

# Oral and Dental Manifestations of Gastroesophageal Reflux Disease in Children: A Preliminary Study

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#### Abstract

Purpose: The aim of this study was to investigate the effects of gastroeophageal reflux disease (GERD) on: (1) erosion; (2) caries formation; (3) salivary function; and (4) salivary microbiological counts.

Methods: Thirty-eight GERD patients with a mean age of 6½ years and 42 healthy children of the same age and gender and social background comprised the study group. All subjects answered a detailed frequency questionnaire related to acidic drinks, foods, and sugar consumption and participated in a clinical dental examination. The caries experience of the children was recorded according to World Health Organization criteria, and erosion was scored according to the Eccles and Jenkins grading scale. The children were also investigated for stimulated salivary flow rate, buffer capacity, and salivary mutans streptococci (MS), lactobacilli, and yeast colonization.

Results: The prevalence of dental erosion and the salivary yeast and MS colonization in GERD children was found to be significantly higher than for healthy subjects (P<.05). The caries experience, salivary flow rate, buffering capacities of the children, and frequency of acidic drinks, foods, and sugar consumption were found to be similar in both groups. Conclusion: This current investigation has shown that GERD children were at an increased risk of developing erosion and caries compared with healthy subjects. (Pediatr Dent 2006;28:279-284)

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as the involuntary passage of gastric contents into the esophagus. 1,2 Symptoms include pyrosis, heartburn, chest pain, hoarseness, asthma, recurrent pneumonia, chronic cough, otitis media, reflux laryngitis, and sore throat. 3 The most important GERD symptom from the dental perspective is regurgitation, which is the appearance of gastric juice in the mouth following a reflux episode. 1

In children with suspected GERD, a clinical history is essential to establish the nature of symptoms and associated respiratory or failure to thrive manifestations. Diagnosis by extended esophageal pH monitoring over 18 to 24 hours is commonly performed.<sup>4</sup> Endoscopy is useful for examining the esophagus for macroscopic signs of inflammation followed by histologic confirmation.<sup>2</sup>

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This disease is particularly interesting from the dental point of view because the acidity of gastric contents may be below pH 1. Thus, regurgitation can be strongly detrimental to the teeth by causing dental erosion.5 Dental erosion was defined by Pindborg<sup>6</sup> as the superficial loss of hard tissues of the teeth by a chemical process that does not involve the action of bacteria. It was first associated with GERD in the reports of Bodecker,7 Bargen and Austin,8 and Holst and Lange,9 and a number of articles have suggested a relationship between dental erosion and gastroesophageal disturbances. 10-13 It first begins on the palatal surfaces of the maxillary teeth, which are relatively remote from the major salivary glands. The tongue may also be involved by maintaining contact of the gastric juice against the teeth's palatal surfaces. The lower teeth are not affected in the early stage, as the tongue provides some protection. In more severe cases, however, the erosion pattern may be more widespread.1

Salivary flow rates and pH are believed to exert an important influence on the occurrence and severity of erosion as well as caries. <sup>14,15</sup> Since dental erosion is often a multifactorial phenomenon, the additional insult of poor buffering capacity in a GERD patient is likely to cause more severe and quickly occurring erosion lesions than would otherwise be the case. <sup>16,17</sup>

The association between dental erosion and GERD has been widely accepted in the literature. 18-21 There are doubts, however, about the possibility of a direct influence of the gastric acids in the mouth and about their relationship with caries formation, salivary flow, buffer capacity, and micro-organism colonization. This study's aim was to investigate GERD's effects on erosion, caries formation, salivary function, and salivary microbiological counts compared to healthy controls.

# Methods

# Study population

This study was approved by the Ethical Committee of Ege University, Bornova-Izmir, Turkey, and written informed consent was obtained from each parent. Thirty-eight patients (19 males, 19 females) were selected from pediatric gastroenterology patients in the Department of Hepatology and Nutrition, Ege University, whose GERD diagnoses were firmly established with long-term esophageal pH recordings. The selection criteria also included not taking antibiotics or antimicrobial agents in the previous 3 months. A detailed medical history was obtained from the parent. Gastrointestinal symptoms included recurrent abdominal pain, regurgitation, vomiting, pyrosis, and eructation.

#### Control subjects

Forty-two healthy children of the same age and gender and from similar socioeconomic backgrounds were also invited to attend the dental examination and be included in the study as the control group (21 girls and 21 boys).

