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UVEĆANJE GINGIVE

GINGIVAL OVERGROWTH

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Sažetak

Zdrava gingiva je preduslov zdravlja celokupnog organizma. Izraženo uvećanje gingive ometa govor, žvakanje i ishranu, izaziva estetske smetnje i povećava rizik za razvitak parodontopatije i sistemskih bolesti. Ovaj rad predstavlja sažetak znanja o etiologiji, kliničkom izgledu, klasifikaciji, patogenezi i mogućoj multidisciplinarnoj prevenciji i terapiji uvećanja gingive..
Učestalost uvećanja gingive varira u zavisnosti od socioekonomskog statusa i faktora rizika sa zastupljenošću od 1/9000 odraslih. Najučestalije je inflamatorno uvećanje gingive izazvano lekovima. Histološka ispitivanja uvećane gingive otkrila su normalan nespecifičan nalaz, uprkos mnogobrojnim faktorima rizika, koji se uglavnom karakterišu povećenjem zapremine gingive na račun epitela (ćelijska hiperplazija) i lamine proprije (akumulacija ekstraćelijskog matriksa i ćelija). Terapija uvećanja gingive zavisi od kliničkog izgleda i etioloških faktora, a zasniva se na korekciji estetskih i/ili funkcionalnih problema i deli se na konzervativnu i hirurško terapiju.
Zaključak: *Neophodna su dalja istraživanja u vezi sa etilogijom, kliničkom slikom, patogenezom, prevencijom i multidisciplinarnom terapijom uvećanja gingive, a posebnu pažnju treba posvetiti faktorima koji regulišu vezu između etiologije i izraženosti uvećanja gingive.*

Ključne reči: *uvećanje gingive, lekovi, histologija gingive, parodontalna terapija*

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Abstract

Gingival health is a prerequisite for the health of the whole body. Severe gingival overgrowth affects speech, mastication, and nutrition, causes aesthetic concerns and increases susceptibility to periodontitits and systemic diseases. This article reviews the etiology, clinical manifestations, classification, pathogenesis, prevention and possible multidisciplinary management of gingival overgrowth.
Gingival overgrowth incidence varies according to the socioeconomic status and the risk factors involved, with a reported rate of 1/9000 adults. Most commonly, gingival overgrowth is induced by drugs. Histological studies performed on samples of gingival overgrowth tissues revealed common, non-specific finding, despite numerous risk factors, which is generally characterized by an increase of gums volume to which contribute both the epithelium (cellular hyperplasia) and lamina propria (accumulation of extracellular matrix and cells). The treatment of gingival overgrowth depends on clinical appearance and etiological factors of gingival overgrowth; it focuses on the correction of the aesthetic and/or functional problems and can be divided into nonsurgical or surgical alternatives.
Conclusion*: Further studies are needed regarding the etiology, manifestation, pathogenesis, prevention and multidisciplinary management of gingival overgrowth, and special attention should be paid to factors that regulate the relationship between the etiology and severity of gingival overgrowth.*

Key words: *gingival overgrowth, drugs, gingival histology, periodontal therapy*

Uvod

"Uvećanje gingive" (gingival overgrowth, gingivalno povećanje, hiperplazija ili hipertrofija gingive) predstavlja termin za opis različitih oblika uvećanja gingive, a definiše se kao poremećeno uvećanje parodontalnog tkiva[1,2]. Smatra se da raniji naziv "hiperplazija gingive" nije adekvatan, jer uvećanje gingive nije samo posledica povećanja broja ćelija, već i povećanja zapremine ekstracelularnog matriksa gingive [1,2].

Iako ranije zanemarivana, zdrava gingiva je preduslov za pravilno funkcionisanje celokupnog organizma. Izraženo uvećanje gingive ometa govor, žvakanje i ishranu, izaziva estetske smetnje i povećava rizik za razvitak parodontopatije i sistemskih bolesti[2]. Uvećanje gingive izaziva bol, osetljivost i krvarenje gingive, patološko pomeranje zuba, poremećaj okluzije i govora. Može biti posledica dejstava različitih faktora, kao što su primena lekova ili genetski poremećaji [3-6].

