis; Opioid; Toxicology

1. Introduction

Drug-induced pancreatitis, a diagnosis of exclusion in itself implicates an exhaustive list of drugs. It is said to represent 5% of the cases of acute pancreatitis, making it the third most common cause of pathology once the two most common causes like alcohol consumption and cholelithiasis are ruled out[1]. The incidence of drug-induced pancreatitis is about 5-8 per 100 000 adults worldwide[2]. Opioids are one of the incriminated classes. There

have been case reports about opioids in the form of heroin and codeine leading to acute pancreatitis[3,4]. The cause in such cases has been attributed to the sphincter of Oddi dysfunction but the exact pathophysiology is yet to be established[5]. We report one such rare case of a patient who presented with acute pancreatitis which on further workup was diagnosed to be opioids induced after the rest of the common causes were ruled out.

2. Case report

A 45-year-old, male patient presented to the medicine emergency with complaints of upper abdominal pain for 3 days which was acute in onset, severe in intensity, and radiating to the back. It was also associated with non-passage of stools for the same duration. He had an episode of vomiting before his presentation to the hospital. The vomitus was nonblood-stained and non-bilious. No urinary complaints were present. There was no history of fever, yellowish discoloration of the eyes, alcohol intake, travel, or trauma. No similar complaints were present in the past. He had no history of comorbidities like diabetes, hypertension, joint pains, or congenital problems. Family history was not significant. There was no history of any gastroenterological intervention or surgery done recently

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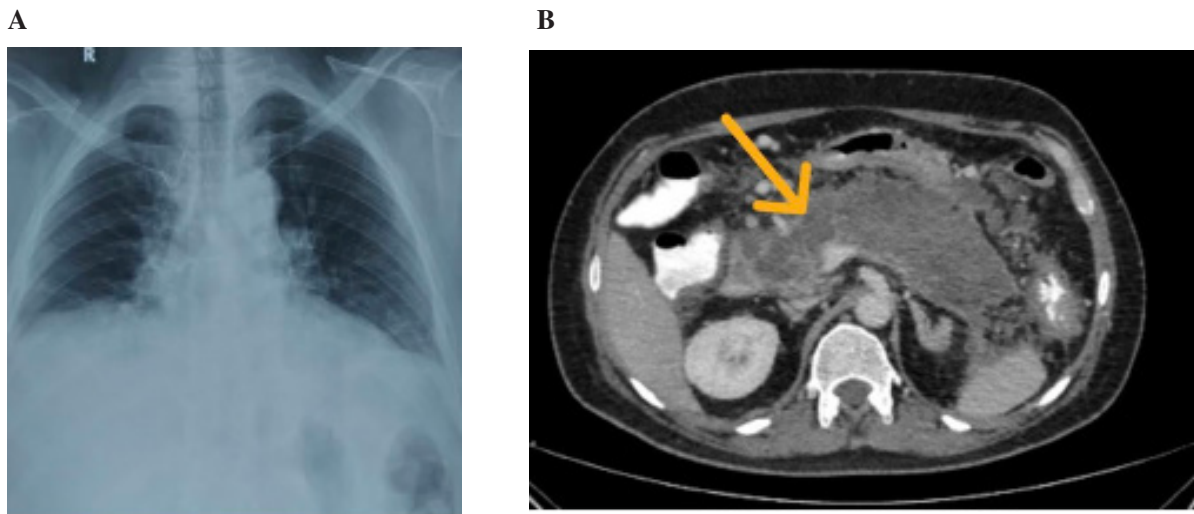


Figure 1. Radiological imaging of a 45-year-old patient. A) Chest roentgenogram showing blunting of the right costophrenic angle; B) Abdominal contrast-enhanced computerized tomography (CECT) showing a bulky pancreas with a well-defined peripancreatic collection (arrow).

before the onset of symptoms. On further history, it was revealed that he was a known case of hyperthyroidism for 2 years and was on carbimazole 5 mg OD for which he had been non-compliant for 5 days. He had been abstinent from alcohol for 1.5 years but had been consuming opium orally for the past 3 years. The last intake of opium was 3 days ago.

A written informed consent was duly signed by the patient. The consent was obtained after explaining to the patient that no identity will be revealed and the case information, including pictures will be used for education purpose only. The patient gave positive consent for publication and authors certify that written patient consent is present, procured for publication.

At presentation, he had stable vitals with blood pressure of 128/88 mmHg, respiratory rate of 18 breaths per minute, pulse rate of 102 beats per minute, temperature of 36.7 degree celsius, and oxygen saturation of 96% by pulse oximetry. On general physical examination, there was no pallor, edema, icterus, koilonychia, lymphadenopathy, clubbing, or tremors. On superficial abdomen examination, tenderness was presented in the left upper quadrant. A deep abdominal examination was not done to avoid causing unnecessary pain to the patient. Auscultation of the abdomen revealed sluggish bowel sounds.

