American Journal of Medicine and Medical Sciences 2024, 14(2): 369-373
DOI: 10.5923/j.ajmms.20241402.45

Modern Aspects of the Etiology and Pathogenesis of Chronic Periodontitis in Children

Safarov Sherzod Umarovich

Bukhara State Medical Institute, Uzbekistan

Abstract The article provides a comprehensive examination of the fundamental causative elements and immunological mechanisms that contribute to the resistance of the oral cavity, leading to the development of inflammatory periodontal diseases. Research has demonstrated that the primary cause of periodontal disease is microbial oral plaque. The quantitative and typological makeup of the specific microflora does not necessarily align with the clinical aspects of the disease. The relationship between the presence of microbes and the strength of the immune system in an organism is most accurately explained by the current understanding of the causes and development of periodontitis.

Keywords Chronic periodontitis in children, Diagnosed using various approaches, Dental and maxillofacial system

1. Introduction

In terms of prevalence in children, periodontitis ranks third after caries and pulpitis. However, this prevalence has not been studied well enough [1]. An individualized examination and evaluation are necessary to analyze the prevalence of apical periodontitis in youngsters. Apical periodontitis is detected according to certain criteria and diagnosed in more than 50% of schoolchildren [2]. The likelihood of developing apical periodontitis is greatly enhanced following root canal treatment and is influenced by the quality of the root filling. The prevalence of apical periodontitis in children was found to be 38.5%, indicating a significant occurrence of the condition. Apical periodontitis often arises from oral trauma, temporary tooth decay, inadequate root filling, and insufficient treatment and healing. The incidence of apical periodontitis in this cohort of children is greater compared to other age groups [3].

Periodontal diseases have a frequency of 90% in youngsters. Presently, inflammatory periodontal diseases are a significant and urgent issue in dentistry, particularly among children [4].

2. The Aim of the Study

The objective of this study is to examine contemporary mechanisms underlying the development and progression of chronic periodontitis in childhood, with the aim of formulating suggestions to enhance dental health.

The significance of studying the etiology and pathogenesis of periodontal diseases is underscored by their high prevalence in different countries. This is due to the fact that periodontal infection acts as a catalyst and supporting mechanism for the emergence of systemic pathology, thereby determining its overall medical and social importance [5].

The microbial component, as a significant causative agent, induces many clinical symptoms of periodontal disorders. The composition and species structure play a crucial role in their pathogenesis simultaneously. The specificity of microorganisms in plaque, its volume, duration of stay in areas of the gingival mucosa and periodontal tissues [6].

Odontogenic infections, such as periodontitis, are widespread among children and adolescents [7]. According to research results, periodontitis is diagnosed in 30-35% of children seeking dental care [8]. Some authors report the occurrence of chronic periodontitis in children in 60.3% of cases [9].

Given that adult diseases sometimes have their roots in childhood, an imperative objective of pediatrics is the prompt identification of cardiovascular problems arising from persistent localized infectious pathology. Currently, there is a widespread consensus that endothelial dysfunction plays a crucial role in the development of many cardiovascular illnesses. Endothelial dysfunction is linked to the progression of atherosclerosis, coronary heart disease, hypertension, chronic heart failure, and poor pulmonary hemodynamics. Dysfunction of the endothelium in children can be an early, preclinical indicator of the development of a severe cardiovascular disease. This warrants additional comprehensive examination of the kid, as well as therapeutic and preventive interventions [10].

Currently, a number of dystrophic-degenerative diseases of the dentoalveolar junction are known, leading to tooth loss. One of the most common complications of caries,

Received: Jan. 31, 2024; Accepted: Feb. 15, 2024; Published: Feb. 17, 2024
Published online at http://journal.sapub.org/ajmms

chronic periodontitis, deserves special attention. The essence of chronic periodontitis is a prolonged inflammatory process in the periapical region of the tooth, causing destruction of the tissues surrounding the tooth [11-13].

