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TEMPORARY TEETH CARIES AND ITS COMPLICATIONS IN CHILDREN AS A SOCIALLY SIGNIFICANT INFECTIOUS DISEASE

ABSTRACT

The article contains the analysis of literature of more than 50 years. It gives the ground to identify the caries of children's temporary teeth as infectious process. Under favorable conditions, it can turn into an infectious disease with natural peculiarities of epidemiology, etiology, pathogenesis, pathomorphology, clinical manifestations, complications, immunological changes, with the possibility of specific and nonspecific prevention. The decay is viewed as the globally common infection with high social value.

Keywords: caries of temporary teeth, children, the carious process, infectious disease, opportunistic infection, cariogenic bacteria, biofilms.

Epidemic of carious illness among the population has enlarged from isolated cases in ancient times to 100% nowadays [1]. According to WHO data, the prevalence of caries in different countries varies from 17 to 94 %. It is a problem of public health in general [14], and according to the data [27] caries of teeth is one of the most widespread infectious diseases, and the most expensive illness in the world. The severity of tooth decay and their complications at an early age is increasing [14], the prevalence of caries of teeth of one year old children is 15 %, by 3 years this indicator reaches 46, by 6 years – 96 % [6, 7, 11, 16].

Terms Caries Status (CS), Caries Free (CF) children, the Early Childhood Caries - ECC, a Severe Early Childhood Caries (S-ECC) are often applied. ECC is characterized by the presence of one or more carious, by a removed tooth because of the complicated caries or the sealed-up surface in any temporary children's tooth at the age of 71 months and earlier [34]. ECC is recognized as a clinical syndrome, described by Beltrami in the 1930th as "Les dents noire de tout-petits" (Black teeth in toddlers) [19]. The susceptibility of enamel to the influence of acids in the period of an incomplete mineralization is the greatest. At the age of 2-3 years after teething the high prevalence of a focal demineralization can take place. The probability of emergence of this disease increases with insufficient hygienic care of an oral cavity and consumption of a large amount of carbohydrates [4, 6, 20]. At this time teeth especially need thorough and effective care [4]. The role of parents increases in ensuring prophylaxis of the oral cavity of the child. Their own sanological culture and their active position in providing a healthy lifestyle to their children is very important, however local all-hygienic and social and economic aspects are important as well [12].

The fact of high prevalence of S-ECC among children of early age is alarming. It leads to notable medical and medico-social consequences, significantly influencing a condition of physical and mental health of a child and also family welfare in general [6]. Society has recognized interrelation between ECC and inadequate childcare [21]. The authors [35] related untreated or neglected caries to the crime basis - child abuse. Mostly ECC is connected with a low economic level in the country of child's accommodation. In such countries small children have weight reduction because of impossibility of good nutrition: children at the age of 3 years with early caries weighed 1 kg less, than children in a control group as the toothache complicates the process of meal taking, promotes a sleep disorder, slowing down metabolic activity [28].

The aggressive course of caries leads to complications, the foci of a chronic infection and their further sensibilization influences the functioning of an organism and growth of the child, defining his psychoemotional state [13, 18].

In modern socially we have recognized the official status of important infections such as: HIV, hepatitis's B, C, D, tuberculosis and others, which in some cases lead to death. The infectious disease of teeth caries is a silent pandemic. It doesn't threaten life, but considerably reduces its quality, leading to global social and economic consequences. It requires - up to 5 percent of expenses on a medical care in the world, up to 70 percent of stomatologists working time [18], and can confidently be recognized as a socially significant infection of the 21st century.

Classifications of caries are made on the basis of clinical and morphological features of a lesion of teeth. According to the classification of ICD-10 caries belongs to: class XI - Illnesses of digestive organs; to section 1.1. - Illnesses of an oral cavity, sialadens and jaws; K02 - Caries of teeth. Also caries is distinguished on lesion depth: initial, superficial, average and deep caries. These classifications allow us to judge a carious lesion of a separate tooth; however the main task is a treatment of the child with caries of teeth, but not only one tooth. Therefore in pediatric dentistry it is important to use one more classification by T.F. Vinogradova. This classification is based on a degree of illness activity: the compensated, subcompensated, and decompensated form [4]. The conceptual framework of an infectology is successfully applied in cariology: according to the clinical state there can be peracute, acute and chronic caries; mild, average, and severe forms; uncomplicated and complicated forms of caries.

