ORIGINAL ARTICLE

Drug-Induced acute Pancreatitis: A Real-World Pharmacovigilance Study Using the Tunisian Databases

Pancréatite aiguë médicamenteuse : Une étude de pharmacovigilance à partir des bases de données tunisiennes

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Abstract

Background: Acute pancreatitis (AP) is acute inflammatory process of the pancreas, commonly attributed to biliary or alcoholic causes. Drug-induced AP, although rare, is increasingly recognized as a potential etiology. However, there is limited literature on this topic.
Aim: This study aims to review the epidemiological, clinical, radiological, and management characteristics of drug-induced AP based on data from Tunisian databases.
Methods: A retrospective study was conducted at the Department of Digestive and Visceral Surgery, spanning five years. Thirteen cases of drug-induced AP out of 1580 admitted patients with AP were analyzed. Data collection included clinical characteristics, diagnosis methods, therapeutic approaches, and outcomes.
Results: We report a series of 13 cases of drug-induced acute pancreatitis (AP). Due to the rarity of this event, our small sample size limits the statistical power of our findings. However, each case provides valuable insight into potential drug associations with AP.
The mean age of affected patients was 55.62 years, with a male predominance (77%). Common symptoms included sudden onset epigastric abdominal pain (100% of cases) and elevated lipase levels (92.3%). Implicated medications included captopril (38.5%), atorvastatin (23.1%), azathioprine (23.1%), metformin, and olanzapine (7.7% each). Management involved pain management and proton pump inhibitors, with favorable outcomes in 84% of cases.
Conclusion: Drug-induced pancreatitis is a recognized entity requiring multidisciplinary management. Early recognition and reporting of suspected cases are essential for improved pharmacovigilance.

Keywords: Acute pancreatitis, drug-induced, epidemiology, management, outcomes.

Résumé

Introduction: La pancréatite aiguë est le plus souvent d'origine biliaire ou alcoolique. La pancréatite aiguë d'origine médicamenteuse, bien que rare, est de plus en plus reconnue comme une étiologie possible. Cependant, les données sur ce sujet restent limitées dans la littérature.
Objectif : Cette étude vise à analyser les caractéristiques épidémiologiques, cliniques, radiologiques et thérapeutiques de la pancréatite aiguë d'origine médicamenteuse à partir des bases de données tunisiennes.
Méthodes : Une étude rétrospective a été menée au sein du service de chirurgie digestive et viscérale sur une période de cinq ans. Parmi 1580 patients admis pour pancréatite aiguë, 13 cas de pancréatite d'origine médicamenteuse ont été identifiés et analysés. La collecte des données a porté sur les caractéristiques cliniques, les méthodes diagnostiques, les approches thérapeutiques et les évolutions.
Résultats: L'âge moyen des patients atteints était de 55,62 ans, avec une prédominance masculine (77%). Les symptômes les plus fréquents étaient une douleur abdominale épigastrique brutale (100% des cas) et une élévation des taux de lipase (92,3%). Les médicaments incriminés comprenaient le captopril (38,5%), l'atorvastatine (23,1%), l'azathioprine (23,1%), la metformine et l'olanzapine (7,7% chacun). La prise en charge reposait principalement sur le traitement symptomatique, incluant les antalgiques et les inhibiteurs de la pompe à protons, avec une évolution favorable dans 84% des cas.
Conclusion: La pancréatite aiguë d'origine médicamenteuse est une entité bien décrite nécessitant une prise en charge multidisciplinaire. Une reconnaissance précoce et une déclaration systématique des cas suspectés sont essentielles pour améliorer la pharmacovigilance.

Mots-clés: Pancréatite aiguë, origine médicamenteuse, épidémiologie, prise en charge, évolution.

