Clin Oral Invest (2007) 11:175–180
DOI 10.1007/s00784-007-0100-9

ORIGINAL ARTICLE

Dental erosion in chronic renal failure

Pervin Imirzalioglu · Emel Olga Onay · Erhan Agca · Ersin Ogus

Received: 30 June 2006 / Accepted: 10 January 2007 / Published online: 2 February 2007

Abstract Dental erosion is a common oral finding in chronic renal failure and may develop due to extrinsic and/or intrinsic causes. The aim of this study was to compare the salivary calcium and phosphorus levels, salivary flow rate, salivary buffer capacity, salivary pH, serum calcium and phosphorus levels and parathyroid hormone levels in chronic renal failure patients with and without dental erosion. Twenty-four men and 19 women who had had chronic renal failure for at least 2 years were studied. Twenty-two subjects displayed erosion-like patterns on their teeth and the other 21 patients showed no signs of erosion. Two closely age- and sex-matched control groups (control groups 1 and 2) were enrolled in this study because of the age disparity between the erosion and non-erosion groups. The data were analyzed by Mann–Whitney *U* test, Student *t* test, Pearson's and Spearman's correlation tests. None of the comparisons were statistically different between the erosion and non-erosion groups. There were statistically significant differences in salivary calcium ($P<0.01$) and phosphorus ($P<0.01$) levels, serum phosphorus level ($P<0.01$) and serum PTH level ($P<0.01$) for the erosion group and control group 1 and also for the non-erosion group and control group 2. There was also a significant difference in salivary flow rate ($P<0.05$) for the erosion group and control group 1. There was a positive significant correlation between saliva buffer capacity and salivary phosphorus level ($r=0.454$, $P<0.05$) in the erosion group.

Keywords Chronic renal disease · Dental erosion · Salivary flow rate · Salivary buffer capacity · Secondary hyperparathyroidism

P. Imirzalioglu
Department of Prosthodontics,
Baskent University Faculty of Dentistry,
Ankara, Turkey

E. O. Onay
Department of Endodontics,
Baskent University Faculty of Dentistry,
Ankara, Turkey

E. Agca
Department of Nephrology, Konya State Hospital,
Konya, Turkey

E. Ogus
Department of Biostatistics,
Baskent University Faculty of Medicine,
Ankara, Turkey

E. O. Onay (✉)
Baskent Universitesi Dis Hekimligi Fakultesi,
11. sok. No: 26,
06490 Bahcelievler, Ankara, Turkey
e-mail: eonay@baskent.edu.tr

Introduction

Dental erosion is defined as the irreversible loss of dental hard tissues due to a chemical process that does not involve microorganisms [7]. The condition may develop due to extrinsic and/or intrinsic factors. One important extrinsic cause is the demineralizing acid in citrus fruits, acidic beverages [1, 7, 23], medicines such as effervescent vitamin preparations, chewable vitamin C tablets [8, 16] and iron tonics [11]. However, dietary habits are not the only factors in dental erosion. Exposure to acid contaminants in the working environment [26] and frequent swimming in chlorinated pool water were also associated with dental erosion [3].

Some intrinsic causes of dental erosion include recurrent vomiting in psychological disorders such as anorexia and bulimia [10, 14] and regurgitation of stomach contents due to gastrointestinal problems [6, 18]. Finch [9] reported one erosion case that was associated with diabetes insipidus. The patient's condition had been caused by high intake of citrus fruit drinks. Low unstimulated salivary flow rates result in insufficient rinsing and buffering of demineralizing acids, which is also believed to contribute to dental erosion [12, 28]. Jarvinen et al. [12] recorded lower salivary calcium and phosphorus levels in patients with erosion compared to the controls, but the low levels were found to be associated with low stimulated salivary flow rate.

Chronic renal failure (CRF) is characterized by bilateral progressive deterioration of functioning nephrons. The nephron loss possibly compromises renal function, leading to the accumulation of waste products in the patient's system [29]. The various clinical manifestations of CRF may involve the cardiovascular, hematological, neuromuscular, endocrine, gastrointestinal and dermatological systems. These patients also exhibit altered blood and urinary biochemistry and altered bone metabolism [4].

