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Drug-Induced Acute Pancreatitis Following Etoricoxib Use: A Case Report

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Abstract

Drug-induced acute pancreatitis (DIAP) is a rare but important condition linked to various medications, including NSAIDs. This report highlights a rare case of Etoricoxib-induced acute pancreatitis in a 59-year-old male who presented with severe abdominal pain and elevated serum amylase and lipase levels. Diagnostic imaging confirmed acute pancreatitis, with common causes such as gallstones and alcohol use excluded. The patient had recently taken Etoricoxib for back pain, and his symptoms resolved following drug discontinuation and supportive care.

This case underscores the importance of considering DIAP in unexplained pancreatitis and highlights Etoricoxib as a potential causative agent. Clinicians should maintain a high index of suspicion and carefully review medication histories in similar cases. Enhanced awareness and documentation of DIAP are critical for improving the understanding of drug safety profiles and guiding clinical practice.

Keywords: Etoricoxib, Drug-induced acute pancreatitis (DIAP), Selective COX-2 inhibitors, Hypersensitivity reaction, Pancreatic inflammation.

Introduction

Drug-induced acute pancreatitis (DIAP) is a rare but significant cause of pancreatic inflammation triggered by various medications, accounting for approximately 0.1%–2% of acute pancreatitis cases [1,2]. The condition has gained recognition over the years with advancements in diagnostic methods and reporting systems [3]. DIAP occurs through mechanisms such as direct cytotoxicity, hypersensitivity reactions, and disruptions in metabolic pathways, with individual susceptibility playing a crucial role [2,4]. It is associated with diverse drug classes, including diuretics, antibiotics, immunosuppressants, antiepileptics, and NSAIDs [2,3]. The global prevalence of DIAP varies, influenced by regional patterns of

medication use [4]. Among NSAIDs, Etoricoxib—a selective COX-2 inhibitor used for managing pain and inflammation—has been rarely linked to pancreatitis, with few documented cases. The exact mechanism remains unclear, though hypotheses include direct toxicity and hypersensitivity reactions, highlighting the need for heightened clinical awareness [1,4].

This report presents a rare case of Etoricoxib-induced acute pancreatitis, highlighting the importance of identifying drug-related causes when common etiologies like gallstones or alcohol are ruled out. It contributes to the limited data on such cases and underscores the need for vigilance in evaluating drug histories in unexplained pancreatitis.

Case report

A 59-year-old male presented with complaints of upper abdominal pain for the past week. The pain was sudden in onset, severe, located in the upper abdomen, non-radiating, and aggravated by food intake, with no relief from medications. The patient also experienced multiple episodes of non-projectile vomiting, containing food particles.

The patient reported a recent intake of Etoricoxib 90 mg for back pain, with the last dose taken three days prior to the onset of abdominal pain. There was no history of alcohol intake or other medications. Upon admission, he had tachycardia but was otherwise hemodynamically stable. Per abdominal examination revealed mild distention and tenderness over the epigastric and supraumbilical regions.

Laboratory investigations revealed Serum Amylase 1184 U/L (reference range: 25-115 U/L),Serum Lipase: 1220 U/L (reference range: 10-140 U/L),Hemoglobin: 12.2 gm/Dl, Total White Blood Cell Count: 17,400 cells/cu mm (Neutrophils: 76.6%, Lymphocytes: 16.9%, Monocytes: 6.5%) and Platelet Count: 179,000 cells/cu mm.

Ultrasound performed of the abdomen revealed mild fatty liver, a small umbilical hernia, a bulky pancreas with minimal peripancreatic fat stranding, and minimal ascites, suggesting acute pancreatitis. A contrast-enhanced CT scan showed a normal-sized pancreas with diffuse peripancreatic fat stranding, predominantly along the distal body and tail of the pancreas, and reactive thickening of the left Gerota's fascia. The pancreatic duct appeared normal, confirming the diagnosis of acute pancreatitis.

The diagnosis of acute pancreatitis was made based on the clinical presentation, laboratory findings, and imaging studies. The absence of other common causes such as gallstones or alcohol intake raised suspicion for a drug-induced etiology, specifically related to Etoricoxib.

Management included intravenous fluids, Nil per oral status, and paracetamol as an alternative analgesic. Good hydration was ensured, and the patient showed significant improvement after discontinuation of Etoricoxib. Oral intake was gradually resumed, and the patient tolerated it well. Follow-up showed normalization of serum amylase (110 U/L) and lipase (44 U/L).

The patient's condition improved significantly, with full recovery, as demonstrated by the normalization of clinical and laboratory parameters during follow-up. The discontinuation of Etoricoxib and supportive management led to the resolution of the acute pancreatitis.

Discussion

Drug-induced acute pancreatitis (DIAP) remains a diagnostic challenge due to its rarity and the wide range

of medications implicated in its development. This case highlights a rare instance of Etoricoxib-induced acute pancreatitis, contributing to the limited data on this adverse reaction. While NSAIDs are known to cause DIAP, most documented cases have involved non-selective COX inhibitors, such as diclofenac and ibuprofen, with selective COX-2 inhibitors like Etoricoxib being far less frequently reported.

Etoricoxib is a selective COX-2 inhibitor widely used for the management of osteoarthritis, rheumatoid arthritis, ankylosing spondylitis, and acute gouty arthritis due to its efficacy in reducing pain and inflammation with lower gastrointestinal side effects compared to non-selective NSAIDs [4]. Studies have emphasized its favourable gastrointestinal safety profile while acknowledging its potential for adverse events, including hypersensitivity and hepatic toxicity [4,6]. Such hypersensitivity reactions may extend to pancreatic inflammation, as seen in this case.

The exact mechanisms by which Etoricoxib triggers acute pancreatitis remain unclear. Proposed hypotheses include direct cytotoxicity to the pancreatic acinar cells, hypersensitivity reactions, and disruptions in cellular or metabolic pathways [1,2]. The absence of common etiologies such as gallstones, alcohol use, hypertriglyceridemia, or trauma strongly implicates Etoricoxib as the likely offending agent in this case. The temporal relationship between drug intake and symptom onset, coupled with symptom resolution after withdrawal, supports causality.

The findings of this case have significant clinical implications. Acute pancreatitis of unknown etiology requires careful evaluation of drug history, especially when common causes have been excluded. Early recognition and withdrawal of the offending medication are essential for favourable patient outcomes. This emphasizes the importance of avoiding rechallenge with the suspected drug to prevent recurrence.

Conclusion

This case demonstrates a rare occurrence of Etoricoxib-induced acute pancreatitis, emphasizing the importance of evaluating drug-related causes when common etiologies are excluded. Early recognition, drug withdrawal, and supportive management ensured a favourable outcome.

Further awareness and documentation of such cases will enhance the understanding of DIAP and refine the safety profiles of medications like Etoricoxib.

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