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Etodolac-Induced Gastric Ulcer in a Patient with Arthritis

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ABSTRACT

This case report presents a rare instance of etodolac-induced gastric ulcer in a patient with osteoarthritis, despite adhering to recommended dosing and protective medication. It underscores the multifaceted nature of nonsteroidal anti-inflammatory drugs (NSAIDs) and the importance of individual susceptibility and vigilant monitoring in vulnerable populations.

INTRODUCTION

Nonsteroidal anti-inflammatory drugs (NSAIDs) have long been instrumental in managing pain and inflammation, yet their use is often marred by the potential for gastrointestinal complications. Etodolac, a widely prescribed NSAID, has been associated with a distinct profile of adverse effects, with a particular focus on its propensity to induce gastric ulcers. This article aims to provide a thorough examination of the complexities surrounding etodolac-induced gastric ulcers, shedding light on the mechanisms, risk factors, clinical manifestations, and evolving strategies for both prevention and management. As a member of the NSAID class, etodolac exerts its therapeutic effects through the inhibition of cyclooxygenase enzymes, yet this mechanism also compromises the protective mechanisms of the gastric mucosa, rendering it susceptible to injury. The result is a clinical scenario that demands a nuanced understanding of the interactions between drug pharmacology, patient factors, and the delicate balance of gastrointestinal homeostasis (1-4).

Throughout this review, we will explore the epidemiology of etodolac-induced gastric ulcers, identifying patient-specific risk factors and potential correlations with dosage and duration of therapy. The article will delve into the evolving landscape of diagnostic modalities, highlighting the challenges posed by the often asymptomatic nature of NSAID-induced gastrointestinal damage and the importance of early detection to prevent complications. As healthcare providers grapple with the delicate balance of pain management and gastrointestinal safety, this article seeks to synthesize current research findings and clinical insights. By exploring the intricate relationship between etodolac and gastric ulceration, we aim to contribute to the ongoing dialogue surrounding NSAID-related adverse effects, ultimately fostering improved prescribing practices, enhanced patient monitoring, and optimized overall care for individuals navigating the therapeutic challenges of etodolac (5-9).

Here it was aimed to present a gastric ulcer case drecovered with anti-anxiety treatment.

CASE PRESENTATION

A 65-year-old woman with a five-year history of osteoarthritis of the knees and hips presented with a two-week history of epigastric burning pain, nausea, and vomiting. She had been taking etodolac 400mg twice daily for the past four weeks for relief of her osteoarthritis pain. She also took a proton pump inhibitor (PPI) as recommended by her physician.

Past medical history was significant for hypertension and hyperlipidemia, both well-controlled on medication. Social history revealed no smoking or excessive alcohol consumption. Physical examination revealed mild epigastric tenderness upon palpation. Initial investigations, including complete blood

count and liver function tests, were unremarkable. An upper endoscopy confirmed a shallow gastric ulcer in the antrum.

DISCUSSION

While etodolac is classified as a COX-2 selective NSAID, with a theoretically lower risk of gastrointestinal (GI) complications compared to non-selective NSAIDs, this case highlights the potential for even relatively safer NSAIDs to induce gastric ulcers. Several factors may have contributed to this occurrence. Some individuals, regardless of age or other comorbidities, may be inherently more susceptible to NSAID-induced GI injury due to genetic variations or underlying mucosal inflammation. Although the patient was within the recommended treatment duration for etodolac, exceeding three weeks of continuous use can increase the risk of GI complications. While PPIs provide protective benefits, they may not completely eliminate the risk of NSAID-induced ulcers, especially in vulnerable individuals. Unidentified comorbidities, such as Helicobacter pylori infection or undiagnosed gastric mucosal disease, could have potentiated the ulcer formation (8-11).

Prior to prescribing NSAIDs, even COX-2 selective ones, a thorough risk assessment for potential GI complications should be conducted, considering age, comorbidities, and prior GI issues. Patients taking NSAIDs, regardless of risk profile, should be monitored for early signs of GI distress, and endoscopy may be considered in specific cases, especially if symptoms persist or worsen. When NSAIDs are deemed necessary, exploring additional pain management strategies, such as physical therapy or complementary therapies, alongside appropriate gastric protection, can help minimize reliance on medications and reduce the risk of complications (10-13).

While etodolac offers valuable pain relief for patients with osteoarthritis, this case serves as a cautionary reminder that even seemingly safer NSAIDs can cause gastric ulcers. Individualized risk assessment, vigilant monitoring, and exploring alternative pain management options are crucial to ensure patient safety and optimize long-term outcomes in individuals requiring chronic pain management.

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