As a result of these body system shifts in chronic renal disease, several oral changes occur. Xerostomia is the most common oral symptom in this patient group and it develops due to the decrease in fluid intake. Retrograde parotitis may also occur and is believed to result from a combination of direct involvement of the gland, chemical inflammation, dehydration and mouth breathing. CRF patients may also show increased dental calculus due to elevated serum calcium and phosphorus levels [4, 29]. Further, Sampson and Meister [19] have suggested that tooth erosion may also be related to regurgitation during dialysis.

The aim of this study was to compare salivary calcium and phosphorus levels, salivary flow rate, salivary buffer capacity, salivary pH, serum calcium and phosphorus levels and parathyroid hormone (PTH) concentrations in chronic renal failure patients with and without dental erosion. The null hypothesis tested was that there is no relationship between dental erosion and chronic renal failure.

Materials and methods

Ethical approval was obtained from the Local Ethics Committee at Baskent University. The investigation included 24 men and 19 women who had had chronic renal disease for at least 2 years. Patient age ranged from 19 to 75 years and the mean age was 44 (Table 1). Twenty-two subjects displayed erosion-like patterns and the other 21 showed no signs of dental erosion. Medical and dental histories were obtained to identify individual behavioral habits that might be associated with tooth erosion. The pathologies causing chronic renal failure are listed in Table 2. Patients with CRF were taking only routine medications like phosphate-binding drugs and multivitamin supplements (vitamin B, C, E) for renal failure and hemodialysis. In addition, some of these patients were also prescribed acetylsalicylic acid and antihypertensives. None of the patients had a history of bruxism, horizontal brushing, excessive intake of acidic juices or alcohol, or regurgitation at any time of the day. All of the patients exhibited poor oral hygiene and dental calculus and most had other medical problems related to CRF. All the patients were undergoing medical treatment and were on a highly restricted diet that was low in protein and high in calories with carefully balanced sodium, potassium and fluid intakes. None of the subjects reported problems with vomiting, stomachache, heartburn or acid taste in the mouth; however, nearly all of them had gastric disease that had been diagnosed by a gastroenterologist.

Two closely age- and sex-matched control groups were enrolled in this study because of the age disparity between the erosion and non-erosion groups. The control group 1 compared to the erosion group consisted of 22 healthy hospital staff volunteers (12 men, 10 women; mean age 48.4±6.22 years). The control group 2 compared to the non-erosion group consisted of 21 healthy hospital staff volunteers (11 men, 10 women; mean age 38.23± 5.02 years) who were not being treated for any systemic

Table 1 Characteristics of the erosion and non-erosion groups

	Erosion group (N=22)	Non-erosion group (N=21)
Gender	10 F, 12 M	9 F, 12 M
Age (years)	49.40±11.95 (23–75)	39.85±14.45 (19–67)
Duration of hemodialysis (years)	7.54±3.93 (2–16)	7.09±1.92 (2–10)

Table 2 Primary kidney disorders in patients with chronic renal failure

Primer diagnosis	Erosion group (N=22)	Non-erosion group (N=21)
Unknown etiology	6	9
Chronic pyelonephritis	6	3
Chronic glomerulonephritis	5	2
Vesicourethral nephritis	1	4
Nephrolithiasis	2	
Nephrocalcinosis		1
Nephrotic syndrome	1	
Ig A nephropathy		1
Congenital polycystic kidney		1
Nephritis	1	

illness or taking any prescribed medication. Controls were also free of xerotomic symptoms.

Dental erosion was classified and scored according to the criteria of Eccles and Jenkins [7] and the following findings characterized these cases: (1) absence of developmental ridges on the enamel, resulting in a smooth glazed enamel surface; (2) concavities in the cervical region on the labial enamel surfaces with the concavity width greatly exceeding depth, thus distinguishing from cervical abrasion lesions; (3) edges of amalgam restorations raised above the level of the adjacent tooth surface; and (4) depression of the cusps of the posterior teeth, producing cupping.

Salivary collection

Stimulated whole saliva was collected at room temperature in a quiet, isolated room. The participants were fully informed how saliva was going to be collected and they were instructed not to drink, eat, smoke or brush their teeth for at least 1 h before saliva collection. The subjects were seated in an upright position and asked to relax during the collection. For pre-stimulation, they chewed standard weight 1 g paraffin wax (Vivadent, Lichtenstein) for 60 s and swallowed the whole forming saliva. Thereafter, while chewing the same paraffin intensively, the subjects spat their saliva into sterile containers during the next 5 min and the salivary flow rate was expressed as ml/min.

