Chapter 2

Early Childhood Caries (ECC) — Etiology, Clinical Consequences and Prevention

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Additional information is available at the end of the chapter

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1. Introduction

Primary teeth are also known as milk or deciduous teeth. The 20 primary teeth start to appear in a baby's mouth around the sixth month and they stay in the mouth until they are gradually replaced by the permanent teeth between the ages of six to twelve years.

Primary teeth start to develop from the 6^{th} to 7^{th} week of fetal life from epithelial cells of the mouth that form the tooth buds. The cells of these initial tooth germs continue to differentiate during pregnancy, and, when the baby is born most teeth are already partially formed in the jaws.

The primary teeth play an important role in giving facial fullness and aesthetically pleasant facial shapes. Absence of teeth, due to any reason, not only hampers the masticatory activity of the individual, but also affect the facial features to great extent, affecting the concerned person physiologically, emotionally and socially.

Unfortunately, the primary teeth's function is disrupted when the demineralization process of hard tooth structures is involved – dental caries.

The oral health of children is especially aggravated with the occurrence of the so-called early childhood caries(ECC). ECC is an acute, rapidly developing dental disease occurring initially in the cervical third of the maxillary incisors, destroying the crown completely.

The presence of dental caries, especially of ECC, may reflect on the oral health status of children in countries with insufficient health system and inefficient primary dentistry. Early Childhood Caries (ECC) is a public health problem with biological, social and behavioral determinants.

The preventive activities must start at an early age. Home-care methods are more than necessary.

1.1. Primary Teeth

The eruption of the primary teeth starts around the sixth month with the central incisors of the lower jaw and it is fully completed by the 3rd year of age with the appearance of the upper second molar. Normally, the first teeth that erupt are the two front teeth of the lower jaw (mandibular central incisors). After a few of months they are followed by the four front teeth of the upper jaw (maxillary central and lateral incisors). The last primary teeth that erupt are the upper second molars which are expected to appear around the age of 2½ years and not later than the completion of the 3rd year.

Teeth eruption is the process during which they move towards the surface of the jawbone and break through the gums, until they take their final position in the mouth with their crown fully visible. At this point the crown is completely formed, but the root of the tooth will continue to form for one more year.

The process of tooth eruption is usually accompanied with pain and discomfort for the baby. The associated symptoms such as drooling, disruptions in eating or sleeping patterns, irritability, swollen gums are referred as 'teething'.

When the primary dentition is completed, children have a set of 20 primary teeth, ten at every jaw. They belong to 3 different teeth types: 8 incisors, 4 canines, and 8 molars.

Eruption of the first permanent molar (age of 6 years), marks the end of the primary dentition period and the start of mixed dentition.

The anatomy and morphology of the primary teeth is also generally similar with that of permanent teeth. Externally the tooth is covered by a layer of enamel at the crown area and by cementum at the root area. Under the enamel there is a layer of dentine which surrounds the soft and alive dental pulp at the center of the tooth.

However some distinctive features of primary teeth are: smaller size, thinner and more translucent enamel, less mineralized enamel (which makes primary teeth more vulnerable to cavities, especially for early childhood caries), larger pulp chambers, narrower and smaller roots, etc.

1.2. Role/importance of the primary teeth

Parents commonly ask why they should worry about cavities in baby, since they will be replaced by the permanent teeth? The role of the primary teeth is just as important as the role of the permanent ones.

Humans use teeth to tear, grind, and chew food in the first step of digestion. Teeth also play a role in human speech. Additionally, teeth also provide structural support to muscles in the face and form the human smile and other facial expressions. So, broadly the main functions of the teeth can be summarized as follows: role in mastication (helps eating), aids in articulation and speech, role in aesthetics (gives shape and beauty to the face).