#### Dental and dietary histories

Details of each child's dental history were obtained from the parent regarding dental treatment, systemic fluoride exposure, and oral hygiene habits.

Dietary history and dietary analysis were recorded for each subject to eliminate the possibility of dietary causes for the erosion. This diet analysis was examined for the number of times per week each child consumed sugar and the number of acid exposures per week. Acid exposures included for analysis were those foods and drinks known to have a pH<5, such as fruit juices, carbonated soft drinks, and citrus fruits. Other types of foods were also included, such as apples, oranges, bananas, grapes, tomato ketchup, and yogurt. The amount of consumption of drinks, foods, and fruits per week were categorized into 4 groups: (1) no consumption at all; (2) low consumption (1-7 times/week); (3) medium consumption (8-21 times/week); and (4) high consumption ( $\geq$ 22 times/week).

# Dental examination

The caries experience and the presence of erosion and its degree were recorded by 2 calibrated examiners in the Pediatric Clinic at the University of Ege using dental mirrors and explorers under daylight. Training and calibration exercises were conducted on 20 children 4 to 6 years old prior to the study

by the same 2 examiners. Results yielded a kappa value of 0.95 for intraexaminer reproducibility for surfaces diagnosed as carious and 0.90 for surfaces diagnosed as having erosion. Wear confined to incisal surfaces was excluded.

Caries was recorded using World Health Organization criteria.<sup>23</sup> Erosion was charted using the Eccles and Jenkins index<sup>24</sup> for classification of tooth erosion caused by GERD. In this index:

- 1. grade 0=no erosion present;
- 2. grade 1=loss of surface detail, with change confined to the enamel;
- 3. grade 2=exposure of dentin affecting less than one third of the crown; and
- 4. grade 3=exposure of dentin affecting one third or more of the crown.

When a subject had different grades of erosion on different teeth, the highest grade of erosion was recorded for that subject.

#### Saliva analysis

Saliva assessment included salivary flow rate, salivary buffer capacity, mutans streptococci (MS), lactobacilli, and yeast counting. Whole stimulated saliva samples were collected between 9 a.m. and 11 a.m. to control for circadian rhythms. The individuals were instructed not to eat for at least 2 hours before collection. A stimulated saliva sample was collected by asking the children to chew a standard piece of paraffin wax. Collection was started after chewing for 30 seconds. Salivary flow rate was recorded as milliliters per minute after a 5-minute collection period. Buffering capacity was immediately assessed from the stimulated saliva by using the CRT buffer test (CRT buffer test, Vivadent, Lichtenstein).

The results were classified according to the manufacturer's instruction as: (1) low (pH ≤4); (2) normal (pH 4.5-5.5); and (3) high (pH ≥6). For MS and lactobacilli counting, sampled saliva was immediately spread on both sides of the plastic strip by using a pipette from a commercially available test kid (CRT bacteria, Vivadent, Lichtenstein), according to the manufacturer's instructions. A tablet containing NaHCO3 was placed at the bottom of the collection vial. After incubation for 48 hours at 37°C, the samples were examined. Standard charts, supplied by the manufacturer for estimation of colony-forming units of bacteria, were used to read results. For yeast cultivation, the samples were inoculated onto Sabouraud's glucose agar and incubated for 72 h at 37°C. The yeast colonies were counted and then identified by using the germ-tube test, ricemeal-Tween 80 chamidospore test, and API Candida (Biomeriux SA, Marcy-l'etoile, France) carbohydrate assimilation test. For final analyses, the yeast counts were recorded as positive vs negative.

# Statistical analysis

Mann-Whitney and Spearman rank correlation tests were used for the comparisons and correlations between groups. Chi-square or the Fisher exact test was used for the analysis of the categorical variables. Logistic regression models were

		· ( ap)			34 (CD) P	Salivary buffering capacity n (%)		
	n	Mean (±SD) age (ys)	Girls	Boys	Mean (±SD) salivary flow rate (mL/min)	High	Medium	Low
GERD subjects	38	6.5±3.6	19	19	0.70±0.5	25 (66%)	7 (18%)	6 (16%)
Control subjects	42	6.9±2.8	21	21	0.84±0.5	33 (79%)	8 (19%)	1 (2%)
Toral	80	67+32	40	40	0.78±0.5	58	15	77

used to assess and estimate the odds ratios of caries experience, erosion, and salivary parameters. The statistical level of significance was set at P<.05.

