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Case Report

Diclofenac Induced Gastritis: A Case Report

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ABSTRACT

Non-steroidal anti-inflammatory drugs are extensively used as anti-inflammatory agents, antipyretics and analgesics by many patients worldwide. The evidence based mechanism of therapeutic effects of NSAIDS is due to inhibition of Cyclooxygenase (COX) enzyme. However, the chronic usage of NSAIDS will result in plethora of adverse effects which includes Gastrointestinal, renal and cardiovascular systems. A scrupulous literature search revealed that diclofenac can cause GI adverse effects, but no diclofenac associated gastritis report has been published till date. In this back drop, we describe a case of 50 year old man, developed gastritis and anaemia as an adverse event who was taking OTC medication of diclofenac for six months duration. After administration of intravenous antibiotic, intravenous Proton pump inhibitor, anti-ulcer agent and fluid therapy the patient was recovered and discharged.

Key Words: NSAIDS, COX enzyme, gastritis

INTRODUCTION

Non-steroidal anti-inflammatory drugs (NSAIDs) are used chronically to reduce pain and inflammation in patients with arthritic illnesses, and also minor ailments as analgesics by many patients. However, regardless of their COX selectivity, reports are still appearing on the GI side effect of NSAIDs particularly on the lower gastrointestinal (GI) tract. [1] Diclofenac is a phenyl acetic acid derivative that is relatively non-selective COX inhibitor. This drug is used in various conditions as an analgesic and anti-inflammatory drug. [2] Gastritis is often produces no symptoms but may cause dyspepsia, anorexia, nausea or vomiting, and haematemesis or melena. [3] We would like to report a case of NSAID induced gastritis which was favoured by the presence of co-morbid conditions namely, asthma and left leg cellulitis.

CASE REPORT

A 50 year old male patient had visited the inpatient department presenting with continuous vomiting, abdominal pain, dyspepsia, anorexia since one month. He was known asthmatic since 2 years for which he was on regular anti-asthmatic medication. The case was known left leg cellulitis and underwent surgery 7 months ago. He was taking diclofenac 50mg as OTC medication for the post surgical pain for 6 months daily once a day.

Currently he admitted in general medicine department and had been diagnosed as NSAID induced gastritis. On examination, the patient was alert and appeared dehydrated. His body temperature was 38.9°C, blood pressure was 110/70 mm Hg, pulse rate was 80 beats per minute, and respiratory rate was 20 breaths per minute. He had no icterus, lymphadenopathy or clubbing pedal edema but he was pallor. His

cardiovascular and central nervous systems were within normal limits.

His laboratory data revealed his haemoglobin was decreased to 6gm/dl and RBS was 90mg/dl. All other laboratory parameters were found to be normal. Following deranged report and patients' history he was asked to stop OTC medication tablet diclofenac 50mg. The length of hospital stay was seven days and the patient was kept on treatment with antacid medication syrup sucralfate, H2 receptor antagonist ranitidine and an antibiotic amoxicillin + clavulanic acid. Along with these, intravenous fluids were administered on the day of admission and anti-asthmatics were continued without any changes.

DISCUSSION

Adequate pain management is a widespread clinical indication, and both prescription and over-the-counter (OTC) nonsteroidal anti-inflammatory drugs (NSAIDs) are frequently used for pain relief. [4] In a study conducted by Gor AP et al, various adverse effects were developed due to the usage of NSAIDs in the orthopaedic patients involving gastrointestinal system, cardiovascular system and renal system. [5] Another study conducted by B.K. Shah et al. concluded that use of fixed dose Combinations of diclofenac and paracetamol caused adverse reactions related to gastrointestinal tract. [6] Regardless of their safety and efficacy in most cases at prescribed dosages and for short durations, these drugs cause gastrointestinal toxicity in a large number of cases and also they can cause serious toxicity requiring hospital admission and immediate management. The gastrointestinal tract (GIT) is the main target of NSAID toxicity. [7]

The mechanism behind NSAID induced gastric problems is inhibition of COX pathway.COX enzyme is responsible for synthesis of Prostaglandins (PGs) which are known to be mucoprotective and ulcer healing agents. [8] Prostaglandins shield GI mucosa by forming a cytoprotective layer and increasing the secretion of bicarbonate ions that neutralise the gastric acidity. All therapeutically useful NSAIDs act by inhibiting the synthesis of PGs. [9] Cyclooxygenase has two isoforms, one constitutive (COX-1) and another inducible (COX-2). NSAIDs are now divided into selective (those inhibiting COX-2) and non-selective (inhibiting both COX- 1 and COX-2). [10] Most NSAIDs are weak organic acids and have low pKa. Therefore, they remain unionised in stomach and are absorbed appreciably from stomach. [11]