The pH and buffer capacity of the saliva from each collection were measured immediately after collection. The acidity was measured with pH strips (Urine reagent strips, Cliawaived, CA, USA). The CRT Buffer Test (Vivadent, Lichtenstein) was used to determine the buffer capacity of saliva using a colorimetric test strip.

Salivary calcium and phosphorus were measured with the Modular P Roche/Hitachi automated clinical chemistry analyzer (Roche Diagnostics, Mannheim, Deutschland). The whole saliva was used to determine the salivary calcium and phosphorus concentrations. The test principle for the calcium content was based on colorimetric assay with endpoint determination and sample blank. The color intensity of the purple complex formed was directly proportional to the calcium concentration and was measured photometrically.

$$\text{calcium} + \text{o-cresolphthalein complexone} \xrightarrow{\text{alkaline solution}} \text{calcium-o-cresolphthalein complex}$$

The test principle for the phosphorus content was based on the endpoint method with sample blanking. Inorganic phosphate forms an ammonium phosphomolybdate complex having the formula $(NH_4)_3[PO_4(MoO_3)_{12}]$ with ammonium molybdate in the presence of sulfuric acid. The complex was determined photometrically in the ultraviolet region (340 nm).

Measurement of serum calcium, phosphorus and parathyroid hormone levels

All of the serum samples were taken just before the dialysis and on empty stomach. Serum calcium and phosphorus were measured with the Modular P Roche/Hitachi automated clinical chemistry analyzer. The serum PTH levels were measured by immunometric assay (Immulite Analyzer, Diagnostic Products, CA, USA). The normal range for serum phosphorus was 2.50–4.50 mg/dl, serum calcium was 8.50–10.50 mg/dl and serum PTH level was 12–72 pg/ml.

Results were analyzed statistically using the Mann–Whitney *U* test and Student *t* test. Correlations between the all parameters were determined by the Pearson's and Spearman's correlation tests. All analyses were performed using the SPSS statistical package for Windows version 11.5.0 (September 6, 2002), license number 3816436, Baskent University (SPSS, Chicago, IL, USA). All levels of significance were set at $P<0.05$.

Results

Erosion of the occlusal and incisal tooth surfaces was more common in the mandible compared to the maxilla. Furthermore, nearly one half of the affected tooth surfaces had erosions involving the dentin.

Table 3 summarizes the findings in the erosion and non-erosion groups. None of the comparisons were statistically different. Salivary flow rate was lower in the erosion group compared to the non-erosion group, but this difference was not significant.

Tables 4 and 5 summarize the findings in the erosion and non-erosion groups compared with the age- and sex-matched controls. There were statistically significant differences in salivary flow rate ($P<0.05$), salivary calcium ($P<0.01$) and phosphorus ($P<0.01$) levels, serum phosphorus level ($P<0.01$) and serum PTH level ($P<0.01$) for the erosion group and control group 1.

When comparing the non-erosion group and control group 2, there were statistically significant differences in salivary calcium ($P<0.01$) and phosphorus ($P<0.01$) levels,

Table 3 Buffer capacity, flow rate, pH, calcium and phosphorus levels of paraffin-stimulated saliva and serum calcium, phosphorus and parathyroid hormone concentrations in chronic renal failure patients with and without dental erosion

	Erosion group (mean±SD)	Number of patients	Non-erosion group (mean±SD)	Number of patients	Significance
Salivary buffer capacity	High	22	High	21	NS
Salivary flow rate ml/min	1.42±3.24	22	1.84±5.21	21	NS
pH	8.28±0.75	21	7.61±1.87	21	NS
Salivary Ca mg/dl	2.29±1.95	22	1.87±1.35	21	NS
Salivary P mg/dl	21.51±10.11	22	19.48±8.67	21	NS
Serum Ca mg/dl	9.47±0.86	22	9.27±0.83	21	NS
Serum P mg/dl	5.65±1.38	22	5.59±1.43	21	NS
Parathyroid hormone pg/ml	365.27±423.71	22	456.32±436.57	21	NS

Ca: calcium, *P*: phosphorus, *NS*: no significance.

serum phosphorus level ($P<0.01$) and serum PTH level ($P<0.01$).