Recently, there has been a growing consensus on the genesis of chronic periodontitis, with two distinct perspectives emerging: one suggests that specific bacteria might trigger inflammation at the tooth's apex, while the other proposes that local protective mechanisms in the body are compromised. Given these pathogenetic pathways, it is challenging to conceive that they occur without the participation of the immune system. The authors' focus in this work is on investigating the connection between innate and adaptive immune responses in chronic periodontitis. Specifically, they examine the role of immune systems, such as cytokines and complement, at both the systemic and local levels. Attributing these systems solely to either innate or acquired immunity is challenging, as most effector reactions cannot occur without their involvement [14].

The development of "immune deficiency" in chronic inflammatory process, including in conditions of dental pathology [15]. It is the basis for searching for ways to effectively correct existing violations. At the same time, drugs that can affect the activity of cytokines and the complement system, their main producers, as well as accelerate the processes of reparative tissue regeneration come into view [16-18].

Periodontitis is a result of untreated pulpitis or damage. Both a blow and an expanded seal can cause injury to the periodontium. If the filling disrupts the normal chewing process, it might cause excessive stress on the tooth, ultimately leading to the development of a disease. The symptoms of periodontitis include odontalgia upon occlusion and the sensation of dental elongation. This occurs due to the accumulation of purulent exudate near the apex of the root. The dentist will perform an incision on the tooth, extract the accumulated pus, and provide a course of anti-inflammatory medication. Once the pain from biting subsides, the dentist will proceed to cleanse and disinfect the tooth canals before sealing them. If the channels are not well sealed at the tops, the pathological process will persist and eventually lead to an aggravation. Healing teeth with inadequately sealed channels is highly challenging, and in some cases, it is outright impossible. If it is not feasible to close or block these channels, the tooth must be extracted. Untreated or inadequately treated periodontitis can result in the emergence of sinusitis and periostitis. Sinusitis is the inflammation of the air-filled cavities of the upper jaw, where a pus-filled infection can extend to the ends of the teeth used for chewing. Periostitis manifests as the presence of swelling and agonizing pain. Failure to perform an incision may lead to the emergence of serious consequences such as abscess (localized purulent inflammation), phlegmon (widespread purulent inflammation), or osteomyelitis (death of bone tissue). The numerous complexities present a significant risk to both health and survival [19].

Chronic periodontitis primarily impacts adults, although aggressive periodontitis may also manifest in children. The onset and progression of periodontal disease occur due to an imbalance in the normal oral microbiota (dental plaque), which then interacts with the host's immune defenses, resulting in inflammation and the development of the disease. This pathological condition continues during periods of activity and inactivity, until the diseased tooth is removed or the microbial biofilm is effectively eliminated and the inflammation diminishes. The severity of periodontal disease is determined by several environmental and host risk factors, which can be classified as either modifiable (such as smoking) or non-modifiable (such as inherited vulnerability). To prevent this, individuals should practice regular dental hygiene and have the bacteria biofilm removed by a professional every three to six months [20,21].

The incidence of gingivitis in children can be comparable to or higher than dental caries, however it has been relatively overlooked in terms of comprehending its lasting effects on general well-being. Oral health clinicians should be aware that the clinical signs of gingivitis progression or severity in baby teeth are only visible when there is a significant amount of inflammatory cells present in the gums, which can be observed through inflamed tissues. In addition, although the clinical appearance of chronic inflammation of the periodontal tissues in childhood may seem harmless, it has the potential to cause local tissue destruction, leading to periodontitis. Furthermore, it can create an environment in the tissues that increases the risk of negative effects on tissue health throughout a person's life. The manuscript provides essential insights into the features of chronic inflammation in the gingival tissues of children and adolescents. It also discusses the potential long-term consequences of gingival and periodontal infections during childhood on oral and systemic health in adulthood [22].

The mouth cavity serves as an ecological habitat that harbors a diverse and abundant population of microorganisms. Typically, the mouth cavity contains between 300-400 distinct species of bacteria. They maintain continuous interaction with the external environment, utilize diverse food sources for essential functions, and thrive under optimal conditions for microbial reproduction (such as humidity, temperature, pH levels, etc.) [23].

The microbiota of the oral cavity is subject to constant influence from a range of protective and regulatory factors, including neurogenic and immunological mechanisms. The oral cavity's microbiocenosis possesses the capacity to autonomously manage and uphold ecological equilibrium. This ability is predominantly influenced by both overall and specific factors pertaining to the interaction with oral cavity tissues and secretions [24].

