Original Research Article

A study on endoscopic abnormality in drug induced UGI bleeding

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Abstract

Background: Non-steroidal anti-inflammatory drugs (NSAIDs) including aspirin are among the most frequently prescribed drugs worldwide and are available 'Over-The-Counter' (OTC) also. Though reasonably safe in most cases in prescribed dosages and for short durations, these drugs cause serious gastrointestinal toxicity in a large number of cases. They can affect all segments of the gastrointestinal tract causing ulcers, severe bleeding, perforation, and obstruction.
Aim: To study the endoscopic abnormalities in fifty cases of drug induced UGI bleeding.
Materials and methods: Fifty patients (32 males and 18 females) admitted with drug-induced hematemesis and or malena were studied with respect to age group, number of bouts of hematemesis, approximate quantity of total blood loss, causative drug responsible for UGI bleeding, risk factors of GI bleeding, number of individual risk factors in each patients. Thorough clinical and laboratory investigations were done. UGI scopy was done for all patients.
Results: NSAIDs were the commonest cause for UGI bleeding. Age $\geq$ 50 years (66%) was the commonest risk factor for UGI bleeding. It was observed that ten patients were having normal endoscopic study, twenty five patients were having lesions in stomach only, ten patients were having lesions in duodenum only and five were having lesions in both stomach and duodenum. None of the patients studied, had lesions in the esophagus. It was found that ulcers were more common than erosions.
Conclusion: Stomach is the commonest site affected (50%) and ulcers (64%) were common than erosion.

Key words

UGI bleeding, NSAID, Hematemesis, Malena, UGI scopy.

Introduction

Peptic Ulcer Disease (PUD) results from the imbalance between the defensive factors that protect the mucosa and offensive factors that disrupt the important gastric mucosal barrier. Primary ulcers seen in teenagers are associated with Helicobacter pylori infection and the secondary ulcers are due to NSAIDs including aspirin [1]. NSAIDs including aspirin are the common drugs responsible for peptic ulcer. Dyspepsia is present in 10 to 20 percent of patients and the true prevalence may range from 5 to 50 percent. Incidence of gastric ulcer cases following NSAID intake, ranges from 10% to 40% and the incidence of duodenal ulcer was 5% - 15%. Most patients are asymptomatic [1]. Drug induced UGI bleeding was common in above 50 years of age, nearly 70% [2].

Materials and methods

The study was conducted in the department of general medicine in KarpagaVinayaga Institute of Medical Sciences and Research from March 2015 to March 2016. Fifty patients satisfying the following inclusion criteria and not having any of the exclusion criteria were taken up for the study.

Inclusion criteria

- All adult patients of both sexes who were giving definite history of intake of drugs and subsequently developed vomiting of frank blood or coffee ground coloured vomit and/or passed dark coloured stools were chosen for this study.
- Inpatients admitted for other illnesses and who subsequently developed UGI bleeding following prescription with drugs like aspirin, other NSAIDs, steroids, anticoagulants and other gastro toxic drugs were also included.
- Standard definitions of hematemesis and malena were used when abstracting data from the clinical records.

Exclusion criteria

- Patients with past history of hematemesis and or malena
- UGI endoscopy finding of other causes of UGI bleeding (e.g. Varices, Mallory weiss syndrome etc.)
- Bleeding and clotting disorders
- Cirrhosis of liver with portal hypertension
- Hematological disorders
- Critically ill patients with life expectancy < 72 hr.

Detailed history regarding the UGI bleeding like, number of times of hematemesis, approximate quantity of blood vomited each time, associated with malena or presenting with malena alone and past history of hematemesis and or malena were obtained. Symptoms of GI toxicity of the drugs, symptoms of common diseases that can lead to UGI bleeding and symptoms due to blood loss were recorded in the questionnaire and detailed history regarding the drug and risk factors were asked. Routine general and systemic examination of the patients was carried out with the aim of assessing the general condition of the patient, confirmation of UGI bleeding by Ryle's tube aspiration and/or per rectal examination and assessing severity of blood loss and Ruling out other common causes of gastrointestinal bleeding like cirrhosis of liver with portal hypertension.

