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Research Article

A STUDY ON ASSOCIATION OF SMOKING AND GASTRIC CARCINOMA IN THE RESIDENTS OF WEST BENGAL

Ashis Kumar Saha¹, Goutam Chatterjee¹, Subhas Chandra Hazra²

¹Assistant Professor, ²Professor & Head, Department of General Medicine, K P C Medical College, Jadavpur, Kolkata

*Corresponding author email: asissaha2008@gmail.com

ABSTRACT

Objectives: The aim of the study is to know the association of tobacco intake in the form of smoking and chewing with gastric carcinoma in West Bengal. **Materials and methods:** Total 28860 patients (smokers and tobacco chewer 17240, nonsmokers 11620) were interrogated before performing upper gastrointestinal endoscopy. Among the smokers and tobacco chewers, isolated bidi and cigarette smokers were 5067, 9323 and 2850 respectively. Among 542 gastric cancer cases, smokers were 301 (165 cigarette and 136 bidi smokers) and tobacco chewers 82 respectively. Then comparisons were done: 1. to know the incidence of smokers and nonsmokers in total number of patients, the influence of bidi and cigarette smoking on gastric carcinoma, 3] Effects of the early starters and number of cigarettes/bidi per day on gastric carcinogenesis. Again, comparisons were done to know influence of bidi and cigarettes on the sites of gastric carcinoma. **Results:** Bidi smokers, earlier starters of smoking and significantly ($P < 0.0001$) suffered from gastric carcinoma. Heavy drinkers were mostly affected ($P < 0.0001$). **Conclusions:** Bidi smokers, young heavy smokers were mostly affected. So there were strong associations between bidi smoking and gastric carcinoma in the residents of West Bengal.

Keywords: Tobacco smoking, tobacco chewing, gastric carcinoma, residents, West Bengal

INTRODUCTION

Stomach cancer is the second most common cause of death due to cancer only throughout the world¹ following lung cancer.² It is the 2nd and 4th most common cancer in males and females respectively.^{3, 4} Case fatality ratio is higher than other malignancies, like, colon, breast and prostate cancers⁵. Tobacco smoking has been identified as recognized risk factor as observed in different epidemiological studies⁶, but some studies failed to identify tobacco smoking as risk factor^{7, 8}. Risk factors for gastric cancer include high intake of alcohol, tobacco smoking and tobacco chewing, high intake of pickled and salted food⁹. Complex interaction between genetic factors and environmental factors are responsible for the genesis

of gastric cancer. Genetic factors include polymorphism in inflammatory cytokine genes, xenobiotic metabolic genes – these factors play a major role.^{10, 11}. Whereas major environmental factors are alcohol, tobacco smoking, tobacco chewing, Helicobacter pylori infection, low intake of fruits and green vegetables and a high intake of salted and pickled food. The association between smoking and gastric carcinogenesis has been studied for several years, since, first cohort studies conducted by Khan¹² and Hammond¹³. The risk of gastric cancer among young adult and adult smokers, higher than in non-smokers was shown in a meta-analysis published in 1997.¹⁴ The blood group of the patients suffering

from gastric cancer is “A”. Our present study was to demonstrate the association of tobacco smoking (in the form of bidi and cigarette) and chewing in the genesis of gastric cancer in the Gangetic areas of West Bengal and to update with the systemic review of the available epidemiological evidences on the relationship between tobacco smoking and chewing and gastric carcinogenesis.

MATERIALS AND METHODS

After the IEC approval and inform consent from the patients, the present study was conducted in the department of Medicine in K P C Medical College from the year 2007 to 2013.

Inclusion criteria: The patients undergone upper gastrointestinal endoscopy for evaluation of symptoms (pain abdomen, vomiting, indigestion, hematemesis with/without melena, dysphagia, weight loss, anorexia) in the age-group of 18 to 85 years and in both sexes were included in our study.

Exclusion criteria: Obviously, who were not willing to give consent for endoscopy excluded from the study. In our study, no patient suffered from HIV disease or active tuberculosis.