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One of the main functions of teeth is the mastication of the food. The first step of digestion involves the mouth and teeth. Each type of tooth serves a different function in the chewing process. Depending on the shape, teeth enable cutting, grinding, chewing and preparing food for swallow and further digestion in the digestive tract. The Incisors cut foods when you bite into them. The sharper and longer canines tear food, while the wider molars grind the food.

Masticatory function, besides stimulating the development of the jaws, allows the child to learn the right way of eating. Toothache during mastication can affect the child's nutrition. According to some studies it has been found that children with more decayed teeth have less than 80% of average weight, which they are expected to have for their age (Acs et al 1999, Acs et al 1992). Children with toothache often after their recovery reach their normal weight and have tranquility during their sleep (Elice & Fields 1990).

The role of healthy primary teeth consists in clearly speaking and emphasizing the correct letters and sounds. The mouth, especially the teeth, lips, and tongue are essential for speech, one of the very important functions of teeth. The teeth, lips, and tongue are used to form words by controlling airflow through the mouth. Especially, the front teeth enable correct pronunciation of consonants: t, th, d, f, etc.

Primary teeth, among other roles, have one more extremely important role. As long as they are in the oral cavity, until their physiological loss, they will serve as space retention for permanent teeth. Their premature loss can be a cause of malocclusions in children. If we achieve to prevent their premature loss, malocclusion frequency will be reduced for 30%. Although early loss of primary incisors would not have a major consequence, a premature loss of primary molar and canine will be marked by a significant disorder in the development of occlusion during the eruption of the permanent teeth (Marković 1976).

Healthy teeth and full realization of their function, in fact, will allow a normal psycho-physical development of children, which for their age, is very important.

2. Dental caries — Definition, etiology and epidemiology

Dental caries is one of the most prevalent diseases in children worldwide. The Center for Disease Control and Prevention reports that dental caries is perhaps the most prevalent infectious diseases in children. Dental caries is five times more common than asthma and seven times more common than hay fever in children (US Department of Health and Human Services 2000).

Tooth decay is localized progressive disease, whose character consists in the destruction of tooth structures mainly under the influence of metabolic products of the oral microflora.

Dental caries is pathological destruction of tooth hard tissue with progressive effluence. Initially it appears in enamel, the dentin is involved after that, and later the pulp and the periodontium, with the possibility of complications that will affect the general health. The consequences of caries may be numerous, ranging from the morphological changes to

functional ones, e.g. complete crown destruction (early childhood caries), chewing difficulties, speech impediments, digestive tract disorders, odentogenic focal points (Raiç 1985, Stosic 1991).

Dental caries usually begins as small, shallow holes; if left untreated, these holes can become larger and deeper and potentially lead to tooth destruction or loss. Complications of dental caries include: pain, dental abscess, difficulties during chewing, tooth damage or loss, tooth sensitivity.

There are numerous definitions on caries, depending on what is taken in consideration: etiology, pathogenesis, clinical features.

Dental caries may be defined as a bacterial disease of the hard tissues of the teeth characterized by demineralization of the inorganic and destruction of the organic substance of tooth (Soames & Southam 1999).

According to Douglas, dental caries is the most common chronic infectious disease of childhood, caused by the interaction of bacteria, mainly *Streptococcus mutans*, and sugary foods on tooth enamel. *S. mutans* breaks down sugars for energy, causing an acidic environment in the mouth and result in demineralization of the enamel of the teeth and dental caries (Douglass et al. 2004).

Since *S. mutans* is transmitted to the child, another definition is based in the transmissibility. Dental caries is defined as a transmissible localized infection caused by a multi-factorial etiology. In order for dental caries to develop, four interrelated factors must occur: the patient (host), substrate (carbohydrates), dental plaque (*S. mutans*), and the time factor.

2.1. Etiology

Dental caries is a disease that is not caused by one factor. If only one factor would cause this disease, its prevention would have been much easier and more controllable. Many studies like clinical studies, but mostly longitudinal epidemiological studies, show convincing evidence for a multi-factorial nature of this disease. Numerous factors affecting the appearance of caries act team-wise and not separately, make caries pathogenesis very complex but also hinder the possibility to undertake effective preventive measures.