A negative significant correlation were found between salivary buffer capacity and salivary flow rate ($r=-0.485$, $P<0.05$), salivary buffer capacity and serum phosphorus level ($r=-0.456$, $P<0.05$), salivary phosphorus level and salivary pH ($r=-0.5$, $P<0.05$) and also serum calcium and serum phosphorus levels ($r=-0.427$, $P<0.05$) in the erosion group. There was also a positive significant correlation between saliva buffer capacity and salivary phosphorus level ($r=0.454$, $P<0.05$) in the erosion group.

A negative significant correlation were found between saliva buffer capacity and salivary pH ($r=-0.506$, $P<0.05$), salivary flow rate and salivary phosphorus level ($r=-0.603$, $P<0.01$) in the non-erosion group.

There were not any significant differences between control group 1 and control group 2.

Discussion

Dental erosion may develop due to extrinsic and/or intrinsic causes. Anorexia nervosa, bulimia [10, 14] and gastrointestinal disturbances that involve frequent regurgitation [6, 18] are some examples of intrinsic factors. In addition, although their relative degrees of influence are not clear, salivary flow rates, salivary pH, salivary buffer capacity and salivary calcium and phosphorus levels are all thought to be important in dental erosion [12].

In the present study, the relation between chronic renal failure and dental erosion was searched by studying a sample of CRF patients with dental erosion compared to another sample of CRF patients without dental erosion, was found to be irrelevant.

Xerostomia, retrograde parotitis, enlarged tongue and metallic taste before dialysis are some of the oral symptoms that CRF patients experience [29]. Xerostomia may be a significant contributor to erosion in CRF patients because saliva and its components protect the dentition through various mechanisms. In the normal protective processes that occur, increased salivary flow helps dilute acids in the mouth; salivary buffers partially neutralize the acids in the oral fluid and salivary mucins and other organic components form a pellicle on the tooth surface, which inhibits or slows mineral loss during acid dissolution [17]. Widely accepted normal values for stimulated flow rates are 1.0–3.0 ml/min. Values below 0.7 ml/min are considered as hyposalivation and values 0.7–1.0 ml/min low [27]. Although there was a significant difference between the erosion group and control group 1 in the present study, the

Table 4 Buffer capacity, flow rate, pH, calcium and phosphorus levels of paraffin-stimulated saliva and serum calcium, phosphorus and parathyroid hormone concentrations in chronic renal failure patients with dental erosion compared to control group 1

	Erosion group (mean±SD)	Number of patients	Control group 1 (mean±SD)	Number of patients	Significance
Salivary buffer capacity	High	22	High	22	NS
Salivary flow rate ml/min	1.42±3.24	22	2.63±9.54	22	S ($P<0.05$)
pH	8.28±0.75	21	7.9±0.87	22	NS
Salivary Ca mg/dl	2.29±1.95	22	3.95±1.06	22	S ($P<0.01$)
Salivary P mg/dl	21.51±10.11	22	10.69±2.28	22	S ($P<0.01$)
Serum Ca mg/dl	9.47±0.86	22	9.64±0.46	22	NS
Serum P mg/dl	5.65±1.38	22	3.78±0.52	22	S ($P<0.01$)
Parathyroid hormone pg/ml	365.27±423.71	22	47.94±24.21	22	S ($P<0.01$)

Ca: calcium, *P*: phosphorus, *NS*: no significance, *S*: significant.

Table 5 Buffer capacity, flow rate, pH, calcium and phosphorus levels of paraffin-stimulated saliva and serum calcium, phosphorus and parathyroid hormone concentrations in chronic renal failure patients without dental erosion compared to control group 2

	Non-erosion group (mean±SD)	Number of patients	Control group 2 (mean±SD)	Number of patients	Significance
Salivary buffer capacity	High	21	High	21	NS
Salivary flow rate ml/min	1.84±5.21	21	2.26±6.15	21	NS
pH	7.61±1.87	21	8.11±0.65	21	NS
Salivary Ca mg/dl	1.87±1.35	21	3.80±0.79	21	S ($P<0.01$)
Salivary P mg/dl	19.48±8.67	21	10.81±1.92	21	S ($P<0.01$)
Serum Ca mg/dl	9.27±0.83	21	9.44±0.22	21	NS
Serum P mg/dl	5.59±1.43	21	4.02±0.43	21	S ($P<0.01$)
Parathyroid hormone pg/ml	456.32±436.57	21	41.31±18.55	21	S ($P<0.01$)

Ca: calcium, *P*: phosphorus, *NS*: no significance, *S*: significant.

mean stimulated salivary flow rates in the erosion and non-erosion groups were in the normal range. Our findings are also in agreement with the study of Bots et al. [2] in which the mean unstimulated and stimulated salivary flow rates of hemodialysis patients were found to be relatively normal.