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Introduction

Acute pancreatitis (AP) is an acute inflammatory process of the pancreas linked to self-digestion of the gland by its own enzymes, with the two main etiologies being biliary and alcoholic[1].Two forms of AP are distinguished based on the type of lesions observed: Interstitial pancreatitis (or edematous), often benign, without visible lesions of acinar cells and pancreatic tubules, or capillary signs and Necrotizing acute pancreatitis (NAP) (or necrotico-hemorrhagic) is characterized by necrosis of glandular tissues. While the positive diagnosis of AP relies on the association of epigastric abdominal pain and elevated lipase levels, abdominal CT scan confirms the presence of pancreatic necrosis. The severity of AP is determined using clinical data and clinical-biological and radiographic scores [2]. Indeed, it is a common indication for hospitalization for gastrointestinal disorders. Each year, it accounts for up to 275,000 hospitalizations in the United States [3]. It is a potentially life-threatening disease [1]. Although its overall mortality is estimated at 5%, it can reach up to 30% in cases of severe AP and <1% in mild AP. [8]

In the vast majority of cases, AP is due to chronic alcoholism or biliary disease. It can also be caused by obstruction of the pancreatic duct or a disease of the descending duodenum [2]. In Tunisia, the lithiasic etiology is the most frequent.The involvement of a drug in the onset of acute pancreatitis (AP) is a rare eventuality (less than 2%) but not exceptional in adults. It is defined by the occurrence of an episode of pancreatitis shortly after the introduction of a drug or after an increase in its doses.[3] In recent years, drugs have been increasingly recognized as an etiological factor with more than 260 implicated medications. Indeed, drugs account for 0.1 to 2% of AP cases and are usually diagnosed by excluding other causes. [3] Currently, there are few studies on drug-induced pancreatitis. Drug-induced pancreatitis (DIP) remains a diagnostic challenge. The causality is often difficult to establish due to retrospective bias, lack of standardized evaluation, and the influence of confounding factors.

The aim of this work is to review the epidemiological, clinical, radiological, and management characteristics of drug-induced acute pancreatitis allowing early detection of local or distant recurrence

Methods

We conducted a retrospective study in the Department of Digestive and Visceral Surgery at Fattouma Bourguiba University Hospital in Monastir. This study spanned a period of 5 years

This study involved patients who presented with drug-induced acute pancreatitis. Data were collected from medical records and pharmacovigilance reports. We assessed causality using the Naranjo scale where sufficient information was available (see Supplementary Table 1).

This study has no financial support, and we do not have any conflict of interest to disclose.

Patients meeting the following criteria were included in this study:

-Hospitalized and admitted through the emergency department.

-Presenting with pancreatic-type abdominal pain confirmed by lipase levels > 3 times the normal range and/or with a CT scan showing signs of acute pancreatitis.

Data collection was done on a canvas that included the clinics characteristics, the circumstances of discovery, the means of diagnosis, the therapies methods, monitoring, diagnosis and management.

We calculated simple frequencies and relative frequencies (percentages) for qualitative variables. We calculated means and medians and determined the range (extreme values) for the quantitative variables.

Table 1. Naranjo Scores for Drug-Induced Pancreatitis Cases

Case Number	Implicated Drug	Naranjo Score	Probability Category
1	Azathioprine	7	Probable
2	Captopril	5	Probable
3	Atorvastatin	4	Possible
4	Olanzapine	3	Possible
5	Metformin	2	Possible
6	Hydrochlorothiazide	6	Probable
7	Furosemide	4	Possible
8	Valproate	8	Probable
9	Mesalazine	6	Probable
10	Isotretinoin	3	Possible
11	Enalapril	5	Probable
12	Omeprazole	2	Possible
13	Paracetamol	1	Doubtful

Results

Over a period spanning from January 2016 to January 2021, we managed 13 cases of drug-induced acute pancreatitis out of 1580 patients admitted for acute pancreatitis from the Department of Digestive and Visceral Surgery, representing 0.8 % of acute pancreatitis ethology in our department.

The age range varied from 16 to 89 years, with a mean age of 55.62 years. The most affected age group was between 66 and 75 years, accounting for 4 patients, or 30% of cases. (figure 1).

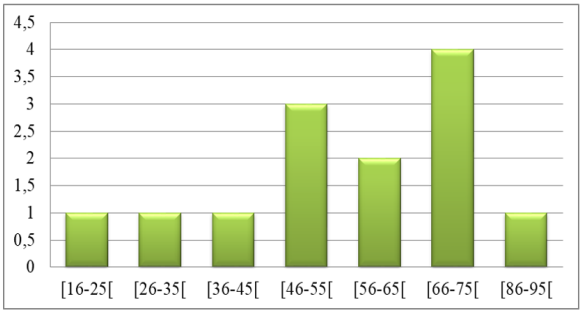

Figure 1. Distribution of patients by age.

We observed a male predominance with 10 men (77%) and 3 women (23%), resulting in a male-to-female ratio of 3.33. 12 of our patients had associated comorbidities (92.3%).(figure 2).