The baseline blood investigations revealed hemoglobin of 12.7 g% (13 g%-16 g%), hematocrit of 38.6% (40%-50%), total leucocyte count of $11.4 \times 10^9/L$ ($4.0 \times 10^9/L$ - $10.0 \times 10^9/L$), and platelet count of $170 \times 10^9/L$ ($150 \times 10^9/L$ - $450 \times 10^9/L$). No hepatic or renal derangements were seen. The total protein was 6.0 g/dL (6.0-8.0 g/dL) and serum albumin was 3.3 g/dL (3.5-5.5 g/dL). The lipid profile had slightly elevated serum triglycerides (205 mg/dL) (less than 150 mg/dL) with the rest of the parameters within normal limits. The coagulogram was normal. The thyroid function tests were within normal limits. A urine toxicology screen was done

as the patient gave a history of intake of opioids which came out to be positive for opiates (>300 ng/mL). Serologies for hepatitis A, B, C, E, and human immunodeficiency virus came out to be negative. Chest radiography showed blunting of the right costo-phrenic angle (Figure 1A). Ultrasonography of the abdomen revealed normal bowel status, Grade I fatty liver with a heterogenous hypoechoic collection of $3.7 \text{ cm} \times 3.5 \text{ cm}$ in relation to the head of the Pancreas with no cholelithiasis or ascites along with minimal right pleural effusion. The patient underwent abdominal contrast-enhanced computerized tomography which revealed a bulky pancreas with a fluffy outline with a well-defined peripancreatic collection with an enhancing wall of the size $4.9 \text{ cm} \times 4.3 \text{ cm}$, along with peripancreatic and mesenteric fat stranding pancreatic duct was not dilated and no parenchymal or ductal calcification (CTSI 10/10) with mild bilateral pleural effusion (Figure 1B).

The patient was managed conservatively with non-opioid analgesics acetaminophen 15 mg/kg every 6 hour and intravenous fluids like ringer lactate for 5 days. Psychiatry and psychologist consultations were taken for deaddiction related to opioid intake.

3. Discussion

Acute pancreatitis is a potentially life-threatening condition that affects approximately 34 in 100 000 population annually[6]. The most common etiological factors are cholelithiasis and alcohol consumption. Drug-induced pancreatitis though rare, attributes to an incidence of 0.1%-2.0%[7]. Another study documents its incidence to be 5.0%-8.0%[2]. The diagnosis of drug-induced pancreatitis is a diagnosis of exclusion and must be considered only after ruling out the evidence of gallstones, alcoholism, hypertriglyceridemia, trauma, tumors, or anatomical variations like pancreatic divisum or

annular pancreas[7].

Badolev *et al.*[8] classified drugs into 4 classes as per their level of evidence available in causing pancreatitis. Class I includes medications with at least 1 case report about recurrence of acute pancreatitis with a rechallenge of the drug. Class II includes drugs with a consistent latency in 75% or more of the reported cases. Class III includes drugs that had 2 or more case reports published, but neither a rechallenge nor a consistent latency period. Class IV were similar to Class III drugs, but only one case report had been published. Opiates are incriminated under Class I. Hastier *et al.* published a case series of four patients who developed pancreatitis after ingestion of codeine, an opioid. Unintentional rechallenge in three of the four studied cases led to the recurrence of symptoms[9]. The pathophysiology behind opioid-induced pancreatitis has been attributed to the sphincter of Oddi dysfunction which is further believed to be secondary to increased phasic amplitude and frequency of sphincter contractions mediated by mu-opioid receptor[10]. It has further led to explain that opium addicts, like in the present case, may have dilated bile or pancreatic duct leading to the pathology[5].

In the present case, we believe that opium ingestion was the culprit behind acute pancreatitis in the patient. As the drug was withdrawn from consumption, the patient improved gradually with supportive management. In a disease where opiates are very commonly used for pain management, caution must be practiced during the same as the practice can be counterproductive in certain cases.

In conclusion, drug-induced pancreatitis though a diagnosis of exclusion, should actively be considered as a possible differential diagnosis as an early diagnosis in patients with pancreatitis can help reduce further complications and sequelae. Opioids which have such an intimate relation with the management of pancreatitis must also be used with a word of caution.

Conflict of interest statement

The authors report no conflict of interest.

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Authors' contributions

AK, HS: Case presentation, data collection, investigations and writing of original draft; JS: Literature review, writing of original draft including conclusion, references; SA, NA: Intellectual content, literature search, manuscript final editing and review.

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