JUNK FOOD ASSOCIATION WITH THE MORPHOLOGICAL CHANGES OF GASTRITIS-A CROSS-SECTIONAL STUDY AMONG RURAL CHILDREN OF MELMARUVATHUR

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ABSTRACT

BACKGROUND AND AIM

Junk food consumption is common among children that have much adverse effect on the growth of the children and health. This study is aimed to assess the correlation of frequency of junk food intake with the morphological changes of gastric antral biopsies in dyspeptic children.

MATERIALS AND METHODS

This cross-sectional observational study was carried out in Melmaruvathur Medical College Hospital, Melmaruvathur, during the year 2014-2015. The participants were dyspeptic children between 10-15 years of age. They were interviewed using various junk food frequency questionnaires and antral biopsy was taken for histopathological assessment. The morphological changes of gastric antral biopsies were recorded and their association with junk food was analysed.

RESULTS

Out of 37 children studied, the predominant age group affected were between 5-10 years (56.8%) with female predominance (67%). Among the frequency, daily usage of junk food constitutes 57% of cases and the remaining 43% were taking intermittently. Biscuits, chocolates, packed chips were regularly used by daily user and bottled drinks, ice cream were used intermittently. Morphological assessment of gastritis showed significant association of junk food intake with increased intensity of mononuclear cell infiltration with a P value of 0.05, presence of Helicobacter pylori with a P value of 0.02 and presence of regenerative atypical changes with a P value of 0.006.

CONCLUSION

There is a significant association between the junk food intake and the severity of gastritis. The intensity of inflammatory changes, regenerative atypical glands and Helicobacter pylori presence and load were more among those having the habit of daily junk food intake than those taking intermittently.

KEYWORDS

Junk Food, Fast Food, Gastritis, H. Pylori, Dyspepsia in Children.

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INTRODUCTION: Junk foods are energy dense foods with high sugar, salt, fat content and low nutritive value of protein, fibre, vitamins and minerals.(1) Consumption of junk food is an increasing trend nowadays especially among growing children who are the investments of future community. The potential consequences of excessive consumption of junk food are both the nutritional deficiencies due to lack of essential nutrients, early satiety and obesity due to high-positive energy balance.(2) It is also well known that dietary factors play an important role in most of the gastrointestinal pathology. Gastritis is one such disease characterised by inflammation of gastric mucosa.(3) The introduction and universal use of endoscopy and targeted biopsy has increased the documentation of gastritis.(4) Chronic pain abdomen and dyspepsia is one of the common complaint among paediatric population and various studies shows the presence of gastritis as well as Helicobacter pylori by biopsy among dyspeptic children. Both the diet and Helicobacter pylori are the risk factors for gastric cancer(4,5) and the density of Helicobacter pylori is more in antrum due to its alkaline PH. We have attempted to assess the morphological changes of gastritis according to Sydney system in antral biopsy alone among dyspeptic children.(6) The aim of this study is to assess the frequency and type of junk food intake among the dyspeptic children and the morphological changes of gastritis by histopathological findings in relation to junk food consumption.

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MATERIALS AND METHODS: This cross-sectional study was carried out in Melmaruvathur Medical College Hospital, Tamilnadu, during the year 2014-2015. The inclusion criteria were children between the age group of 5 to 15 years presenting with symptoms of chronic dyspepsia for more than one month duration. Exclusion criteria were children with a history of antibiotics, proton pump inhibitors and other drugs like steroids intake over the past month. Questionnaire was prepared regarding the type of junk food used like biscuits, chocolates, packed chips, fryums, bottled drinks, ice cream, noodles, cakes, their quantity and frequency. In addition to the routine clinical and laboratory examination, all the children were subjected to upper gastroduodenal endoscopy after getting proper consent. Antral mucosal biopsy was taken and sent for histopathological examination. Haematoxylin and eosin stain was done to assess histopathologic changes and Giemsa staining was done to identify the presence of Helicobacter pylori and its density. All biopsy sections were viewed by single pathologist for avoiding subjective variation. The histopathological findings of the gastric mucosa in chronic gastritis were graded based on the Sydney system of grading of gastritis, which includes five criteria namely chronic inflammation by mononuclear cell infiltration, polymorphonuclear cell infiltration to assess the activity, glandular atrophy, intestinal metaplasia and presence of Helicobacter pylori. In addition to that, we have recorded other associated findings such as regenerative atypia and mucosal erosion.