#### Results

# Demographics

The age and sex distribution of the 80 subjects were shown in Table 1. The most common gastrointestinal symptoms were regurgitation (16%) and vomiting (11%) in the GERD patients. The past dental treatments, systemic fluoride exposures, and oral hygiene habits were also similar in both groups.

#### Saliva analysis

Three GERD group children and 1 control group child were unable to provide a saliva sample for salivary flow rate and yeast counting. The difference in mean stimulated salivary flow rates between the 2 groups was not statistically significant. The buffering capacities were also similar in both groups (Table 1).

## Erosion prevalence

When the overall number of teeth affected by erosion between the 2 groups was considered, there were significantly more teeth in the GERD group showing erosion than in controls. In the GERD group:

- 1. 19% of the primary teeth (120/603) were affected compared to only 5% in the control group (34/664; P<.05); and
- 2. 10% of the permanent teeth (20/200) were affected compared to 2% (6/264) of the permanent teeth affected in the control group (P<.05).

In both the primary and permanent dentition there were significant differences between the number of affected teeth in the GERD and control groups.

When numbers of affected subjects were considered, significant differences were observed in the prevalence of erosion between the groups. In the GERD group, 76% of the children had erosion, while 24% of the control group subjects had erosion (P<.05).

#### Erosion severity

Teeth affected by erosion in the GERD group showed more severe erosion compared to the teeth in the control group. There were greater numbers of teeth with grade 2 and grade 3 erosion in the GERD group compared to teeth in the control group, as shown in Table 2 (37% vs 5%; P<.05).

# Caries prevalence

The number of caries-free subjects in the GERD group was similar to the control group (29% vs 26%) and the difference was not statistically significant. When the total number of decayed, missing, or filled teeth (defs/ DMFS) was examined, a significant difference was found between GERD and control subjects when F scores were compared. GERD subjects had significantly more permanent tooth surfaces filled due to caries compared to control subjects (Table 2).

# Prevalence of MS and lactobacilli and yeast and their associations with erosion

There was a significantly higher percentage of subjects with ≥105 MS CFU/ml of saliva in the GERD group (32 children; 84%) compared to the control group (17 children; 41%; P<.05). Erosion was not found more commonly in the subjects with ≥105 MS CFU/ml of saliva than subjects with <105 MS CFU/ml of saliva within the GERD group and the control group (Table 3). The prevalence of lactobacilli was similar in both groups, and erosion was not found more commonly in subjects with ≥105 lactobacilli CFU/ml of saliva than subjects with <105 lactobacilli CFU/ml of sa-

Table 2. The Prevalence of Erosion, Severe Erosion (Grades 2 and 3), and Caries Prevalence in Children With Gastroesophageal Reflux Disease (GERD) Compared to Control Subjects							
	n	Erosion prevalence n (%)	Severe erosion (%)	Caries prevalence n (%)	dfs (mean±SD)	DMFS (mean±SD)	F (mean±SD)
GERD subjects	38	29 (76%)* P<.0001	37* P<.05	27 (71%)* P=.020	5.6±6.9	0.8±1.8	0.45±1.35* P<.0001
Control subjects	42	10 (24%)	5	31 (74%)	4.1±5.5	0.4±0.8	0.02±0.2

<sup>\*</sup>Shows different significant results.

Table 3. Associations of Salivary Mutans Streptococci, Lactobacilli, and Yeast and Erosion in Gastroesophageal Reflux Disease (GERD) Subjects Compared to Controls

		GERD n (%)			Control n (%)		
		Erosion	No erosion	Total	Erosion	No erosion	Total
Mutans streptococci (CFU/ml)	≥10 <sup>5</sup>	25 (86%)	7 (78%)	32* (84%)	4 (40%)	13 (41%)	17 (41%)
	<105	4 (14%)	2 (22%)	6 (16%)	6 (60%)	19 (59%)	25 (59%)
Lactobacilli	≥10 <sup>5</sup>	19 (66%)	5 (56%)	24 (63%)	3 (30%)	18 (56%)	21 (50%)
	<105	10 (34%)	4 (44%)	14 (37%)	7 (70%)	14 (44%)	21 (50%)
	(+)	5 (19%)	2 (25%)	7† (20%)	0	2 (6%)	2 (5%)
Yeast	(-)	22 (82%)	6 (75%)	28 (80%)	9 (100%)	30 (94%)	39 (96%)

<sup>\*</sup>P<.0001; †P=.046.

liva in both groups. The presence of yeast was significantly higher in the GERD group compared to the control group, but there was no association between the presence of yeast and erosion in either group.