Ovaj rad predstavlja sažetak znanja o etiologiji, kliničkom izgledu, klasifikaciji, patogenezi, prevenciji i mogućoj multidisciplinarnoj terapiji uvećanja gingive [7].

Klinički izgled

Uvećanje gingive obično zahvata labijalnu površinu gingive prednjih zuba. Klinički, najčešće počinje u predelu interdentalne papile, koja se uvećava i širi lateralno sve dok se ne spoji sa papilom susednog zuba. Ako je kontrola oralnog biofilma na zadovoljavajućem nivou, krvarenje na provokaciju biće minimalno, a uvećano tkivo gingive čvrsto i zdrave ružičaste boje. Ako je oralna higijena loša, biće prisutna inflamacija, a tkivo gingive je crveno. Postoji značajna pozitivna veza između incidencije i/ili intenziteta uvećanja gingive i količine prisutnog oralnog biofilma i kamenca. Takođe, uvećanje zavisi od koncentracije leka u plazmi i trajanja terapije. Uvećanje je bezbolno i sporo napreduje, a u težim slučajevima može u potpunosti prekriti zube [8-11].

Učestalost uvećanja gingive varira u zavisnosti od socioekonomskog statusa i faktora rizika sa zastupljenošću od 1/9000 odraslih. Najučestalije je inflamatorno uvećanje gingive izazvano lekovima. Fenitoin povećava zapreminu gingive u 57% slučajeva, cefalosporini u 30-46%, a blokatori kalcijumovih kanala u 10%[9]. Nasledno uvećanje gingive je najređi oblik uvećanja

Introduction

"Gingival overgrowth" (GO; gingival enlargement, gingival hyperplasia or hypertrophy) is the term used to describe different forms of gingival overgrowth and can be defined as an abnormal growth of the periodontal tissue [1,2]. Formerly used term “gingival hyperplasia” is inappropriate because enlargement is not only the result of an increase in the number of cells, but rather an increase in extracellular tissue volume [1,2].

Even though it seemed as an issue of little importance, the health of gums is a prerequisite condition for the normal function of the whole body. Severe GO affects speech, mastication, and nutrition, causes aesthetic concerns and increases susceptibility to periodontitits and systemic diseases[2]. GO produces pain, tenderness and gingival bleeding, abnormal tooth movement, dental occlusion problems and speech disturbances. It may be caused by many different factors such as medications or genetic abnormalities [3-6].

This article reviews the etiology, clinical manifestations, classification, pathogenesis and possible multidisciplinary management and prevention of gingival overgrowth [7].

Clinical manifestations

Gingival overgrowth (GO) usually affects gingival tissues around the labial surfaces of the anterior teeth. Clinically, it most commonly begins in the region of the interdental papillae, which gradually increases in size and extends laterally until adjacent papillae coalesce. If the condition of the oral biofilm is at the satisfactory level, there will be minimal bleeding and the enlarged tissues will be of a firm consistency with a healthy pink color. If plaque control is not good, the inflammation will be present and the gingival lesion will be red. There is a significant correlation between the incidence and/or severity of gingival overgrowth and the level of plaque and calculus accumulation. The extension of gingival enlargement is related to plasmatic levels of the patient's drugs and therapy duration. The enlargement is painless and slowly progressive, but in severe cases it can increase to the point of full-tooth coverage[8-11]. GO incidence varies according to the socioeconomic status and the risk

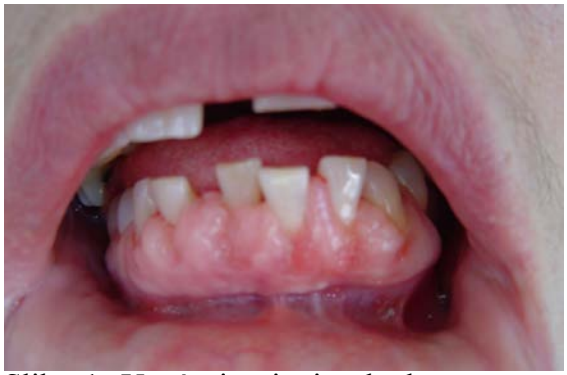
gingive i javlja se u 1/750 000 ljudi sa istom zastupljenošću u oba pola[8.] Kod bezubih pacijenata i dece sa mlečnom denticijom uvećanje gingive se veoma retko javlja, ali je njegovo pojavljivanje uočeno uz titanijumske dentalne implante osoba koje uzimaju fenitoin[8,9].

