

## DENTAL AND PERIODONTAL INVOLVEMENT IN PATIENTS WITH LARYNGOPHARYNGEAL REFLUX.

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### ABSTRACT

Little is known about the relationship between laryngopharyngeal reflux (LPR) and periodontal or dental lesions. This study investigates the association of dental and periodontal involvement in patients with LPR compared to a control group.

A prospective study was carried out on 102 patients complaining of LPR symptoms. The authors used the Reflux Symptom Index (RFS), Reflux Findings Score (RSI) and 24-h ambulatory pH monitoring to diagnose LPR disease. Two study groups were established, the LPR group included patients with LPR diagnosis and a control group selected from excluded patients. All patients underwent an oral examination for the evaluation of dental and periodontal status. Plaque index, gingival index and hemorrhage index were collected too.

LPR was revealed to be associated with both an increased incidence of chronic periodontitis with significantly higher values of plaque index ( $p=0.018$ ), hemorrhage index ( $p=0.048$ ), gingival recession ( $p=0.039$ ) and higher tooth wear scores when compared to those of the control group.

We support the view that laryngopharyngeal reflux might play a considerable role in the manifestation of periodontal and dental lesions.

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### 1. Introduction

Laryngopharyngeal reflux disease (LPRD) is caused by the back-flow of stomach contents and/or gastric acid into the laryngopharynx [1].

It is currently estimated that 4–10 % of patients evaluated by otolaryngologists may have reflux-associated symptoms such as hoarseness, sore throat, throat-clearing, chronic cough, globus sensation, dysphagia, postnasal drip, sense of a foreign body in the pharynx and halitosis. However, only in the last few years have the accompanying laryngopharyngeal symptoms related to acid reflux been investigated by ENT specialist [2-5].

Gastroesophageal reflux disease (GERD) has been shown to be associated with periodontitis as an independent risk factor for chronic periodontitis, regardless of the presence of other established risk factors such as dental caries, tobacco use, HIV infection or a history of calcium

channel blocker, cyclosporine, or phenytoin use [6-8]. It has also been demonstrated that patients with inflammatory periodontal diseases associated with GERD were marked by a more pronounced therapeutic effect of combined treatment in comparison with isolated local treatment for chronic inflammatory periodontal disease [9]. The association between acid reflux and dental erosion was first described by Howden in 1971 [10] and confirmed in later studies [11,12]. However, little is known concerning a hypothetical relationship between acid reflux disease and periodontal or other dental lesions, i.e., caries [13]. Only one study, performed by Katunaric et al. [14], has analysed this relationship and concluded that, compared to healthy subjects, dental and gingival hygiene were worse in patients with acid reflux. Significant associations between GERD, tooth loss, and functional dyspepsia (FD) have been observed in women, but not in men. Furthermore, a significant relationship exists between tooth loss and components of FD, such as early satiety [15]. Despite these reports, the association between dental lesions or

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periodontitis and LPRD remains unclear. Therefore, in this study, we aimed to investigate the relationship between dental lesions, chronic periodontitis and LPR, observing the prevalence of dental and periodontal lesions in patients with LPR.

## 2. Materials and Methods

The study was conducted prospectively among outpatients who had visited our ENT Unit, Acireale Hospital, between September 2016 to July 2017. The study was approved by the Institutional Review Board Hospital and it was conducted in accordance with the ethical principles out-lined in the Declaration of Helsinki. Informed consent was obtained from all patients.

One hundred and two consecutive patients who referred to our ENT Unit, because of atypical GERD symptoms (hoarseness, throat clearing, “globus” sensation, throat itching, cough) that lasted more than 2 months, were examined.

All subjects underwent an otolaryngology and gastroenterology evaluation. Parameters obtained from this examination were: a diagnosis of LPR based on history (RSI), physical examination (RFS) and ambulatory 24-h double pH probe (simultaneous oesophageal and pharyngeal) monitoring. The RSI was used to assess the presence of LPR. RSI which consists of 9 questions with 5 possible answers ranging from 0 to 5. It was considered positive when a score of more than 13 was obtained [16]. All patients then underwent a transnasal laryngopharyngoscopy to examine the larynx and hypopharynx. The diagnosis was confirmed with RFS, which consists of 8 questions with 4 possible answers ranging from 0 to 4. It was considered positive when a score of more than 7 was obtained [17]. Subsequently the diagnosis was validated with ambulatory 24-h double pH probe (simultaneous oesophageal and pharyngeal) monitoring [18].