Regarding saliva acidity, we found no statistically significant difference between the erosion and non-erosion groups regarding salivary pH and the mean findings were in the normal range (Table 3). Also, when comparing the erosion and non-erosion groups to the control groups, there were not any statistically significant differences in salivary pH (Tables 4 and 5). Further, with respect to high buffer capacity of stimulated saliva, there was no significant difference between patients with erosion and non-erosion and also between the matched controls. This may be because urea is converted to ammonia by oral bacteria [21, 22]. Stimulation itself increases pH, buffer capacity and flow rate. This effect might mask the changes that are due to the disease condition [13]. The previous studies have described that the increased salivary phosphate concentration could partially contribute to the higher buffer capacity. In line with this, a positive significant correlation was found between saliva buffer capacity and salivary phosphorus level in the erosion group.

Mannerberg [15] reported that although the amount of saliva, its pH value, buffering capacity and calcium and phosphorus contents did not differ between cases with erosion and controls, the salivary mucin content did vary. Human salivary mucins have a multifunctional role in the oral cavity in that they lubricate oral surfaces, provide a protective barrier between underlying hard and soft tissues and the external environment and aid in mastication, speech and swallowing [25]. The role of salivary mucins against erosion needs re-consideration and should be further studied.

Another common oral finding in CRF patients is increased dental calculus due to elevated serum calcium and phosphorus [4, 29]. Our patients all showed poor oral hygiene and calculus deposits. The testing showed that serum calcium was in the normal range in both the erosion and non-erosion groups (Table 3), but both groups had elevated serum phosphorus levels. When the group findings were compared to each other, serum phosphorus was higher in the patients with erosion than patients with non-erosion, but the difference was not statistically significant (Table 3). When the erosion and non-erosion groups were compared to the control groups, the serum phosphorus levels were significantly higher in the erosion and non-erosion groups than the matched controls (Tables 4 and 5). Although it is difficult to make strong conclusions, these results seem to indicate that poor oral hygiene may be more important than elevated serum calcium and phosphorus levels in relation to calculus formation.

Sampson and Meister [19] suggested that tooth erosion may be linked to regurgitation during dialysis. However, Stafne and Lovestedt [24] have stated that the enamel usually does not erode until regurgitation has continued for at least 2 years. None of the CRF patients in our study had problems with regurgitation, stomach ache, heartburn or acid taste in the mouth, but almost all of them had gastric disease that had been diagnosed by a gastroenterologist. The fact that gastric disease was highly prevalent in our patients who showed no dental erosion indicates that this condition is not strongly associated with dental erosion in CRF patients.

Secondary hyperparathyroidism is also a major problem in hemodialysis patients because calcium balance must be maintained on strict levels. As secondary hyperparathyroidism progresses in chronic renal failure, PTH acts mainly on calcium metabolism. The net result of PTH will be Ca reabsorption on tubular calcium transport and raising the calcium level in intracellular fluid. For that, on other body secretions, PTH acts as lowering the calcium levels on secretions as on the parotid gland and the net result would be lowered calcium secretion in saliva. Lowered calcium in saliva might ease dental erosion. In literature, experimental studies showed that PTH would increase plasma concentrations of calcium, whereas in saliva there was no change in calcium levels [5, 20]. The present study showed that the

PTH level was significantly elevated in both the erosion and non-erosion groups than the matched healthy controls. Although the serum and the salivary calcium levels in the erosion group were not significantly different from the non-erosion group, there were significant differences in salivary calcium levels when the erosion and non-erosion groups were compared to the control groups.

The pathogenesis of dental erosion is not completely understood. Within the limitations of this study, it is concluded that there is no relationship between dental erosion and chronic renal failure. The protective functions of saliva and its various components against erosion should be further studied.