There are several important factors that make up the dental caries etiological circle. Host respectively tooth, dental plaque respectively bacteria, and substrate respectively saliva carbohydrates, all in co-operation with the time factor, are vicious chain of dental caries development.

The hard tooth structure, the enamel, is the forefront part that undergoes the demineralization process, respectively caries. The development of caries in enamel surface as much as it is affected by the internal structure of the hard tissue build of the tooth, equally, perhaps even more it depends on the strength of external factors affect.

Sometimes for various reasons: local, general or even hereditary, tooth structure can be so poorly mineralized, that it would need a very small amount of external factors to cause dental caries.

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The general opinion regarding the etiology of dental caries nowadays is that it is a very complex multifactorial disease, presented with high prevalence in all age groups. It has already been established that dental caries is a chronic infectious process with a multifactorial etiology. Dietary factors, oral microorganisms that can produce acids from sugars, and host susceptibility all need to coexist for caries to develop (Konig & Navia 1995).

There are some important factors that comprise the etiological circles of the dental caries: host or the tooth, dental or bacterial plaque, substrate – carbohydrates and saliva, and altogether co-react with the time factor. The hard dental structures, initially the enamel, undergo the demineralization process, respectively the caries. The caries development in the enamel surface is equally dependent from the inner hard dental structure and from the intensity of the extrinsic factors' action.

The newest concept in dentistry explains the cause of dental caries as a consequence of disruption of "Caries Balance" (Featherstone 2004). Dr. John Featherstone introduced the concept of the Caries Balance in 2002. The theory of "Caries Balance" defines dental caries as a disease of hard dental tissues, and the destruction of the enamel surface as a result of the disruption of the balance of demineralization and remineralization.

This misbalance may be manifested in the beginning of demineralization or during the process of remineralization. The defect in the enamel surface is a result of the domination of the demineralization process and such process has progressive course directed towards pulpar space. Which process will dominate depends on the proportions of the factors that constitute "Caries Balance", i.e. protective and pathological factors.

The balance disorder will be manifested with early demineralization process or eventually with remineralization process. This concept is that dental caries can be viewed as a balance of healthy or protective factors (factor of remineralization), and disease or pathogenic factors (factor of demineralization). Cavities are caused by an imbalance between risk factors for the disease and protective factors.

Pathological factors (risk factors) are: acid-producing bacteria, frequent eating/drinking of fermentable carbohydrates, sub-normal saliva flow and "function".

Protective factors are: saliva flow and its components; fluoride, calcium, phosphate remineralization; antibacterials (chlorhexidine, xylitol), etc.

The level of risk for dental caries depends on the domination of the certain group of factors that participate in the "Caries Balance". If there is domination of the pathological factors, the risk for dental caries will be higher and the treatment needs will require larger restorative interventions, as well as other consequences. If there is a domination of protective factors, then the invasive restorative dentistry will have fewer burdens, and concentrate in minimal restorations of superficial caries. Biological factors tend to be similar within all cultures and populations, although habit/environmental factors tend to be influenced specifically by the culture in place.

2.2. The prevalence of dental caries

It has already been mentioned that dental caries is the mostly spread disease in the world. Dental caries is a disease that affects all age groups, most commonly children.

Epidemiological data derived from the Oral Health Promotion Group of Kosovo showed a high prevalence of caries among children in Kosovo (89.2% among preschool children and 94.4% among school children). The mean dmft/DMFT index was 5.6 for preschool children and 4.9 for all school children (Begzati et al. 2011).