However, once they breach the cell membranes of stomach cells and reach within, they encounter a basic pH (e.g., 7.1). This causes so called "trapping" of the drugs inside the cell. This topical effect is considered an important mechanism of gastro-duodenal damage associated with their use. [12] Even short-term (<1 week) use of aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs) can precipitate ulcer-related bleeding. Conventional NSAIDs cause non-selective inhibition of cyclooxygenase, which leads to reduction in bicarbonate secretion and reduced mucous production. Coupled with it is vasoconstriction that occurs due to NSAIDs, which causes hypoxia and consequent formation of ulcer. [12]

The risk factors for NSAID induced gastrointestinal damage are older age, prior history of upper GI events, use of corticosteroids or anticoagulants, and high-dose or multiple NSAIDs (including NSAID plus low-dose aspirin). [13] The preventive strategies for NSAID induced gastrointestinal damage include anti-secretory agents, gastro protective agents, alternative NSAID formulations, and non-pharmacologic therapies. Greater awareness of the risk factors and potential therapies for GI complications resulting from NSAID use could help improve outcomes for patients requiring NSAID treatment. [14,15] Adding an adjuvant like H2 receptor antagonist or proton pump inhibitor to the non-steroidal therapy can improve the gastrointestinal

damage associated with the use of NSAIDs. [16]

REFERENCES

1. Harirforoosh S et al. Adverse effects of nonsteroidal antiinflammatory drugs: an update of gastrointestinal, cardiovascular and renal complications. *J Pharm Pharm Sci*.2013;16(5):821-47.
2. Bertram G. Katzung, Basic and clinical pharmacology 14th edition, McGraw-Hill publishers, Pg No: 647
3. Davidson's Principles and practice of medicine 21st edition, Elsevier publishers, Pg no: 870
4. Wilcox CM, Cryer B, Triadafilopoulos G. Patterns of use and public perception of over-the-counter pain relievers: focus on nonsteroidal antiinflammatory drugs. *J Rheumatol*. 2005;32(11):2218–2224.
5. Alpa Pragnesh Gor et al, Adverse drug reactions of nonsteroidal anti-inflammatory drugs in orthopedic patients, *J Pharmacol Pharmacother*. 2011 Jan-Mar; 2(1): 26–29.
6. B.K.Shah et al, Evaluation of prescribing pattern and adverse effects of fixed dose combination of non-steroidal anti-inflammatory drugs**,** *International Journal of Basic & Clinical Pharmacology*,May-June 2016:Vol 5, Issue 3 :1029-33.
7. Vikas Dhikav et al, Non-Steroidal Drug-induced Gastrointestinal Toxicity: Mechanisms And Management, *Journal, Indian Academy of Clinical Medicine* _ Vol. 4, No. 4 _ October-December 2003: 315-22.
8. Goodmann and Gillman's Mannual of Pharmacology and Therapeutics 12th edition, McGraw-Hill Publishers, Pg No: 423
9. Tamblyn, Robyn et al. Unnecessary prescribing of NSAIDs and the management of NSAID-related gastropathy in medical practice. *Ann Intern Med* 1997; 127: 429-38.
10. Charles Craig, Robert E.stitzel's Modern Pharmacology with Clinical Applications 5th edition, Williams and Wilkins Publishers, Pg No:425
11. Brahmankar's Bio pharmaceutics an Pharmacokinetics, 3rd edition, Vallabh publishers, Pg No: 44
12. Raskin JB. Gastrointestinal effects of NSAID therapy. *Am J Med* 1999; 106 (S 5B): 3-12
13. Laine L. GI risk and risk factors of NSAIDs, *J Cardiovasc Pharmacol.* 2006;47 Suppl 1:S60-6.
14. Simon LS, Weaver AL, Graham DY. Anti-inflammatory and upper gastrointestinal effects of celecoxib in rheumatoid arthritis: a randomised controlled trial. *JAMA* 1999; 282 (20):1921-8.
15. Jay L Goldstein, Gastrointestinal injury associated with NSAID use: a case study and review of risk factors and preventative strategies, *Drug, Healthcare and Patient Safety* 2015:7 31–41.
16. Goldstein JL. Challenges in managing NSAID-associated gastrointestinal tract injury. Digestion. 2004;69 Suppl 1:25-33.

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