

Relationship between Periodontal Pathogens and Fundamental Ailment of Systematic Diseases

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Abstract

A developing collection of writing proposes that there is a connection among periodontitis and foundational illnesses. These illnesses incorporate cardiovascular sickness, gastrointestinal and colorectal malignant growth, diabetes and insulin obstruction, and Alzheimer's ailment, just as respiratory tract contamination and antagonistic pregnancy results. The nearness of periodontal pathogens and their metabolic side-effects in the mouth may in actuality tweak the resistant reaction past the oral depression, therefore advancing the improvement of fundamental conditions. A circumstances and logical results relationship has not been set up yet for the greater part of the ailments and the middle people of the affiliation are as yet being distinguished. A superior comprehension of the fundamental impacts of oral microorganisms will add to the objective of utilizing the oral depression to analyze and potentially treat non-oral foundational sickness.

Keywords: Oral pathogens; Systemic ailment; periodontal sickness; chronic infection; Inflammation; Dentistry

Introduction

Periodontal infection is one of the most widely recognized incendiary illnesses in grown-ups. In 2010, 3.9 billion individuals overall were accounted for to have periodontal ailment, with the pervasiveness of mellow periodontitis being 35% and moderate to serious periodontitis, 11% [1]. As the worldwide populace ages, periodontal infection has become a noteworthy general wellbeing concern and a mounting trouble on the medicinal services framework [2]. As indicated by the US Centers for Disease Control and Prevention, periodontal malady is viewed as an overall pandemic, causing incapacity, discourse debilitation, low confidence, and diminished personal satisfaction [2].

The investigation of periodontal pathogens and aggravation has pulled in the consideration from scientists outside of dentistry because of the expected impact of periodontitis on commencement or potentially movement of a few foundational ailments. Throughout the years, proof has aggregated that joins oral maladies with numerous non-oral and fundamental sicknesses, including malignant growth, cardiovascular ailment, type 2 diabetes, respiratory tract contamination, unfriendly pregnancy results, and neurodegenerative illness [3].

Generally, in any case, it stays to be set up whether explicit periodontal pathogens animate improvement of the fundamental illness, or if the foundational sickness makes the wealth of periodontal pathogens change. On the off chance that the pathogens cause non-oral sickness, at that point they would speak to clear focuses for remedial mediation. However, as a base, the nearness of periodontal pathogens could be utilized as demonstrative markers to foresee defenselessness to non-oral ailment. Periodontal pathogens could advance improvement of non-oral ailment legitimately or in a roundabout way. For instance, around 30 bountiful species in the oral pit, predominantly Gram-negative anaerobic microbes, are known to deliver endotoxins, which could straightforwardly add to foundational malady [4-6]. Movement of oral pathogens to the circulatory system could likewise happen now and again, for example, following surgeries. Bacterial aggregation on the teeth because of helpless dental cleanliness or potentially ecological elements actuates a host provocative reaction, which may bring about periodontitis and bone misfortune [7] yet could likewise be hurtful to the host foundationally.

Dental Plaque, Periodontal Pathogens and Bacteremia

The most widely recognized Gram-negative bacterial genera in the oral cavity incorporate Treponema, Bacteroides, Porphyromonas, Prevotella, Capnocytophaga, Peptostreptococcus, Fusobacterium, Actinobacillus, and Eikenella [8]. Early examinations recognized Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans and Tannerella forsythia as causative operators in periodontal malady [9], and a great part of the exploration on periodontal infection keeps on concentrating on these microorganisms. Nonetheless, later examinations have verified that the oral pit contains around 500–700 predominant taxa; this microbial network is alluded to as the oral micro biota, oral micro-flora, or oral micro biome [10]. The oral micro biota is available in salivation, on gingival epithelium and other inward surfaces of the oral hole and amassed in dental plaque.

The dental plaque is a sorted out biofilm of microorganisms that are either joined to the tooth surface or to different microorganisms in a manner that permits the microorganisms to endure and oppose have safeguard systems or anti-infection treatment [11]. As the biofilm develops, microbial dysbiosis happens, causing a dynamic move from Gram-positive to dominatingly Gram-negative anaerobic species and coming about in biofilm arrangement under the gingival surface [12]. Also, sugar digestion by the dental plaque biofilm prompts creation of natural acids, which assume a significant job in pH decrease and demineralization of the tooth surface [13]. Accordingly, visit sugar utilization is likewise known to prompt dysbiosis of the supragingival microbiota, advancing advancement of carious injuries [12].