Mononuclear cell infiltration, polymorphonuclear cell infiltration, regenerative atypia, Helicobacter pylori load were graded into mild, moderate, severe based on the extent and intensity of involvement as follows;

1. **Mononuclear cell infiltration - Intensity and extent:**
   - Mild (+) - Mononuclear cell infiltration 6-10/HPF; Less than 1/3 of tissue involvement.
   - Moderate (++) - Mononuclear cell infiltration 10-20/HPF; 1/3 to 2/3 of tissue involvement.
   - Severe (+++) - Mononuclear cell infiltration >20/HPF; More than 2/3 of tissue involvement.

2. **Polymorphonuclear cell infiltration - Intensity and extent:**
   - Mild (+) - Scattered in the lamina propria only.
   - Moderate (++) - Many neutrophils infiltrating few gastric pits.
   - Severe (+++) - Many neutrophils forming clusters infiltrating majority of gastric pits.

3. **Regenerative atypia - Intensity and extent:**
   - Mild (+) - Mild atypical nucleus involving less than 1/3 of total glandular tissue received.
   - Moderate (++) - Moderate atypical nucleus involving 1/3 to 2/3 of total glandular tissue received.
   - Severe (+++) - Severe atypical nucleus involving more than 2/3 of total glandular tissue received.

4. **Helicobacter Pylori - Intensity:**
   - Mild (+) - Rare to scattered.
   - Moderate (++) - few small clusters in few fields.
   - Severe (+++) - Many small clusters in many fields.

The other findings like glandular atrophy, intestinal metaplasia, erosion were recorded as either present or absent. All these histopathological findings were correlated with frequency of junk food consumption and statistically analysed.

RESULTS AND OBSERVATION: Among the 37 children studied, 56.8% were between the age group of 5-10 years and 43.2% were between 11-15 years with a female predominance (Table-1). The frequency of various types of junk food intake showed daily consumption of junk food were by 57% of cases and the remaining 43% were taking intermittently (Figure-1). The predominant type of junk food used daily were biscuits, chocolates and packed chips in descending order whereas bottled drinks, ice cream were taken intermittently. Morphological grading of gastritis showed 100% involvement of chronic gastritis with activity by mononuclear cells, neutrophils infiltration of varying intensity along with lymphoid follicles (Figure 3,4). Regenerative atypia was observed in 94.6% of cases and 81.1% were found to be Helicobacter pylori positive in biopsy (Table-2). The presence of Helicobacter pylori infection was more common and the load was also high among those who were taking junk food daily (Figure-2). Helicobacter pylori were seen as small rod-shaped structure distributed in glandular lumen and surface. Even occasional scattered forms of Helicobacter pylori, which was not well-appreciated in Haematoxylin and Eosin stain in our study were also picked up nicely by Giemsa stain (Figure 5). Regenerative atypical glands showed glandular epithelial cells with hyperchromatic, anisokaryotic nucleus and altered polarity in contrast with small basally located nucleus of normal mucous glands (Figure 6). Chi-square statistical analysis of junk food intake frequency in association with the gastritis grading was done. It showed significant correlation of daily intake of junk food with increased intensity of mononuclear cell infiltration, presence of Helicobacter pylori and regenerative atypical glands with a P value of 0.05, 0.02 and 0.006, respectively (Table 3). Intestinal metaplasia was not appreciated in all cases and glandular destruction & atrophy of glands was seen in 23.8% of daily junk food users and 12.5% of intermittent users.

Age * Sex					
			Female	**Male**	**Total**
Age	Up To 10 Years (5-10) Years	Count	14	7	21
		%	56.0%	58.3%	56.8%
	11-15 years	Count	11	5	16
		%	44.0%	41.7%	43.2%
	Total	Count	25	12	37
		%	100.0%	100.0%	100.0%

