

Factors associated with localization of dental erosion in patients from two French medical centers

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Abstract. Introduction: The occurrence of dental erosion (DE) involves the prolonged contact of acids of extrinsic and intrinsic origin with the tooth tissues, without intervention of pathogenic bacteria. Gastro esophageal reflux disease (GERD) is one of the most common disorder of digestive tract causing several extra-esophageal manifestations including impacting the hard dental structures. Objective: We investigate the distribution/severity and the relationships of DE with associated factors in patients from two French medical centers involved in an international AUF project: Digestive Diseases Institute, University of Nantes and Faculty of Dentistry Victor Segalen, University of Bordeaux. Material and method: A total of 119 patients comprising 77 (64.7%) female and 42 (35.3%) male, mean age 43.81 ± 14.42 years were included in the study. Two questionnaires regarding alimentary habits, lifestyle, general and digestive diseases and medications associated that favor the occurrence of DE and gastro-esophageal reflux were completed. Oral examination to quantify the severity of DE was done using the Basic Erosive Wear Examination Index. Buccal (B), palatal/ lingual (P/L) and occlusal (O) / incisal (I) surfaces are examined and the highest score was recorded. Results: DE scores were higher on the palatal/ lingual and incisal surfaces of the anterior teeth and the palatal/lingual and occlusal surfaces of the posterior teeth in patients with GERD. No statistically differences were found between DE scores and age, gender, diet or medications. Conclusion: The most affected surfaces by DE in the presence of GERD were the palatal/ lingual and incisal surfaces of the anterior teeth and the palatal/lingual and occlusal surfaces of the posterior teeth.

Key Words: dental erosion, risk factors, gastro-esophageal reflux.

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Introduction

Dental erosion (DE) is defined as a progressive, irreversible loss of dental hard tissues due to a chemical process, without bacterial involvement, and is not directly associated with mechanical or traumatic factors or with dental caries (Bamise et al 2008, Imfeld 1996). Anyway, the mechanism of DE induces fragile surfaces of dental tissues, increasing tooth wear by mechanical mechanism (Lasserre 2003).

A multifactorial condition, with higher prevalence in the recent decades (Lussi 2006, 2012) DE is caused by the presence of acidic source which may be either of intrinsic origin or extrinsic source or a combination of both (Barron et al 2003; Lussi et al 2008, 2012).

Prolonged contact between extrinsic or intrinsic acids with tooth surfaces determinate the dissolution of mineralized teeth structures. Critical pH of the enamel is 5.5. According to Lussi, pH of the acid is less important than contact duration (Lussi 2006). Although, the critical pH below which enamel dissolves is not constant, but it is rather inversely proportional to the concentrations of calcium and phosphate in saliva (Dawes 2003). The most devastating acid is gastric juice, which contains hydrochloric

acid and low concentrations of calcium and phosphate and has a pH of less than 2 thus having great potential to cause DE. An enamel surface eroded by acidic attacks cannot be remineralized because there is no suitable matrix for crystal growth (Dawes 2003). DE produced by acidic exposure, typically progresses very slow over a period of years. The DE is a slowly progressive process, with periods of activity and inactivity ranging from a minimal loss of surface enamel to the partial and complete exposure of dentine.

A variety of extrinsic and intrinsic factors are associated with DE. Extrinsic factors include most commonly dietary acids (citrus fruits, acidic drinks and foods), environment (industrial chemicals-sulfuric, nitric and chromic acid exposure, chlorinated pools) and medication in particular, the use of vitamin C tablets, non steroidal anti-inflammatory or some asthma drugs. Intrinsic factors of DE are acids of gastric origin regurgitated into esophagus and oral cavity and come in contact direct with the teeth in different pathological conditions: gastro-esophageal reflux disease (GERD), regurgitation, excessive vomiting related to eating disorders such as anorexia nervosa or bulimia, chronic vomiting during pregnancy, drug’s effects and alcoholism (Bartlett et al 2011, 2013; Dawes et al 1995).

GERD is a common digestive disease of the upper gastrointestinal tract, considered an important cause of DE. Gastroesophageal reflux has been classified into two types: physiologic and pathologic. The physiologic form occurs postprandial and is associated with eructation or belching. Usually, it is temporary and does not require medication (Dawes 2003). The pathological gastric reflux is associated to GERD and to the other pathological conditions: hiatal hernia, duodenal and gastric ulcers, esophagitis. When the pathological gastro-esophageal reflux is associated with regurgitation or vomiting the erosive lesions can be severe (Jarvinen et al 1991; Picos et al 2014). An association between gastrointestinal disorders and DE has been reported many years ago.

