Erozije u pacijenata s anoreksijom i bulimijom nervozom

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Sažetak

Dentalne erozije spadaju u skupinu kemijski uzrokovanih kroničnih lezija tvrdih zubnih tkiva. Zajedničko obilježje takvih oštećenja jest da se pojavljuju na površinama zuba bez plaka te da imaju nekarijesnu i nebakterijsku prirodu. Kao potencijalni rizični čimbenici nastanka dentalnih erozija spominju se: promjena načina života i prehrane, povećano konzumiranje kisele hrane i pića, te različiti gastrointestinalni poremećaji. Dugotrajno djelovanje izrazito kisela sadržaja uzrokuje postupnu demineralizaciju anorganske tvari tvrdih zubnih tkiva i nastanak erozijskih lezija. Ti defekti uzrok su bržem abrazijskom trošenju i smanjivanju sveukupnog volumena zuba.

Ključne riječi: *dentalne erozije, anoreksija, bulimija.*

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Uvod

Erozija je rezultat bezbolnog, kroničnog, lokaliziranoga gubitka tvrdoga zubnog tkiva kemijski otopljenog kiselinom ili kelacijom bez bakterijskog djelovanja. Prvi su je opisali Darby 1892., Miller 1907. i Pickeril 1923. (1,2). Čimbenike koji uzrokuju eroziju možemo podijeliti u tri skupine: vanjske, unutarnje i idiopatske.

Vanjski čimbenici posljedica su djelovanja kiselina koje na zub dospijevaju aerosolima iz kiselina onečišćenoga zraka u tvornicama, zatim hranom ili napitcima (klorirane vode). Hrana s niskim pH vrijednostima i razni kiseli napitci koji snizuju pH ispod 2 (npr. tonik, acetil salicilna kiselina, vitamin C) također su mogući uzročnici erozije zubne krune (3).

Djelovanjem unutarnjih čimbenika povećava se količina želučane kiseline u usnoj šupljini. Kliničke promjene nastaju postupno svakodnevnim povraćanjem, gastroezofagealnim refluksom ili ruminacijom, a posljedica su poremećaja u gornjem dijelu gastrointestinalnog trakta, metaboličkih i psihosomatskih poremetnji (anoreksija nervosa i bulimija) (4,5,6).

Idiopatske erozije rezultat su djelovanja kiselina čije podrijetlo nije moguće odrediti. Erozije uzrokovane hranom i pićem najčešće postoje labijalno na prednjim zubima, odnosno okluzalno i vestibularno na pretkutnjacima i kutnjacima. Zdjelastog su oblika ili u obliku slova U, plitke su i široke udubine s glatkim rubovima (7,8,9,10).

Erozijsko djelovanje nekoga kiselog napitka uvelike ovisi o količini i čestoći konzumacije. Zakiseljeli napitci nisu toliko štetni ako se uzimaju normalno, a ne stalno i u kratkim vremenskim razmacima. Za erozijski učinak nekog napitka važna je i ukupna količina kiseline otopljene u napitku, tj. titar kiseline. No, potencijalno jako erozivni mogu biti za pacijente s poremećenom salivacijom (11).

Anoreksija i bulimija nervoza

Anoreksija nervoza je psihosomatski poremećaj koji se očituje u odbijanju hrane, što naravno uzrokuje znatan gubitak tjelesne težine. Ovisno o tome kako pacijenti gube tjelesnu težinu, razlikujemo "restriktivnu" anoreksiju nervosa (gubitak tjelesne težine suzdržavanjem od jela) i "bulimijsku" anoreksiju nervoza (gubitak tjelesne težine suzdržavanjem od jela s povraćanjem i uporabom laksativa).