The mucosal membrane is protected from colonization by normal microflora [25] and the activation of nonspecific and immunological protection responses.

Bacteria infiltrate tissues, infecting cells and generating poisons and metabolic byproducts. Bacterial virulence can manifest through both direct toxic effects, which lead to inflammation and destruction, and indirect mechanisms,

such as the stimulation of immunopathological destructive processes [26]. One instance of bacteria's direct harmful effect is leukotoxin, an exotoxin generated by the periodontopathogenic Actinobacillusactinomycetemcomitans. This toxin leads to the destruction of polymorphonuclear leukocytes through lysis. The indirect pathogenic action of bacteria is exemplified by the production of proteolytic enzymes such as phosphatase, aminopeptidase, protease, phosphoamidase, and glycosidase, as well as hyaluronidase, chondroitin sulfatase, and fibrinolysin. Additionally, bacteria can induce the production of similar enzymes by periodontal cells and tissues. The culture fluid of Actinomyces viscosus was shown to include proteases that degrade IgG4, IgA1, IgA2, IgM, and IgE. Certain pathogenic bacteria produce S-IgA proteases, as well as proteases that degrade complement factors [27].

An instance of "multifaceted" virulence can be observed in R. gingivalis, which generates lethal extracellular hydrolytic enzymes and fibrillar antigens that eliminate immunoglobulins. Furthermore, it has been confirmed that the culture of R. gingivalis induces lymphocyte death, which could play a significant role in the development of periodontitis and contribute to the incidence of immunodeficiency. Immunocompetent cells are additionally hindered in their activity by bacterial components such as peptidoglycans, lipopolysaccharides, and short-chain toxic fatty acids [28].

Saliva contains the enzyme lysozyme, which plays a crucial role in the local nonspecific defense of the oral cavity. Lysozyme, also known as muromidase, is a mucolytic enzyme mostly produced by neutrophils and blood monocytes. Lysozyme mostly targets gram-positive coccal germs, although there have been documented cases of its impact on gram-negative species. This enzyme has the ability to break down the complex carbohydrates in the cell wall of microorganisms. Lysozyme is believed to enhance the innate immune response by working together with other components that provide humoral protection [29]. Lysozyme boosts the process of phagocytosis and promotes the ability of the Ig SA complex with the C3 complement fraction to destroy gram-negative bacteria. As stated by the authors [30], the collaboration between lysozyme and IgA leads to the destruction of bacteria that are resistant to other circumstances through a process called lysis.

Lactoferrin is a protein that can attach to iron and has the ability to inhibit the growth of bacteria. By sequestering iron, it renders it unavailable for bacterial metabolism. Lactoferrin is present in the crevices of the gums and is produced in that area by a kind of white blood cell called polymorphonuclear neutrophils. Saliva serves as a protective element by aiding in the mechanical removal of harmful substances from the mouth mucosa. The presence of enzymes, immunoglobulins, and leukocytes in it gives it strong bactericidal characteristics. The composition of the body's saliva is highly intricate and can vary based on the body's response, the existence of different diseases, and pathological processes occurring in the oral cavity [31].

Periodontitis is a long-lasting inflammatory condition that causes the gradual deterioration of bone and connective tissue in the mouth. This destruction is caused by the microorganisms present in the oral cavity [32]. The current research is focused on investigating the processes via which microorganisms impact bone resorption. The treatment principles for periodontitis are intricately linked to the concept of the etiology and pathogenesis of inflammation in periodontal tissues. Currently, there is no established understanding of the causes and development of periodontitis, and there are still many unresolved debates around this topic [33].

Nevertheless, the inflammatory process in the periodontium is unable to occur without the corresponding response from the host organism. Not all instances of introducing microbes and other foreign agents into the body lead to the occurrence of an infectious or pathogenic condition. This phenomenon does not occur if foreign chemicals are eradicated or removed by mechanisms of both systemic and localized immune defense. Inflammation is triggered by the body when its normal mechanisms for maintaining homeostasis are no longer effective [34].