Table 1: Age, Sex Distribution

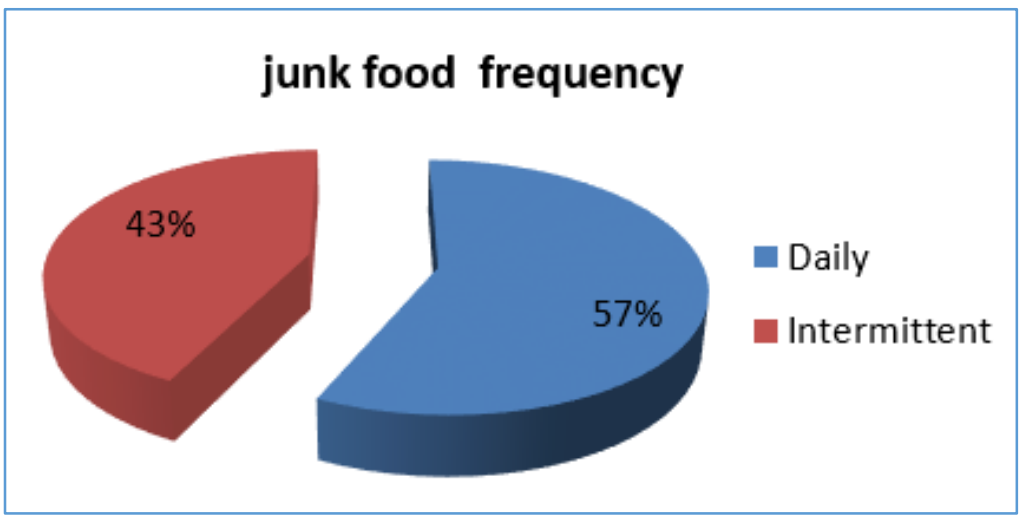

Figure 1: Frequency of Junk Food Intake

Morphological changes of gastritis	Grade	Number of patients	Percentage
Mononuclear cell infiltration	+	0	0
	++	21	56.8%
	+++	16	43.2%
Neutrophil infiltration - Extent	+	10	27.0%
	++	17	45.9%
	+++	10	27.0%
Neutrophil infiltration - Intensity	+	29	78.4%
	++	7	18.9%
	+++	1	2.7%
Helicobacter pylori	+	27	73.0%
	++	3	8.1%
	Absent	7	18.9%
Erosion	Present	36	97.3%
	Absent	1	2.7%
Glandular atrophy	Present	7	18.9%
	Absent	30	81.1%
Regenerative atypia - Extent	+	15	40.5%
	++	20	54.1%
	Absent	2	2.4%
Regenerative atypia - Intensity	+	32	86.5%
	++	3	8.1%
Intestinal metaplasia	Absent	37	100%

Table 2: Biopsy Grading of Gastritis Changes

Morphological changes of gastritis		Junk food in take					
		Daily		Intermittent			
		Count	Column N %	Count	Column N %	Chi-square	P value
Mononuclear cell infiltration - Extent	+++	21	100.00%	16	100.00%	-	-
Mononuclear cell infiltration - Intensity	++	9	42.90%	12	75.00%	3.824*	0.05
	+++	12	57.10%	4	25.00%		
Neutrophil infiltration - Extent	+	6	28.60%	4	25.00%	0.258	0.879
	++	10	47.60%	7	43.80%		
	+++	5	23.80%	5	31.20%		
Neutrophil infiltration - Intensity	+	17	81.00%	12	75.00%	1.354	0.508
	++	3	14.30%	4	25.00%		
	+++	1	4.80%	0	0.00%		
Helicobacter pylori	-	1	4.80%	6	37.50%	7.854*	0.02
	+	17	81.00%	10	62.50%		
	++	3	14.30%	0	0.00%		
Erosion	-	0	0.00%	1	6.20%	1.349	0.245
	+	21	100.00%	15	93.80%		
Glandular atrophy	-	16	76.20%	14	87.50%	0.757	0.384
	+	5	23.80%	2	12.50%		
Regenerative atypia - Extent	-	0	0.00%	2	12.50%	10.381*	0.006
	+	5	23.80%	10	62.50%		
	++	16	76.20%	4	25.00%		
Regenerative atypia - Intensity	+	19	90.50%	13	81.25%	2.834	0.242
	++	2	9.50%	1	6.25%		
Intestinal metaplasia	-	-	-	-	-		
* The chi-square statistic is significant at the 0.05 level							