Klasifikacija

Uvećanja gingive su obicno klasifikovana u odnosu na klinički izgled i etiološke faktore .

• *Zapaljenska uvećanja gingive.* Kliničkim pregledom uočava se inflamacija, gingiva je crvena, mekana, sjajna i lako krvari na provokaciju. Zapaljenje je obično izazvano lošom oralnom higijenom i akumulacijom oralnog biofilma i izaziva reaktivno uvećanje gingive (fokalno reaktivno uvećanje gingive, inflamatorna hiperplazija ili epulis). Naziv epulis se sada smatra neadekvatnim za ovakva uvećanja bez histološkog opisa lezije. Preporučuje se naziv reaktivna lezija gingive [12]. Pušenje i sistemske bolesti (diabetes mellitus, HIV infekcija) takođe izazivaju zapaljenske promene gingive.

• *Nezapaljenska uvećanja gingive.* Ovakva uvećanja imaju tamnije crvenu ili ljubičastu boju, mogu biti čvrsta ili meka, kada i lakše krvare. Predisponirajući faktori su mnogobrojni: loša oralna higijena, specifična hormonska stanja (pubertet, trudnoća...), nutritivni deficit (skorbut), krvne diskrazije (akutna leukemija, limfomi, aplastična anemija), genetski faktori (epulis ili Nojmanov tumor), lekovi (antikonvulzivni lekovi, fenitoin), imunosupresivi (ciklosporin A), antihipertenzivi blokatori kalcijumovih kanala (verapamil, diltiazem, nifedipin), sistemske bolesti (sarkoidoza, Kronovo oboljenje, akromegalija, primarna amiloidoza) [13]. (Slika 1)



Slika 1. Uvećanje gingive kod pacijenta na terapiji nifedipinom

Figure 1. Gingival overgrowth in patient with nifedipine therapy

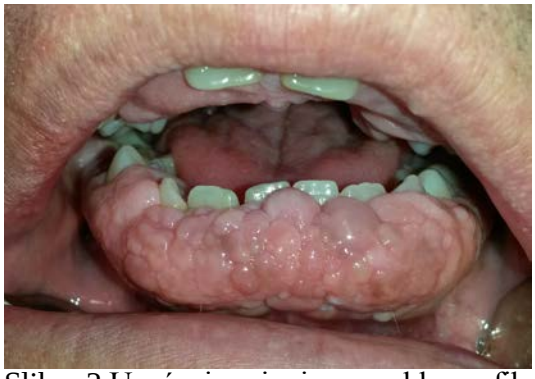
factors involved, with a reported rate of 1/9000 adults. Most commonly, gingival overgrowth is induced by drugs**.** Phenytoin increases the gingival volume in 57% of cases, cephalosporin in 30-46% and calcium channel blockers in 10%[9]. HGF is the rarest type of GO and is estimated to affect 1/750.000 people with the same incidence in both sexes[8]. GO has only been reported occasionally in edentulous patients and in primary dentitions, but has been documented adjacent to titanium dental implants with phenytoin use [8,9].

Classification

Usually, gingival overgrowth (GO) is classified according to the clinical appearance and the etiological factors into:

• *Inflammatory gingival overgrowth.* Clinical examination displays an inflammatory aspect and the gums are red, soft, shiny, and they bleed easily. Inflammation is induced frequently by poor dental hygiene resulting in bacterial plaque, and it causes reactive GO (focal reactive GO, inflammatory hyperplasia or epulis). The epulides is the term considered unsuitable without a histological description of the lesion. Nowadays, the preferred term is gum reactive lesion[12]. Smoking and systemic diseases (diabetes mellitus, HIV infection) also determine inflammatory gum lesions.

