TPM Vol. 32, No. S3, 2025
ISSN: 1972-6325
https://www.tpmap.org/

DRUG-INDUCED PANCREATITIS: A CASE SERIES HIGHLIGHTING RARE ETIOLOGIES

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Abstract

Background:

Drug-induced pancreatitis (DIP) is an uncommon cause of acute pancreatitis (AP), accounting for 0.1–2% of cases. Early recognition of offending medications is vital to reduce morbidity. Herein, we present two illustrative cases involving diuretic- and oral contraceptive-related DIP—both rare and under-reported triggers.

Case Presentation:

• Case 1: A 68-year-old female with atrial fibrillation and mitral valve replacement experienced acute-onset abdominal pain and vomiting. Laboratory values revealed markedly elevated amylase (6,942 U/L) and lipase (94,245 U/L). CT imaging confirmed interstitial pancreatitis with bilateral pleural effusions. Exclusion of gallstones, alcohol use, hypertriglyceridemia, and hypercalcemia bolstered the suspicion on recently initiated loop diuretic (furosemide). Discontinuation, along with supportive care from a multidisciplinary team, led to full recovery.
• Case 2: A 42-year-old woman on prolonged oral contraceptive pill (OCP) therapy for abnormal uterine bleeding presented with epigastric pain. Elevated pancreatic enzymes and CT imaging confirmed mild AP. Other common causes were excluded. OCPs were ceased, and conservative management resulted in clinical and laboratory resolution.

Conclusion:

These cases underscore that commonly prescribed medications like furosemide and OCPs—though rarely implicated—can cause DIP. Clinicians must maintain a high index of suspicion for DIP, obtain detailed medication histories, and promptly withdraw the culprit drug. Multidisciplinary coordination enhances patient recovery and prevents recurrence.

Keywords :

Drug-induced pancreatitis; Furosemide; Oral contraceptives; Acute pancreatitis; Etiology

INTRODUCTION

Acute pancreatitis (AP) is a frequent gastrointestinal emergency, most commonly caused by alcohol consumption or gallstones. Drug-induced pancreatitis (DIP), accounting for approximately 0.1–2% of AP cases, remains underrecognized while the list of implicated agents continues to expand ([1], [4]).

Diuretics—including loop (e.g., furosemide) and thiazide agents—have been reported in association with AP. Proposed mechanisms include direct cellular toxicity, ischemia secondary to hypovolemia, hypersensitivity reactions, and even

hypercalcemia or hyperlipidemia with certain thiazides ([4]). A recent case series has documented diuretic-related pancreatitis with furosemide and thiazide use, with causality assessed as “probable” using Naranjo scale.

Estrogen-containing medications, such as oral contraceptives (OCPs), may precipitate AP by causing hypertriglyceridemia, inducing a hypercoagulable state, or exposing patients with familial lipid disorders to higher risk ([4], [5], [7], [2], [3]). Though rare, case reports of OCP-induced AP, including among patients with underlying lipid metabolism disorders, substantiate this association ([2], [3], [6], [7]).

This case series adds valuable clinical insight into these rare but clinically significant DIP triggers, emphasizing the need for vigilance, prompt identification, and management.