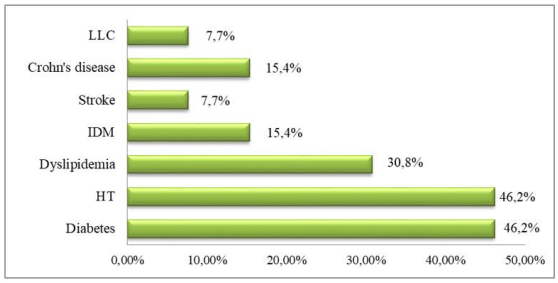

Figure 2. Distribution according to associated defects

While the positive diagnosis of AP relies on the association of epigastric abdominal pain and elevated lipase levels. In our series Pancreatic-type abdominal pain was present in all our patients, accounting for 100% of cases. The pain onset was sudden, and it was epigastric and transfixing in 10 cases (77%). (figure 3).

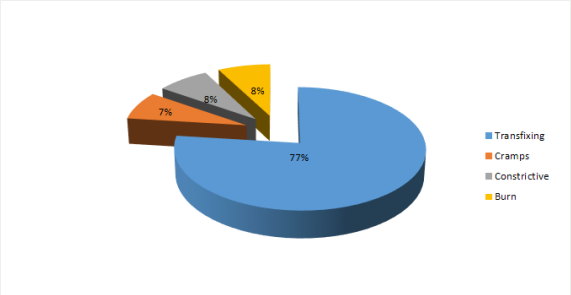

Figure 3. Distribution according to type of abdominal pain

Fever was noted in 3 patients (23.1%) ranging between 38.5 and 39 degrees Celsius. Epigastric tenderness was observed in all patients, accounting for 100% of cases. Lipase levels were elevated (>3 times the normal) in 12 patients (92.3%).

The SIRS score at admission was calculated for all patients. (figure 4).

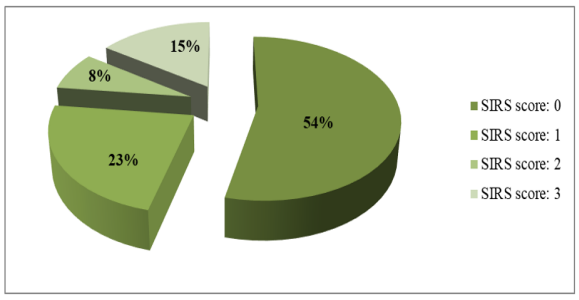

Figure 4. Distribution according to SIRS Score

The mean in hospital length of stay was 7 days.

Abdominal ultrasound was performed in 10 patients (76.9%), revealing a swollen pancreas in 1 patient (7.7%).12 patients underwent abdominal CT scans, with 46.2% classified as Balthazar grade C and 30.8% as grade E.

Implicated Medications:

Captopril (LOPRIL) was the most implicated medication in our series (38.5%). Atorvastatione in 23.1 % of cases , azathiopirine in 23.1 % of cases ,metformine and olanzapine in 7.7 % of cases. (Figure 5)

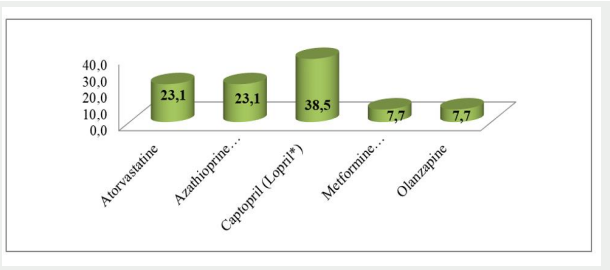

Figure 5. Distribution according to etiology

All patients received medical treatment as the sole part of their medical strategy.Pain Management using Injectable paracetamol (PERFALGAN) in 77% of cases. PERFALGAN + ACUPAN (Nefopam) combination in 15.3%. PERFALGAN + ACUPAN + Tramadol (TRAMADIS) combination in 7.7%. Injectable proton pump inhibitor (MOPRAL) was prescribed in 77% of cases.

Prophylactic anticoagulation was prescribed in 61.5% of cases. The final outcome was favorable in 84% of cases, with complications observed in 16% of cases, including recurrence and mortality, resulting in an overall mortality rate of 8%.

