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Original Research Article

NSAID Induced Gastritis and its Prevention through Education

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Abstract: Aim of this study is to study the prevalence of drug induced gastritis in general population and to take measures to prevent NSAID induced Gastritis through proper counseling and education. More than 15000 rural population patients were studied prospectively during December 2015 to March 2017 who were on NSAIDS for a period of 2 years to more than 10 years. Patients on NSAID treatment for more than 2 years were included in the study. This was a prospective study undertaken in a Community Health Centre; where patients from peripheral villages come for treatment and were addicted for NSAIDS. Patients were maximum labourers dependent on analgesics to get rid of Generalized Body Ache [GBA] due to extensive labour. The combined data showed that though the percentage of patients taking NSAID was less, yet the prevalence of NSAID induced gastro deodenal damage was very high in patients who were on NSAID treatment. Judicious use of drugs specially NSAIDS is required to prevent its untoward side effects. In this study it was evident that this drug induced gastrodeodenal damage was preventable by proper indication and counseling.
Keywords: Gastritis, NSAID, Generalized Body Ache, deodenal damage.

INTRODUCTION

Non-steroidal anti-inflammatory drugs (NSAIDs) are a broad class of agents with analgesic and anti-inflammatory properties that inhibit the two recognized isoenzymes of prostaglandin G/H synthase (also known as cyclo-oxygenase (COX))—namely, COX 1 and COX 2 Nonsteriodal anti-inflammatory Drugs(NSAIDS) are among the most commonly and overused drugs[1]. It is well known phenomenon that NSAID cause gastric mucosal damage resulting in NSAID Induced Gastropathy[2]. The mucosal injury and ulceration can be anywhere in Gastrointestinal tract from oesophagus to deodenum and can even end in complications like Erosive colitis, Peptic ulceration , Gastritis, Analgesic nephropathy, Hypersensitivity, Sodium and water retention leading to edema, hypertension and heart failure are complications of NSAIDS. Out of these most commonest causes, our study was focussed on NSAID induced gastritis and its prevention[3,4].

The mechanisms of NSAID-induced GI injury are not fully understood. Topical damage occurs in acidic NSAIDs such as acetylic-salicylic acid (ASA) and includes the accumulation of ionized NSAID in the gastric epithelial cell called ‘ion trapping’ effect, the reduction of the hydrophobicity of the gastric mucosal surface and uncoupling of oxidative phosphorylation. Disruption of the epithelial barrier allows back-diffusion of acid into the mucosa[5].

By inhibiting cyclo-oxygenases (COX) NSAIDs block the formation not only of proinflammatory but also of gastroprotective prostaglandins . This is a key element in NSAID gastropathy as prostaglandins maintain gastric mucosal blood flow and increase protective mucus as well as bicarbonate production. Leukotrienes are supposed to contribute to gastric mucosal injury by promoting tissue ischaemia and inflammation. Increased expression of adhesion molecules such as intercellular adhesion molecule-1 by proinflammatory mediators such as tumour necrosis factor-α leads to an increased neutrophil–endothelial adherence and activation .NSAID-induced neutrophil adherence might contribute to the pathogenesis of gastric mucosal damage by two principal mechanisms: (i) occlusion of gastric microvessels by

microthrombi leading to reduced gastric blood flow and ischaemic cell damage; (ii) increased liberation of oxygen-derived free radicals. Free oxygen radicals react with poly unsaturated fatty acids of the mucosa leading to lipid peroxidation and tissue damage.

Acute gastritis occurs when the lining of the stomach is damaged . This allows digestive acids to irritate the stomach. There are many things that can damage the stomachlining. The commonest causes include medications such as nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroids

Common symptoms include:

- loss of appetite
- indigestion
- black stools
- nausea /vomiting
- bloody vomit that looks like used coffee grounds
- pain in the upper part of the abdomen
- a feeling of fullness in the upper abdomen after eating.

MATERIALS AND METHOD

In this prospective study from December 2015 to March 2017, total 15023 patients were examined . Detailed clinical history and complete physical examination was done. Study was taken in Rural area at a Community Health Centre, where labour was the main source of earning and people were more dependent and already addicted to pain killers to get rid of their GBA. Our study involved patients taking drugs for more than 2 years. Surprisingly patients taking NSAIDS from Decade were also found. Patients on NSAIDS were studied.

Out of them NSAID induced Gastritis was our targeted group for study. 6712 were male patients while 8311 were female patients.

Factors that have been identified as placing patients at increased risk for NSAID-related GI complications include the following:

1. Prior history of gastrointestinal event (ulcer, hemorrhage)
2. Age .60 yr
3. High dosage
4. Concurrent use of corticosteroids
5. Concurrent use of anticoagulants.

Drugs that were most commonly prescribed

1. Diclofenac sodium
2. Brufen
3. Paracetamol.

Out of 15023 patients 1830 patients (12.18%) were on NSAIDS for Generalised Body Ache.[GBA]. Surprisingly patients were on more than one analgesic.