Tissue injury, flossing, dental strategies, or in any event, biting food may instigate breakage of veins in closeness to the dental plaque, which can bring microscopic organisms into the fundamental circulatory system [14]. Bacteremia has in certainty been watched following some dental or clinical strategies, and a few microbes were detached from the blood after endodontic treatment [15, 16].

Connection among oral and non-oral foundational malady

Numerous on-going investigations investigate the interrelationship between oral wellbeing, irritation, and foundational illness Oral microbiota can cause oral irritation however may likewise legitimately add to fundamental aggravation, expanding aggravation through the arrival of poisons or spillage of microbial items into the circulation system. The relationship between oral aggravation and foundational irritation is basic to understanding the inconvenient impacts of oral irritation on a few organ frameworks and the capacity of oral illness to build the danger of creating non-oral ailment. We consider proof connecting oral illness with some major fundamental non-oral sicknesses in the areas beneath.

Cardiovascular illness

Cardiovascular illness is viewed as the main source of death in the U.S. what's more, is a significant reason for handicap as per the CDC. Given its high monetary and social effect, the relationship among cardiovascular and periodontal malady has stood out of numerous analysts. Albeit different epidemiological examinations have recommended that there might be a relationship among periodontitis and cardiovascular ailment, the effect of oral contamination on cardiovascular infections has stayed indistinct.

A meta-investigation that joined 5 companion contemplates (86,092 patients) indicated that people with periodontal malady had 1.14 occasions higher danger of creating coronary illness than the controls, freely of puzzling variables. The case-control examines (1423 patients) demonstrated a significantly more serious danger of creating coronary illness (2.22 occasions). This investigation demonstrated that both commonness and frequency of cardiovascular malady are essentially expanded in patients with periodontitis. In addition, a relationship among edentulousness and serum antibodies against P. gingivalis and A. actinomycetemcomitans with coronary illness was seen in an investigation with 1163 men [16]. An extra examination affirmed the nearness of bacterial DNA species in 42 atheromatous plaques recovered by endarterectomy [9]. The bacterial species most usually found in this examination were P. gingivalis, trailed by A. forsythia, Eikenella actinomycetemcomitans, T. corrodens, Fusobacterium nucleatum and Campylobacter rectus [9]. Along comparative lines, DNA from periodontal pathogens, for example, P. gingivalis, A. actinomycetemcomitans, Prevotella intermedia, and T. forsythia, was found in human atherosclerotic plaques, proposing that these oral pathogens may move from the oral cavity to inaccessible locales of the body [2].

In synopsis, a few oral pathogens are related with a higher danger of cardiovascular malady in people, and studies in mice bolster the likelihood that contamination with the oral pathogens may prompt the ailment.

Respiratory tract contamination and pneumonia

Pneumonia is a huge reason for dreariness and mortality in patients everything being equal, particularly in the old and immune compromised. The lung contaminations can be brought about by microbes, growths, infections and parasites. Microorganisms can contaminate the lower respiratory tract by inward breath of irresistible mist concentrates, spread of disease from touching locales, and spread from extra pulmonary destinations. The oral hole particularly spit and dental plaque in patients with periodontal malady by all accounts a sensible hotspot for pathogens to aggregate and spread to the lower aviation routes. A few oral pathogens have just been embroiled in lung diseases, including A. actinomycetemcomitans, Actinomyces israelii, Capnocytophaga spp, Chlamydia pneumoniae, E. corrodens, F. nucleatum, Fusobacterium necrophorum, P. gingivalis, P. intermedia and Streptococcus constellatus.

Respiratory pathogens segregated from dental plaque and bronchoalveolar lavage liquid from similar patients in the emergency unit demonstrated to be hereditarily the equivalent, which fortifies the view that dental plaque could fill in as a noteworthy repository for respiratory pathogens [3]. Indeed, people with periodontitis are multiple times bound to create nosocomial pneumonia, contrasted and patients without periodontitis [1]. In an intratracheal mouse model of contamination, P. gingivalis was liable for steady provocative reactions in the lungs including cell enrollment and proinflammatory cytokine creation [7]. Curiously, an examination with 40 subjects experiencing orotracheal intubation indicated huge amounts of A. actinomycetemcomitans, P. gingivalis and T. forsythia in toothed and innocuous patients, recommending that the oral condition, even without teeth, presents positive conditions for pathogenic bacterial collection [2].