Bulimija nervoza očituje se pretjeranim konzumiranjem jela te povraćanjem i uzimanjem laksativa. Žene su češće zahvaćene anoreksijom i bulimijom nego muškarci; oni čine samo 10% slučajeva. Istraživanja provedena u zapadnoj Europi i sjevernoj Americi pokazala su da 5% žena u dobi između 18 i 35 godina pati od bulimije nervoza. Najveći broj oboljelih od anoreksije nervoza jesu osobe između 12 i 20 godina, što čini oko 2% svih slučajeva. Općenito gledano, anoreksične osobe imaju 15% manje tjelesne težine od idealne, a bulimične pretežito 10% ispod ili čak nešto više od idealne težine (12,13,14).

Rana dijagnoza bolesti iznimno je važna jer od obaju poremećaja nastaju mnoge i ozbiljne somatske komplikacije. Budući da pacijenti s bulimijom nervoza imaju normalnu tjelesnu težinu, obično treba mnogo godina da bi se poremećaj točno dijagnosticirao. Pacijenti se obično stide svojih abnormalnih oblika ponašanja u vezi s hranom, pa je upravo stomatolog često prvi u prigodi dijagnosticirati poremećaje na temelju erozija nastalih kroničnim povraćanjem (15,16,17).

Razdioba i lokalizacija erozija

Postoje mnoge podjele koje opisuje dentalne erozije. Jednu od prikladnijih dao je Eccles. On erozije dijeli u nekoliko skupina:

RAZRED I - obuhvaća površinske lezije - zahvaćena je samo caklina,

RAZRED II - obuhvaća lokalizirane lezije - zahvaćen je dentin manje od 1/3 površine,

RAZRED III - generalizirane su lezije - zahvaćen je dentin više od 1/3 površine.

Razred III dijeli se na nekoliko podrazreda:

III a - zahvaćena labijalna/bukalna površina,

III b - zahvaćene su lingvalna i palatinalna površina,

III c - zahvaćene su incizijska i okuzijska površina,

III d - više zahvaćenih površina se isprepleće.

Eccles smatra da lezije razreda I mogu biti tretirane promjenom pacijentovih navika pa se time smanjuje izlaganje usne šupljine kiselinama. Razred II može se tretirati kompozitnim materijalima, a lezije razreda III zahtijevaju veće rekonstrukcije ili krunice (18).

U pacijenata s restriktivnom anoreksijom nervoza one erozije koje su uzrokovane vanjskim dijetetskim čimbenicima većinom su na vestibularnoj plohi zuba, a u pacijenata s bulimijom nervoza obično je zahvaćena palatinalna ploha maksilarnih zuba, osobito sjekutića. Ako povraćanje stalno traje duže od pet godina, labijalna i bukalna ploha također su zahvaćene erozijama (5). Postoji, dakle, razlika u zahvaćenosti zuba gornje i donje čeljusti. Zahvaćene su palatinalne i okluzijske plohe svih zuba gornje čeljusti, te bukalne i okluzijske polohe zuba donje čeljusti. Bukalne plohe gornjih zuba nisu u kontaktu s kiselinom, a osim toga zaštićene su parotidnom žlijezdom. Lingvalne plohe donjih zuba pokrivene su jezikom i vlažne od sekreta submandibularne i sublingvalne žlijezde te su time sačuvane od djelovanja kiseline (19).

Svojevoljno izazvano povraćanje povećava čestoću erozija na palatinalnim plohama.

Čestoća, trajanje i broj povraćanja nisu linearno povezani s erozijama i nije utvrđena razlika u veličini erozijskih promjena u onih pacijenata koji rjeđe ili češće povraćaju.

Erozije cakline mogu uzrokovati eksponiranje dentina, a posljedica toga je povećana osjetljivost na mehaničke i toplinske podražaje. Reaktivna papilarna hiperplazija može biti rezultat kronične iritacije kiselinom tijekom povraćanja, a nađeno je i proširenje izvodnoga kanala parotidne žlijezde (20,21).

Uloga sline

Protok sline poveća se prije povraćanja jer je medularni centar za kontrolu povraćanja povezan s *nu-*

cleus salivarii. Mučnina nakon prejedanja također utječe na veći protok sline. Kao posljedica poremećenoga konzumiranja hrane može biti pojava angularnog heilitisa, kandidijaze, glositisa i ulceracije oralne mukoze (12).