# Association of the oral microflora with caries

As shown in Table 4, when the GERD subjects with MS and lactobacilli counts of greater than 10<sup>5</sup> CFU/ml saliva were examined, there was a positive correlation between this level of MS, lactobacilli, and caries experience (P<.05). No correlation, however, could be demonstrated in the control group. In addition, there was no correlation between erosion and caries in either group (Table 5).

# Dietary sugar and acid exposure

When the children's diets were analyzed, no difference was found in the mean number of sugar exposures per day between GERD subjects and controls.

Similarly, no difference was found in the mean number of acid exposures. The highest rates of drink consumption per week (22 or more) were seen with drinks such as orange juice and cola.

According to the logistic regression, any child with erosion was at lower risk for caries (OR <1), though this difference was not statistically significant. Low salivary flow rate was protective against caries (OR <1), though this, too, was not statistically significant. This finding is also at odds with the known protective effects of saliva.

# Discussion

In spite of its common occurrence, there is little reported in the literature on the oral health of GERD children. <sup>25</sup> The majority of published reports deal only with the prevalence of dental erosion. Thus, the present study examined the caries experience, salivary flow rate, and buffer capacity and the salivary levels of MS, lactobacilli, and yeast in a group of GERD subjects compared to healthy controls.

This study's results showed that, compared to healthy children, GERD subjects had more dental erosion, which presumably was directly related to the reflux. The prevalence of 76% of GERD subjects showing erosion in the present study was higher than reported by O'Sullivan et al<sup>26</sup> and Meurmann et al,<sup>5</sup> but similar to that found in the study of Aine et al (87%). The difference in results among the studies may be due to differences in age and sample sizes. In the study of O'Sullivan et al,<sup>26</sup> the children were relatively young, with a mean age of only 4.9 years. In the study of Aine et al,<sup>18</sup> the children were of similar ages to those of the present study, but their sample size of only 17 was considerably smaller. The relative lengths of time teeth were exposed to gastric acid may also account for the differences in severity noted in the different studies.

According to Hellström<sup>27</sup> and Ruff et al,<sup>28</sup> it is highly likely that erosive lesions become clinically evident only after a period of gastric acid exposure of several times a week for at least 1 to 2 years. It is not known, however, whether the gastric contents reach the mouth and to what extent GERD

Table 4. Associations of Salivary Mutans Streptococci, Lactobacilli, and Yeast and Caries in Gastroesophageal Reflux Disease (GERD) Subjects Compared to Controls

	Mutans strep		Lactobac		Yeast n (%)	
GERD	≥10 <sup>5</sup>	<105	≥105	<105	(+)	(-)
Caries	25*(78%)	2 (33%)	23† (96%)	4 (29%)	6 (86%)	20 (71%)
Caries free	7 (22%)	4 (67%)	1 (4%)	10 (71%)	1 (14%)	8 (29%)
Control	≥10 <sup>5</sup>	<105	≥10 <sup>5</sup>	<10 <sup>5</sup>	(+)	(-)
Caries	11w (65%)	20 (80%)	18 (86%)	13 (62%)	2 (100%)	28 (72%)
Caries free	6 (35%)	5 (20%)	3 (14%)	8 (38%)	0	11 (28%)

<sup>\*</sup>P=.047; †P<.0001.

causes a decrease of oral pH. In this regard, salivary factors such as flow rate and buffering capacity may affect the influence of acid on the teeth. <sup>29</sup> Saliva buffering capacity has been suggested to be low in patients with dental erosion. <sup>16,29</sup> Moazzez et al<sup>12</sup> also found poor buffering capacity of saliva in GERD patients in conjunction with the higher prevalence of erosion. In this study, the number of patients who had low buffering capacity in the GERD group was higher compared to the healthy group, but it was not statistically significant.

There are also controversial results about the relationship between salivary flow rate and erosion. In one study, it was shown that an unstimulated salivary flow of less than 0.1 mL/min was associated with a 5 times increased risk of erosion. The Astudy by Wöltgens et al had also shown that dental erosion may be associated with a reduced saliva flow. Other studies, however, did not find any correlation between salivary flow rate value and erosions. The Sonnerberg et al also found no difference in resting saliva volume between GERD patients and controls. In the present study, no significant difference in salivary flow rate was found between GERD and control subjects, but the salivary flow rate was found to impact the presence of erosion.