Case Presentations

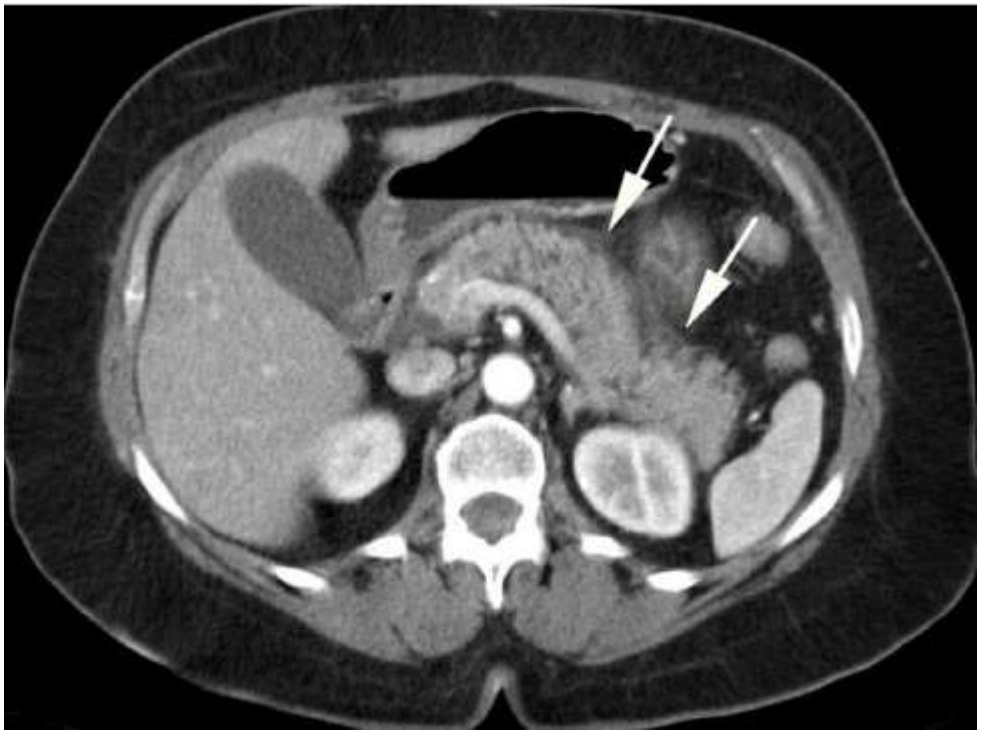
Case 1: Furosemide-Induced Pancreatitis

Patient Profile:

A 68-year-old female with a history of atrial fibrillation, rheumatic heart disease, and mitral valve replacement presented with acute-onset severe epigastric pain, vomiting, and dyspnea.

Clinical Findings & Investigations:

Epigastric tenderness without guarding was noted. Serum amylase: 6,942 U/L; lipase: 94,245 U/L. CT abdomen revealed interstitial pancreatitis with bilateral pleural effusions. (Figure-1 and 2) Gallstones were absent on ultrasound;



lipids, calcium, and liver enzymes normal. No alcohol exposure.

Figure-1

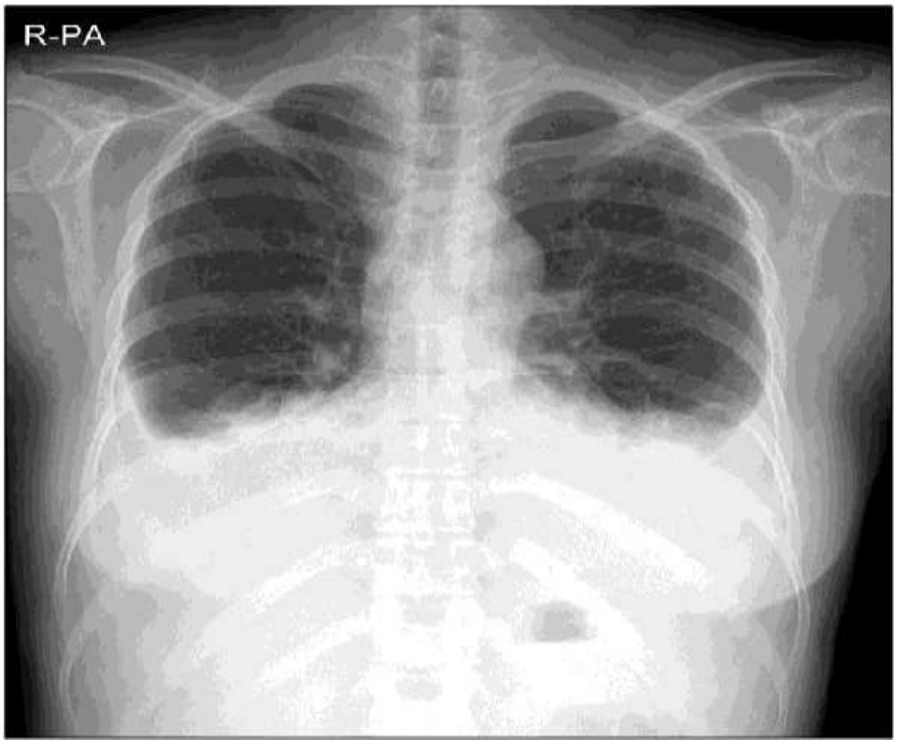


Figure-2

Three weeks prior, she began taking furosemide. Recognizing the timing and exclusion of other etiologies, a diagnosis of DIP due to furosemide was made.

