



Association between Smoking and Silent Peptic Ulcer Disease in the General Population of Kashmir

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ABSTRACT

Objectives: To assess the occurrence of silent peptic ulcer disease (PUD) in patients who smoke and try to define the relationship between the two.

Methods: The study was carried out between 10th January 2014 to 28th November 2014 in the general surgical department of SMHS hospital in Srinagar, Kashmir. The patients with Smoking exposure of at least one pack-year, and current smoker, Negative serology for H. Pylori, No (or occasional) history of NSAID intake/ alcohol consumption were included in the study. The end point of the study was one year follow-up.

Results: The evidence of peptic ulcer disease on endoscopy was found in 23 subjects, 19 of these occurred in male subjects while 4 were in female subject.

Conclusion: Based on our study, we conclude that silent peptic ulcer disease is fairly common in smokers than previously thought, and the risk of acquiring it increases with increase in smoking exposure. We conclude that upper gastrointestinal endoscopy should be a routine at 10 pack-years of exposure irrespective of age. Also, PPI/H₂ blockers should be considered in smokers irrespective of symptoms.

Keywords: Kashmir, peptic ulcer, smoking, H. pylori, endoscopy.

INTRODUCTION

Smoking is a common social problem world over, and the population of subcontinent is no exception to it. In Kashmir, we have seen it in about 60% of the total OPD patients visiting our tertiary care hospital. Among different forms of smoking, filtered cigarettes account for roughly 70%, while

hookah accounts for about 24% of forms of smoking. The health impacts of smoking are vast with notables being lung cancer, COPD, peptic ulceration, oesophageal cancers etc.

Smoking seems to be a risk factor of major importance for ulcerperforation. A study from Norway showed a strong association between

ulcer perforation and smoking¹. One study also shows that it may be a stronger factor than H. Pylori in causation of PUD in certain populations². However not all peptic ulcer diseases are symptomatic. Asymptomatic ulcers are common in patients who take aspirin and other NSAIDs. They are also common in patients with H. Pylori infestation³. Smoking also plays an important role in the occurrence of silent peptic ulcer disease.

In this study, we will assess the occurrence of silent peptic ulcer disease (PUD) in patients who smoke and try to define the relationship between the two.

MATERIALS AND METHODS

The study was carried out between 10th jan.2014 to 28th November 2014 in the general surgical department of a tertiary care hospital in Srinagar, Kashmir. A total of 134 endoscopies were performed during this period for the purpose of this study.

The selection criteria of subjects for the study were:

- Age ≥ 45 years
- No history of previous peptic ulcer disease/ PPI (or H₂antagonist) use.
- No previous endoscopies

- Smoking exposure of atleast one pack-year, and current smoker.
- Both sexes
- Negative serology for H.Pylori
- No (or occasional) history of NSAID intake/ alcohol consumption.
- No alcohol consumption/ steroid use.

In Kashmir, screening endoscopies are fairly common from 45 years of age; hence this age group was chosen for this study to increase “yield”. A thorough history was taken from each participant of the study to exclude any peptic ulcer disease in the past. Similar history was taken to rule out any aetiologies of PUD like NSAIDs, Alcohol, H.Pylori, stressful lifestyles etc. None of the study subjects in the group has had any previous upper GI endoscopies.

The smoking history was quantified in terms of pack-years. Because in India cigarettes are dispensed in a pack of 10, unlike in western countries where its 20 per pack, therefore, each Indian brand of cigarettes pack was considered ½ pack. This was done for the standardisation of results to be obtained thereof. None of the study subjects in our study consumed foreign brands or smoked *hookah*. An informed consent was taken from each participant of the study.

The demographic features of the study population are as shown in table 1.

	male	female
<i>n</i>	90	44
age	52.1± 5.25	50.56±4.80
BMI	22.6±2.49	25.11±2.97
smoking	58(64.4%)	8(18.18%)

The low incidence of smoking trends in women subjects above can be attributed to the fact that in conservative societies smoking by women is considered a taboo, and hence discouraged.

RESULTS

The evidence of peptic ulcer disease on endoscopy was found in 23 subjects. 19 of these occurred in male subjects while 4 was in female subject. Most of these lesions were duodenal erosions (fig. 1).