References

1. Asher C, Read MJ (1987) Early enamel erosion in children associated with the excessive consumption of citric acid. Br Dent J 162:384–387
2. Bots CP, Brand HS, Veerman EC, Valentijn-Benz M, Van Amerongen BM, Valentijn RM, Vos PF, Bijlsma JA, Bezemer PD, Ter Wee PM, Amerongen AV (2004) Interdialytic weight gain in patients on hemodialysis is associated with dry mouth and thirst. Kidney Int 66:1662–1668
3. Centerwall BS, Armstrong CW, Funkhouser LS, Elzay RP (1986) Erosion of dental enamel among competitive swimmers at a gas-chlorinated swimming pool. Am J Epidemiol 123:641–647
4. De Rossi SS, Glick M (1996) Dental considerations for the patient with renal disease receiving hemodialysis. J Am Dent Assoc 127:211–219
5. Dua K, Abbas SK, Care AD (1995) The secretion of parathyroid hormone-related protein in the saliva of sheep and its effects on the salivary clearance of phosphate, calcium, magnesium, potassium and sodium ions. Exp Physiol 80:589–595
6. Eccles JD (1978) Erosion of teeth by gastric contents. Lancet 2:479
7. Eccles JD, Jenkins WG (1974) Dental erosion and diet. J Dent 2:153–159
8. Eriksson J-H, Angmar-Mansson B (1986) Erosionsskador av C-vitamintuggtabletter. Tandlakartidningen 78:541–544
9. Finch LD (1957) Erosion associated with diabetes insipidus. Br Dent J 103:280–282
10. Hellstrom I (1977) Oral complications in anorexia nervosa. Scand J Dent Res 85:71–86
11. James PM, Parfitt GJ (1953) Local effects of certain medicaments on the teeth. Br Med J 4848:1252–1253
12. Jarvinen VK, Rytomaa II, Heinonen OP (1991) Risk factors in dental erosion. J Dent Res 70:942–947
13. Kho HS, Lee SW, Chung SC, Kim YK (1999) Oral manifestations and salivary flow rate, pH, and buffer capacity in patients with end-stage renal disease undergoing hemodialysis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 88:316–319
14. Knewitz JL, Drisko CL (1988) Anorexia nervosa and bulimia: a review. Compendium 9:244–247
15. Mannerberg F (1963) Saliva factors in cases of erosion. Odontol Revy 14:157–166
16. Meurman JH, Murtomaa H (1986) Effect of effervescent vitamin C preparations on bovine teeth and some clinical and salivary parameters in man. Scand J Dent Res 94:491–499
17. Meurman JH, ten Cate JM (1996) Pathogenesis and modifying factors of dental erosion. Eur J Oral Sci 104:199–206
18. Pope CE (1982) Gastroesophageal reflux disease. In: Wyngaardenand JB, Smith LH Jr (eds) Cecil textbook of medicine, Saunders, Philadelphia, pp 623–624
19. Sampson E, Meister F Jr (1984) Dental complications in the end stage of renal disease. Gen Dent 32:297–299
20. Schneider P, Paunier L, Sizonenko PC, Wyss M (1977). Effects of parathyroid hormone on total protein, calcium, magnesium, phosphorus, sodium and potassium concentrations of normal human parotid saliva. Eur J Clin Invest 7:121–126
21. Singer DL, Kleinberg I (1978) Ammonia and urea content of human incisor tooth plaque. Arch Oral Biol 23:1083–1087
22. Sissons CH, Cutress TW, Pearce EI (1985) Kinetics and product stoichiometry of ureolysis by human salivary bacteria and artificial mouth plaques. Arch Oral Biol 30:781–790
23. Smith BG, Knight JK (1984) A comparison of patterns of tooth wear with aetiological factors. Br Dent J 157:16–19
24. Stafne EC, Lovestedt SA (1943) Dissolution of tooth substance by lemon juice, acid beverages and acids from some other sources. J Am Dent Assoc 34:586–592
25. Tabak LA (1995) In defense of the oral cavity: structure, biosynthesis, and function of salivary mucins. Annu Rev Physiol 57:547–564
26. ten Bruggen Cate HJ (1968) Dental erosion in industry. Br J Ind Med 25:249–266
27. Tenovuo J, Lagerlof F (1994) Saliva. In: Thylstrup A, Fejerskov O (eds) Textbook of clinical cariology, 2nd edn. Munksgaard, Copenhagen, Denmark, pp 17–43
28. Woltgens JH, Vingerling P, de Blieck-Hogervorst JM, Bervoets DJ (1985) Enamel erosion and saliva. Clin Prev Dent 7:8–10
29. Ziccardi VB, Saini J, Demas PN, Braun TW (1992) Management of the oral and maxillofacial surgery patient with end-stage renal disease. J Oral Maxillofac Surg 50:1207–1212