Caries is a localized, transmissible pathological infectious process [25]. The targets of a carious infection are the solid dentine and cement of teeth while in other infectious diseases, soft organs and tissues are damaged. Various scientists at various times defined caries first of all as an infectious disease, and only secondly as the progressing destruction of tissues of tooth. Recognition of caries an infectious disease gives the prospect of integration of cariology with the general medicine. Its infectious nature focuses attention of experts on elimination of a microbial plaque on teeth and decrease risk factors promoting acid dissolution of mineral substrate [7].

Treatment-and-prophylactic measures at tooth decay are based on Miller's chemical and parasitological theory however there are "white maculae" in etiology and pathogenesis of "illness of a civilization". There are indisputable data of interrelation between the epidemic of caries and climate-geographical and medico-social factors [7]. The most

reasonable is the concept of ecology of a microbic flora of the dental plaque according to which pathological process develops if there is the interaction of two main cariogenic factors — the influence of substrate for a certain period of time and the existence of acid-forming bacteria [13]. The most recognized and dangerous are cariogenic bacteria, Gram-positive coccuses of phylum *Firmicutes*, species of *Streptococcus mutans*, *Streptococcus sobrinus*, the Kingdom of *Bacteria*, the Class *Bacillaceae*, the Family *Streptococcaceae*, and the Genus *Streptococcus*.

The hypothesis of a role of streptococci in development of caries has been accepted in the USA for practical guidance since 1960 after the research works conducted in the country from the 40th of the previous century [29]. Streptococci make an considerable share of micro flora of a tooth plaque, up to 80 percent of total amount of microorganisms in an oral cavity [5]. They can be met in various quantitative ratios which depend on a diet, individual hygiene, structure and peculiarities of saliva and other factors [20]. Scientists study the role of *S. gordonii, S. sanguinis*, and other Viridians Group Streptococci (VGS), which are the residents of an oral microbiota. We should mention that 7 indigenous species according to modern classification are related to the ecological *mutans* group: *S. cricetus, S. rattus*, *S. mutans, S. sobrinus, S. downei, S. macacae, S. ferus*. Also scientists found in a tooth plaque *S. sanguinis* type. It interacts on a tooth enamel surface with *S. mutans*, making the environment for it less hospitable which is important for caries status of a child [9].

Streptococcus mutans is the key etiological agent initiating infectious cariogenic process. It can have serotypes c, e, f and k. Types f and k are often found in persons with an infectious endocarditis [31]. Specific virulent types of *S. mutans* have unique adhesive proteins SpaP and Cnm which promote their resistance to antibacterial properties of saliva, and children may have “impetuous” or “rampant” caries. It is connected with the increased adhesive ability of *S. mutans* development [9]. It is one more scientific proof of the infectious theory which has great practical value.

Thus, caries is a nonspecific polyetiological infectious process which under certain conditions develops into a chronic infectious disease. It is inapparant (asymptomatic, latent), having the nature of an opportunistic infection which develops and progresses under favorable conditions of external and internal environment. Caries originators are ubiquitous bacteria - the VGS of the *mutans* group. The most virulent of them of *S. mutans, S. sobrinus* are constantly living in an oral cavity of a person from the moment of primary infection and further on during all person’s life. They are opportunistic representatives of an autokhtonny local microbiota of an oral cavity.

The most important factor of virulence of *Streptococcus mutans* is their prosperity in acidic environment [9, 22]. Unlike the majority of microorganisms in an oral cavity such viridans streptococci as *S. sanguis*, *S. mitis*, *S. oralis*, whose metabolism is considerably slowed down by low pH, the metabolism of *S. mutans* and *Lactobacillus spp.* in such conditioned only increases. That makes them the dominating bacteria in cariogenesis [22]. These properties allow streptococci to be the first to occupy various biotopes in an oral cavity of the child [5]. It was considered earlier that *Streptococcus mutans* infect a child in the period between 19 and 31 months, in a so-called discrete window of infection [23]. But nowadays it is proved that infection with cariogenic types of *S. mutans* and their colonization in tongue grooves, are possible before teething [20]. It confirms the way of their transfer from a mother to a child [4]. At the same time Tanzer writes that solid surfaces are necessary for colonization of *S. mutans.* So their fast emergences before babies’ teething lead to the use of obturators for correcting a cleft lip [32].