The implicated drugs included azathioprine, captopril, atorvastatin, olanzapine, and metformin, among others. These associations must be interpreted with caution as prescription bias may explain the frequency of some drugs rather than a strong causal link. Other risk factors for pancreatitis were systematically evaluated and excluded when possible; details are provided in Table 1.

Given the retrospective design, data collection may have been incomplete due to recall and reporting bias. Some mild or undocumented cases may have been missed.

There was no control group, which limits our ability to infer statistical significance regarding age or symptomatology.

Discussion

Acute drug-induced pancreatitis is a rare but potentially serious condition.[3] It accounts for approximately 2 to 5% of acute pancreatitis cases. Its incidence is currently increasing.[1] There is no current data on prevalence in Tunisia. The average age of onset of drug-induced acute pancreatitis (AP) in several studies varies between 53 and 59 years, with a mean of 54 years,[3] ranging from 25 to 64 years.[2] It is interesting to note that drug-induced AP accounts for 9 to 30% of cases of AP in children, alongside traumatic causes,[2] and cases have been observed at earlier ages than for AP of other common etiologies.[4] In our series, the mean age is 55.62 years, with the most affected age group being between 66 and 75 years. In the literature, 52% of patients are male and 48% are female, with a sex ratio of 1.1.[2] In our series, male predominance is noted with a percentage of 77%. In the literature, 38.7% of patients had a history of alcoholism, 19.3% were tobacco smokers, and 12.9% were cannabis users.[2] In our series, a history of alcohol consumption was noted in 15.4% of cases. Epidemiological studies

have suggested a predisposition of type 2 diabetes in the occurrence of AP, associated in 8.6%.[2] The exact nature of this role remains poorly understood. In our series, type 2 diabetes was found in 46.2% of cases. Inflammatory bowel disease (IBD) was reported in 11% of cases. Indeed, studies have suggested that IBD is associated with a risk of developing pancreatitis, especially for Crohn's disease, due to common pathogenic mechanisms, decreased enterohepatic circulation of bile acids in patients with ileal involvement or who have undergone surgical ileal resection, and mechanical factors in the duodenal locations of the disease.[5] Two patients in our series (15.4%) have Crohn's disease. Furthermore, drug-induced pancreatitis is more common in populations recognized to be at risk of AP, such as [2,5]: HIV infection in 6.6% of cases; Acute lymphoblastic leukemia especially in children; Hyperlipidemia/hypercholesterolemia/ hypertriglyceridemia in 4.6% of cases. Dyslipidemia was noted in 4 patients in our series (30.8%); History of cholecystectomy in 6.6% of cases, noted in 15.4% of cases in our study; - History of pancreatitis in 0.8% of cases, found in one patient in our series (7.7% of cases). No symptomatology is formally specific to drug-induced AP.[3] The association of an evocative clinical picture and an elevation of pancreatic enzymes, amylases and/or lipases, more than three times the upper limit of normal, can reasonably allow the diagnosis of acute pancreatitis. [6] The clinical manifestations of drug-induced pancreatitis are nonspecific. There is no symptomatology that allows differentiation of drug-induced pancreatitis from other origins.[6] Pain Abdominal pain represents the inaugural symptom of AP in nearly 100% of cases. It is considered "typical", and characteristic when it is epigastric, of major intensity, transfixing, or radiating to both hypochondria, progressively settling in a rapid and permanent manner to reach its maximum within a few hours and lasting beyond 24 hours. [5, 6] In our series, pain was present in almost all of our patients, and it was epigastric and transfixing in 77% of cases.

The search for the etiologies of acute pancreatitis (AP) can be challenging, yet it is crucial for both the treatment of the current episode and the prevention of its recurrence. As more medications are utilized in clinical practice, the incidence of drug-induced AP continues to rise. This condition is still underreported in the literature, making its diagnosis difficult. Often, it is a diagnosis of exclusion with highly variable onset times. Indeed, awareness of medications capable of causing AP can help clinicians identify this uncommon etiology, thus avoiding the re-administration of the offending drug. Available literature on drug-induced pancreatitis is primarily based on reported cases and small series. Mallory et al. in 1980 proposed four criteria in their review to attribute a drug as the cause of acute pancreatitis[7,11]:

- The pancreatitis develops during treatment with the drug.
- Absence of other causes of pancreatitis.
- Resolution of pancreatitis upon discontinuation of the drug.
- Recurrence of pancreatitis upon reintroduction of the drug.