Management & Outcome:

Furosemide was discontinued. Supportive care—IV fluids, analgesia, electrolyte monitoring—was provided with the assistance of cardiology and gastroenterology teams. Clinical symptoms and enzyme levels normalized over a week. She was discharged in stable condition.

Case 2: Oral Contraceptive-Induced Pancreatitis

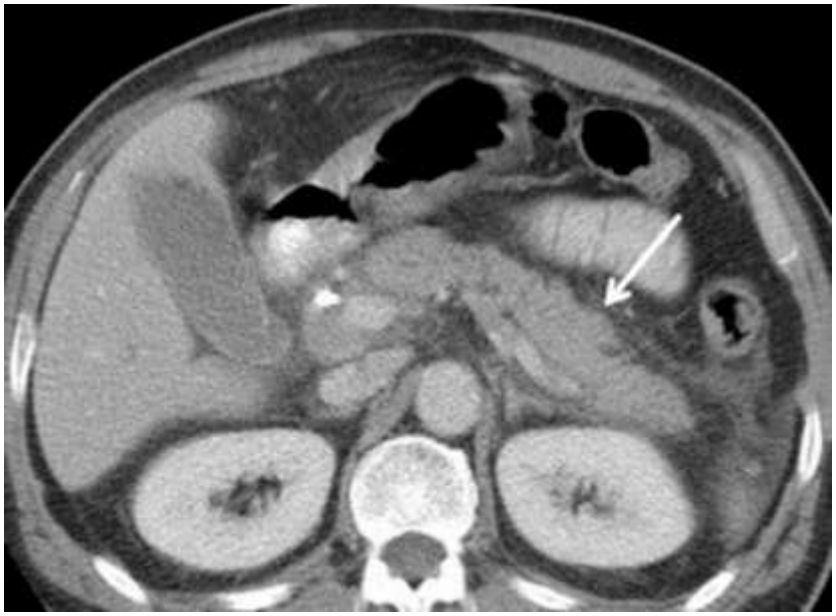
Patient Profile:

A 42-year-old woman with bronchial asthma and abnormal uterine bleeding treated with combined OCPs for four months presented with dull epigastric pain for several days.

Clinical Findings & Investigations:

Epigastric tenderness without peritoneal signs. Serum amylase: 842 U/L; lipase: 1,210 U/L. CT abdomen showed mild pancreatic swelling, peripancreatic fat stranding. (Figure-3) Workup excluded gallstones, alcohol, hypertriglyceridemia (>500 mg/dL), hypercalcemia, hyperlipidemia, or autoimmune causes.

Figure-3



Given the OCP use and absence of alternative causes, DIP from estrogen exposure was suspected.

Management & Outcome:

OCP therapy was withdrawn. The clinical course was benign with supportive treatment. Symptoms and enzyme levels resolved by hospital day 3, and she remained well at follow-up.

DISCUSSION

DIP remains a rare but clinically important cause of AP, demanding careful medication review and exclusion of more common etiologies ([1]). The mechanisms implicated include:

- Furosemide: May provoke AP via direct pancreatic toxicity, ischemia from hypovolemia, or hypersensitivity; loop and thiazide diuretics have been reported in multiple cases, categorized as “probable” causes based on Naranjo assessment ([4]). A case of rehospitalization after bumetanide challenge further supports this ([1]).
- Oral Contraceptives (Estrogens): Can precipitate AP by inducing hypertriglyceridemia or a hypercoagulable state; case reports describe severe triglyceride elevation and recurrent AP episodes during OCP therapy, particularly in patients with underlying lipid sensitivity ([2], [3], [5], [6], [7]).

In both presented cases, the temporal relationship, lack of alternative causes, and resolution upon withdrawal of the suspected drug strongly support causality.

Management principles include:

1. Early identification and cessation of the offending agent.
2. Supportive care according to standard AP protocols.
3. Multidisciplinary coordination—especially important when comorbid conditions are present, as in Case 1.
4. Patient education regarding avoidance of the causative drug to prevent recurrence.

CONCLUSION

This case series highlights two rare but clinically important causes of DIP: furosemide and OCPs. When evaluating AP of unclear etiology, a meticulous medication history is essential. Recognizing DIP allows for prompt drug withdrawal and favorable outcomes, emphasizing the need for clinician awareness and timely, multidisciplinary intervention.

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