The purpose of the study was to investigate the distribution/severity and the relationships of DE with associated factors in patients from two French medical centers: Digestive Diseases Institute, University of Nantes and Faculty of Dentistry Victor Segalen, University of Bordeaux.

Material and method

A total of 119 patients comprising 77 (64.7%) female and 42 (35.3%) male, mean age 41.78 ± 14.29 years were included in this study. All subjects were referred to two medical centers, partners in an international project of Francophone Universities Association coordinated by "Iuliu Hatieganu" University of Cluj-Napoca, Romania: Digestive Diseases Institute, University of Nantes and Faculty of Dentistry Victor Segalen, University of Bordeaux. The research protocol was approved by the Medical Ethic Committee of the University of Medicine and Pharmacy Iuliu Hatieganu Cluj-Napoca, Romania. Informed consent was obtained from all patients and two separate questionnaires regarding medical and dental situations were completed by each patient.

The first questionnaire included data on lifestyle, dietary, more specifically the consumption (frequency, quantity intake and duration of consumption) of acidic drinks and fruits, medical history including the diseases and medication (aspirin, antacid, etc) associated with potential implication in DE and dental evaluation. The second questionnaire completes the first one by noting specific signs and symptoms of gastro esophageal reflux, their frequency and severity.

A detailed dental examination was performed by the dentists, part of the research project for each patients, in order to investigate the presence/absence of DE, distribution on tooth surface and severity of DE on all teeth, excepting wisdom molars. The distribution and severity of DE was determined by using the Basic Erosive Wear Examination Index (BEWE) having diagnostic criteria of hard tissues loss < 50% or > 50% of the surface, respectively (Table 1). It is a tool for screening DE and to quantify loss of tooth structures that assist the diagnosis and clinical management. We considered score 0 absence of DE and the scores 1, 2 and 3 presence of DE. Missing teeth, restored dental surfaces, carious teeth were not scored.

For the most severely affected dental surfaces (buccal/labial, palatal/lingual, occlusal/ incisal) a cumulative score was determined on each sextant during dental exams.

The patients with symptoms of GERD were investigated by high resolution manometry and 24-h esophageal pH-tests considered as gold standard in diagnosis of GERD (Dawes et al. 1995). These

investigations were performed by gastroenterologist and the results were used to establish the presence/absence of GERD.

Table 1. Criteria for grading DE in BEWE score (Bartlett et al 2008)

Score	Description
0	No surface loss
1	Initial loss of surface texture
2	Distinct defect, hard tissues loss < 50% of the surface area
3	Hard tissue loss > 50% of the surface area
With score 2 and 3, dentin is often involved	

For all patients the teeth were scored in six sextants (S1-S6) (Table 2) on the buccal (B), palatal/ lingual (P/L) and occlusal (O) / incisal (I) surfaces as follows:

Table 2. Sextants for evaluation DE

S1 (17-14) maxillary posterior right sextant
S2 (13-23) maxillary anterior sextant
S3 (24-27) maxillary posterior left sextant
S4 (37-34) mandibular posterior left sextant
S5 (33-43) mandibular anterior sextant
S6 (44-47) mandibular posterior right sextant

The buccal/labial, palatal/lingual, occlusal/ incisal surface of every tooth were examined in the same order for each patient. Data were recorded and analyzed with the software Medcalc version 14.8.1. Categorical data were shown as frequency and percent. Continuous data was presented as mean \pm standard deviation. Chi-square or Fisher test was used to determine differences between groups regarding the frequency of a categorical data. P-values less than 0.05 were considered statistically significant.

Results

The distribution and severity of DE on all surfaces: buccal (B), palatal/ lingual (P/L) and occlusal (O) / incisal (I) in six sextants are shown in Table 3.