The drug is then classified into three categories based on these criteria: definite, probable, or possible association with pancreatitis. A definite association implies that all four criteria are met. A probable association requires all criteria except re-exposure. Possible association occurs when there are incomplete or contradictory levels of evidence.

Therefore, drug-induced origin should always be suspected even if other predisposing factors for AP are present (such as alcoholism, gallstones, liver cytolytic activity, or chronic pancreatitis). Many reported cases are due to the use of a single drug, with few caused by a combination.

There is no specific clinical presentation to distinguish drug-induced pancreatitis from other origins. Currently, there are no known individual predisposing factors. The pathophysiological mechanism is not clearly understood and could involve immune-allergic reactions, cytotoxicity, accumulation of toxic metabolites or intermediates, and hypersensitivity reactions.Given the absence of a specific clinical presentation, excluding other causes of acute pancreatitis is a key element in the diagnostic process. [8] Considering the frequency of biliary and alcoholic acute pancreatitis, these two diagnoses should be ruled out before considering a drug-induced origin. Diagnostic criteria for drug-induced AP should include evidence of drug intake shortly before AP onset, a direct correlation between increased risk of AP and dose. The onset of AP is independent of the dose, and the shorter the interval between drug intake and AP onset, the more probable the causality. Confirmation of drug overdose biologically unquestionably supports the diagnosis of drug-induced AP when the toxicity of the product is identified as dose-dependent.Generally, the interval between onset and drug intake ranged from 0 to 9 days. The exact chronology of symptom evolution concerning drug administration, cessation, and possibly reintroduction is essential in the causality assessment. Quantifying the interval between AP onset and drug intake is crucial; the shorter this interval, particularly in cases of immune-allergic reactions, the more likely the drug's responsibility. Interpreting symptom evolution after drug cessation is necessary. If symptoms regress rapidly after discontinuing the implicated therapy and reappear upon reintroduction, the drug is more likely implicated. Clinicians must remain vigilant and improve screening to reduce these complications. This pathology underscores the need for advanced pharmacovigilance research.[9]

In the future, genetic tests could contribute to improving prescription decisions. Similar to what has been done regarding drug-induced hepatotoxicity, a computerized database of drug-induced pancreatic side effects (Pancréatox) was created in 1985. Initially based on the registry of the regional pharmacovigilance center of Saint-Antoine Hospital in Paris, this database collects bibliographic references from authoritative sources. The first update, collating 261 international nonproprietary names with 1,115 corresponding bibliographic references, was published in 2001. This regularly updated database consolidates bibliographic references from authoritative sources.

The first reported cases of drug-induced AP date back to the 1950s and mainly involved opioids or corticosteroids. Over the years, the number of implicated drugs has gradually increased. Antimitotics remain the most common drugs associated with this condition.[10]
The five drugs studied in our research are as follows:

*__Azathioprine__ (Imuran): Azathioprine, an immunosuppressant, is indicated for treating inflammatory bowel diseases and is also the first-line drug for other gastrointestinal diseases such as autoimmune hepatitis. Since 1980, azathioprine and its metabolite, 6-mercaptopurine, have been strongly implicated as agents responsible for AP in patients with Crohn's disease. The timeline between treatment initiation and the onset of characteristic clinical symptoms, typically 2 to 4 weeks later, supports this etiology.

*__Metformin__ (Biguanides): an oral hypoglycemic commonly used in type 2 diabetes, is considered a safe drug with minimal side effects. Additionally, some studies have shown a certain association between the onset of pancreatitis and biguanide agents. Among published case reports, postulated mechanisms include drug overdose, drug accumulation, and acute renal failure triggered by vomiting. Therefore, metformin has been classified as a possible contributor to AP. For our study, only one case was detected.

*__Captopril__ (Lopril): Angiotensin-converting enzyme inhibitors (ACEIs) are one of the most commonly prescribed classes of drugs for hypertension, heart failure, and proteinuria. The first reported case of ACEI-induced pancreatitis was observed with enalapril. Other cases of pancreatitis induced by lisinopril, captopril, ramipril, and perindopril have also been published. The risk increased with higher daily doses and was highest during the first 6 months of treatment. A possible mechanism of action for ACEI-induced acute pancreatitis is proposed to follow the mechanism of local angioedema of the pancreatic duct. However, ACEIs, particularly captopril, have shown an important role in attenuating vascular permeability in severe experimental acute pancreatitis in rats by reducing matrix metalloproteinase expression. Captopril was the most implicated drug in our series, with a frequency of 38.5%.

