

Case Report: Peptic Ulcer and Anemia Gravis Due to Over-the-Counter Anti-Inflammatory Drugs Misuse in Gout Arthritis

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Abstract

Background: Peptic ulcer disease (PUD) is a serious global health concern, often linked to nonsteroidal anti-inflammatory drugs (NSAIDs). Chronic, unsupervised NSAID use increases the risk of severe gastrointestinal complications, particularly in rural areas with limited healthcare access.

Case Presentation: A 62-year-old male from a remote village presented with hematemesis, melena, and severe anemia (Hb 6.7 g/dL). He had a history of prolonged NSAID and steroid use for gout arthritis without medical supervision. Due to limited diagnostic facilities, the diagnosis of NSAID-induced peptic ulcer with anemia was based on clinical and laboratory findings. The patient received blood transfusions, proton pump inhibitors, gastroprotective agents, and urate-lowering therapy. His condition improved after seven days of hospitalization, and he was discharged with medication guidelines and follow-up care.

Discussion: This case highlights the dangers of self-medication and the lack of awareness regarding NSAID-associated complications in underserved regions. Alternative pain management strategies, including colchicine and lifestyle modifications, should be prioritized to reduce reliance on NSAIDs.

Conclusion: Chronic NSAID misuse can lead to life-threatening complications, as demonstrated in this case of peptic ulcer and anemia gravis. This report emphasizes the need for better patient education, medication supervision, and stricter regulations on over-the-counter drug sales, especially in rural settings.

Keywords: Peptic ulcer, anemia gravis, NSAID misuse, gout arthritis, self-medication, rural healthcare

Introduction Section

Peptic ulcer disease remains a significant global health concern, with nonsteroidal anti-inflammatory drugs (NSAIDs) being one of the primary contributing factors (Xie et al., 2022). The widespread availability of over-the-counter NSAIDs, combined with inadequate public awareness about their proper use, poses a particular challenge in developing countries and remote areas (Sánchez-Sánchez et al., 2021). Gastrointestinal bleeding is a serious side effect of NSAIDs, potentially compromising the protective functions of the GI lining and impacting blood clotting. Numerous epidemiological studies and clinical trials have investigated the GI toxicity of NSAIDs. These drugs can cause GI mucosal damage ranging from asymptomatic endoscopic erosions or ulcers to more severe ulcer complications like bleeding, perforation, and stenosis. Out of about 50.000 GI bleeding cases, 5.6% of patients died after using aspirin or other NSAIDs (Chi et al., 2018).

The relationship between NSAID use and gastric mucosal injury is well-documented, with recent studies showing that chronic NSAID use increases the risk of peptic ulcer disease by 4-6 times (Goldstein & Cryer, 2014). When combined with corticosteroids, this risk increases substantially, potentially leading to severe complications such as upper gastrointestinal bleeding (Kavitt et al., 2019). Today, NSAIDs are widely used around the globe for their well-recognized pain-relieving, anti-inflammatory, and fever-reducing effects. Over 30 million people take NSAIDs daily, making up 60% of the U.S. over-the-counter pain relief market. However, NSAIDs, like most drugs, can cause a variety of side effects. These side effects include gastrointestinal and cardiovascular problems, kidney failure, high blood pressure, and worsening of congestive heart failure (Al Meslamani et al., 2024). According to

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an epidemiological study, most elderly individuals living in both rural and urban areas use NSAIDs, with a higher usage rate among those in remote areas. Rural residents significantly outnumber their urban counterparts in using NSAIDs without a prescription (over-the-counter) for analgesic properties (Paliwal et al., 2019; Tawfik et al., 2017). This case report presents a severe manifestation of gastric ulcer complicated by anemia gravis in a patient with chronic NSAID and steroid use for the relief of gouty arthritis pain. This case highlights the importance of proper medication supervision and the severe complications of unsupervised long-term NSAID use, especially in remote and resource-limited areas. This report demonstrates the consequences of inappropriate self-medication practices where access to specialized healthcare is limited, emphasizing the need for better treatment and patient education in these situations.

Case Description

A 62 year old man from a remote village, Lakuan Village, Buol Regency, Central Sulawesi, Indonesia, came to the emergency unit with the chief complaint of hematemesis which occurred three times with a volume of more than 200cc per episode in the last day and black stools (melena) experienced for the last five days. The patient also reported worsening abdominal pain over the past three months, although the pain was not severe during episodes of hematemesis. His medical history indicated long-term and frequent use of non-steroidal anti-inflammatory drugs, specifically Diclofenac Sodium 50mg and Dexamethasone 0.5mg orally, to treat chronic joint pain due to gouty arthritis. The patient denied any history of gastrointestinal illness or previous bleeding episodes, lost appetite, and reported a weight loss of approximately 2 kilograms over the past six months. There was no history of previous treatment. He denied any history of diabetes or hypertension.