The results from the same previous study show that dental health of these children in Kosovo is worse than that of children in other European countries. Specifically, the mean dmft of five-year-olds at preschools in Kosovo (8.1) was found to be higher than the same value of preschool children in USA (1.7) and in many other European countries (1991-1995), including Ireland (0.9), Spain (1.0), Denmark (1.3), Norway (1.4), Finland (1.4), Netherlands (1.7), United Kingdom (2.0), France (2.5), and Germany (2.5). Our results are only comparable to the rates in Belarus (7.4), Sarajevo, Bosnia (7.53) (ages 5-7) and Albania (8.5), (Marthaler 1996, Kobaslia 2000). The low treatment rate of children in Kosovo (<2%) indicates a high treatment need. Also, the mean DMFT (5.8) of school children in Kosovo (age 12) was higher in comparison with school children (age 12) of the following developed countries: Netherlands (1.1), Finland (1.2), Denmark (1.3), USA (1.4), United Kingdom (1.4), Sweden (1.5), Norway (2.1), Ireland (2.1), Germany (2.6) and Croatia (2.6) (16). The mean DMFT of Kosovo's children (age 12) was similar to the mean values in Latvia (7.7), Poland (5.1) and a group of 12- to 14-year-olds in Sarajevo, Bosnia (7.18), (Marthaler 1996, Kobaslia 2000).

3. Early Childhood Caries (ECC)-definition

ECC is an acute, rapidly developing dental disease occurring initially in the cervical third of the maxillary incisors, destroying the crown completely. Early onset and rampant clinical progression makes ECC a serious public health problem. Due to varying clinical, etiological,

localization, and course features, this pathology is found under different names such as labial caries (LC), caries of incisors, nursing bottle mouth, rampant caries (RC), nursing bottle caries (NBC), nursing caries, baby bottle tooth decay (BBTD), early childhood caries (ECC), rampant early childhood dental decay, and severe early childhood caries (SECC) (James 1957, Goose 1967, Fass 1962, Winter et al.1966, Derkson & Ponti 1982, Ripa 1988, Arkin 1986, Bruered et al. 1989, Kaste & Gift 1995, Tinanoff et al. 1998, Horowitz 1998, Drury et al. 1999).

According to Davis, the definition of this pathology has always been complex and "difficult to be described, but when it is seen, you know what it's about" (Davis 1998).

In 1862, an American physician, Abraham Jacobi (Jacobi 1862) was the first to describe the clinical appearance of early childhood caries, which he observed in one of his own patients. Whereas, in 1932 Beltrami described this form of caries, as "Les dentes noires des tout petits" (black teeth in small children), (Beltrami 1952). Author Fass, created the term *nursing bottle mouth* (Fass 1962).

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The literature contains a variety of other terms used to describe early childhood caries and its diagnostic criteria. Most of them relate to the use of a feeding bottle or prolonged breastfeeding (feeding bottle tooth decay, feeding bottle syndrome, nursing caries, nursing bottle mouth, and so on). The authors wish to highlight the danger of excessive drinking from a baby bottle, if it contains sweetened liquids, or prolonged on-demand breastfeeding (Schroth et al. 2007).

To inform the scientific community with internationally comparable data on the incidence of early childhood caries, delegates to a conference at the Centers for Disease Control and Prevention, invented the term *early childhood caries* in order to better the multi-factorial pathogenesis of the disease" (Kaste & Gift 1995).

Unfortunately, this term was seen to have its limitations. Three years later, a further conference on early childhood caries, organized by the National Health Institute (USA), added two further definitions/descriptions, which were *rampant infant caries* and *early childhood dental decay* (RIE, CDD), (Quartey & Williamson 1998). These differences in definition were due above all to diversity in diagnostic criteria.

3.1. Clinical diagnostic criteria of Early Childhood Caries (ECC)

Due to the early appearance, typical localization, rapid destruction of the hard tissue of tooth, early childhood caries is a specific form of primary tooth decay. Childhood caries appears in caries resistant regions, such as: labial surfaces of the upper incisors, in the upper and lower molars, more rarely in the upper canine, and even less or not at all in the lower canine and incisors. In addition, during bottle-feeding with sugar-containing drinks, the upper incisors bathe in these sugar-containing drinks but the saliva from minor salivary glands in the area of these teeth has only limited remineralising properties, whereas the lower incisors remain largely protected by the tongue during bottle-feeding.