An ongoing immune imbalance in the body exacerbates the chronicity of a specific system or organ's pathology, which in turn worsens the prognosis and clinical course. Conversely, the progression of the pathology and the subsequent increase in compensatory reactions deplete the metabolic resources, adaptive mechanisms, and processes, leading to an increase in secondary immune deficiency [35].

Multiple studies have substantiated the significant influence of microbial factors in the development of periodontitis. However, the primary focus for investigating the causes and mechanisms of this illness lies in the alterations of the body's immune system [36].

Chronic generalized periodontitis is now understood to be more than just inflammation of the tissues surrounding the teeth. It is also a response by the body to bacterial infection in the teeth and the area below the gums. This infection is caused by various nonspecific factors that have a negative impact on oral health [37].

3. Conclusions

This review suggests that a more robust multidisciplinary approach is required to evaluate the short and long-term impact of gingival inflammation in children and adolescents. A clearer understanding of individual responses to standard prevention and treatment approaches needs to be developed. Moreover, investigation of gingival and periodontal diseases in children and adolescents should incorporate a broader view of the role of variations in the oral microbiome throughout the life of the individual patient. Couple this with molecular variations in age dependent gingival inflammatory responses to these oral microbial biofilms, will enable a better understanding of the individual’s potential for future destructive oral disease and the associated risk for systemic sequelae later in life.

REFERENCES

[1] Nowzari H, Botero JE, Rich SK. The impact of early-in life periodontal infection on the smiles of children: a worldwide view. Compend Contin Educ Dent, 31: 154, 156-8, 160 passim, 2020.

[2] Oh T-J, Eber R, Wang H-L: Periodontal diseases in the child and adolescent. J Clin Periodontol, 29: 400–10, 2022.

[3] Dibart S. Children adolescents and periodontal disease. J Dent, 25: 79-89, 2017.

[4] Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 2008-2014. J Periodontol, 70: 13-29, 2019.

[5] Montandon A, Zuza E, Toledo BE. Prevalence and reasons for tooth loss in a simple from a dental clinic in Brazil. Int J Dent 2012 Available on line August 29 at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3437633/. Accessed August 15 2013.

[6] Department of Health and Human Services, Centers for the Disease Control and Prevention. Available at: http://www.cdc.gov/oralhealth/publications/factsheets/dental_caries.htm. Accessed August 15, 2013.

[7] American Academy of Pediatrics. Policy Statement. Section on Pediatric Dentistry. Oral health risk assessment timing and establishment of the dental home. Pediatrics, 111: 1113-6, 2013.

[8] American Academy of Pediatric Dentistry, Pediatric Dentistry Reference Manual. Guideline on periodicity of examination, preventive dental services, anticipatory guidance/counseling. And oral treatments for infants, children and adolescents. Pediatr Dent, 34: 110-6, 2012.

[9] Stamm JW. Epidemiology of gingivitis. J Clin Periodontol, 13: 360-6, 1986. 11. Bhat M. Periodontal health of 14-17-years-old US schoolchildren. J Pub Health, 51: 5-11, 2021.

[10] Page RC. Oral health status in the United States: prevalence of inflammatory periodontal diseases. J Dent Educ, 49: 354-64, 2015.

[11] Mackler SB, Crawford JJ. Plaque development and gingivitis in the primary dentition. J Periodontol, 44: 18-24, 2013.

[12] Matsson L. Development of gingivitis in the preschool children and young adults. J Clin Periodontol, 5: 24-34, 2018.

[13] Bimstein E, Matsson L. Growth and development considerations in the diagnosis of gingivitis and periodontitis in children. Pediatr Dent, 21: 186-91, 2019.

[14] Löe H. principles of aetiology and pathogenesis governing the treatment of periodontal disease. Int Dent J, 2:119-26, 2013.

[15] Brown LJ, Löe H. Prevalence, extent, severity and progression of periodontal disease. Periodontol 2020, 2:57–71, 2023.

[16] Mariotti AJ. Gingival diseases. In: Bimstein E, Needleman HL, Karimbux N, Van Dyke TE, eds. Periodontal and gingival health and diseases. Children, adolescents and Young adults. Martin Dunitz Ltd, London; 31-48, 2021.

[17] Parfitt GJ. A five year longitudinal study of the gingival condition of a group of children in England. J Periodontol, 28: 26-32, 2017.