Two study groups were established. The LPR group comprised of patients diagnosed with LPR and a control group selected from excluded patients (healthy subjects without any LPR-related symptoms and who had an RSI score less than 13 and an RFS score less than 7). All patients of the two groups then underwent an oral examination by the same dentist, blind to the diagnosis. The diagnosis of chronic periodontitis was based on clinical findings of gingival inflammation, loss of attachment in excess of 1 mm, and probing pocket depths  $\geq 4$  mm at three or four sites for more than four teeth per quadrant [19].

Plaque index, gingival index and hemorrhage index were collected too. Plaque was assessed using the Silness and Loe plaque index: grade 0 (absence of plaque), grade 1 (thinned plaque on gingival edge that can only be detected with the dental examination tube), grade 2 (moderate quantity of plaque on gingival edge with inter-dental spaces clean), and grade 3 (great quantity of accumulated plaque on both gingival edge and inter-dental spaces). The hemorrhage index evaluated the degree of inflammation in the gingival groove: grade 0 (absence of inflammation), grade 1 (gingival inflammation without bleeding), grade 2 (bleeding caused by the tube of examination), and grade 3 (spontaneous bleeding).

Gingival status was determined with the Loe and Silness gingival index, calculating the length of the gingival recessions using a tube inserted from the amelocementary junction to the bottom of the gingival groove and was classified as grade 0 (3 mm), grade 1 (3–6 mm) and grade 2 (6 mm) [20]. Dental condition, was evaluated by applying the Tooth Wear Index on the basis of depth and area, whereby all four visible surfaces (buccal, cervical, lingual and occlusal/incisal) of all teeth present are scored for wear,

irrespective of how it occurred [21]. The cervical, buccal/ labial, occlusal/incisal and lingual/palatal surfaces of each tooth were examined by using Community periodontal index & treatment need (CPITN) probe, that was run over the tooth surface to check for the loss of enamel surface characteristics.

Statistical analyses were performed using SPSS version 16.0 for Windows (SPSS Inc., Chicago, IL, USA). The distribution of the data was assessed by a Shapiro-Wilk test. Characteristics of the patients were determined by descriptive statistics. Categorical data were described as a frequency and percentage. Normally distributed parameters were reported as a mean  $\pm$  standard deviation. Statistical differences between the two groups were explored using chi-square or Student t-test. For non-normally distributed parameters, a Mann-Whitney U test was used.

Comparisons were considered statistically significant if the p value was  $< 0.05$ .

## 3. Results

Of the 102 examined patients, there were 55 that belonged to the LPR group and 47 to the control group. There was no significant difference ( $p > 0.05$ ), between the LPR and control groups in terms of age and gender. The mean values of RSI and RFS between the two groups are shown in (Table 1).

	LPR group (n=55)	Control group (n=47)	p value
Gender (M/F n, %)	35/20 (63.6/36.4)	26/29 (47.3/52.7)	0.712
Age (mean $\pm$ SD)	42.13 $\pm$ 10.69	43.09 $\pm$ 9.85	0.071
RSI (mean $\pm$ SD)	22.11 $\pm$ 5.19	6.21 $\pm$ 2.22	<0.001
RFS (mean $\pm$ SD)	16.83 $\pm$ 6.47	5.75 $\pm$ 3.02	<0.001

**Table 1** - Descriptive statistics of the study groups

A significantly higher prevalence and severity of periodontal lesions was noted among LPR group, the difference between the groups was statistically significant regarding Plaque index (pathological values prevalence 72.7% vs. 31.9% respectively,  $p = 0.018$ ), Hemorrhage index (pathological values prevalence 63.6% vs. 42.6% respectively,  $p = 0.048$ ) and gingival recession (pathological recession prevalence 69.1% vs. 38.3% respectively,  $p = 0.039$ ) (Table 2).

	LPR group (n=55)	Control group (n=47)	p value
Gingival recession			
<3 mm	17 (30.9%)	29 (61.7%)	0.039
$\geq 3$ mm	38 (69.1%)	18 (38.3%)	
Hemorrhage index			
Normal	20 (36.4%)	27 (57.4%)	0.048
Pathological	35 (63.6%)	20 (42.6%)	
Plaque index			
Normal	15 (27.3%)	32 (68.1%)	0.018
Pathological	40 (72.7%)	15 (31.9%)	

**Table 2** - Prevalence and severity of periodontal lesions in laryngopharyngeal reflux group (LPR) and Control group

In the CPITN probe was run over labial and lingual surfaces of incisors and buccal, lingual, occlusal surfaces of molars to check for loss of enamel surface. According to the depth of erosion, out of 55 patients with GERD, 11 (20%) patients have normal surfaces, 26 (47.3%) patients have loss of surface characteristics and 18 (32.7%) have loss of enamel exposing dentin (Table 3).