Regarding the distribution of DE according to gender, there were significant statistically differences between females and males. On the sextant S1-P 54 (60%) females did not have DE and 36 (40%) male did not have DE. On S1-P 60% of the patients with score 0 were females (54) and 40% of the patients with score 0 were males (36). On S1-P 84% of the patients with score 1 were females (21) and 36% of the patients with score 1 were males (4). On S1-P 50% of the patients with score 2 were females (2) and 50% of the patients with score 2 were males (2) ($p=0.07$). On S4-O 70% of the patients with score 0 were females (47) and 28.4% of the patients with score 0 were males (19). On S4-O 37% of the patients with score 1 were females (13) and 62.9% of the patients with score 1 were males (22). On S4-O 72.2% of the patients with score 2 were females (13) and 27.8% of the patients with score 2 were males (5) ($p=0.03$). On S5-L 57.5% of the patients with score 0 were females (61) and 42.5% of the patients with score 0 were males (45). On S5-L

63.2% of the patients with score 2 were females (12) and 31.6% of the patients with score 2 were males (6). On S5-L 60% of the patients with score 3 were females (6) and 40% of the patients with score 2 were males (4) ($p=0.03$).

Table 3. Frequency of BEWE scores on all dental surfaces in investigated patients

Variables	Score 0	Score 1	Score 2	Score 3
S1-B	117	-	2	-
S1-P	90	25	4	-
S1-O	69	42	8	-
S2-B	134	-	2	-
S2-P	72	12	36	16
S2-I	73	34	25	4
S3-B	109	-	7	-
S3-P	93	20	2	1
S3-O	77	31	8	-
S4-B	116	2	3	-
S4-L	98	20	3	-
S4-O	67	35	18	1
S5-B	141	2	2	-
S5-L	106	10	19	10
S5-I	54	34	50	7
S6-B	110	2	4	-
S6-L	96	17	3	-
S6-O	62	32	21	1

On S6-L 57.3% of the patients with score 0 were females (55) and 42.7% of the patients with score 0 were males (41). On S6-L 76.5% of the patients with score 1 were females (13) and 17.6% of the patients with score 1 were males (3). On S6-L 66.7% of the patients with score 2 were females (2) and 33.3 of the patients with score 2 were males (1) ($p=0.05$).

No statistically differences between gender and distribution and severity of DE were observed on the other sextants depending on the scores obtained in every sextant ($p>0.05$).

70 (47.29%) patients were diagnosed with GERD and 78 (52.70%) without symptoms of GERD.

On S1-P 31(34.4%) patients with score 0 have GERD and 59 (65.6%) did not have GERD. 19 (76%) patients with DE score 1 have GERD and 6 (24%) did not have GERD. Patients with score 2 did not have GERD, while 4 (100%) patients with score 2 were without GERD confirmed ($p<0.001$).

On S2-P 22(30.6%) patients without DE have GERD and 50(69.4%) did not have GERD. 8(66.7%) patients with score 1 have GERD and 4(33.3%) patients with score 1 did not have GERD. 21(58.3%) patients with score 2 have GERD and 15(41.7%) patients with score 2 did not have GERD. 9(56.2%) patients with score 3 have GERD and 7(43.8%) patients with score 3 did not have GERD ($p=0.008$).

On S2-I 41(56.2%) patients without DE have GERD and 32(43.8%) did not have GERD. 6 (17.6%) patients with score 1 have GERD and 28 (82.4%) patients with score 1 did not have GERD. 9 (36%) patients with score 2 have GERD and 16(64%)

patients with score 2 did not have GERD. 4(100%) patients with score 3 have GERD and no patients without GERD ($p<0.001$). On S3-P 32(34.4%) patients without DE have GERD and 61(65.6%) did not have GERD. 17(85%) patients with score 1 have GERD and 3(15%) patients with score 1 did not have GERD. 1(50%) patients with score 2 have GERD and 1(50%) patients with score 2 did not have GERD. No patients with score 3 have GERD and 1(100%) patients with score 3 did not have GERD ($p<0.001$).

On S4-L 38(38.8%) patients without DE have GERD and 60(61.2%) did not have GERD. 14(70%) patients with score 1 have GERD and 6(30%) patients with score 1 did not have GERD. 2(66.7%) patients with score 2 have GERD and 1(33.3%) patients with score 2 did not have GERD ($p=0.02$).

On S5-L 40(37.7%) patients without DE have GERD and 66(62.3%) did not have GERD. 8(80%) patients with score 1 have GERD and 2(20%) patients with score 1 did not have GERD. 14(73.3%) patients with score 2 have GERD and 5(26.3%) patients with score 2 did not have GERD. 5(50%) patients with score 3 have GERD and 5(50%) patients with score 3 did not have GERD ($p=0.004$).