Out of 1830 patients who were on treatment with NSAIDS, 697 Patients(38%) suffered from gastritis.

RESULTS

Out of total 15023 patients examined 45% were male and 55% were female (Fig-1). Out of total study population of 15023 the patients on treatment with analgesics were 12.18% whereas 4.64% developed Drug Induced Gastritis.

Though out of total population only 4.64% patients suffered from drug induced gastritis, but when compared with only NSAIDS taking patients; the incidence of gastritis in patients on NSAIDS, this study gives estimate of 38% cases of drug induced gastritis(Fig-2).

Further confirmation of gastritis could not be done as patients were not ready to go for higher centre for Endoscopic Studies. The most important observation during study was that patients were not aware of the side effects of NSAIDS and were getting dependent . In almost more than 50% drugs were misused and prescribed only for daily labour induced GBA, which could have been managed by proper counseling. and relaxation techniques**.** Also in patients on NSAIDS, may be complications could have been reduced by use of PPIS[6].

Table-1: Compilation of data during study time

Sn	Month/Year	Total Patients Examined	Pts On Analgesics	Drug Induced Gastritis
1.	DEC 2015	64	21	5
2	JAN 2016	1005	152	72
3	FEB 2016	835	112	64
4	MAR 2016	1059	180	72
5	APRIL 2016	988	109	49
6	MAY 2016	907	94	50
7	JUNE 2016	1101	81	28
8	JULY 2016	1331	170	54
9	AUGUST 2016	1021	121	53
10	SEPT 2016	1274	132	44
11	OCT 2016	551	76	25
12	NOV 2016	1107	91	40
13	DEC 2016	1120	118	18
14	JAN 2017	1050	154	42
15	FEB 2017	910	124	47
16	MAR 2017	700	95	33

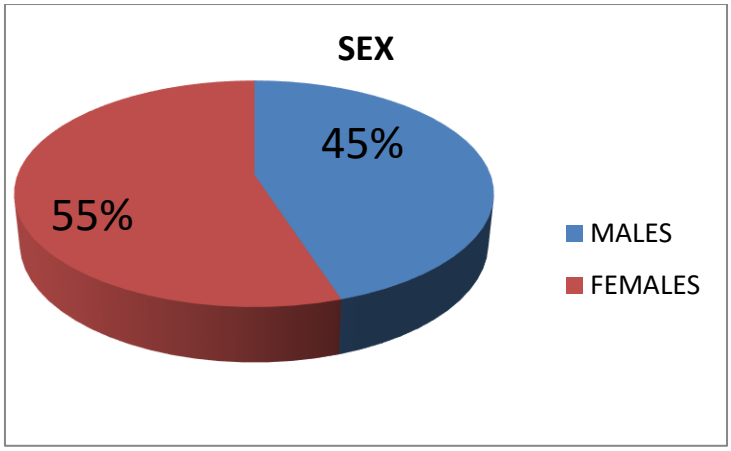

Fig-1: Distribution patients as per sex

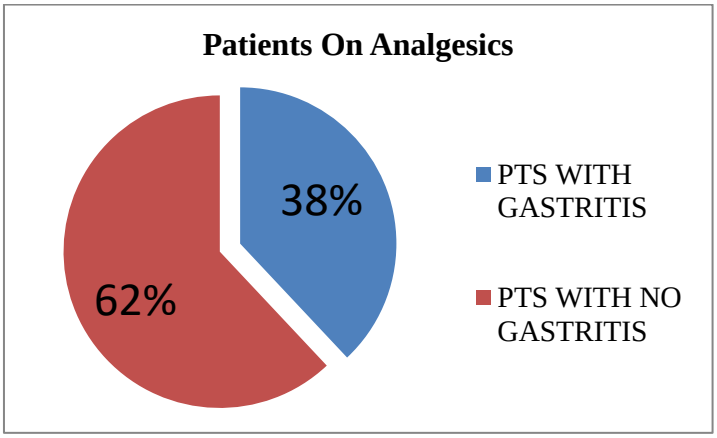

Fig-2: Patients On Analgesics

CONCLUSIONS

The best way to prevent NSAID gastropathy is to avoid these drugs. This is, of course, not possible in most cases. When using nonselective NSAIDs, it is important to reduce the doses to a minimum, as most of the adverse events occur dose-dependently. Drugs with a low GI toxicity profile such as ibuprofen should be preferred. It is crucial to identify patients at high risk for NSAID-induced GI complications . At least these patients require a gastroprotective Comedication with Vitamin C, where the prevention of NSAID gastropathy has been only little studied .The first step in the treatment of NSAID-associated ulcers lies in a reduction in the dosage of the NSAID[7] or discontinuation of the drug. If NSAID treatment cannot be withdrawn, a proton pump inhibitor [8] appears to be the most effective treatment in healing ulcers, accelerating the slow healing observed with H_2 antagonists.

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