[18] Bimstein E, Garcia-Godoy F. The significance of age, proximal caries, gingival inflammation, probing depths and the loss of lamina dura in the diagnosis of alveolar bone loss in the primary molars. ASDC J Dent Child 6:125-8, 2014.

[19] Turnbaugh PJ, Ley RE, Hamady M, Frase-Liggett CM, Knight R, Gordon JI. The human microbiome project. Nature, 449: 804-10, 2017.

[20] International Human genome sequencing consortium, Initial sequencing and analysis of the human genome. Nature, 409: 860-921, 2021.

[21] Collins FS, Morgan M, Patrinos A. The human genome project: Lessons from large-scale biology. Science, 300: 286-90, 2023.

[22] Chen T, Yu W-H, Izard O, Baranova OV, Lakshmanan A, Derwhirst FE. The human oral microbiome database: a web based accessible resource for investigating oral microbe taxonomic and genomic information. Available at: http://www.homd.org Database 2020; baq013 doi10.1093/databasebaq013. Accessed August 15, 2023.

[23] Dewhirst FE, Chen T, Izard J, Paster BJ, Tanner AC, Yu WH, Lakshmanan A, Wade WG. The human microbiome. J Bacteriol, 192:5002-17, 2020.

[24] Devaraj S, Hemarajata P, Versalovic. The human gut microbiome and body metabolism: implications for obesity and diabetes. Clin Chem, 59: 617-28, 2013.

[25] Pimentel GD, Micheletti TO, Pace F, Rosa JC, Santos RVT, Sira FS. Gut-central nervous system axis is a target for nutritional therapies. Nutr J, 10; 11: 22. doi: 10.1186/1475-2891-11-22, Available at http://www.ncbi.nlm.nih.gov/pubmed/22490672. Accessed on August 15, 2013.

[26] Ohlrich RJ, Cullinan MP, Seymour GJ. The immunopathogenesis of periodontal disease. Aust Dent J, 54(Suppl 1):S2-10, 2019.

[27] Teng T-YA, The role of acquired immunity and periodontal disease progression. Crit Rev Oral Biol and Med, 14: 237-52, 2013.

[28] Berglundh T and Donati M. Aspects of adaptive host response in periodontitis. J Clin Periodontol, 32(suppl6): 87-107, 2015.

[29] Papapanou PN, Abron A, Verbitsky M, Picolos D, Yang J, Qin J, Fine JB, Pavdilis P. Gene expression signatures on chronic and aggressive periodontits: a pilot study. Eur J Oral Sci, 112: 216-23, 2014.

[30] Albandar JM. Epidemiology and risk factors of periodontal disease. Dent Clin North Am, 49: 517-32, 2015.

[31] Tonetti MS, Mombelli A. Early-onset periodontitis. Annals Periodontol, 4:39-52, 2019.

[32] Bimstein E. Extended kindred with 10 children with periodontitis: a sevenyear follow-up report. Pediatr Dent, 25: 389-96, 2023.

[33] Shaddox L, Wiedley J, Bimstein E, Magnuson M, Clare-Seltzer M, Aukhil I, Wallet SM. Hyper-Responsive Phenotype in Localized Aggressive Periodontitis. J Dent Res, 89: 143-8, 2020.

[34] Shaddox LM, Wiedey J, Calderon NL, Magnusson I, Bimstein E, Bidwell JA, Zapert EF, Aukhil I, Wallet SM. Local inflammatory markers and systemic endotoxin in

aggressive periodontitis. J Dent Res, 90: 1140-4, 2021.

[35] Suvan J, D'Aiuto F, Moles DR, Petrie A, Donos N. Association between overweight/obesity and periodontitis in adults. A systematic review. Obes Rev, 12: e381-404, 2011.

[36] Meisel P, Wilke P, Biffar R, Holtfreter B, Wallascohofski H, Kocher T. Total tooth loss and systemic correlates of inflammation: role of obesity. Obesity, 20: 644-50, 2012.

[37] Suvan J, D'Aiuto F, Moles DR, Petrie A, Donos N. Association between overweight/obesity and periodontitis in adults. A systematic review. Obes Rev, 12: e381-404, 2018.