We started our extensive study the influence of tobacco smoking and tobacco chewing on the genesis of gastric cancer. During the last six years, total 28860 patients from different districts of West Bengal (involving Malda, Murshidabad, Nadia, Howrah, Hoogly, North and South twenty-four Parganas, Midnapore and Kolkata) were sent for upper gastrointestinal endoscopy (UGIE) to evaluate the different presenting symptoms. Before performing UGIE, informed written consent were taken from patients’ parties followed by taking a proper history in the form of a structured questionnaire. This included demographic data (age, sex and religion) and “substance use” (tobacco smoking and chewing) data. Under the heading of “substance use” data, following histories were included – 1. Age at which smoking and chewing have been started. 2. Number of bidi or cigarette per day was taken. 3. The form of tobacco used – tobacco chewing, bidi or cigarette

smoking. UGIE were performed using 15% xylocaine as local anesthesia. From the suspected lesion in the stomach, eight bits of tissues were taken and were sent in 10% formalin at room temperature for histopathological examinations.

Statistics: All the analyses were done at 95% confidence interval and probability values (p-values) were observed to identify the significance of the results. Mean values with standard deviation were used to detect the age at which the smoking was started and the number of bidi or cigarette per day.

1. P value indicates the maximum probability for a given level of significance.
2. 95% CI for difference of percentage:

$$(p_1 - p_2) \pm 1.96SE (p_1 - p_2), \text{ where } SE (p_1 - p_2) = \left\{ \left[\frac{p_1(1-p_1)}{n_1} \right] + \left[\frac{p_2(1-p_2)}{n_2} \right] \right\}^{1/2}$$

Calculations were done by using Graphic pad software.

RESULTS

Among 28860 patients underwent endoscopy, 17240 patients were smokers and tobacco chewers and 11620 patients were non-smokers and non-chewers. Total 542 patients were diagnosed as gastric carcinoma, some tumors were well differentiated, and some were poorly differentiated (fig 1, 2 and 3). Smokers and tobacco chewers were significantly affected than non-smokers and non-chewers (383 vs. 159, $p < 0.0001$) [Table 1]. Smokers were significantly affected than tobacco chewers (301 among 14390 vs. 82 among 2850 patients, $p < 0.005$). [Table 2]. Again, bidi smokers were significantly affected than cigarette smokers (165 in 9323 patients vs. 136 in 5067 patients, $p < 0.0001$) [Table 3]. Early starters as well as, heavy smokers were significantly affected (23.2 ± 5.8 vs. 12.3 ± 5.1 in case of early starters, $p < 0.0001$, and 13.1 ± 7.5 vs. 20.5 ± 9.2 , $p < 0.0001$) [Table 4]. Again, antral and incisural mucosa were significantly involved in smokers and non-smokers respectively (214 in 383 vs. 58 in 159 patients, $p < 0.002$ and 39 in 383 and 37 in 159 patients, $p < 0.01$ respectively) [Table 5].

Table: 1 Incidence of gastric carcinoma in smokers and nonsmokers (n=28860)

Smoker and tobacco chewers	patients undergone endoscopy	persons affected	% affected
Smoker & tobacco chewer	17240	383	2.221
Non smoker	11620	159	1.368

Table: 2 Relation between isolated smoking and tobacco chewing with gastric carcinoma (n=383):

Smokers+ Tobacco chewer (17240)	UGIE performed	Cases (383)	%
Smoking	14390	301	2.09
Tobacco chewer	2850	82	2.87

Table: 3 Relationship of bidi & cigar with gastric carcinoma (smokers =301):

Smoker (cigar + bidi) (14390)	pts performed	Cases (420)	%	95% CI	P value
Cigarette smoker	9323	165	1.76	-0.01, 0.004	<0.001
Bidi smoker	5067	136	2.68		