Depth of erosion in permanent incisor	LPR group (n=55)	Control group (n=47)	Total
Normal	11	20	31
Loss of enamel surface characteristics	26	18	44
Loss of enamel exposing dentin	18	8	26
Loss of enamel & dentin exposing pulp	0	0	0
Assessment could not be made	0	1	1
Total	55	47	102

**Table 3** - Distribution of depth of erosion in permanent incisors in the two groups

According to the area of erosion, out of 55 patients who have GERD, 5 (9.1%) patients have normal surfaces, 19 (34.5%) patients have less than one-third of the enamel surface involved, 21 (38.2%) patients have teeth between one-third and two-thirds of the surface involved, 9 (16.4%) patients have more than two-thirds of the surface involved and in only 1 (1.8%) patient, assessment was not possible due to calculus (Table 4).

Area of erosion in first permanent molar	LPR group (n=55)	Control group (n=47)	Total
Normal	5	20	25
Less than one third surface involved	19	13	32
Between one third & two third surface involved	21	11	32
More than two third surface involved	9	2	11
Assessment could not be made	1	1	2
Total	55	47	102

**Table 4** - Distribution of area of erosion in first permanent molar in the two groups

#### 4. Discussion

Gastric acid reflux was originally identified by Cherry and Margulies (1968) as an etiologic factor in laryngeal disease [22]. Since that time, studies on laryngopharyngeal reflux have continued, especially after understanding that this disorder can be the etiology of many extraesophageal disorders, i.e., ear, nose, throat, and oral disorders as demonstrated in some reports [23-27].

Among these investigations, the presence of oral cavity lesions in patients with acid reflux has been studied less and only the association of gastro esophageal reflux disease with dental erosion has been established in a number of studies in adults [28]. The relationship of other dental lesions, such as caries and periodontal lesions, in patients with acid reflux disease remains unclear especially for confounding factors such as a correct LPR and periodontitis diagnosis. Thus, for our study, we used RFS, RSI scores and 24-h ambulatory pH monitoring, which are validated methods to evaluate LPR with reliability [16-18], showing that there was a significantly higher prevalence of chronic periodontitis in LPR patients with greater pathological prevalence and severity values of Plaque index, Hemorrhage index and gingival recession compared to non-LPR patients. These findings are in agreement with those suggested by the study of Dzhambaldinova [9], where it was suggested that there was a pathogenic link between chronic inflammatory periodontal disease and the presence of pathological acid reflux, since it can induce poor oral conditions through poor salivary function or microbial colonization. Other results from the present study indicate that patients with symptoms of LPR have more tooth wear than subjects without symptoms. It appears that the enamel erosions were more common and severe in patients with laryngopharyngeal reflux disease with higher values of both depth and area of erosion than in control subjects, highlighting the erosion action caused by regurgitated gastric acid. This correlation is in agreement with the results of Katunaric et al. [14], who demonstrated more periodontal and gingival damage in patients with GERD.

Our study has several limitations that should be mentioned. It is thought that this discrepancy between the two groups may be due to individual factors other than acid reflux, which could influence the evolution and severity of both periodontal lesions and dental erosion. Although ambulatory 24-h dual-probe pH monitoring is considered the gold standard for LPR diagnosis, this method can be misleading; false-positive outcomes can occur due to artifacts in the upper probe, and false-negative outcomes can occur as a result of the intermittent character of reflux episodes.

Lastly, this is not a large-scale study. Findings regarding the relationship between chronic periodontitis and dental erosion with LPR may result from the small sample size. In future studies, large epidemiological studies are necessary to confirm the association between dental erosion and chronic periodontitis with LPR.

#### 5. Conclusion

In conclusion, although chronic periodontitis and dental erosion have been rarely reported in association with LPR, there is little doubt that acid reflux associated with periodontal or dental lesions are not a rare combination. They could be one of the several symptoms or manifestations of LPR. Because of their impact on quality of life, they

should be taken into consideration in clinical practice and carefully addressed in future studies.

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