In addition to the erosive effects of stomach acid on the teeth, it may be postulated that the acidic oral environment induced by GERD is likely to encourage the growth of the acidophilic *S mutans*, <sup>35,34</sup> which is in accordance with this study's results. In this study, the prevalence of high MS counts was statistically higher in GERD children. Linnett et al<sup>35</sup> also observed that there were more subjects with *S mutans* in the GERD group, but the difference in comparison with the control group was not statistically significant. O'Sullivan and Curzon<sup>29</sup> also found significant differences in *S mutans* counts between children with erosion and a control group.

In this study, although the presence of yeast was statistically higher in the GERD group, the level of yeast colonization was low (GERD group=18%; control group=5%), with a predominance of *Candida albicans*. In the literature, the salivary level of yeast colonization seems to be extremely variable.<sup>36</sup> This variability might be attributed to different age groups studied and also depends on the country of origin. It has been reported that the lower the saliva flow, the more the fungal colonization.<sup>37</sup> The greater yeast colonization in GERD subjects could be explained by the altered salivary parameters, although it did not reach significant values. In the present study, no correlation was found between caries

and yeast and the role of oral fungal flora as a risk factor for dental caries was not clearly established.<sup>36</sup>

To date, there is little information available on the caries experience of GERD children. Linnett et al<sup>35</sup> found that GERD children have more dental caries compared to healthy controls. In the present study, the prevalence of caries was higher in both den-

titions, but they were not significantly different compared with the control group. The only significance was found in the filled surfaces of permanent dentition in GERD subjects.

# Conclusions

This current investigation—in which the effects of gastroesophageal reflux disease (GERD) was investigated on erosion, caries formation, salivary function, and salivary microbiological counts compared to healthy controls—showed that:

- Children with GERD are at increased risk of developing erosion compared with healthy subjects.
- 2. Although GERD children have no significant altered salivary parameters, higher salivary micro-organism colonization increases their susceptibility to caries.
- 3. If dental erosions are detected in asymptomatic children, they should be evaluated for GERD.

## References

- 1. Bartlett DW, Evans DF, Smith BG. The relationship between gastroesophageal reflux disease and dental erosion. J Oral Rehabil 1996;23:289-297.
- 2. Davies A, Sandhy B. Diagnosis and treatment of gastroesophageal reflux. Arch Dis Child 1995;73:82-86.
- 3. Deschner WK, Benjamin SB. Extraesophageal manifestations of gastroesophageal reflux disease. Am J Gastroenterol 1989;84:1-5.
- 4. Johnson L, DeMeester T. Twenty-four-hour pH monitoring of the distal esophagus. A quantitative measure of gastroesophageal reflux. Am J Gastroenterol 1974;62:325-332.
- 5. Meurman JH, Toskala J, Nuutinen P, Klemetti E. Oral and dental manifestations in gastroesophageal reflux disease. Oral Surg Oral Med Oral Pathol 1994;78: 583-589.
- 6. Pindborg JJ. Pathology of dental hard tissues. Copenhagen, Denmark: Munksgaard; 1970:312-325.
- 7. Bodecker CF. Dental erosion: its possible causes and treatment. Dent Cosmos 1933;75:1056-1062.
- 8. Bargen JA, Austin LT. Decalcification of teeth as a result of obstipation with long continued vomiting: report of a case. J Am Dent Assoc 1937;24:1271-1273.
- 9. Holst J, Lange F. Perimylolysis. A contribution towards the genesis of tooth wasting from non-mechanical causes. Acta Odontol Scand 1939;1:36-48.

Table 5. Association of Erosion and Caries in Gastroesophageal Reflux
Disease (GERD) Subjects Compared to Controls

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	Erosion (+)	Erosion (-)	Erosion (+)	Erosion (-)
Caries	21 (72%)	6 (67%)	6 (60%)	25 (78%)
Caries free	8 (28%)	3 (33%)	4 (40%)	7 (22%)

<sup>\*</sup>Spearman rank correlation test; P>.05.