*__Atorvastatin__ (Statins): Statins are generally well tolerated. However, acute pancreatitis has been documented to be induced by statins, primarily atorvastatin, fluvastatin, rosuvastatin, simvastatin, and pravastatin. A study by Singh et al. suggests a class effect for statin-based medications. The onset of statin-induced AP can occur at any time but seems to be very rare at the beginning of treatment. It has been observed from a few hours to several years after treatment initiation. Due to the variance in latency period, the mechanism may be linked to a direct toxic effect on the pancreas and accumulation of a toxic metabolite. Other proposed mechanisms of action for statin-induced AP are associated with rhabdomyolysis, myalgia, and/or drug metabolism or interactions via cytochrome P-450 3A4 (CYP3A4). However, the mechanism of action remains poorly defined. Recent studies have rather demonstrated a slight protective effect in statin users in animal models of severe acute pancreatitis, where statins seem to reduce inflammatory cytokines and pulmonary neutrophil activation in severe acute pancreatitis models. In our series, we noted 3 cases of AP induced by atorvastatin.

*__Olanzapine__ (Antipsychotics): an antagonist of dopaminergic and serotonergic receptors, is an atypical antipsychotic belonging to the thienobenzodiazepine class. It is used to treat schizophrenia. The mechanism of olanzapine-induced pancreatitis remains uncertain. It is unclear if olanzapine's effect on the pancreas is directly related to drug-induced lesions or if it is a consequence of global metabolic changes, particularly elevated triglycerides. Since 2000, several cases of pancreatitis associated with olanzapine have been reported. A study by Emmanuel et al. reported a case of necrotizing pancreatitis, probably due to a significant elevation in serum triglycerides. In the study by Thomas et al., three cases of AP induced by olanzapine were described. The episodes decreased or resolved after discontinuation of the drug. In our series, we noted only one case of AP induced by olanzapine.

In the vast majority of cases, drug-induced acute pancreatitis is predominantly edematous in type, with a rapidly favorable outcome after discontinuation of the implicated treatment. In 10 to 15% of cases, progression to necrotizing pancreatitis is described, with morbidity and mortality most often dictated by the patient's context (e.g., age, immunosuppression, and advanced cancer). Indeed, the severity of acute pancreatitis appears not to depend on the drug class responsible.
The prognosis for drug-induced pancreatitis is generally excellent. In the review by Lankisch et al., 19 out of the 22 patients described presented with edematous pancreatitis. There were no cases of extensive necrosis observed on imaging, and there were no fatalities. The few reported deaths occurred in patients with multiple comorbidities in a fragile clinical setting.[10] While our findings suggest possible associations, drugs such as olanzapine and metformin have a weaker causality profile based on literature. The addition of the Naranjo scale strengthens the causality assessment in this study. However, we acknowledge the need for prospective studies or case-control designs to better establish risk factors and causative links.

Conclusion

Drug-induced pancreatitis has undergone significant evolution, leading to improvements in its prognosis. The diagnosis of pancreatitis relies on the presence of suggestive abdominal pain and elevated lipase levels. This diagnostic process typically includes abdominal ultrasound, which remains highly valuable for exploring the biliary tract.

The severity of acute pancreatitis is assessed based on the presence of organ failure and the risk of severe progression using the SIRS score. The Ranson score should be abandoned in this context.

Drug-induced pancreatitis is now a well-recognized entity, accounting for 2% of acute pancreatitis cases. It is crucial to consider this condition when common causes of acute pancreatitis such as alcohol or biliary migration have been ruled out. It is a condition with a formidable prognosis requiring multidisciplinary management. The timeline of symptoms, the initiation of an implicated treatment, as well as clinical evolution and the kinetics of biological tests, are key to diagnosis. A delay of several months does not, however, exclude a drug etiology. Detailed lists of commonly implicated medications are available in the literature; however, it is the responsibility of every healthcare professional to report any suspicion of drug-induced pancreatitis to a pharmacovigilance center. Our findings, despite limitations such as small sample size, retrospective bias, and lack of controls, highlight the need for more robust, prospective studies and pharmacovigilance awareness.

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