Pacijenti s bulimičnom anoreksijom nervoza imaju niži protok stimulirane i nestimulirane sline, a time i veće erozijske defekte nego pacijenti s bulimijom nervoza i istim trajanjem poremećaja prehrane. Puferski kapacitet sline kod pacijenta s bulimičnom anoreksijom nervosa nešto je veći nego kod onih s bulimijom nervosa. U pacijenata s bulimijom reducirana je količina bikarbonata u slini, ali je povećana njezina viskoznost (5).

Histološke promjene cakline i dentina

Zbog demineralizacije na površini cakline i trenutačnog uklanjanja rastopljenih minerala caklina gubi sloj po sloj. Taj je proces ireverzibilan. Djelovanje kiseline na prizmatsku caklinu bitno se razlikuje od djelovanja na aprizmatsku caklinu zubnoga vrata. U prizmatskoj caklini gubitak tkiva uzrokom je anizotopnog otapanja prvo sredine prizme, potom tzv. "prizmatske ovojnice", a zatim i interprizmatske cakline što uzrokuje nastanak saćaste strukture u caklini. Aprizmatska caklina sklonija je nepravilnoj destrukciji zbog morfoloških razlika i nije toliko podložna erozivnom razaranju kao prizmatska (22). Na površini cerviksne cakline mogu se opaziti manje ili veće erozije nepravilnog oblika i gruboga neravnog dna. Što je vrijeme djelovanja duže to su erodirane površine veće. Od dugotrajna djelovanja kiseloga sadržaja smanjuje se sveukupni volumen zuba i povećavaju se interdentalni prostori. Erozije dentina prvo zahvaćaju prostor između intertubulusnog i peritubulusnog dentina, a zatim u kasnijoj fazi uzrokuju potpuno otapanje peritubulusnog dentina zbog čega se proširuju dentinski tubulusi (22). Iako otpornost dentina na kiseline varira od mjesta do mjesta, erozivne destrukcije otvaraju dentinske tubuluse te se tako uspostavlja komunikacija s dubljim i vitalnim dijelovima dentina. To objašnjava bol i osjetljivost eksponiranoga dentina kod erozija. Površinska demineralizacija dentina nikada ne ide dublje od 100 μm u dubinu, što je mnogo manje od demineralizacije uzrokovane karijesom. No neke lezije nisu osjetljive i taj nedostatak osjetljivosti te sjajna glatka površina najvjerojatnije su rezultat uspješne skleroze i zatvaranja dentinskih tubulusa (23,24,25).

Naputci pacijentima s erozijskim defektima uzrokovanih dijetnim režimom prehrane

Osobi s klinički dijagnosticiranom erozijom uzrokovanom načinom prehrane preporučuje se prihvatiti sljedeće naputke:

- smanjiti čestoću konzumiranja kiselih jela i pića
- ograničiti kisela jela kao glavno jelo
- završiti jelo s neutralnom hranom (npr. sirom, mlijekom) radije nego kiselom (kao npr. voćna salata)
- kisela pića ispiti brzo, a ne pijuckati
- nakon konzumacije kiselog usta isprati vodom
- za oralnu higijenu rabiti meku ili srednje tvrdu četkicu, nisko abrazivnu pastu s fluoridima i bikarbonatima
- ne četkati zube neposredno nakon izlaganja kiselim iritansima
- rabiti gume za žvakanje bez šećera, naročito one koje sadrže bikarbonate ili ureu te fluoride kako bi stimulirali lučenje sline (26).

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Dental Erosions in Patients with Anorexia and Bulimia Nervosa

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Summary

Dental erosions belong to a group of chemically caused chronic lesions of hard tooth tissue. A common characteristic of these lesions are the appearance of a tooth surface without plaque as well as noncarious and nonbacterial etiology. As potentional risk factors for dental erosions are new life style, increased consumption of acid food and drinks and different gastrointestinal disorders. Long-lasting exposure to acid substances leads slowly to demineralization of the anorganic component of hard tooth tissue and the appearance of dental erosion. These defects lead to faster abrasion and decrease of whole tooth volume and greater tooth sensitivity.