Table: 4. Among the smokers (Mean±SD)—14390

Criteria of smoking	Subjects not affected (14089)	Subjects affected (301)	95% CI	t- test	P value
Age at which smoking started	23.2 ± 5.8	12.3 ± 5.1	10.24, 11.56	32.33	<0.001
No. of cigars/day	13.1 ± 7.5	20.51 ± 9.2	-8.27, -6.55	-16.87	<0.0001

Table: 5 Among the affected persons (542) relation of smoking and tobacco chewing with site of gastric carcinoma

Type of persons	Fundus	95% CI	P value	Body	95% CI	P value	Antrum	95% CI	P value	Incisura	95% CI	P value
Smokers & tobacco chewer (383)	59 (15.4)	-0.11, 0.03	0.42	66 (18.53)	-0.09, 0.05	0.37	219 (55.8)	0.04, 0.22	0.01	39 (10.18)	-0.19, -0.06	0.01
Non smokers (159)	31 (19.4)			34 (21.3)			58 (36.4)			37 (22.64)		

NS*= Not significant, S**= Significant

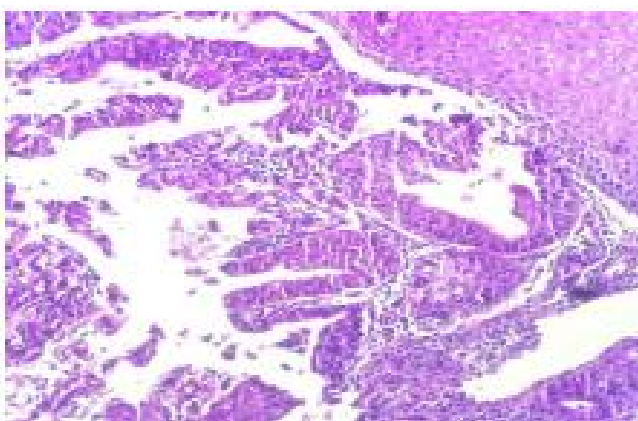


Fig 1: Stomach GEJ (bx): Moderately differentiated adenocarcinoma

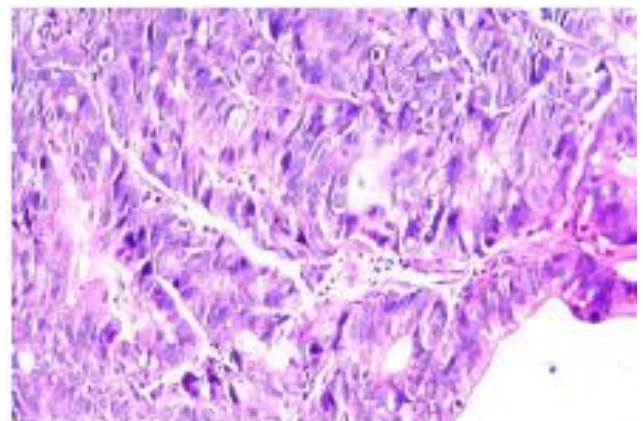


Fig 2: Stomach (bx) : Well differentiated adenocarcinoma.

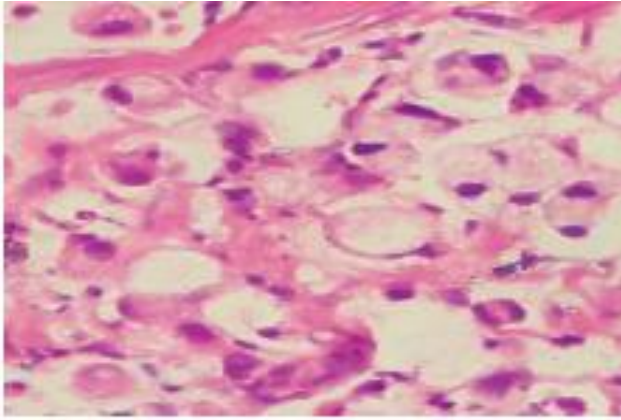


Fig 3: Stomach (GEJ) (bx): Signet ring cell carcinoma.