Research papers have shown that 119 Afro-American children in their early age have 315 genotypes of strains of *S. mutans.* One child had from 1 up to 9 genotypes at the same time. Usually they have more than one genotype. 33 percent of children allocated only one highly transmissible genotype which wasn’t bound to a streptococcus genotype of a mother or any family member, and at least one genotype is identical to maternal or to one of family members. It shows strains’ high ability to transfer. Children attending one and the same kindergarten have identical strains of bacteria in their saliva and children who are on home education have identical strains of streptococci with their mother and father [20]. At the same time the genotypes of strains are different as well as their quantity [30]. Transmission can be direct and indirect. Direct transmission takes place when we have the contact and household mechanism of transfer. Indirect transmission goes through general toys, ware, nutrition, and dirty hands.

The earlier S. mutans appear in an oral cavity of a child the higher the prevalence of caries by 4 years of life. Children, whose mothers have a high level of Streptococcus mutans, have a larger risk of receiving microbes, than children whose mothers have low level [18]. And if food contains a high level of easily fermented carbohydrates, then S. mutans, being in symbiotic interaction with Lactobacillus spp., synthesizes the extracellular polysaccharides helping to stabilize a tooth plaque matrix [34].

Frequent consumption of sugars is recognized as the trigger mechanism: in an etiopathogenesis of caries microorganisms are emphasized as secondary, the main material is sucrose – the only carbohydrate which uses S. mutans. It makes a sticky extracellular polysaccharide on the basis of dextran which allows them to bind with each other and form a dental plaque. S. mutans makes dextran by means of dextransucrase enzyme, decomposing sucrose: n molecules of sucrose (glucose) n + n fructose [31]. Sucrose is the most significant aggressive factor as it transforms anticariogenic products into cariogenic and causes “metabolic explosion” in an oral cavity [4]. It increases the proportion of *Streptococcus mutans* and lactobacilli, at the same time the level of *Streptococcus sanguini* decreases [24, 33]. All this in general leads to a cariogenic situation – acidic environment promotes a → demineralization of highly mineralized tooth enamel, it becomes speckled, and vulnerable for destruction [4].

Streptococcus mutans are supplied with specialized receptors for adhesion to the surface of teeth. Using the enzymes making key matrixes, its glucosyltransferases - GtfB, GtfC, GtfD, produce sticky glucosylglucan polymers which facilitate the attachment of bacteria to the surface of tooth. Glucans are the main components of a biofilm matrix which protects the microbial community from mechanical and oxidizing stresses, and also they organize cariogenic biofilms. Besides, they receive lactic acid in great amount. It is a by-product of bacteria consumption of sucrose, together with the community of mature biomembranula. It finally leads to a demineralization of a surface of teeth and to cariogenesis. Thus, glucosyltransferases B, C and D provide the mechanism of formation of cariogenic biofilms. It is a key factor of *S. mutans* virulence [31]. The formation of a plaque on the teeth surface made of biological biofilms contained microorganisms of a mouth biota prevents the enamel of temporary teeth from physiological maturity. It blocks getting macro elements - and minerals from saliva. Bacteria in a biofilm are always metabolic active, causing fluctuations of pH level in saliva [17]. According to the conceptual model of tooth biofilms by Jill S. Nield-Gehrig (2003) it consists of bacteria: micro colonies, units of

micro colonies of bacteria, extracellular polysaccharide matrix of EPS substance, epitheliocytes, components of saliva and nutrition, blood cells. A microbiological portrait of the healthy biomembranula is associated with the state of health of teeth and parodont (Health-associated dental plaque): *S. sanguinis, S. mitis*, *S. oralis*, *S. salivarium*, *Veillonella spp*., *Actinomycetaceae*, *Haemophilus spp*., *Bacteroides spp.* Biomembranula associated with caries is presented by Disease-associated dental plaque with disturbance of a microbial homeostasis: dominance of acidogenic and acid tolerant species of *Streptococcus spp*. and *Lactobacillus spp*., when losing dominant positions of the main symbiotic microflora and augmentation of representation of a transitional microflora [8]. The "social behavior of microorganisms" received the special name: - "quorum sensing" [3]. It is important to notice that pathogenic bacteria don't show aggression against the owner's body until they reach a certain quantity / a critical dose and the degree of virulence don't reach the necessary level in order to overcome the owner's protection. This fact also strengthens positions of the infectious theory of caries of teeth.