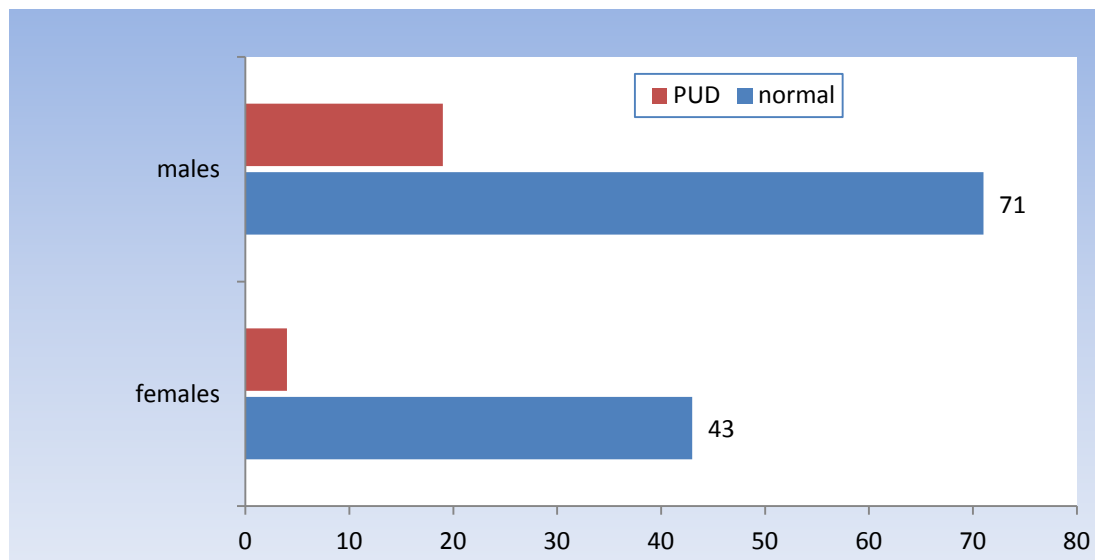


Figure 1

Table 2 and fig. 2 depicts the relationship between smoking and PUD.

		PUD		No PUD	* <i>p</i> -value
smoker	Male	58	16	42	< 0.05
	Female	8	3	5	
No smoking	Male	32	3	29	
	Female	36	1	35	

Table 2 (* Fischer's exact)

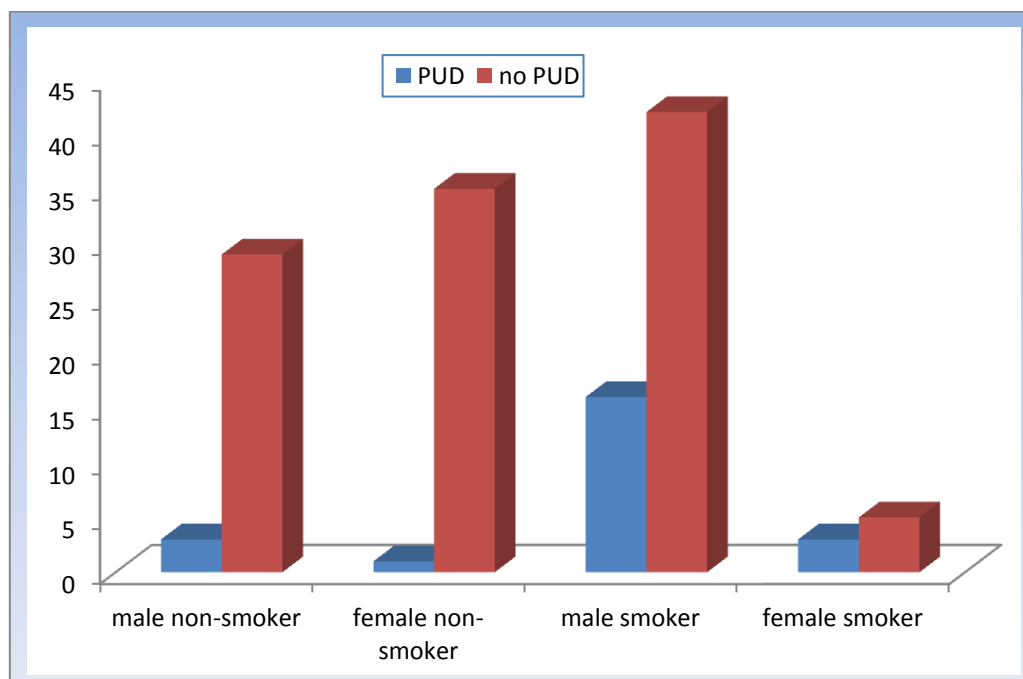


Figure 2

Smoking exposure was further stratified in various groups and the data extrapolated as shown below (table 3):

Quantity of smoking	PUD	No. of smokers	RR
<2 pack-years	02	10	3.4
2-5 pack-years	02	24	1.41
5-10 pack- years	04	12	5.66
>10 pack years	11	20	9.35
TOTAL	19	66	

Table 3

As calculated from the table above, we can see that the relative risk (RR) of developing silent PUD at >10 pack-years is 9.35 (~9.4). Also, relative risk at >5 pack-years is 5.66.

