ALCOHOL AND GASTRIC CANCER IN THE RESIDENTS OF WEST BENGAL

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INTRODUCTION

Second most common cause of cancer death after lung cancer in the world is adenocarcinoma of stomach (Peter Boyle et al, 2008). In males and females gastric cancer is the 2nd and 4th common cancer respectively (Danaei et al, 2005, Catalano et al, 2009). Case fatality ration of the cancer is higher than colon, breast and prostate cancer (Jemal et al, 2011). Japan, China, Korea, Central and South America are the highest risk areas; whereas, Southern Asia, North America and Africa are the low risk areas form the epidemiological point of view (Parkin et al, 2005). Smokers, tobacco chewers, alcoholics and high intakers of salted and prickled foods are at highest risk of gastric cancer (IARC 2004).

But some studies denied this observation (Ray et al, 2007, Bagnard et al, 2001). Genetic and environmental factors interact with each other in the development of gastric cancer. Among the genetic factors, polymorphism are seen in the inflammatory cytokine and xenobiotic metabolic genes, whereas, among environmental factors, tobacco smoking and chewing, alcohol consumption, high intake of salted and prickled foods and lack of refrigeration seem to play major roles (Correa et al, 2005; La Torre ey al, 2005).

Our present study was to observe an association of drinking of alcohol and the development of gastric carcinogenesis and to update with the systemic review of the available epidemiological data on the relationship between alcohol drinking and gastric carcinogenesis published till date.

MATERIALS AND METHODS

We started our original and honest study on the relationship of alcohol drinking and the gastric carcinogenesis only after getting permission from our ethical committee. This was our extensive seven years study. Total 28860 patients from different districts from West Bengal, like, Malda, Nadia, North and South 24 parganas, Howrah, Hoogly, Midnapore and Kolkata, were sent for upper gastrointestinal endoscopy for evaluation of different presenting symptoms. After taking informed consent from all the patients’ parties, all the subjects were interviewed by the trained interviewer to collect demographic data, like, age, sex and religion, and “Substance use data” of alcohol using a structured standard questionnaire. “Alcohol intake” history included: 1. Type of alcohol intake, 2. Age at which drinking has been started, 3. Number of years of drinking, 4. Numbers of pegs per day. Endoscopy was performed using 15% xylocaine as local anesthesia. During the procedure, eight bits of tissues were taken from suspected lesion in any area of stomach and was sent for histopathological examination fixed in 10% formalin at room temperature.

Statistics

Analyses were done at 95% confidence interval to get the probability value (p-value) to detect the level of significance. Median value with standard deviation were also calculated to detect the age at which drinking has been started, numbers of years of drinking, number of pegs per day.

95% CI for difference of percentage:

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RESULTS

Total 28860 patients were underwent endoscopic procedure, of which, 11532 were alcoholic and 17328 non-alcoholic. Alcoholic patients were significantly affected than non-alcoholics (p<0.0001) [Table I]. Early starter of alcohol (34.5±12.8 vs. 25.6±6.5, p<0.0001), long time drinker (21.9±9.3 vs. 32.6±9.6, p<0.0001) and heavy drinkers (3.6±4 vs. 6.2±8.8, p<0.0001) were significantly affected [Table II]. Again, wine and liquor drinker were significantly affected than other beverages including beer drinker (p<0.0001) [Table III].

<table>
<thead>
<tr>
<th>Alcohol intake</th>
<th>Total no. of patients undergoing endoscopy</th>
<th>Total numbers affected</th>
<th>% affected</th>
<th>95% Confidence interval</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcoholic</td>
<td>11532</td>
<td>348</td>
<td>3.01</td>
<td>0.79 – 0.97</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Non alcoholic</td>
<td>17328</td>
<td>194</td>
<td>1.11</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table II Among the alcoholic person (11532) -- Mean±SD