Laboratory investigations

Routine urine and blood investigations to find out diabetes, renal failure, hepatic failure, bleeding and clotting disorders and hematological disorders were carried out. Blood grouping and typing was done not only for transfusion of blood but also to find out the role of blood group 'O' in drug- induced UGI bleeding. Serological test for H. pylori (demonstration of anti-H. pylori IgG) was done to find out the association of this bacterium with drug-induced UGI bleeding.

Upper gastrointestinal endoscopy

Endoscopy was done for all the patients after overnight fasting using PENTAX video endoscopic system to directly visualize the side effects of the drugs on the mucosa of the esophagus, stomach and duodenum, like mucosal hemorrhages, erosions, superficial ulcers and deep ulcers. The number of ulcers, site and location of ulcers, size of ulcers, bleeding or not, healing ulcer or not, clean base of the ulcer or adherent blood clot, oozing of blood from the ulcer base and about visible blood vessel were studied.

Results and Discussion

NSAIDs including aspirin induced ulcers are usually asymptomatic unless complicated. Early symptoms are mild and benign like dyspepsia, nausea, vomiting, and anorexia. Pain is usually a late feature. When the ulcer starts bleeding hematemesis and/or malena occurs in about 1 to 3 % of patients. Most cases of NSAID-induced gastrointestinal ulcers can heal spontaneously, even when the drug is continued [2]. Moreover, it is not usually possible to diagnose these ulcers on the basis of clinical features alone, as symptoms suggestive of the ulcers can occur frequently in their absence. Elderly patients usually have painless gastric ulceration and NSAIDs can mask the symptom of pain [3]. In fact, most elderly patients are referred to physicians for iron deficiency anemia due to fecal blood loss. Few features, however, can be suggestive of NSAID-induced ulceration – like absence of H.pylori infection, anorexia rather than abdominal pain, antral location of gastric ulcers, known risk factors and prolonged self-medication of the large doses of NSAIDs. Chronic NSAID use can increase the risk of ulcer development by 10-30 folds [4, 5]. Elderly patients are particularly prone to develop GI toxicity and unfortunately they are the most frequent users of this group of drugs [5].

Oesophagus

Prolonged use of aspirin and most NSAIDs can result in ulceration, oesophagitis and even strictures more commonly caused by reflux rather than direct action of NSAIDs.

Stomach and duodenum

In acute injury (1-2 weeks) mucosal erythema, superficial erosions and submucosal hemorrhage are seen in endoscopy. In Chronic inury (>4 weeks), gastric antral erosions and ulcers, duodenal ulcers and erosion are seen.

Age

In our study, 66% of patients were in the age group of equal to or above 50 years of age (**Table – 1**). We found that the elderly patients $\geq$ 50 years of age were frequently taking NSAIDs for their orthopedic problems. The relative risk was 2.0 times higher than the others. In the early study by Griffin, et al. (1991), the relative risk for elderly patients with age group of $\geq$ 65 years was 3.8 times than the others [6]. A Sri Lankan study found that majority of the patients were in the middle age or elderly group, prone to high risk of NSAID induced gastric injury [7]. In Vikas, et al. study, seventy percent of the patients were $\geq$ 50 years of age. Indian population were more prone for drug induced UGI bleeding due to various factors like lesser average life span, early onset of arthralgia, arthritis, low backache, sciatica, spondyloses and coronary artery heart diseases [7, 8].

Sex

In this study, 32 were male patients and 18 were female patients and the male: female ratio was 1.77:1 (**Table – 1**). In Oliver, et al. study, incidence of drug induced UGI bleeding was twice as high among men as among women [8]. In a Sri Lankan study it was discussed that the incidence of drug induced UGI bleeding among male and female patients was in the ratio of 1: 2.125 [8].

Endoscopic findings

In the present study, endoscopy was normal in 20% of cases (**Table – 2, 3**). They had undergone delayed UGI endoscopy by two or three days and they had only minor UGI bleeding. On follow up they didn't develop further bleed. NSAID-

induced peptic ulcers usually heal very rapidly once the offending drug is stopped [9]. No one had findings in esophagus. Gastric lesions alone were found in 50% of cases, duodenal lesions alone were found in 20% of cases and in 10% of cases, findings were seen in both stomach and duodenum. Erosions in stomach were seen in 10% of patients while ulcers in stomach were seen in 40% of patients. Similarly, erosions in duodenum were seen in 4% of patients while duodenal ulcers were found in 16% of cases. Among the patients with lesions in both stomach and duodenum, erosions were seen in 2% of patients and ulcers were found in 8% of cases. Hence, ulcers were found to be more common (64%) than erosions (16%). In an Indian study, incidence of new ulcer cases following NSAID intake, ranges from 10% to 40% for gastric ulcers and 5% - 15% for duodenal ulcers [9].