• *Non-inflammatorygingival overgrowth.* This kind of overgrowth is of a dark red or purple color, it is either firm or soft, and bleeds easily. The predisposing factors are numerous: subjects with poor dental hygiene, specific hormonal states (puberty, pregnancy), nutritional deficiency



Slika 2.Uvećanje gingive u sklopu fibrom-atoznog gingivita

Figure 2. Gingival overgrowth during gingival fibromatosis

Fibromatozni gingivitis (GF) je termin koji se često koristi za bilo koje uvećanje gingive kada se sumnja na naslednu komponentu (nasledna gingivalna fibromatoza, HGF), na fibromatozu u sklopu nekog sindroma ili kada je etiološki faktor nepoznat – idiopatska gingivalna fibromatoza (IGF). (Slika2) U nedostatku imunohisto-hemijskih markera, dijagnoza HGF se zasniva isključivo na kliničkom pregledu, istoriji bolesti i porodičnoj anamnezi. Preporučuje se upotreba termina „idiopatska fibromatoza" samo za GF koji nije u vezi sa genetskom predispozicijom, kako bi se izbegla konfuzija u klasifikaciji [13].

Nasledna gingivalna fibromatoza (heriditarna gingivalna fibromatoza, HGF) ranije je zvana gingivalna elefantijaza, idiopatska gingivalna fibromatoza, nasledna gingivalna hiperplazija, gingivalni gigantizam ili samo hiprtrofična gingiva[14,15]. Može se klasifikovati na sledeći način:

A) *HGF u sklopu sindroma* prisutna je kod sindroma izazvanih hromozomskim anomalijama (duplikacijama, delecijama) kao što su [15-20]:

- Kros sindrom – HGF, mikroftalmija, mentalna retardacija, hipopigmentacija;
- Marej-Puretik-Drešer sindrom – HGF, oboljenja kostiju, hrskavice, kože i mišića;
- Ramon sindrom – HGF, hipertrihioza, mentalna retardacija, konvulzije, zaostajanje u rastu, juvenilni reumatoidni artritis;
- Cimerman - Laband sindrom – HGF i deformiteti lica, promene na nosu i ušima, distrofija noktiju, hipoplazija, epilepsija, hepato-splenomegalija, gluvoća, mentalna retardacija, kornealna distrofija, agresivno ponašanje, mentalna retardacija;
- Džons sindrom – HGF, progresivna gluvoća, maksilarne odontogene ciste;
- Kovden sindrom – lokalizovana HGF sa multiplim hemartrozama [15,21-28].

B) *Nasledna ili izolovana HGF* nazvana i nesindromska GF je posledica mutacije SOS1 (Sun of sevenless-1) gena na hromozomu 2. SOS1 je onkogen uključen u rast ćelija[29,30]. Smatra se da je prisustvo zuba u alveolama preduslov za razvitak naslednog uvećanja gingive koji se javlja u primarnoj denticiji i nestaje ili se smanjuje nakon ekstrakcije zuba[13]. Drugi tip HGF sa familijarnom zastupljenošću je posledica mutacija na hromozomu 2 (ali ne na SOS1 onkogenu) sa systemic diseases (sarcoidosis, Crohn disease, acromegaly, primary amyloidosis)[13]. (Figure1).

Gingival fibromatosis (GF) is the term frequently used for any GO when hereditary pattern is suspected (hereditary gingival fibromatosis, HGF), as part of a syndrome or when the etiologic factor is unknown - idiopathic gingival fibromatosis (IGF) (Figure 2). Lacking specific immunohistochemical markers, the diagnosis of HGF is based exclusively on clinical examination, patient medical history and family history. It was recommended to use the term „idiopathic fibromatosis" only for GF that does not incriminate genetic and hereditary causes mentioned above in order to avoid these confusions of classification[13].