- 10. Howden GF. Erosion as the presenting symptom in hernia. Br Dent J 1971;131:455.
- 11. Scheutzel P. Etiology of dental erosion: Intrinsic factors. Eur J Oral Sci 1996;104:178-190.
- 12. Moazzez R, Barlett D, Anggiansah A. Dental erosion, gastroesophageal reflux disease, and saliva: How are they related? J Dent 2004;32:489-494.
- 13. Dahshan A, Patel H, Delaney J, Wuerth A, Thomas R, Tolia V. Gastroesophageal reflux disease and dental erosion in children. J Pediatr 2002;140:474-478.
- 14. Jarvinen V, Meurman JH, Hyvarinen H, Rytomaa I, Murtomaa H. Dental erosion and upper gastrointestinal disorders. Oral Surg Oral Med Oral Pathol 1988;65:298-303.
- 15. Meurman JH, ten Cate JM. Pathogenesis and modifying factors of dental erosion. Eur J Oral Sci 1996;104:199-206.
- Gudmundsson K, Kristleifsson G, Theodors A, Holbrook WP. Tooth erosion, gastroesophageal reflux, and salivary buffering capacity. Oral Surg Oral Med Oral Pathol 1995;79:185-189.
- 17. Woltgens JH, Vingerling P, de Blieck-Hogervorst JM, Bervoets DJ. Enamel erosion and saliva. Clin Prev Dent 1985;7:8-10.
- 18. Aine L, Baer M, Maki M. Dental erosions caused by gastroesophageal reflux disease in children. J Dent Child 1993;60:210-214.
- 19. Bartlett DW, Evans DF, Anggiansah A, Smith BG. A study of the association between gastroesophageal reflux and palatal dental erosion. Br Dent J 1996;181:125-131.
- Bartlett DW, Evans DF, Anggiansah A, Smith BG. The role of esophagus in dental erosion. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89:312-315.
- 21. Gregory-Head BL, Curtis DA, Kim L, Cello J. Evaluation of dental erosion in patients with gastroesophageal reflux disease. J Prosthet Dent 2000;83:675-680.
- 22. Al-Dlaigan YH, Shaw L, Smith AJ. Is there a relationship between asthma and dental erosion? A case control study. Int J Paediatr Dent 2002;12:189-200.

- 23. World Health Organization. Oral Health Surveys: Basic Methods. 4th ed. Geneva, Switzerland: WHO; 1997.
- 24. Eccles JD, Jenkins WG. Dental erosion and diet. J Dent 1974;2:153-159.
- 25. Linnett V, Seow WK. Dental erosion in children: A literature review. Pediatr Dent 2000;23:37-43.
- 26. O'Sullivan EA, Curzon ME, Roberts GJ, Milla PJ, Stringer MD. Gastroesophageal reflux in children and its relationship to erosion of primary and permanent teeth. Eur J Oral Sci 1998;106:765-769.
- 27. Hellström I. Oral complications in anorexia nervosa. Scand J Dent Res 1977;85:71-86.
- 28. Ruff SG, Koch MO, Perkins S. Bulimia: Dentomedical complications. Gen Dent 1992;40:22-25.
- 29. O'Sullivan E, Curzon ME. Salivary factors affecting dental erosion in children. Caries Res 2000;34:82-87.
- 30. Jarvinen V, Rytomaa I, Heinonen O. Risk factors in dental erosion. J Dent Res 1991;70:942-947.
- 31. Wöltgens JHM, Vingerling P, de Blieck-Hogervorst JMA, Bervoets DJ. Enamel erosion and saliva. Clin Prev Dent 1985;7:8-10.
- 32. Sonnenberg A, Steinkamp U, Weise A, Berges W, Wienbeck M, Rohner HG. Salivary secretion in reflux esophagitis. Gastroenterology 1982;83:889-895.
- 33. Bretz W, Krahn D, Drewnowski A, Loesche W. Salivary levels of putative cariogenic organisms in patients with eating disorders. Oral Microbiol Immunol 1989;4:230-232.
- 34. Holtta P, Aine L, Maki M, Ruuska T. Mutans streptococcal serotypes in children with gastroesophageal reflux disease. J Dent Child 1997;63:201-204.
- 35. Linnett V, Seow WK, Connor F, Shepherd R. Oral health of children with gastroesophageal reflux disease: A controlled study. Austr Dent J 2002;47:156-162.
- 36. Moliac E, Gestalin A, Quinio D, Gest PE, Zerilli A, Le Flohic AM. The extent of oral fungal flora in 353 students and possible relationships with dental caries. Caries Res 2001;35:149-155.
- 37. Gergerly L, Uri J. Day-by-day variation in the mycotic flora of the mouth. Arch Oral Biol 1966;11:15-19.