Table – 1: Prevalence of drug-induced UGI bleeding related to age group and sex.

Age group	Male patients	Female patients	Total	%
Age ≤ 19	0	0	0	0%
Age 20 – 34	6	3	9	18%
Age 35 – 49	5	3	8	16%
Age 50 - 64	16	8	24	48%
Age 65 – 79	4	4	8	16%
Age ≥ 80	1	0	1	2%
Number of patients	32	18	50	100%

Table – 2: Prevalence of site of lesions on endoscopic study.

Endoscopic findings	Number of patients (%)
Normal study	10 (20%)
Lesions in esophagus	0 (0%)
Lesions in stomach only	25 (50%)
Lesions in duodenum only	10 (20%)
Lesions in both stomach & Duodenum	5 (10%)

Table – 3: Prevalence of nature of lesions on endoscopic study.

Nature of lesions	Number of patients
Erosions	8 (16%)
Ulcers	32(64%)
Normal endoscopy	10 (20%)

Studies have demonstrated increased intestinal permeability with SR and EC formulations of all NSAIDs but not with conventional release tablets and lower GI bleeding are also more common with them [10]. In the present study only one female patient was prescribed ‘sustained release’ tablet diclofenac 100 mg, and on endoscopy she was found to have lesions in both stomach and duodenum.

Conclusion

The present study on endoscopic abnormality in drug-induced UGI bleeding concludes that ‘Gastric lesions only’ was found in 50% (highest percentage); next to that ‘duodenal lesions only’ in 20% and both gastric and duodenal lesions in 10%. Endoscopy was normal in 20%. Regarding the nature of lesions, ulcers were more common (64%) than erosions (16%).

References

1. Simon L.S., Weaver A.L., Graham D.Y., Kivitz A.J., Lipsky P.E., Hubbard R.C., Isakson P.C., Verburg K.M., Yu S.S., Zhao W.W., Geis, G.S. Anti-inflammatory and upper gastrointestinal effects of celecoxib in rheumatoid arthritis: A randomized controlled trial. Journal of American Medical Association, 1999; 282: 1921 – 1928.

2. Vikas D., Sindhu S., Swati A. C., Kuljeet S., Anand K. Non-steroidal Drug-induced Gastrointestinal Toxicity: Mechanisms and Management. Journal of investigational allergology and clinical immunology, 2003; 4: 315 – 322.
3. Dhikav V. Aspirin misconceptions. Drugs News and Views, 2001; 6: 64 - 65.
4. Andrade S.E., Gurwitz J.H., Fish L.S. The effect of an Rx-to-OTC switch on medication prescribing patterns and utilization of physician services: the case of H_2-receptor antagonists. Medical Care, 1999; 37: 424 - 430.
5. Yeoman N.D. Approaches to healing and protection of NSAID induced ulcers. American journal of Medicine, 2001; 110: 24S - 28S.
6. Griffin M.R., Piper J.M., Daugherty J.R., Snowden M., Ray W.A. Non-steroidal anti-inflammatory drug use and increased risk for peptic ulcer disease in elderly persons. Annals internal Medicine, 1991; 114: 257 - 263.
7. Satarasinghe R.L., Jayamaha D.H. An audit on the prescription habits of non-steroidal anti-inflammatory drugs (NSAIDS) in the medical clinics of a Base Hospital in Sri Lanka. Journal of the Ceylon College of Physicians, 2000; 33: 23 – 32.
8. Oliver B., Lindsay A. D., William R. M., Mary B., Jill P. Acute upper gastrointestinal haemorrhage in west of Scotland: case ascertainment study. British Medical Journal, 1997; 315: 510 – 514.
9. Loren L. Approaches to Non-steroidal Anti-inflammatory Drug Use in the High-Risk Patient. Gastroenterology, 2001; 120: 594 – 606.
10. Choi V.M.I., Coates J.E., Chooi J., Thomson A.B.R., Russell A.S. Small bowel permeability - a variable effect of NSAIDs. Clinical and investigative medicine. Medecine clinique et experimentale, 1995; 18: 357 - 361.