An important series of experiments was conducted in 1950-1960th on the gnotobionts. They proved the interrelation of a carious lesion with biomembranula. Scientists didn't find caries of teeth in the sterile rats receiving a cariogenic diet, but the rats that were artificially infected with *S. mutans* arose and developed caries. O. Fejerskov, E. A. V. Kidd [7, 26] in 2004 proved the infectious theory of caries. According to their research pathological process is initiated within biomembranula, shown in the liable enamel or dentine. Reflecting the activity of biomembranula the lesion can be active or passive, and regular destruction of biofilms can stop a lesion of caries [26].

Thus, children's caries of teeth needs to be viewed as an infectious disease. It is more often chronic, with the appearance of a certain sequence of distinctive signs typical to inherent infection. It has the nature of an opportunistic infection with the contact and domestic mechanism of transfer from an infectious source to healthy child bacteria, residents of an oral cavity. *S. mutans* and other cariogenic microorganisms initially occupy an oral cavity of the child, since the neonatal period and later on. They are normal inhabitants of an oral cavity of a person. Before teething they appear on the mucosa of the mouth, gums, and the root of the tongue and show their pathogenic potential only in the conditions promoting their activation and endogenous diffusion on solid surfaces of an enamel of teeth and further on in organs and body tissues of an individual.

Moreover, the carious infectious disease has its own, epidemic features: it proceeds acyclicly, it has no clear incubation interval, with the distinct etiological factor - an infecting agent - *Streptococcus mutans*, with expansion of the clinical symptoms characteristic only for caries. Also it has formation of pathomorphologic substrate in a target organ - a solid tissue of tooth and no development of specific antibodies. However this infection doesn't come to an end with the originator eradication from a macroorganism. According to the second strategy of a parasitism, acyclic infectious process - AIP [15] the resident flora of an oral cavity perishes together with the owner's organism. AIP begins with penetration of a pathogenic microorganism into a human body but it doesn't come to an end in a habitual time interval - within a week, a month, or a year. It remains until the end of human life. The microorganisms causing AIP belong to different taxonomical groups; they have a different mechanism of penetration into target cells. In this case illness can have different clinical picture. The fundamental difference of AIP from any cyclic infectious mono process is that clinical improvement as the seeming recovery isn't followed by biological recovery. It is shown by decrease or even disappearance of clinical implications of illness, by formation and existence of high levels of specific antibodies in blood serum, but an infecting agent doesn't leave an organism. The existence of a pathogen in a body forms not only a basis for exacerbations and a recurrence, but also it is the evidence of continuous infectious process. Its duration is equal to the life expectancy of the person. The defining factor is a disability of T - and B-cells of immune system to control the infected macrophages and to block infectious process. All herpes virus, viruses of *Rubella*, HBV, HCV and HDV, HIV, the T-cellular leucosis, adenoviruses and other, not yet open microorganisms belong to the pathogens causing AIP [2].

The infectious theory of caries development doesn't contradict to all earlier existing theories, including the neurotrophical, physical and chemical theory of D.A. Entin (1928), the metabolic, proteolysis-chelation, fundamental chemical and parasitological theory of Miller and et al (1890). It does not contradict them, moreover, it enriches them, supplements and synthesizes. The infections caused by the opportunistic microorganisms which are a part of normal microflora of an organism are called opportunistic infections. Opportunistic or potential pathogenic microorganisms, cause diseases when the protective forces of an organism decrease in adverse conditions of the environment. Such diseases can be caused by more than one hundred species of opportunistic microorganisms. Among them there are bacteria, including a genus of streptococci, viruses, mushrooms, protozoa; bacteria: *Staphylococcus spp*., *Streptococcus spp*., *Enterococcus spp*., family *Enterobacteriaceae*, *Pseudomonas spp*., *Acinetobacter spp*.; viruses: *HBV, HCV*; *HSV*-1, 2; *CMV*; *Papovaviruses*; *Adenoviruses*; *Coxsackieviruses* and *ECHO*, etc.; mushrooms: *Candida*; *Histoplasma*; *Aspergillus*, etc.; protozoa: *Pneumocystis*, *Toxoplasma*, *Cryptosporidium*, etc. [2].