The regular oral pathogens F. nucleatum and F. necrophorum were found to cause an unmistakable condition starting with pharyngitis and prompting respiratory tract contamination called Lemierre's disorder [8, 9]. Strangely, a cross-sectional investigation of understudies by introducing intense sore throat recognized F. necrophorum in 20.5% of subjects and 9.4% of asymptomatic people, which was roughly twice as frequently as the typically researched bunch A β -hemolytic streptococcus (10.3% of patients and 1.1% of asymptomatic subjects) [3]. These examinations propose that Fusobacterium is an expected pathogen of the lungs and ought to be considered when exploring aviation route difficulties.

Then again, C. pneumoniae is very much concentrated as a respiratory pathogen and has been related with asthma, bronchitis and interminable obstructive aspiratory infection [4]. This pathogen has additionally been found in the oral cavity [6] and could likely translocate from the oral cavity to the lower aviation routes, from where it could disperse fundamentally to different destinations, for example, spleen, heart, and aorta by means of monocytes through blood dissemination, as recommended in a mouse model [7]. Besides, C. pneumoniae contamination has been related with an expanded danger of atherosclerosis advancement [8], which could give another instrument whereby pathogens in the oral pit add to atherosclerosis.

Together, these investigations exhibit that undesirable oral holes can incline to respiratory contaminations and propose that oral or nonoral pathogens present in the oral depression could add to respiratory sickness.

Respiratory tract contamination and pneumonia

Disease represents one out of four passings consistently, costing the US roughly \$100 billion in medicinal services other than the passionate and clinical weight that it speaks to families and society. In the mid-1990s, Helicobacter pylori was perceived as a causative specialist of human gastric malignancy [9], turning into the main major bacterial pathogen to be related with human disease [4]. In this survey, we will concentrate on the connection between periodontal bacterial pathogens and malignant growth.

A meta-examination study including 3183 subjects indicated that patients with periodontal malady have an expanded vulnerability to oral malignant growth [2]. Later investigations found a positive connection between are periodontal illness and pancreatic, head and neck, and lung diseases [4]. Another investigation inspected one million arbitrarily chose protection cases in Taiwan, and found that patients in the periodontitis companion showed a higher danger of creating malignant growth than those in the gum disease partner [3].

In addition, the periodontal pathogen P. gingivalis was found at fundamentally raised levels in Oral Squamous Cell Carcinoma (OSCC) [4] and Throat Squamous Cell Carcinoma (TSCC) patients, yet not in sound mucosa [1]. The expected job for periodontal pathogens in the enlistment of oral malignancy was affirmed in an oral-explicit concoction carcinogenesis creature model [5]. The investigation indicated that the periodontal pathogens P. gingivalis and F. nucleatum animate tumorigenesis by means of direct collaboration with oral epithelial cells, and that the impact is interceded by the host inborn resistant framework [5]. On account of OSCC, it was shown that P. gingivalis, however not F. nucleatum, advances attack and metastasis of oral squamous cells by initiating framework metalloproteinase 9 (professional MMP9) articulation [4]. Another investigation demonstrated that drawn out and redundant introduction to P. gingivalis builds forcefulness of oral disease cells by means of epithelial-mesenchymal progress like changes in the cells [6]. Hence, oral pathogens, explicitly P. gingivalis and F. nucleatum, have been appeared to decidedly associate with improvement of oral malignant growth, proposing that they could be biomarkers for beginning phases of the sickness, or even focuses for counteraction of oral diseases in people.

ColoRectal Carcinoma (CRC) is the fourth driving reason for malignant growth passings around the world, and has been related with a high plenitude of F. nucleatum and Clostridium difficile in the intestinal microbiota of colorectal carcinoma patients [7]. A polymicrobial mark of Gram-negative anaerobic microorganisms was related with colorectal carcinoma in 130 tissues investigated [8]. Gram-negative anaerobic oral pathogens, for example, Fusobacterium, Leptotrichia and Campylobacter species were recognized in people with tumors [8]. It is believed that oral F. nucleatum could relocate to and colonize the human intestinal tract, to cause harmful provocative contaminations [9]. Moreover, F. nucleatum was found in higher numbers in human colonic adenomas comparative with encompassing tissues and in feces tests from patients with CRC, contrasted and subjects without CRC [5]. Also, in a mouse model of intestinal tumorigenesis, F. nucleatum expanded tumor assortment and specifically selected tumor-invading myeloid cells, which can advance tumor movement [10]. Another robotic examination indicated that

degrees of F. nucleatum steadily expanded during the colorectal adenoma-carcinoma arrangement in human fecal and mucosal examples [5]. F. nucleatum capacity to adjust the creation of the lumen microbiota just as its capacity to intervene discharge of cytokines and enact tumorigenesis-related pathways was exhibited in mouse models of CRC [1].