Hereditary gingival fibromatosis (HGF), previously known as gingival elephantiasis, idiopathic gingival fibro-matosis, hereditary gingival hyperplasia, gingival gigantism or just hypertrophic gums[14,15]. It can be classified as follows:

A) Syndromic HGF is present in some syndromes caused by chromosomal abno-rmalities (duplications, deletions) like [15-20]:

- Cross Syndrome – HGF, microphthalmia, mental retardation, hypopigmentation;
- Murray-Puretic-Drescher Syndrome – HGF, bone, cartilage, skin and muscle diseases;
- Ramon Syndrome – HGF, hypertrichosis, mental retardation, convulsions, growth retardation, juvenile rheumatoid arthritis;
- Zimmerman-Laband Syndrome - HGF and facial deformities, changes of nose and ears, nail dystrophy, hypoplasia, epilepsy, hepato-splenomegaly, deafness, mental retardation, corneal dystrophy, aggressive behavior, mental retardation;
- Jones Syndrome – HGF, progressive deafness, maxillary odontogenic cysts;
- Cowden Syndrome - Localized HGF with multiple hamartomas [15,21-28].

B) *Hereditary or isolated HGF* named also non-syndromic GF seems to be determined by the mutation of SOS1 (Sun of sevenless-1) gene on chromosome 2 SOS1 is an oncogene involved in cell growth [29,30]. The

kliničkom manifestacijom kod odraslih osoba [23].

Histološki aspekti uvećanja gingive

Histološka ispitivanja uvećane gingive otkrila su normalan nespecifičan nalaz uprkos mnogobrojnim faktorima rizika. Nalaz se uglavnom karakteriše povećenjem zapremine gingive na račun epitela (ćelijska hiperplazija) i lamine proprije (akumulacije ekstraćelijskog matriksa i ćelija)[1,2]. Mikroskopskim pregledom uočava se prisustvo tkivne hipertrofije i ćelijska hiperplazija. Klinički i histološki izražena je akumulacija proteina ekstraćelijskog matriksa i osnovne supstance, sa parakeratiniziranim epitelnim slojem i dubokim brazdama koje penetriraju u vezivno tkivo ispod sebe. Razni tipovi uvećanja gingive se karakterišu različitom zastupljenošću proinflamatornih ćelija [4,11,12].

Kod uvećanja gingive izazvanog lekovima vezivno tkivo je bogatije proinflamatornim ćelijama. Prisustvo bakterijskih naslaga i genetska predispozicija smatraju se etiološkim kofaktorom. Otkriveno je da se kod pacijenata koji imaju uvećanu inflamiranu gingivu nakon uvođenja terapije cefalosporinima gingiva još izraženije uveća. Kod prisutnog genetskog polimorfizma u ekspresiji IL-1A obično se javlja uvećanje gingive nakon terapije cefalosporinima [31]. Uvećanje gingive izazvano primenom fenitoina karakteriše se uglavnom fibroznim promenama, dok ono izazvano primenom cefalosporina ili nifedipina zapaljenskim reakcijama [32-35].

Histološke promene kod uvećanja gingive u sklopu sindroma, HGF i uvećanja gingive izazvanog fenitoinom su slične: prisutna je epitelijalna hiperplazija sa hiperkeratozom i produženim papilama, zadebljanje kolagenih snopova, povećana diferencijacija tkiva i varirajući broj fibroblasta [36]. Epitelijalna hiperplazija je posledica akantoze, ali se javlja samo na mestima hronične inflamacije [36,37]. Zastupljenost fibroblasta je diskutabilna, neki autori ističu povećan broj fibroblasta [22,27,35,38,39], dok drugi, naprotiv, navode smanjenje njihovog broja [23,33]. Ovakav nalaz skreće pažnju na različite molekularne mehanizme u toku fibroznih procesa u gingivi.

Uloga mezenhimnih ćelija u uvećanju gingive

Glavne ćelije vezivnog tkiva gingive odgovorne za povećanu sintezu kolagena su

presence of teeth in alveoli seems to be a condition for hereditary GO development which occurs in the primary dentition and disappears or reduces after tooth extraction[13]. Another type of HGF with family aggregation is a mutation also mapped on chromosome 2 (but not to the SOS 1) with the manifestation in elderly [23].