DISCUSSION

The molecular genetics and the pathogenesis responsible for the development of gastric carcinogenesis are poorly understood. The relationship between gastric carcinogenesis and tobacco smoking and chewing is poorly evaluated. Recent review by Tredaniel et al¹⁴ containing meta-analysis of the 40 studies demoes quantitative estimation of association between tobacco smoking and genesis of gastric cancer. In this review, all categories of smoking, e.g. current smoker and non-smoker, smoker and non-smoker and smoking dose relationship (ODDS RATIO=1.49 for smokers up to 20 cigarettes per day and ODDS RATIO=1.67 for heavy smokers) had been properly evaluated. Lauren system classifies gastric cancer into two types: type I is intestinal type (expansive and epidemic type of gastric cancer) and type II is diffuse type (infiltrative and endemic type). This study demonstrated that rise in gastric cancer was higher in current smokers than ever smokers – indicating decreasing trends in the risk after quitting smoking. Similarly, increased risk of gastric cancer in smokers and tobacco chewers were demonstrated by Phukon et al¹⁵ as well as studies performed in South India¹⁶ Gajalakshmi et al¹⁷ Our study similarly demonstrated the higher incidence of gastric cancer in smokers. Sung et al demonstrated a weak association between tobacco smoking and gastric cancer.¹⁸ Symptoms of gastric carcinoma are anorexia, anemia, asthenia, vomiting, pain abdomen, weight loss. Again, Laroia I et al demonstrated that tobacco smoking and chewing were frequently seen in case than the controls, but these differences were not significant.¹⁹ Moreover, case-control study demoes reduced risk (OD=0.52, 95% CI: 0.3 – 0.89) in current smokers as compared

to non-smokers.¹⁹ The study led by E.C. Smith of Memorial Sloan-Kettering Cancer Centre and Colleagues found men and women who had ever used hundred cigarettes per day in their life time were 1.45 times as likely as non tobacco users to die from gastric cancer even after curative operation. But after operation vitamin B₁₂ lack is responsible for lowering of the quality of life in the patients survived. On the other hand, vitamin D is responsible for blocking the growth of the tumor, lowering the blood supply to the tumor and preventing its spread.

Again, Mizoram study¹⁴ showed higher incidence of gastric cancer in tobacco chewers than tobacco smokers, which was similar to our study, where tobacco smokers were significantly affected.

Our study demonstrated that distal parts of the stomach like antrum, incisura were significantly affected in smokers and non-smokers respectively, which was similar to the study done by Chao et al.²⁰ Studies in India showed a strong association between bidi smokers and cancer in pharynx, larynx, oral cavity and esophagus²¹ Again, Gajalakshmi et al showed threefold increase in incidence of gastric carcinogenesis in bidi smokers as compared to cigarette smokers. It is true that amount of tobacco in bidi (0-0.3 gm.) is less as compared to cigarette (1 gm.)²² but rise in gastric carcinogenesis is higher in bidi smokers, which may be attributed to poor combustibility as a result of low porosity of the negligee (Tendu leaf), which causes accumulation of higher concentration of volatile phenol (neoplasm provoking agents), tar, carcinogenic hydrocarbon benz (a), anthracene and benzo (a) pyrene.

Our study demonstrated the significant increase in the incidence of gastric cancer in early starters and chronic heavy smokers as compared to late starters and occasional smokers. Similar findings were shown in the study done by Gajalakshmi et al¹⁷ i.e. The risk of gastric (diagnosed by endoscopic biopsies and histopathological examinations) cancer was decreased with a higher age of onset of smoking. Here, in that study, this trend was shown in case of bidi smokers, and incidence was increased with an increase in the quantity of bidi smoking during their life time.

CONCLUSION

Smokers were significantly affected than non-smokers. Again, bidi smoking was revealed as a significant risk factor for the development of gastric

carcinogenesis. Early starters and chronic heavy smokers were susceptible to gastric cancer. The lower part of the stomach was significantly affected in smokers.

Conflict of interest: Nil

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