Considering caries infectious process as opportunistic, classical approaches such as vaccination, use of antimicrobial chemotherapeutic drugs, antiseptics are restricted and also will not give the expected effect in our fight against caries pandemia. For example, the works on long operating vaccine have been conducted for more than 30 years. The control of an infectious disease – children's caries of teeth is possible on the condition of the use of all potentials of epidemiology and infectology to break the epidemic chain of contact – domestic infection. And now it is necessary to control the mechanisms of endogenic activation of the originator. It is necessary to influence on an etiological factor, after a deep research of its microbiological, molecular and genetic, immunobiological, immunochemical properties as well as on the mechanisms which control the prosperity of cariogenic streptococci in a biological niche; that is their ability to form a tooth plaque on the surface of tooth, to turn digestible carbohydrates into the lactic acid causing damage of a dentine, and their ability to adapt to sudden changes of conditions in a tooth plate [31].

According to the statement of the Director-General of WHO Dr. Tedros Adhanom Ghebreyesus, on December 10, 2017: "The enjoyment of the highest attainable standard of health is one of fundamental rights of every human being without distinction of race, religion, political belief, an economic or social conditions. But when people have an opportunity to be active participants, but not passive recipients in the course of rendering the help to them, results improve, and health care systems become more effective".

On September 28, 2014 in Moscow the Russian experts entered the International alliance for a cavity-free future (ACFF) which urges to recognize caries as continuously proceeding disease, preventable at early stages, and

reversible. They are going to develop comprehensive programs on prophylaxis and treatment [1]. The purposes of Alliance and Russia: by the year of 2020 to integrate a package of measures of prophylaxis suitable to local conditions, to create the system of monitoring of a disease on the local level. One of the tasks is that each child born in 2026 shouldn't get ill with caries throughout the entire life. It inspires hope for decrease of caries and its complications at children of early age [1, 10].

Thus, having recognized the polyetiological and opportunistic nature of carious infectious process, it is necessary to eliminate favorable conditions for the formation of biofilms of a dental plaque, leading to this infectious disease and its complications. The identification of caries as infection will allow us to define the ways of modern nonspecific and specific prophylaxis of an infectious disease – caries of children's temporary teeth. In the absence of effective vaccines, it has the status of an uncontrollable infection with pandemic diffusion. On condition of giving the infection the status of socially important it will be possible to introduce state programs and develop the complex of actions for treatment and prophylaxis for the management of risk factors in caries development. It will help us to involve experts of various profiles and organizers of public health care.

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CLINICAL-EPIDEMIOLOGICAL CHARACTERISTICS OF PATHOLOGICAL PROCESSES OF PERIODONTAL TISSUES OF INFLAMMATORY-DESTRUCTIVE NATURE

ABSTRACT

Today according to Russian and foreign researchers the prevalence of parodontium diseases is high and does not tend to decrease. A wide range of etiological factors and pathogenetic mechanisms of the development of parodontium inflammatory diseases influence on the carrying of complex treatment-and-prophylactic actions. At the same time parodontium diseases often lead to teeth loss, contributing to formation and development of dentoalveolar system disorders and further digestive tract diseases. In this regard, pathological processes of parodontium tissues have both medical and social value as patients are usually of the working-age.

According to the WHO data the prevalence rate of parodontium diseases among various groups of population is characterized by particular features. So, worldwide more than half of the examined patients at the age group of 12-15 years have parodontium diseases, at the same time at the age of 35-44 years it has total character. At the same time in Russia 12-year old patients make 1/3, and 15-year-old teenagers have slightly lower than a half surveyed, at the age of 35-44 years only 1/5 part has rather healthy parodontium. Meanwhile, the high level of parodontium diseases was noted among the North inhabitants due to severe climatic conditions and specific regional environmental and biological risk factors.

It should be noted that quite often treatment of periodontal disease of inflammatory and destructive processes can take several years, and in certain cases it continues during all life. A desire of the patient and dentist to gain most expected clinical effect without damage of the functional activity of dentoalveolar system that demands carrying out further researches for the perfecting of the periodontal help to the population is explained.

Keywords: parodontium diseases, prevalence rate, dentoalveolar system, treatment, prevention.

Nowadays despite broad studying of periodontal diseases, there are still problems of their treatment and prophylaxis [7, 11, 14, 37]. A wide range of etiological and pathogenetic aspects of pathological processes of periodontal tissues and a high level of prevalence among various age groups of the population make particular treatment-and-prophylactic difficulties [9, 17, 19- 21]. In this regard scientists are researching new effective methods of