Key words: *dental erosion, anorexia, bulimia*

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Introduction

Erosions are a result of painless, chronic, localizated loss of hard tooth tissue chemically solvent by acid or chelation without involvement of bacteria. It was first described by Darby 1892, Miller 1907 and Pickeril 1923 (1,2). Factors which cause erosions can be divided into three groups: extrinsic, intrinsic and idiopathic.

Extrinsic factors are a consequence of acids which occur on the tooth through aerosols from acid polluted air in factories, food or drinks (chloride water). Food with low pH and different acid drinks which decrease pH below 2 (tonic water, acetil salicil acid, vitamin C) are also potential factors which can cause erosion of tooth crowns (3).

Intrinsic factors cause a decrease of gastric acid in the mouth. Clinical changes appear slowly with daily vomiting, gastroesophageal reflux or regurgitation and are a consequence of disorders in the upper part of the gastrointestinal area, metabolic and psychosomatic disorders (anorexia and bulimia nervosa) (4,5,6).

Idiopathic erosions are a result of acid with anoun conten. Erosions caused by food and drinks are mostly localised on the labial surface of frontal teeth and occlusal or vestibular on premolars and molars. They are oval or U-shaped, shallow or large holes with smooth edges (7,8,9,10).

Erosion activity of some acid drinks depends on the amount and frequency of consummation. Acid drinks are not dangerous if they are taken normally, not continuously, and for a short period. For the erosive effect of certain drinks, the amount of acid dissolved in such drinks - called acid titre is important. Patients with salivary disorders could have a high erosive effect (11).

Anorexia and bulimia nervosa

Anorexia nervosa is a psyhosomatic disorder which is evident in the rejecting of food consumption which leads to significant weight loss. Depending on how patients lose weight, distinction is made between "restrictive" anorexia nervosa (weight loss through the rejecting of food consumption) and "bulimic" anorexia nervosa (weight loss through the rejecting of food consumption with self-induced vomiting and laxative abuse).

Bulimia nervosa is characterised by binge-eating and self-purging of ingested food by vomiting and laxative abuse. Women are more often affected by anorexia and bulimia nervosa, while male patients account for less than 10%. The results of epidemiological studies caried out in recent years in West Europe and North America established that 5% of women aged between 18 and 35 years suffer from bulimia nervosa. The majority of patients with anorexia nervosa are aged between 12 and 20 years, with a prevalence in this age group of about 2%. However, anorexic patients are 15% under ideal body weight while bulimic patients are mostly 10% under or even some heavier than ideal body weight (12,13,14).

Early diagnosis of these eating disorders is important because they lead to many serious somatic complications. Due to the normal weight of patients with bulimia nervosa it generally takes many years for their disorder to be correctly diagnosed. Patients are usually a shamed because of their abnormal eating behaviour. For this reason the dentist is often the first to diagnose a bulimic eating disorder, based on the dental erosion induced by chronic vomiting (15,16,17).

Classification and localisation of erosion

There are many classifications of dental erosion. One of them is given by Eccless who divided erosions in several groups:

CLASS I - surface lesions - only enamel is involved

CLASS II - localizated lesions - dentine is involved in less than 1/3 of the surface

CLASS III - generalised lesions - dentine is involved in more than 1/3 of the surface.

Class III is divided into subclasses:

III a - labial/buccal surfaces are involved

III b - lingual and palatinal surfaces are involved

III c - incizal and occlusal surfaces are involved

III d - several involved surfaces are jumbled.

Eccles says that class I lesions may be treated by changing the patient's habits by decreasing acid exposure. Class II may be treated with composite materials, and class III lesions require larger reconstruction or crowns (18).