Histological aspects of gingival overgrowth

Histological studies performed on samples of gingival overgrowth tissues revealed common, non-specific finding despite numerous risk factors. It was generally characterized by an increase of gum volume to which contribute both the epithelium (cellular hyperplasia) and lamina propria (accumulation of extracellular matrix and cells)[1,2]. Microscopically, examination displays the coexistence of tissue hypertrophy and cellular hyperplasia. Clinically and histopathologically, there is an excessive accumulation of extracellular matrix proteins and ground substance, with a parakeratinized epithelium and deep ridges penetrating into the underlying connective tissue. Various types of GO are characterized by different incidence of pro-inflammatory cells [4,11,12].

In drug-induced GO, connective tissue is more rich in pro-inflammatory cells. The presence of bacterial plaque and hereditary predisposition are constantly incriminated as etiological cofactors. It was revealed that patients with inflammatory GO developed more severe forms of GO before the onset of treatment with CsA. It is thought that patients carriers of a genetic polymorphism related to IL-1A expression often develop GO after CsA treatment [31]. A phenytoin-induced GO is characterized mainly by fibrotic lesions, whereas CsA or nifedipine-induced GO has inflammatory changes[32-35].

Histological changes of syndromic GO, HGF and phenytoin-induced GO are similar: epithelial hyperplasia with hyperkeratosis and elongated papillae, thickening of collagen bundles, increase of tissue differentiation and fluctuating number of fibroblasts [36]. Epithelial hyperplasia results from acanthosis, however, it appears only in the areas of chronic inflammation [36,37]. The incidence of fibroblast is disputable - some authors reported numerous fibroblasts [22,27,35,38,39] while others mentioned a decreased number[23,33].

fibroblasti. Oni su zaduženi za. sintezu i razgradnju kolagena. Razumevanje na molekularnom nivou ovih procesa može unaprediti i terapiju uvećanja gingive. Posebnu pažnju treba posvetiti faktorima ekspresije gena koji kontrolišu aktivnost fibroblasta u oralnoj mukozi. Oralna mukoza je fiziološki u stalnom stanju zapaljenja i remodelovanja usled kontakta sa različitim spoljašnjim agensima. Pod normalnim uslovima, a posebno u fibrozi organa, fibroblasti su različitog porekla (od trans-diferencijacije epitelijalnih ćelija, proliferacije lokalnih fibroblasta i stem ćelija koštane srži) [40]. Međutim, u fibroznom uvećanju gingive opisane su dve populacije fibroblasta: jedna sa malo citoplazme (inaktivni fibroblasti) i druga sa obilnom citoplazmom, endoplazmatskim retikulumom i Goldžijevim aparatom (aktivni fibroblasti) [25,41].

U gingivi postoje dva načina stimulisanja proliferacije fibroblasta: povećanom ekspresijom sinteze masne kiseline[42,43] ili povećanom ekspresijom nuklearnih proto-onkogena (c-myc)[44]. Smatra se da je nalaz različite stimulacije posledica genetskih razlika ili malog broja proučavanih slučajeva[42]. Prilikom povrede tkiva, lokalni fibroblasti se aktiviraju pod dejstvom citokina oslobođenih iz inflamatornih ćelija ili usled promena u mikrookruženju. Oni postaju najpre protomiofibroblasti, a zatim diferentovani miofibroblasti (ranije zvani aktivni fibroblasti)[45]. Malo je navoda iz literature koji se odnose na postojanje miofibroblasta u reaktivno fokalnom uvećanju gingive ili HGF [19,46,47]. Istaknuto je da su jedina tkiva koja ne stvaraju posttraumatske ožiljke embrionalna tkiva i gingiva. Ova specifična reaktivnost je posledica toga što gingivalni fibroblasti aktiviraju TGFβ1 različitim signalnim putevima[45]. Smatra se da se ožiljci ne javljaju u gingivi, jer se mehanički stres (normalno stanje za funkcionisanje parodonta) i procesi remodelovanja prenose na proliferaciju fibroblasta i produkciju TGF-β1.