Diabetes mellitus

Diabetes mellitus is an incessant metabolic issue described by hyperglycemia because of a deformity in insulin creation by pancreatic β cells (type 1 diabetes), a reduction in insulin affectability (type 2 diabetes), or a mix of both [2] that can influence grown-ups, young people and kids. Critical, diabetes mellitus and periodontitis present a "two-way" relationship in which one influences the other. Interminable disease during periodontitis can prompt exacerbated and dysregulated incendiary reactions, which may bring about poor metabolic control of glucose and expanded insulin prerequisites [3]. Truth be told, people with intense bacterial and viral disease showed extreme and enduring insulin opposition [4]. This thought was affirmed by an investigation with 124 moderately aged men indicating that the weight of enteroviruses and C. pneumoniae related unequivocally with insulin opposition, most likely in light of the interminable second rate aggravation coming about because of these contaminations [5]. As to Gingivalis disease, a decline in gingival vascular capacity and expanded insulin opposition was seen in a diabetes model in rodents [6]. Strangely, results from a meta-examination study recommended that periodontal treatment prompts an improvement of glycemic control in type 2 diabetic patients, for at any rate 3 months [7]. All in all, despite the fact that the instruments fundamental this affiliation are not finished comprehended, periodontitis appears to build the danger of diabetes because of contamination or potentially incendiary reactions [8].

On the other hand, diabetes can likewise prompt various confusions, for example, helpless injury mending, retinopathy, nephropathy, neuropathy, macro vascular infection and periodontitis. Truth be told, diabetic people have a triple increment in the danger of periodontitis, contrasted and non-diabetics subjects [9]. In another examination, periodontitis was found in 58% of type 1 diabetes patients and in 15% of non-diabetic controls [6]. At the point when periodontal status in youngsters and youths with type 1 diabetes mellitus was analyzed, a pervasiveness of 21% of gum disease and 6% of periodontitis was recognized; additionally, patients having lived over five years with diabetes mellitus type 1 demonstrated progressively influenced locales on periodontal sickness boundaries [1].

In outline, a bidirectional relationship between diabetes mellitus and periodontal malady has been appeared, in which diabetes improves the hazard for periodontitis, and then again, periodontal aggravation contrarily influences glycemic control. Also, periodontal treatment improves diabetes side effects, affirming their affiliation and the significance of oral wellbeing for the general living being.

Alzheimer's infection

Alzheimer's infection is a dynamic neurodegenerative ailment portrayed by a dynamic and irreversible hindrance in memory, thinking, and language and learning limit, which at last finishes in death [2]. The psychological decay has been identified with the development of synaptotoxic β -amyloid plaques and

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hyperphosphorylated tau proteins in the areas of the mind related with cutting edge intellectual capacities [2,3]. As appeared for diabetes mellitus, Alzheimer's malady and periodontitis likewise present a bidirectional relationship that will be talked about in this segment.

An on-going far reaching oral-wellbeing study demonstrated that people with cerebrum injury had a higher pervasiveness of helpless oral wellbeing boundaries and ceaseless summed up periodontitis [4]. The cerebrum, which was thought to have no or lessened resistant reactions on account of its "immunologic benefit" status, can experience diverse fiery procedures that add to advancement of Alzheimer's malady, for example, supplement initiation and cytokine and chemokine articulation [5]. Truth be told, aggravation is seen as the connection among periodontitis and Alzheimer's illness. The nearness of actuated glial cells that produce noteworthy degrees of fiery cytokines is a sign of Alzheimer's illness [6]. Other than the immediate harm brought about by β-amyloid plaques and tau totals, the intrinsic resistant reaction endeavors to cleanse these totals from the cerebrum, however rather irritates neuro-degeneration. Accordingly, an expansion in genius fiery cytokines is identified in old patients with Alzheimer's infection and periodontitis [7]. Studies utilizing distinctive calming medications and cytokines strengthen the theory that aggravation is a significant driver of neuro-degeneration in Alzheimer's malady, recommending that Nasal Non-Steroidal Mitigating Drugs (NSAIDs) may be compelling in easing back the beginning of Alzheimer's illness [6]. What's more, the IL-1 receptor foe and immunosuppressive cytokines can shield the cerebrum from further harm and decline the pace of Alzheimer's illness movement.