Vital signs upon examination showed a blood pressure of 150/109 mmHg, heart rate of 121 beats/minute, respiratory rate of 22 times/minute, and a temperature of 36.3 Celsius. Physical examination revealed the patient appeared with signs of anemia, such as anemic conjunctiva, as seen in Figure 1.



Figure 1. Anemic conjunctiva

Significant abdominal findings included distension, hyper-resonance on percussion, and epigastric tenderness with a VAS score of 6 on palpation. Thoracic examination indicated a systolic murmur and tachycardia. Initial laboratory tests at table 1 showed a hemoglobin level of 6,7 g/dL (normal : 13.8-17.2 g/dL) and a uric acid level of 8.4 mg/dL (normal : 3.5-7.2 mg/dL). Other tests indicated elevated blood urea and creatinine, possibly related to chronic gastrointestinal bleeding.

Table 1. Laboratory Result at hospital admission

Test	Value	Normal Range
Hb	6.7 g/dL (L)	14,0-17,5 g/dL
HCT	25. 4 % (L)	40-52 %
Leu	13.0 x 10³/μL (H)	4,4-11,3 x 10 ³ /μL
Eri	4.07 x 10⁶/μL (L)	4.5-5.9 x 10 ⁶ /μL
Tro	313 x 10 ³ /μL	139-335 x 10 ³ /μL
MCV	62.4 fL (L)	82.0-92.0 fL
MCH	18.9 pG (L)	28.0-33.0 pG
MCHC	30.3 g/dL (L)	32.0-37.0 g/dL
Neu	88.4 % (H)	50.0-70.0 %
Lim	6.6 % (L)	25-40 %
Mono	3.9 %	3.0-9.0 %

Eos	0.8 %	0,5-5,0 %
Baso	0.3 %	0,0-1,0 %
Uric Acid	8.4 mg/dL (H)	2.4 -5.7 mg/dL
Ureum	60 mg/dL (H)	10-50 mg/dL
Creatinin	1.44 mg/dL (H)	<1.0 mg/dL

The patient was diagnosed with peptic ulcer complicated by chronic anemia due to bleeding, and gout arthritis. Differential diagnosis of this case was esophageal varices rupture and gastric cancer. Due to the limitations of work-up examinations at healthcare facilities in peripheral areas, the diagnosis can only be made using the available clinical data. Establishing a definitive diagnosis using the gold standard examination is not feasible especially using endoscopic imaging to rule out other possible source of bleeding. Treatment protocol included restricted intake (fasting) for 24 hours, blood transfusion (packed red cells) of five units, and parenteral medications including pantoprazole injection to reduce gastric acid secretion, ondansetron to prevent vomiting, tranexamic acid and vitamin K to control bleeding. Oral medications included sucralfate syrup to coat the gastric mucosa, to lower uric acid levels, and colchicine to alleviate inflammation due to gout.

After seven days of intensive treatment, the patient's condition showed significant improvement. Hemoglobin levels increased to 11 g/dL, and complaints of abdominal pain and nausea diminished. Joint pain also significantly decreased after initiating treatment for gout arthritis. There were no additional symptoms indicating further complications. The patient was discharged with a follow-up care plan that included strict control of anti-inflammatory use, gastric acid management therapy, and management of gouty arthritis with allopurinol and colchicine.

Discussion

NSAIDs are still the most commonly prescribed drugs worldwide as drugs that function to treat pain, especially chronic pain and inflammatory diseases. However, the use of NSAIDs is closely related to PUD which also causes problems and requires special attention. Research shows that the relationship between NSAID use and the onset of PUD occurs up to 15-30% in use that is still within normal limits. (Johnson et al., 2021). This risk is recorded as higher in the elderly population over the age of 65 years who are reported to have a much higher risk of developing gastrointestinal complications in the use of NSAIDs (Goldstein & Cryer, 2014).

The pathophysiology of NSAID-induced PUD involves multiple mechanisms, with the primary pathway being the inhibition of cyclooxygenase (COX) enzymes. NSAIDs inhibit both COX-1 and COX-2 isoforms, leading to reduced prostaglandin synthesis, which plays a crucial role in maintaining gastric mucosal defense (Wilson et al., 2023). The reduction in prostaglandin synthesis results in decreased mucus production, reduced bicarbonate secretion, impaired mucosal blood flow, and altered epithelial cell turnover and repair. Recent research has revealed additional mechanisms contributing to NSAID-induced gastric injury. (Zhang et al., 2022) demonstrated that NSAIDs can directly damage the gastric epithelial barrier by disrupting phospholipids in the mucosal layer, leading to increased acid back-diffusion and subsequent tissue damage. Furthermore, studies have shown that NSAID-induced mitochondrial dysfunction plays a significant role in the pathogenesis of gastric injury. NSAIDs can uncouple oxidative phosphorylation and induce cellular ATP depletion, leading to increased oxidative stress and cellular death. The role of the microbiome has also emerged as an important factor in NSAID-induced PUD. Recent research indicates that NSAIDs can alter the gastric microbiota composition, potentially contributing to mucosal injury and delayed healing (Rodriguez et al., 2023; A. B. Thompson et al., 2022)