Prevencija i terapija uvećanja gingive

Terapija uvećanja gingive zavisi od kliničkog izgleda i etioloških faktora. Terapija izbora za medicinski izazvano uvećanje gingive bila bi prestanak uzimanja leka izazivača. Ipak, ovo obično nije moguće. Efikasna terapija zasniva se na korekciji

This variable evidence points to the different molecular mechanism underlying gingival fibrotic processes.

The role of mesenchymal cells in gingival overgrowth

The main cells of gingival connective tissue incriminated for increased collagen synthesis are the fibroblasts. They are responsible for collagen synthesis and breakdown. Understanding of these processes can develop an effective therapy of GO. Special attention should be paid to factors of gene expression that control the activity of fibroblasts of oral mucosa. Physiologically, it is normally in a continuous state of inflammation and remodelation due to the contact with different external agents. Under normal circumstances and especially in organ fibrosis, fibroblasts have different origins (from the trans-differentiation of epithelial cells, from local fibroblast proliferation and bone marrow stem cells)[40]. In fibrotic GO, two populations of fibroblasts were described: one with little cytoplasm (inactive fibroblasts), and the other with abundant cytoplasm, endoplasmic reticulum and Golgi apparatus (active fibroblasts)[25,41].

There are two ways of stimulating fibroblasts proliferation in the gingiva: the pathway induced by increased fatty acid synthase (FAS) expression[42,43] or by increased expression of a nuclear proto-oncogene (c-myc)[44]. It is thought that these different mechanisms of fibroblast stimulation can be the result of the genetic heterogeneity or by the small number of the studied cases[42]. In tissue injury, the local fibroblasts become activated by local cytokines released from inflammatory cells or by the change of the microenvironment. These cells become first protomyofibroblasts and then differentiated myofibroblasts (previously called activated fibroblasts)[45]. There are a few reports in the literature related to the presence of myofibroblasts in reactive focal GO or HGF[19,46,47]. It was reported that the only tissues that do not develop post-lesion scars are embryonic and gingival tissues. This special reactivity is due to the fact that gingival fibroblasts activate TGFβ1 by different signaling pathways[48]. It is suggested that the lack of scars in the gingival mucosa is due to the fact that mechanical stress (a normal condition

estetskih i/ili funkcionalnih problema i deli se na konzervativnu i hiruršku[22].

Konzervativna terapija

S obzirom da su oralni biofilm i kamenac faktori rizika za nastanak uvećanja gingive, cilj inicijalne parodontalne terapije je smanjenje inflamacije uz pomoć rigorozne oralne higijene kod kuće i profesionalnog stomatološkog čišćenja naslaga. Profilaksa obuhvata instrukcije o održavanju oralne higijene čestim i redovitim četkanjem zuba, upotrebu konca i vodica za ispiranje. Vodice za ispiranje 0,2% hlorheksidinom jako su efikasne kao dopunska metoda mehaničkom četkanju zuba. Primena folne kiseline (lokalno i/ili sistemski) može umanjiti uvećanje gingive u nekim slučajevima, ali njena specifična uloga još nije rasvetljena. Uočeno je spontano povlačenje uvećanja gingive nakon zamene leka izazivača i održavanjem dobre oralne higijene. Na žalost, ne reaguju svi pacijenti jednako dobro na ovaj vid terapije, pogotovo kada su promene gingive prisutne duži vremenski period [22].

Hirurška terapija

Definitivna terapija obuhvata hirurško odstranjenje viška gingive gingivektomijom ili režanj operacijom. Odluka o tome koji od ova dva terapijska pristupa odabrati donosi se individualno u odnosu na: obim oblasti koja zahteva operaciju, prisustvo parodontopatije i koštanih defekata, količinu keratinizirane gingive, odnos parodontalnih džepova i blizine mukogingivalne linije. U novije vreme, sve se više primenjuju laseri u hirurškoj terapiji uvećane gingive zbog svojih prednosti u postoperativnoj hemostazi[22,49].