In patients with restrictive anorexia nervosa, the erosions which are caused by extrinsic dietary factors are confined to the vestibular tooth surfaces, whereas in patients with bulimic anorexia nervosa or bulimia nervosa, the palatal surfaces of the maxillary teeth are usually involved, especially of the incisors. Only when regular vomiting had persisted over a period of 5 years or more were the labial and buccal surfaces of the teeth also affected by erosions (5). There are differences in affecting teeth of the mandibular and maxillary teeth. Palatinal and occlusal surfaces of all maxillary teeth and buccal and occlusal surfaces of the mandibular teeth are involved. Buccal surfaces of maxillary teeth are not in contact with acid and they are protected by the saliva of the glandula parotis. Lingual surfaces of mandibular teeth are covered with the tongue and moisture of submandibular and sublingual glandular saliva and in this way are protected from acids (19).

Self-induced vomiting increases dental erosions on palatinal surfaces. Duration and number of vomiting are not linearly connected with erosion and there is no difference between the size of erosive changes in those patients who rarely or frequently vomit.

Enamel erosions may cause dentine exposure and as a consequence higher sensitivity to mechanical and heat stimuli. Reactive papillary hiperplasion may be a result of chronic acid irritation during the vomiting and enlargement of the ductus parotis has also been confirmed (20,21).

The role of saliva

Saliva flow is increased before vomiting because the medular centre for vomiting control is con-

nected with nucleus salivarii. Sickness after overeating also influences higher saliva flow. As a consequence of eating disorders angular heilitis, candidiasis, glossitis and ulceration of oral mucosa may be diagnosed (12).

Patients with bulimic anorexia nervosa have a lower mean flow rate of unstimulated and stimulated saliva and higher erosion lesions than patients with bulimia nervosa and the same duration of eating disorder. The salivary buffer capacity in bulimic anorexia nervosa patients is higher than in bulimia nervosa patients. By bulimic patients the amount of bicarbonate in saliva is reduced but its viscosity is higher (5).

Histological changes of enamel and dentine

Because of demineralisation on the enamel surface and loss of dissolved minerals, enamel is lost layer by layer. This process is of an irreversible nature. Acid influence on prismatic enamel is different to acid influence on aprismatic enamel of the tooth cervix. In prismatic enamel loss of tissue leads to anizotropic dissolving of the prism centre first, then the prismatic core and later the interprizmatic enamel which leads to the formation of a honeycomb structure in enamel. Aprismatic enamel is more affected by irregular destruction because of morphological direfences and is not as sensitive to erosive destruction as prismatic enamel (22). A small or large erosion of irregular shape and rude bottom may be noticed on the surface of cervical enamel. If the activity time is longer, the erosive surface is higher. Longlasting acid activity leads to a decrease in whole tooth volume and increase in interdental spaces. Dentine erosions appear to affect primarily the surface of the intertubular dentine with decalcification of the peritubular dentine, limited to the apertures of the dentine tubules (22). Subsurface demineralisation is never found to exceed 100 (m in depth, which is much less than in the case of carious lesions. Erosive lesions are often painfully sensitive and this is probably due to the exposure of vital dentine. However, some lesions are not sensitive and this lack of sensitivity, as well as the polished appearance of many lesions is probably due to formation of a layer of sclerotic dentine and blockage of the apertures of the dentine tubuli (23,24,25).

Advice to patients suffering from dietary erosion

Patients with clinically diagnostic erosion caused by eating regime are advised as follows:

- to diminish the frequency of consumption of acid foods and beverages
- to restrict acid foods to main meals
- to finish a meal with neutral food, (e.g. cheese, milk) rather than acid food (e.g. fruit salad)
- to drink acid beverages quickly and not sip or swish around
- to rinse with water after acid consumption
- to use a soft or medium toothbrush, and low abrasive paste with fluoride and bicarbonate for oral hygiene
- not to brush teeth immediately following acid consumption
- sugar-free chewing gum, preferably products containing bicarbonate and urea-containing gum, used alternatively with fluoride-containing gum to stimulate salivary flow (26).