Table 3: Association of Junk Food with Morphological Changes of Gastritis

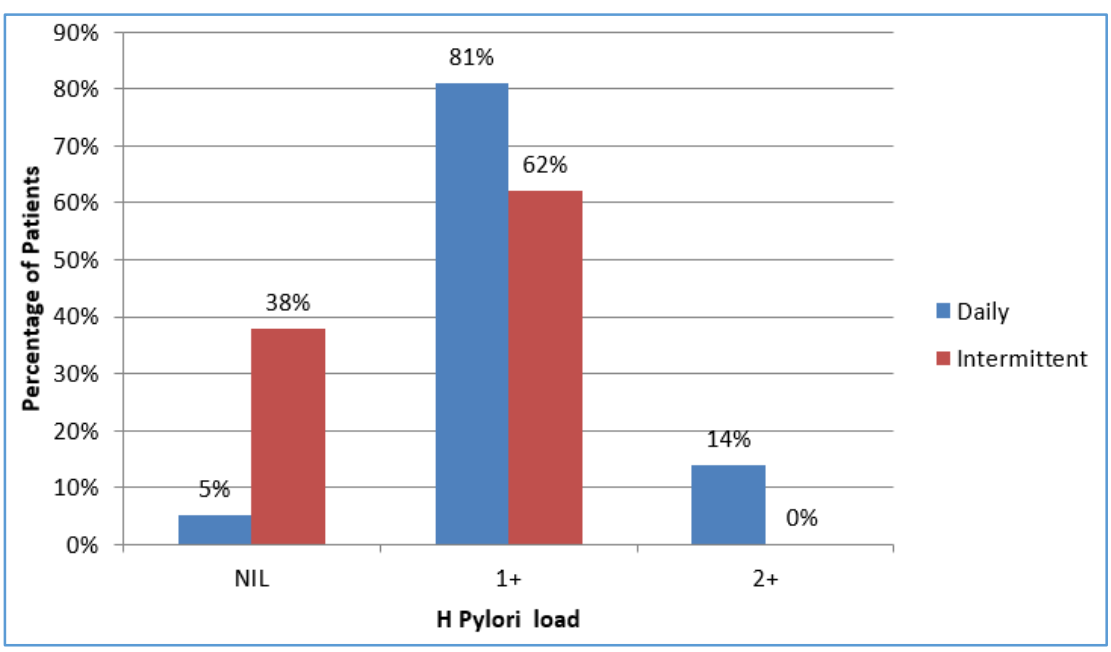

Figure 2: Association of Junk Food with Helicobacter Pylori

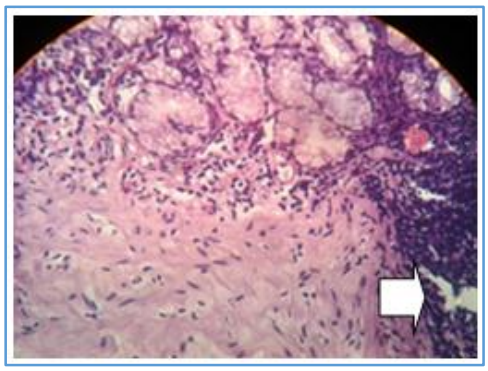

Figure 3: Photomicrograph showing eosinophil, Neutrophil, Plasma Cell Infiltration in the Lamina Propria. Haematoxylin and Eosin x100. Arrow Points Out Lymphoid Follicle

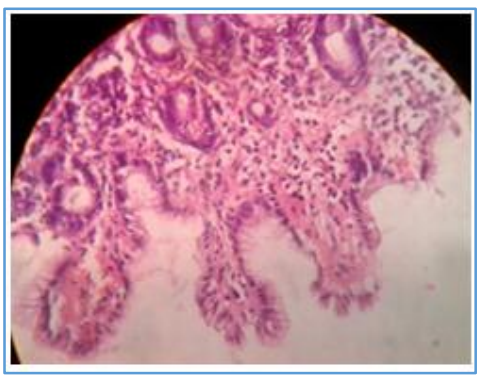

Figure 4: Photomicrograph showing Surface Erosion, Haemorrhage and Neutrophils, Plasma Cells, Lymphocytes in Gastric Pits. Hematoxylin and Eosin x100

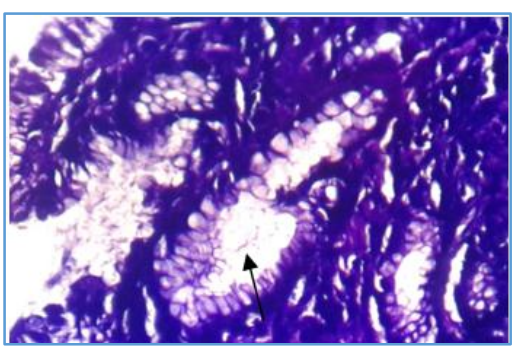

Figure 5: Photomicrograph showing Helicobacter Pylori Over the Luminal Surface (Arrow) Giemsa Stain x400