Iako se recidivi javljaju u određenom broju slučajeva, rigorozna oralna higijena kod kuće, vodice za ispiranje na bazi hlorheksidina i kontrolni pregledi na tri meseca, uz profesionalno uklanjanje naslaga, smanjuju njihovu verovatnoću. Recidiv se može javiti 3-6 meseci nakon hirurške terapije, ali uglavnom remisije traju najmanje 12 meseci [22,49].

for functional periodontal tissues) and remodeling processes are translated into fibroblast proliferation and production of TGF-β1.

Prevention and treatment of gingival overgrowth

The treatment of GO depends on clinical appearance and etiological factors of GO. The treatment of choice for medically induced gingival overgrowth would be the discontinuation of the associated medication. Nevertheless, this is often not possible. Effective treatment focuses on the correction of the aesthetic and/or functional problems, and it can be divided into nonsurgical or surgical alternatives [22].

Nonsurgical therapy

The plaque and calculus are the risk factors for GO development. Therefore, the initial periodontal therapy should be aimed at reducing the inflammation comprising comprehensive oral hygiene at home along with regular professional debridement. Prophylaxis includes oral hygiene instructions with frequent and correct brushing of teeth, use of floss and rinses. The use of a 0.2% chlorhexidine mouthwash is a highly beneficial adjunctive regimen to mechanical oral hygiene methods. The administration of folic acid (topical and/or oral) could ameliorate gingival overgrowth in some cases, but its specific role has not clearly been established. Spontaneous remission of GO has been shown to take place following a change in medication combined with maintaining good oral hygiene. Unfortunately, not all patients respond to this mode of treatment, especially when the gingival lesions have been long-standing [22].

Surgical therapy

Definitive treatment involves surgical elimination of the excess gingival tissue through implementation of either the gingivectomy procedure or periodontal flap approach. The clinician's decision to choose between these two surgical techniques should be made on an individual basis, encompassing careful consideration of the following aspects: the extent of area requiring surgery; the presence of periodontitis and osseous defects; the amount of keratinized gingiva, and the position of the base of the pockets in relation to the existing mucogingival junction. The use of laser surgical therapy is also becoming more

Zaključak

Uvećanje gingive nije retka pojava i od velikog je značaja kao potencijalni pokazatelj sistemskog oboljenja. Može postojati kao samostalno oboljenje, u sklopu sistemskog oboljenja ili kao deo sindroma. Iako je za neke stomatologe pitanje od malog značaja, zdravlje gingive je preduslov za psihičko i fizičko zdravlje pacijenata. U velikom broju slučajeva, klinički izgled različitih tipova uvećanja gingive je sličan i sveobuhvatna terapija može biti pravi multidisciplinarni izazov.

Neophodna su dalja istraživanja u vezi sa etilogijom, manifestacijom, patogenezom, prevencijom i multidisciplinarnom terapijom uvećanja gingive, a posebnu pažnju treba posvetiti faktorima koji regulišu vezu između etiologije i izraženostiuvećanja gingive.

Zahvalnost

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common in the treatment of GO due to its advantages in postoperative haemostasis[22,49].

Although recurrences can occur in some cases, meticulous home care, chlorhexidine gluconate rinses, and close 3-monthly maintenance and professional debridement following surgery will minimize this possibility. Relapse may occur 3-6 months after surgical treatment, but in general, remission last for at least 12 months[22,49].

Conclusion

Gingival overgrowth is not a rare condition and is important because of its potential as an indicator of systemic disease. GO may exist as an isolated abnormality, with systemic disease or as part of a syndrome. Even it seems for some dentists an issue of little significance, the health of gums is a prerequisite condition for a psychological and physical health of patients. Its clinical appearance is similar in most cases and the comprehensive management may be a real multidisciplinary challenge.

Further studies are needed regarding the etiology, manifestation, pathogenesis, prevention and multidisciplinary management of gingival overgrowth and special attention should be paid to factors that govern the relationship between the etiology and severity of gingival overgrowth.

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