<table>
<thead>
<tr>
<th>Criteria of alcohol intake</th>
<th>Persons not affected (11184)</th>
<th>Persons affected (348)</th>
<th>95% Confidence interval (assumed equal variances)</th>
<th>95% Confidence interval (assumed unequal variances)</th>
<th>t-test for equal variances (assumed)</th>
<th>t-test for unequal variances (assumed)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at which alcohol drink started</td>
<td>34.5±12.8</td>
<td>25.6±6.5</td>
<td>7.55, 10.25</td>
<td>8.18, 9.62</td>
<td>12.91</td>
<td>24.12</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Number of years of drinking</td>
<td>21.9±9.3</td>
<td>32.6±9.6</td>
<td>-11.69, -9.71</td>
<td>-11.75, -9.67</td>
<td>21.11</td>
<td>20.49</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Number of pegs per day</td>
<td>3.6±4</td>
<td>6.2±8.8</td>
<td>-3.05, -2.15</td>
<td>-3.53, -1.67</td>
<td>11.306</td>
<td>5.49</td>
<td>&lt;.0001</td>
</tr>
</tbody>
</table>

Table III Comparison of Different types of beverages in the affected patients (348 alcoholic)

<table>
<thead>
<tr>
<th>Types of beverages</th>
<th>Exposed patients 11532</th>
<th>Affected patients 348</th>
<th>95% Confidence interval</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wine &amp; liquor</td>
<td>7919</td>
<td>280</td>
<td>0.01, 0.02</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Other beverages Including beer</td>
<td>3613</td>
<td>68</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

DISCUSSION

Gastric cancer, though one of most common cancers throughout the world, its pathogenesis and molecular genetics responsible for its development has been poorly evaluated. According to different literatures worldwide, alcohol may be carcinogenic to esophagus and cardia, but not to distal part of the stomach. Similarly, studies in Mumbai failed to show alcohol as a risk factor for gastric cancer. In Indian subcontinent, due to social stigma, it is usually not possible to measure the amount of alcohol intake in an individual. Segi et al demonstrated that heavy drinkers suffered most from stomach cancer as compared to controls (Segi et al, 1957), which, on the other hand, were not supported by the prospective study performed by Hirayama(Hirayama 1971). Wynder et al in their studies conducted in Japan and other three countries as well as Gajalakshmi et al in India demoed no significant relation of either type or quantity of alcohol consumption with the gastric cancer as compared to control (Wynder et al, 2005; Gajalakshmi et al, 1996). On the other hand, in a case control study, performed in Chennai, South India, alcohol consumption were demoed as significant risk factor (Sumathi et al, 2005) and few other studies showed it as weak risk factors (Pisters et al, 2005; Correa et al, 2005; La Torre et al, 2005). But in our study, alcohol consumers were detected as significant sufferers from gastric cancer than non-consumers.

In the country of heavy consumption of alcohol in the world, Italy, heavy consumers of wine and liquor suffered from gastric carcinoma, but, there is no association observed with other and other beverages consumers, which was similar to the studies performed in Mexico as well as Portugal (Lopez-Carrillo et al, 1998; Falcao et al, 1994), but no association was found with spirits (Zickutte et al, 2005). Our study also demonstrated strong association of heavy, long time drinkers and early starters with gastric cancer.

Several mechanisms were proposed in the pathogenesis of gastric cancer in relation to alcohol. The principal ingredient of alcohol beverages is carcinogenic because of the following reasons (Lachenmeier et al, 2009): Firstly, many oxygen species and oxidative stress induced by ethanol damage DNA and also affect their repair. Secondly, in case of heavy alcohol consumers, induced cytochrome 450 2E1 (CYP2E1) in various in the body affect conversion of procarcinogens (present in alcoholic beverages, tobacco smoke, diet) into carcinogens (Seitz et al, 2007; Purohit et al, 2005). Thirdly, ethanol acts as solvent for these carcinogens and aids in entering into the cells in the stomach mucosa to produce their direct toxic effects (Seitz et al, 2007). Fourthly, in the body, alcohol will be converted into acetaldehyde by alcohol with the help of alcohol dehydrogenase and CYP2E1, which, in turn, starts point mutation by inducing sister-chromatid exchanges, impairs DNA repair, induces epithelial metaplasia and forms mutagenic adducts with DNA (Purohit et al 2005).

CONCLUSION

Alcoholics were mostly affected from gastric cancer. Wine and liquor showed strong association than beer and other beverages in the genesis of gastric cancer. Early beginners of...
alcohol, heavy and long time consumers were suffered most from gastric cancer.

References