These relative rates are calculated with respect to non-smoker study group (i.e. 4 silent PUD in a group of 68 non-smokers). This is further illustrated graphically in fig.3.

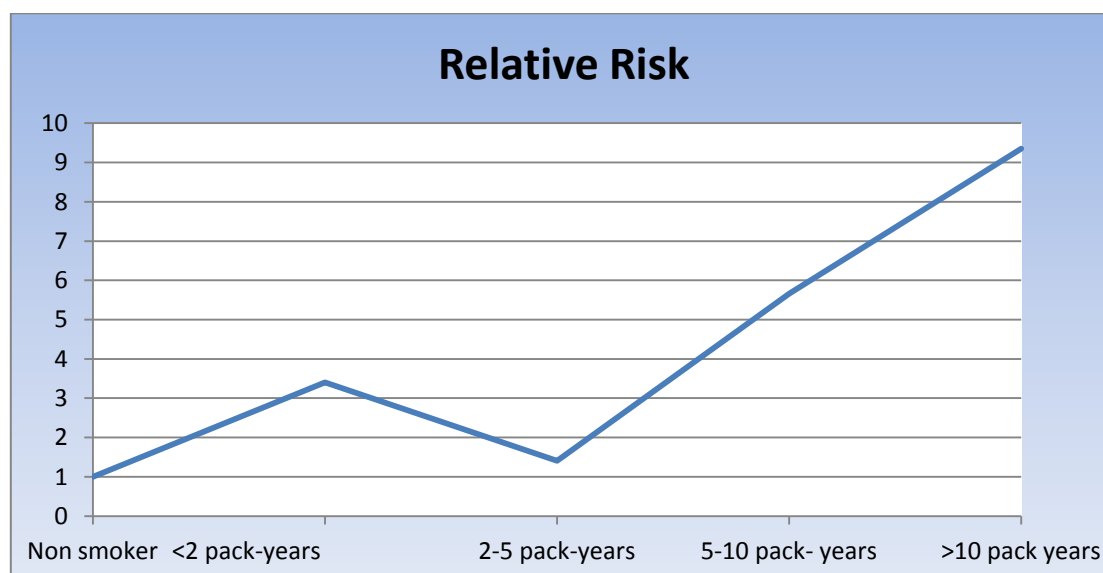


Fig. 3

DISCUSSION

Peptic ulcer disease is fairly common worldwide with fair number of patients in subcontinent. As we all know that the various risk factors for developing PUD are H. Pylori infection, NSAIDS, Alcohol, Smoking, Steroids etc. Although, the association between coffee drinking and PUD is controversial⁴. Smoking is one of the important factors in the causation of PUD. In fact Smoking seems to be a risk factor for complicated PUD like perforation and gastric outlet obstruction^{5,6}.

Although various factors of peptic ulcer causation exist together, we tried to exclude them to in this study to the best of our ability. The main idea was to study smoking in isolation to other risk factors and determine its exact role as a causative agent in silent PUD. Fortunately enough for us, alcohol consumption is a very minor trend in Kashmiri population; and none of our subjects in both the groups had any history of alcohol intake - past or present. As regards NSAIDS, we excluded all the patients with a regular history of NSAIDS intake

and only included those patients who had no history of exposure to NSAIDS (oral or topical); or at the most an occasional exposure to *cox-2* NSAIDS (defined as once in 4-6 months).

H. Pylori was excluded with commercially available serological kits available. Diet plays a role in the development of gastritis, especially in winter months when fresh fruits and fresh vegetables are not available⁷. In addition, smoking of small fresh water fish, called “*pher*” in Kashmiri language, is a winter delicacy in Kashmir. Care was taken to include only those subjects who had either low or no exposure to “*pher*”. Besides, the subjects were allotted to each group keeping all the attributes in mind so as to minimise investigator and observer bias. This study assumed a combined outcome of gastric and duodenal ulcers, gastric and duodenal erosions, all detected by endoscopy in the absence of any symptoms. The results were obtained and subjected to various statistical tests.

CONCLUSION

Based on our study, we believe that silent peptic ulcer disease is fairly common in smokers than previously thought, and the risk of acquiring it increases with increase in smoking exposure. We feel that upper gastrointestinal endoscopy should be a routine at 10 pack-years of exposure irrespective of age. Also, PPI/H₂ blockers should be considered in smokers irrespective of symptoms.

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