Different authors propose different criteria to define or describe the early childhood caries. Author Amidi, studying the publications about ECC, has concluded that: in 27 publications, the criteria for defining ECC was the presence of labial surface caries in at least one frontal maxillary tooth, in 23 studies at least two frontal maxillary teeth while in 9 studies three frontal maxillary teeth (Soames & Southam 1999).

Below are some criteria's for defining early childhood caries from various researchers cited by authors Amid & Woosung 1999:

- involvement of one or more maxillary incisors, without the involvement of mandibular incisors- author Sewint;
- a white or black spot in the labial surface of the maxillary frontal teeth- author Bennitz;
- one or more carious lesions in maxillary incisors, along gingival margin- author Ayhan;
- carious lesions in labial-buccal surfaces at one or more maxillar incisors- author Ramos;
- two or more maxillar incisors- author Harrison.

In the literature we still can find some criteria's, for example:

- one or more frontal maxillary teeth that has evidence that the child was fed with a bottle (Al-Dashti 1995);
- maxillar incisors and the mesial surface of canine (O'Sallivan 1993);
- at least one carious maxillary incisor with the involvement of labial and proximal surface or only proximale surface (Kaste et al. 1996);
- one or more maxillary incisor with cervical crown caries (Lopez 1998).

Author Wynne 1999, classifies early childhood caries into three types:

Type I (moderately easy) - usually involves two upper central incisors.

Type II (moderate, severe) - includes incisors, first molar, canine, and does not include the lower incisors.

Type III (widespread, severe) - including the mandibular incisors.

3.2. ECC — Prevalence

Prevalence of ECC is different and it largely depends on the criteria set by the researcher and the place where the examination takes place. There are differences between the data for urban or rural places, rich or poor places, "flourished" or "non-flourished" places. Furthermore, the prevalence of ECC varies in different countries, which may depend on the diagnostic criteria. While in some developed countries having advanced programs for oral health protection, the prevalence of ECC is around 5% (Derkson & Ponti 1982, Ripa 1988, Kaste et al. 1996, Davenport 1990, Hinds & Gregory 1995). In some countries of Southeastern Europe (neighboring countries of Kosovo) this prevalence reaches 20% (Bosnia) and 14% (Macedonia) (Huseinbegović 2001, Apostolova et al. 2003). Much higher ECC prevalence has been reported for such places as Quchan, Iran (59%) (Mazhari et al. 2007) and Alaska (66.8%) (Kelly & Bruerd 1987). At American Indian children the prevalence is 41.8% (Kelly & Bruerd 1987). Similarly, in North American populations, the prevalence at high-risk children ranges from 11% to 72% (Berkowitz 2003).

Data from relevant literature show different prevalence in different countries, cited by various authors (Berkowitz et al. 1993, McDonald 2000, Wendet 1995, Begzati et al. 2011, Barbakov et al. 1985, Harris & Garsia 1999, Huseinbegović 2001, Holt et al. 1996, Kaste 1991, Pettit et al. 2001, Bruered et al.1989, Reisine 1998, Wyne 1999, Apostolova 2003).

3.3. Etiology

Dental caries is an infectious and transmissible disease. Therefore, early childhood caries is an extremely aggressive form of the disease.

It was suggested that from the biological determinants, the three key causal factors for dental caries were: microorganisms, substrate, and host (Keyes 1962).