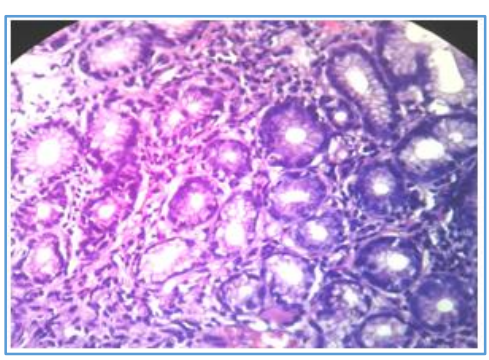

Figure 6: Photomicrograph shows Benign Antral Mucosal Glands (Left) and Reparative Atypical Glands (Right). Haematoxylin and Eosin x400

DISCUSSION: Nowadays, junk food occupies an important part of dietary menu especially among children because of its ready availability and attracted advertisements. Various studies have been done to assess the association of junk food with the growth, nutritional status, mental status and overweight among children.[2,7,8,9,10] Dietary factors also play a role in inducing gastritis due to microbiological contamination as a result of poor hygiene during preparation and storage and many studies were done for assessing dietary factors association with Helicobacter pylori gastritis. One study at Oman showed increased association of soft drinks and Helicobacter pylori infection with a P value of 0.04.[11] Our study also showed increased association of Helicobacter pylori gastritis (81%) with junk food among children with a P value of 0.02. This is also supported by Seyyed Ali et al study who had got a P value of 0.002, 0.001 for burgers and soft drinks associated Helicobacter pylori gastritis, respectively.[5] One study by Davies et al stated that there was a positive association between mucosal reactive oxygen metabolite production, which induces DNA damage and histological (P=0.002) quantitative assessment of Helicobacter pylori.[12] One reason for this increased reactive oxygen metabolites in Helicobacter infection is due to stimulation of macrophage system through L-arginine/nitric oxide pathway that increases endogenous NO formation. Moreover, pathogenic strain of Helicobacter pylori also induces gastric epithelial cells to produce interleukin-B, which is a chemotactic factor for neutrophils. The production of reactive oxygen species by this activated neutrophils also contributed to this increased reactive metabolite production in Helicobacter pylori infection. But, Ladeira et al reported that DNA damage was associated with an increased intensity of inflammation rather than the increased concentration of Helicobacter pylori.[13] Our study shows that all dyspeptic children have taken junk food and morphological findings of chronic gastritis with activity (100%) was appreciated in all antral biopsies. This shows that there is a correlation between junk food and the intensity of inflammatory changes irrespective of the bacterial presence. This may be due to gastric injury caused by chemical constituents of junk food especially increased salt, carbohydrate content, presence of preservative and chemical additives in addition to the microbiological factors, which damage the gastric mucosal barrier. This is supported by presence of gastric erosion in all cases except in one case in our study. Intestinal metaplasia was not appreciated in our study whereas Gisele et al got these findings in 35% of H. pylori positive cases of their study.[14] Their study also showed regenerative cell atypical findings in 68.7% of the biopsy specimens whereas regenerative cell atypia was observed in 94.6% of our cases. Cases have been reported stating the association between regenerative atypia and intestinal metaplasia in the evolution of gastric carcinoma. The increased incidence of these regenerative atypical changes predominantly of mild degree among junk food user in our study is an alarming finding that has to be taken significantly. Though regenerative atypical changes are reversible, repetitive injury and regeneration increases the chances of getting

mutation and promote the transition of a normal cell into a mutant cell, which is the basis for any carcinogenesis. This regenerative changes occur following glandular destruction by inflammatory mediators especially enzymes and reactive oxygen species. Though Helicobacter pylori induce epithelial and inflammatory cells to produce these mediators, the primary initiating event is the gastric injury following the destruction of mucosal barrier. So, a dietary factor especially junk food play an initiative role in gastritis changes. Since, persistent regenerative atypia may become the precancerous lesion later, authors suggesting inclusion of this reparative atypical changes also as one of the criteria for assessing the grade of gastritis.

However, the authors accepting the limitations of this study because of the small sample size and selection bias of assessing the biopsy changes of dyspeptic children alone. The authors also suggesting to carry out this as an observational cohort study for large sample so that the carcinogenic potential of junk food and morphological fluctuations in gastric biopsies can be evaluated.

CONCLUSION: In summary, the present study on junk food consumption and its interaction with the gastric morphological changes showed the frequency of junk food intake among children was high and 57% of children were taking daily and 43% were taking intermittently. Biscuits, chocolates, packed chips were daily used and bottled drinks, ice cream were intermittently used by children. Almost, all the children studied had the habit of junk food intake and all showed features of chronic gastritis with activity. The intensity of morphological changes, presence of Helicobacter pylori, their load, regenerative atypical changes of glandular cells were increased among daily junk food user than intermittent user.

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