On S5-I 31(57.4%) patients without DE have GERD and 23(42.6%) did not have GERD. 9(26.5%) patients with score 1 have GERD and 25(73.5%) patients with score 1 did not have GERD. 22(44%) patients with score 2 have GERD and 28(56%) patients with score 2 did not have GERD. 5(71.4%) patients with score 3 have GERD and 2(28.6%) patients with score 3 did not have GERD ($p=0.01$).

On S6-L 35(36.5%) patients without DE have GERD and 61(63.5%) did not have GERD. 14(82.4%) patients with score 1 have GERD and 3(17.6%) patients with score 1 did not have GERD. 2(66.7%) patients with score 2 have GERD and 1(33.3%) patients with score 2 did not have GERD ($p=0.002$). On the other sextants, no significant statistically differences between GERD and no GERD were observed ($p>0.05$).

The eating disorders were not associated with DE on S1-P. GERD was associated with DE irrespective of eating disorders on S6-L ($p=0.05$). GERD was independent of eating disorders on S5-I ($p=0.05$), S5-L ($p=0.01$), S4-L ($p=0.03$).

No statistically significant differences were found between DE and diet (acidic drinks (juices fruit, soft drinks, sports and carbonated drinks) and fruits like oranges, lemons, apples, grapefruits (frequency, quantity intake and duration of consumption) or medication (aspirin, antacid). We found that DE occurred most frequently on the palatal surfaces of maxillary teeth in GERD patients (42(50%) patients on S1-P, 44.1(60%) patients on S2-P and 43.1(50%) patients on S3-P), following by the lingual surfaces of mandibular teeth and the incisal / occlusal surfaces of teeth.

Discussion

DE is a pathology of dental hard tissues (enamel, dentin and cement), due to the interaction of acids with dental surfaces, in the absence of pathogenic bacteria. In the early stages, calcium and phosphate ions from dental hard tissues are removed by acids from oral cavity leading to enamel surfaces' demineralization. In the advanced stage, the hydroxyapatite crystals of the tooth are destroyed and dissolved layer by layer (Wang et al 2010). Erosions start with minor changes in the enamel surface and

can progress in dentine producing severe and irreversible loss of tooth surfaces. The causes of erosive lesions are multifactorial. As only minor signs/symptoms are present in the early stages, the diagnosis of DE is sometimes difficult. In advanced stages of DE due to dentine involvement the clinical presentation is more severe and the damage of dental tissues is more extensive, therefore complex treatment is required for restorations of the lost tooth tissues.

Gastroesophageal reflux is the passage of gastric content into esophagus. Once pass the upper esophageal sphincter, the gastric acid may pass into oral cavity. The prolonged and repeated acid contact with the teeth may lead to erosive lesions in the teeth. An acid environment below the critical pH of the dental enamel (5.5) dissolves the hydroxyapatite crystals in enamel. Since pH of gastric acid is 1.5 to 3.5 in humans, this may explain the chemical dissolution of the tooth tissues in the presence of an acid reflux.

The refluxed acid, typically dissolve the palatal surfaces of upper incisors. Bartlett *et al* (1996) reported that the gastro-esophageal reflux is strongly associated with palatal erosion and patients presenting with palatal DE should be assessed for gastro-esophageal reflux as a possible cause, even in the absence of clinical symptoms of reflux.

In several studies, association between DE and GERD has been reported. One study found that DE and GERD are associated in adults and confirmed the importance of early diagnosis and treatment of GERD in preventing the dental damage and tooth loss (Firouzei *et al* 2011). Other studies have also investigated groups of patients presenting DE and found associations with gastric reflux. Oginni *et al* 2005 found in their study on 225 adults patients that the prevalence of erosion was statistically significant between GERD patient (16%) and control (5%) $p < 0.05$.

In our study we found that the palatal surfaces of the maxillary incisors and the occlusal surface of the mandibular first molars are the initial surfaces affected by DE. We observed that other surfaces of these teeth are less affected by erosion, owing to their location away from the salivary glands that protect them in accord with reported results of Jaeggi *et al* (2006).

Conclusion

In our study we found that GERD was associated with higher scores of DE in the investigated patients on more surfaces in sextants. This study shows that the most affected surfaces by DE in presence of GERD are the palatal/lingual and incisal surfaces of the anterior teeth and the palatal/lingual and occlusal surfaces of the posterior teeth. A good collaboration between dentist and gastroenterologists would favor for early diagnosis and management of both conditions in order to avoid irreversible lesions in teeth.

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