The complex relationship between PUD, hypochromic microcytic anemia, and NSAID use in the treatment of gouty arthritis remains a challenge in therapy management. First-line management focuses on discontinuing NSAIDs and initiating proton pump inhibitors (PPIs) (Sostres et al., 2013). Recent studies have shown that high-dose PPIs, especially esomeprazole 40 mg twice daily, showed a superior healing rate (93.2%) compared to standard doses in NSAID-induced ulcers (Wang et al., 2019). The gastroprotective effect of PPIs is essential in preventing further mucosal damage and facilitating ulcer healing. The management of iron-deficiency anemia, commonly associated with PUD, requires iron supplementation. Recent evidence supports the use of intravenous iron preparations, particularly ferric carboxymaltose, which has shown rapid hemoglobin improvement within 4-6 weeks and better tolerability compared to oral preparations (Singh et al., 2020). The target hemoglobin level should be monitored until reaching normal ranges (>12 g/dL in women, >13 g/dL in men).

For gout arthritis management, the challenge lies in finding alternatives to NSAIDs. Current evidence supports the use of colchicine for acute attacks, with a loading dose of 1.2 mg followed by 0.6 mg after one hour, showing significant pain reduction with minimal gastrointestinal effects (Khanna et al., 2021). For long-term management, urate-lowering therapy with allopurinol, starting at low doses and gradually titrating up, remains the cornerstone of treatment. Gastroprotection strategies for patients requiring continued NSAID therapy include concurrent PPI use and consideration of COX-2 selective inhibitors. A meta-analysis by (A. E. Thompson et al., 2020) demonstrated that the combination of a COX-2 inhibitor with a PPI reduced the risk of upper gastrointestinal complications by 78% compared to traditional NSAIDs alone. Prevention of recurrence requires ongoing monitoring and follow-up as well as efforts to educate patients. In addition, providing education about lifestyle modification, stopping smoking and drinking alcohol provides benefits in the management of gout arthritis as a meaningful additional therapy (Chen et al., 2021).

A recent qualitative study found that unsupervised NSAID use was higher in older and more rural demographics. This demographic group relied heavily on local stores to purchase pain medication without medical supervision or advice to manage their pain, but this led to complications in its use. This case report highlights the consequences of self-medication itself, especially when access to healthcare is limited. This highlights the urgent need for better medication monitoring and patient's education. Other studies have also found some links in this regard due to several concepts from this demographic group. First, they think that the treatment they are doing is effective based on testimonials they get from their acquaintances, past experiences, or acquaintances who work in the health sector. Second, they think that the treatment they are doing is safe enough and the stomach disease they experience they do not know is related to their drug use habits. Third, they have a mindset to shop for patented drugs compared to generic drugs because they are believed to have better and safer effects (Paliwal et al., 2019). By increasing their understanding of medication risks and safe practices, we can reduce medication side effects and addiction issues. In addition, better regulation of the use of over-the-counter drugs is essential to prevent inappropriate self-medication practices, especially in remote areas (Chi et al., 2018; Tawfik et al., 2017).

Conclusion

This case report examines the severe complications of chronic unsupervised NSAID use in a 62-year-old man from a remote area, which resulted in peptic ulcer disease and anemia gravis. The patient had a history of long-term use of NSAIDs and steroids for pain relief from gouty arthritis without medical supervision. Clinical evaluation revealed hematemesis, melena, and significant anemia, which were confirmed by laboratory findings. Limited access to diagnostic tools in the peripheral health care setting necessitated empiric treatment, including blood transfusion, proton pump inhibitors, and gout management medications. This case highlights the prevalence of chronic unsupervised NSAID use in remote and underserved areas, where limited access to health care transmits severe risk complications such as peptic ulcer disease and anemia. It illustrates the widespread misconception that over-the-counter medications are safe and the reliance on self-medication due to inadequate medical resources. Clinicians are urged to address these challenges through community outreach, early identification of high-risk patients, and implementation of safer therapeutic alternatives. Policies should focus on public health initiatives that promote medication safety awareness and advocate for stricter regulations on over-the-counter drug sales, especially in rural settings, to mitigate such preventable healthcare crises.

Conflict of Interest

The author declares no conflict of interest.

Consent Form

Informed consent for publication was obtained from the patient verbally.

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