However, in the etiology of early childhood caries very special role given to dental plaque, respectively cariogenic bacteria. Of the great interest in the cariogenesis process are only two

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Place (year)	Author	Age	Prevalence
England(1989)	Silver	3 years	4%
Sweden1991)	Wendet	12-14 months	4.7%
Finland (1993)	Paunio	3 years	6%
Irak (1990)	Yagoot	1-5 years	15.6%
Kosova (2011)	Begzati	2-6 y.	17.5%
Indonesia (1979)	Aldy, Siregar	Up to 5 years	48%
Bosnia (2001)	Huseinbegović	5 y.	29%
Nigeria (1985)	Salako	3-7 y.	38.4%
Canada (1987)	Budowski	1-5 y.	7.4%
USA (1976)	Kaste	One years	0.8-2%
USA(1991)	Kaste	5 y.	5%
USA (1991)	Kamp	4 y.	5.3%
USA (1987)	Brured	3-5y.,native Indians Amer., and Alaska's population,	41.8-66.8%
USA (1992)	Weinstetin,	Mexican American, 8-47 m.	29.6%
Italia (2002)	Petti, Iannazzo	3-5 y.	7.6%
Macedonia (2003)	Apostolova	3 y.	13.3%
Australia (1998)	Reisine	Aborigin children	50%
Saudi Arabia	Wynne	Preschool children	15%
Kuwait (1986)	Soparkar	4-5 y.	11.5%

bacterial genera: mutant streptococci and lactobacills (Norman & Franklin 1999). A very important role is attributed to the bacterium Streptococcus mutans-called "the window of infection" (Caufield et al. 1993), in that it is responsible for the primary oral infection in the first phase of ECC (Berkowitz 1980; Berkowitz et al. 1996).

The most important requirement is an early infection, usually with the mother's cariogenic bacteria, for example, between the age of 19-31 months. However, earlier and later infection is a possibility (Caufield et al. 1993, Wan 2001).

After transmission of cariogenic bacteria and a frequent supply of substrate (sucrose) to the plaque, usually given as a sugary drink (juices and so on from a feeding bottle) or in older children, in snacks in the form of solid-cariogenic foods such as sweets, chocolates, cakes, biscuits, the development of early childhood caries occurs. If this loading of the plaque with sugars occurs at bedtime (night) and there is no tooth brushing, caries can progress rapidly.

In addition to the other severe types of early childhood caries, feeding on demand with cariogenic food and liquids is regarded as a co-factor for early childhood caries (Wendt & Birkhed 1995). As mentioned earlier, many social and behavioral determinants are risk factors for early childhood caries.

Favoring risk factors are as follows: low socio-economic status, low educational attainment in parents, chronic non-communicable diseases, inadequate health literacy, are all risk factors for a early childhood caries. Social and behavioral factors have been described in association with early childhood caries in numerous publications (FDI 1988, Horowitz 1998, Reisine & Douglass 1998, Seow 1998).

3.3.1. Cariogenic bacteria

In one of our studies conducted in the clinic of Paediatric Dentistry, it was found that S.mutans had a crucial role in ECC. The prevalence of S.mutans at our children was around 90% (Begzati et al. 2014). These facultative anaerobes are commonly found in the human oral cavity, and are a major contributor of tooth decay. The result of decay can greatly affect the overall health of the individual (Whiley & Beighton 2013).

The mutans streptococci and some Lactobacillus species are the two groups of infectious agents most strongly associated with dental caries. Earlier clinical studies reported that MS could not be detected in the mouths of normal predentate infants (Berkowitz et al. 1975, Berkowitz et al. 1980, Stiles et al. 1976, Catalanotto et al. 1975, Caufield et al. 1993, Karn et al 1998).

More recent clinical investigations have demonstrated that MS can colonize in the mouths of predentate infants (Tanner et al. 2002; Wan et al. 2001).

According to Berkowitz transmission of S.mutans happens in two ways: vertical and horizontal transmission. Vertical transmission is the transmission of microbes from caregiver to child. The major reservoir from which infants acquire S.mutans is their mothers. A study conducted by Berkowitz and co-authors reported that, when mothers harbored greater than 10^5 colony forming units (cfu) of MS per mL of saliva, the frequency of infant infection was 58%. When mothers harbored 10^3 cfu of MS per mL of saliva or more, however, the frequency of infant infection was 9 times less (6%) (Berkowitz et al 1981). These data clearly demonstrate that mothers with dense salivary reservoirs of MS are at high risk for infecting their infants early in life.