The host combines professional fiery cytokines foundationally because of oral bacterial contamination, recommending that periodontal sickness may add to the cerebrum aggravation that portrays Alzheimer's illness [7]. Strikingly, Lipopolysaccharide (LPS) from periodontal pathogens, for example, P. gingivalis and T. denticola was confined from momentary posthumous Alzheimer's infection human minds, recommending that destructiveness factors from these pathogens could assume a job being developed of cerebrum aggravation and Alzheimer's sickness [1]. Additionally, microscopic organisms, for example, the periodontal pathogen T. denticola [2] and C. pneumonia were recognized in after death Alzheimer's infection cerebrums, recommending that other than incendiary middle people, some periodontal pathogens may attack the mind by intersection the mind blood hindrance. This was affirmed in creature considers, which indicated the nearness of P. gingivalis in mouse cerebrums [5]. In addition, more significant levels of antibodies against A. actinomycetemcomitans, P. gingivalis, T. forsythia [7], F. nucleatum and P. intermedia [6] were seen in old patients with Alzheimer's illness, contrasted and sound controls. In spite of the fact that these periodontal pathogens appear to be related with side effects of Alzheimer's illness, further longitudinal examinations will be expected to straightforwardly interface periodontal pathogens (and antibodies against them) with neurodegeneration in Alzheimer's malady.

In rundown, helpless oral cleanliness adds to ceaseless periodontitis and may in a roundabout way increment the hazard for Alzheimer's sickness. On the other hand, patients with Alzheimer's infection present weaknesses in the capacity to keep up legitimate oral cleanliness or even visit a dental specialist for proficient consideration, which upgrades the hazard for periodontitis. In this sense, it is enticing to guess that keeping up great oral wellbeing could turn into a prophylactic measure against Alzheimer's sickness.

Antagonistic pregnancy results

Maternal contaminations are related with unfriendly pregnancy results, including preterm work, preterm untimely break of the layers, pre-eclampsia, premature delivery, intra-uterine development impediment, low birthweight, stillbirth, and neonatal sepsis [7]. Because of hormonal changes in pregnant ladies, they are more defenseless to gum disease and periodontitis than non-pregnant ladies [8]. In reality, around 40% of pregnant ladies exhibit clinical proof of periodontal ailment [8].

Two unique components have been proposed to clarify how oral wellbeing is related with unfavorable pregnancy results. The first recommends that oral pathogens themselves can translocate from an unfortunate oral hole and cross the placenta, coming to the intraamniotic liquid and fetal course [9]. The second theorizes that the foundational scattering of endotoxins or fiery arbiters got from periodontal illness could influence advancement of the hatchling or unconstrained premature birth [8].

Bacterial pathogens, antigens, endotoxins, and star fiery cytokines created during periodontal sickness can cross the placental boundary. bringing about unsettling influences in the maternal-fetal unit that could add to unfriendly pregnancy results. F. nucleatum is the most widely recognized oral pathogen found in an assortment of placental and fetal tissues [7]. A case report of term stillbirth recommended that F. nucleatum could translocate from the mother's mouth to the uterus when her invulnerable reaction was debilitated during a respiratory disease. Reliable with its high obtrusiveness, F. nucleatum is over and over segregated from amniotic liquid and rope blood in instances of preterm birth and neonatal sepsis [5]. Besides, F. nucleatum is frequently recognized alongside other oral subspecies in intrauterine diseases, which are likely from the equivalent irresistible inception, inferring co-translocation from the oral cavity. Other oral pathogens, for example, P. gingivalis (and its endotoxins), were likewise found in the placenta of preterm conveyance patients. Studies in creature models show the capacity of P. gingivalis to affect contrarily on pregnancy: LPS from P. gingivalis instigated placental and fetal development limitation and desorption in rodents and antibodies raised against P. gingivalis caused fetal misfortune when latently regulated into mice.

The maternal-fetal interface speaks to an immunologically exceptional site that must elevate safe resistance to the hatchling while simultaneously keeping up a vigorous host guard against potential contaminations. Despite the fact that little is thought about the job of intrinsic invulnerable receptors during pregnancy, it was realized that the placenta communicates Toll-Like Receptors (TLRs) during typical pregnancy. Periodontal infection or the nearness of periodontal pathogens, for example, T. denticola and P. gingivalis has been appeared to expand the statement of TLRs, recommending expanded natural invulnerable reactions.

Albeit more examinations will be required to build up decisively that there is a circumstances and logical results connection between periodontal ailment and unfriendly pregnancy results, the outcomes so far recommend that preventive measures against periodontal malady in pregnant ladies are justified.

Conclusion

A developing group of proof in the writing shows the immediate and aberrant effect of periodontal pathogens on generally wellbeing.

Late epidemiological, clinical and test considers bolster the connection between bacteraemia or irritation because of periodontal malady and fundamental infection. More examinations are expected to clarify the instruments whereby periodontal pathogens or the resulting irritation cause or add to foundational ailment. Regardless, it is as of now certain that administration of periodontal sickness and legitimate oral consideration can decidedly affect the bleakness, mortality, and medicinal services costs related with non-oral foundational illnesses

Conflicts of Interest

The authors declare that they have no conflicts of interest.

References

- 1. Cummins D (2009) Dentin hypersensitivity: From diagnosis to a breakthrough therapy for everyday sensitivity relief. J Clin Dent 20:1.
- Bubteina N, Garoushi S (2015) Dentine hypersensitivity: A review. Dentistry 330: 2161-1122.
- West N, Seong J, Davies M (2014) Dentine hypersensitivity. Monogr Oral Sci 25:108-122
- 4. Davari AR, Ataei E, Assarzadeh H (2013) Dentin hypersensitivity: Etiology, diagnosis and treatment; a literature review. J Dent 14:136.
- Zeola LF, Soares PV, Cunha-Cruz J (2019) Prevalence of dentin hypersensitivity: Systematic review and meta-analysis. J Dent 81:1-6.
- Porto IC, Andrade AK, Montes MA (2009) Diagnosis and treatment of dentinal hypersensitivity. J Oral Sci 51:323-332.
- 7. Miglani S, Aggarwal V, Ahuja B (2010) Dentin hypersensitivity: Recent trends in management. J Conserv Dent 13:218.
- Kleinberg I. SensiStat (2002) A new saliva-based composition for simple and effective treatment of dentinal sensitivity pain. Dent Today 21:42.

- Lata S, Varghese NO, Varughese JM et.al (2010) Remineralization potential of fluoride and amorphous calcium phosphate-casein phospho peptide on enamel lesions: An in vitro comparative evaluation. J Conserv Dent 13:42.
- Merh A, Singhbal K, Parikh V, Mehta S, Kulkarni G et.al (2015) Comparative evaluation of immediate efficacy of diode laser versus desensitizing paste containing 8% arginine and calcium carbonate in treatment of dentine hypersensitivity: An in vivo study. J Evol Med Dent Sci 4:4346-4356.
- 11. Panagakos FO, Schiff T, Guignon A et.al (2009) Dentin hypersensitivity: effective treatment with an in-office desensitizing paste containing 8% arginine and calcium carbonate. Am J Dent 22:3A-7A.
- Petrou I, Heu R, Stranick M, Lavender S, Zaidel L, et.al (2009) A breakthrough therapy for dentin hypersensitivity: How dental products containing 8% arginine and calcium carbonate work to deliver effective relief of sensitive teeth. J Clin Dent 20:23-31.
- Douglas de Oliveira DW, Marques DP, Aguiar-Cantuária IC, Flecha OD, Gonçalves PF (2013) Effect of surgical defect coverage on cervical dentin hypersensitivity and quality of life. J Periodontol 84:768-775.
- Biagi R, Cossellu G, Sarcina M, Pizzamiglio IT, Farronato G (2015) Laser-assisted treatment of dentinal hypersensitivity: a literature review. Ann Stomatol 6:75.
- 15. de Fátima Zanirato Lizarelli R, Miguel FA, Villa GE, de Carvalho Filho E, Pelino JE, et.al (2007) Clinical Effects of Low-intensity Laser vs. Light-emitting Diode Therapy on Dentin Hypersensitivity. J Oral Laser Applications 7: 129-136.
- Alcântara PM, Barroso NF, Botelho AM, Douglas-de-Oliveira DW, Gonçalves PF, et.al (2018). Associated factors to cervical dentin hypersensitivity in adults: A transversal study. BMC Oral Health. 18:155.

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