Vertical transmission is not the only vector by which MS are perpetuated in human populations.

Horizontal transmission also occurs. Horizontal transmission is the transmission of microbes between members of a group (eg, family members of a similar age or students in a classroom). Based on appearance, ways of transmission and prevention, Berkowitz concludes that: primary oral infection by mutans streptococci (MS) may occur in predentate infants. Infants may acquire MS via vertical and horizontal transmission. Improvements in the prevention of dental caries may likely be realized through intervention strategies that focus on the natural history of this infectious disease.

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Streptococcus mutans (SM)

Streptococcus mutans are gram-positive cocci shaped bacteria. SM is isolated from all tissues of the oral cavity and constitutes the largest number of inhabitants of the oral microflora. This bacteria belongs to the Viridans group of streptococci (Galdvin 2004). Traditionally oral streptococci are differentiated on the basis of simple biochemical and physiological tests. Many recent studies comparing homologous DNA, gave description of the whole protein content and detection of glicosidasis activity clarifying the relationship between many species.

Mutant streptococci represent a group of bacterial species that had previously been classified as serotypes of the same species. These bacteria are characterized by their ability to ferment manitol and sorbitol, producing extracellular glicanes from sucrose with cariogenic activity in animal models. Important for the human population are two species: S. mutans and S. sobrinus. Streptococcus mutans has got this name in 1924 when Clarke in England isolated the microorganisms from human carious lesions. He noted that they are more oval shaped, not round and assumed that they are mutants of streptococcus.

Mutant streptococci, are now considered as the main pathogenic species involved in the caries process. It is noted that if they are seeded in the mouth of animals, including rats, rodents and monkeys, are able to cause caries. Some detailed studies have shown a correlation between the presence of S.mutans and caries. These findings are repeated in longitudinal studies of microbiology and caries incidence. Mutant streptococci are usually found in relatively large numbers in plaque formed immediately after the development of lesions at the superficial soft surfaces. During a longitudinal study samples are taken periodically for analysis of separate parts for S.mutans and teeth were examined simultaneously. Teeth destined to become decayed, showed a significant increase of the ratio of S.mutans 6 to 24 months before the eventual diagnosis of caries. In similar conditions SM isolated from dental plaque terrains on stained white lesions are characterized by a ratio greater than plaque by SM while probing enamel grounds. The increased number of SM in saliva has also gone hand in hand with the development of lesions in smooth surface. In another study of saliva analysis of 200 children showed that 93 percent of them were positive for caries evident S. mutant, while uninfected children were almost always unaffected by decay (Russell 2003).

S. mutans position as the primary agent of caries formation in favor of their certain physiological characteristics. These features include the ability to adhere in tooth surface, producing insoluble polysaccharides from sucrose, rapidly producing lactic acid substrates by a number of sugars, acid tolerance and formation of intracellular stores of polysaccharides. These features help cariogenic SM survival in an environment not suitable in terms of so-called "feast or famine" cycles or due to the low concentration of substrate (i.e between meals) or excess substrate concentrations (e.g during consumption of food rich in sugar). As a general rule, cariogenic bacteria metabolize sugars to produce energy they need for growth and multiplication. The products of this metabolism are acids, which are derived from bacteria in plaque fluid. Damage caused by S.mutans is mainly due to lactic acid, although other acids such as butyric and propionic was found within the plaque. Generally, S.mutans is the most common streptococcal mutant infectious agent in humans and strong evidences are presented as the most virulent cause of odontogenic infections. Another mutans bacteria from the group of so-

called S.sobrinus, differs from S.mutans because they require sucrose for adherence and growth in the dental plaque.

Correlation between caries and S. mutans, based on the data described in the literature and based on experimental models that are performed, and based on certain conclusions (Russell 2003):

- Animal experiments: S. mutans causes caries among gnatobiotik animals in the presence of sugar;
- Virulence: S. mutans has properties that contribute to caries development. These properties are acidogenety, uric acid production, extarcelular production of glicanes and intracellular storage of polysaccharides;
- Cross-sectional studies in humans: an increase in the number of S. mutans found in the initial carious lesions;
- Longitudinal studies in humans: a large number of S. mutans in a number of tooth decays correlates with subsequent caries;
- Streptococci other "non mutans" with similar properties can also be cariogenic.

S.mutans, sugar and caries

Taking large amounts of sugar combined with low values of pH frequently leads to an increased number of S. mutans.These bacteria are characterized by these features:

- Capacity to adhere to tooth structure
- Sugar Transportation system
- Production of lactic acid from sugars
- Production of intra and extracellular polysaccharide
- Tolerance in acidic environment

S. mutans sugar transportation

S. mutans is equipped with a conveyor system more efficient to carry sugars within their cells. During the metabolic process in the cell, they produce different substances, which contribute sufficiently to their pathogenicity. When it received the greatest amount of sugars S. mutans produce mainly Lactic Acid (Hamada and Slade, 1980). Streptococcus mutans produces extracellular and intracellular polysaccharides. Extracellular polysaccharides are also produced during the enzymatic reactions. Their sticky properties are favorable for bacterial adherence capabilities on the surface of the teeth, helping their placement on smooth surfaces (Koga 1986, Loesch 1986).

Polysaccharides also help connectivity and multiplication of dental plaque. Moreover, their insolubility prevents natural protective effect of saliva. Polysaccharides ensure the survival of intracellular bacteria in nutritionally poor intervals, and are used by bacteria to produce acids (Hamada and Slade 1980).

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S. mutans, tolerance to acidic environment

Bacteria multiply under certain environmental conditions and they have obvious advantages compared with other micro-organisms. Diet and lack of suppressive factors determine the composition of the oral flora.

The decrease in pH prevents many bacteria from growth, while streptococci are multiplying in this particular environment (Harper & Loesch 1984). Changes in bacterial flora are in favor of bacteria which can survive in acidic conditions on account of acid no-tolerant microorganisms and acidic production. Pathogenic micro-organisms produce acid, the pH of which is lower enough than the value below which the tooth enamel begins to melt. S. mutans is recognized as the initiator of caries. They affect the initiation of the process leading to loss of minerals, and this facilitates the bacteria to penetrate the tooth structure (Burne 1998).

3.3.2. Substrate (Carbohydrates)

The human body uses glucose as substrate food, while other carbohydrates under the action of relevant enzymes converted into glucose. Cariotic action of sugars depends on their fermenting potential, respectively as far as the highest level of acids produced by the their fermentation. It was found that carbohydrates are the major class fermentabile affecting ecological changes in the mouth. While carbohydrates are transformed into acids, sucrose under the action of bacterial enzymes (glykosiltransferasa-GTF, and fruktosyltransferasa-FTF) turns into two classes polymers (glukan and fruktan).

Glukan plays the role of infectious matter to the surface of the tooth, not dissolved in water. This attribute enables attachment of dental plaque and S.mutans for tooth surface. Levan under the influence of enzymes derived from S. mutans fermented in the acidic product (Pincaham 1994).

Dairy products (milk, cheese) has an influence in the ecology of the area of the mouth. Dairy products can protect teeth from decay. This can happen as a result of buffer capacity of milk proteins or because of decarboxylation of amino acids after proteolysis some bacteria can metabolise kazein. Milk protein (casein) and its derivatives can be absorbed on the surface of the tooth, modify the structure of pelicula which make it unsuitable for adhesion of S. mutans, but also enable establishing of calcium phosphate and initiate the process of remineralisation.

Some sugar substitutes that do not turn into acid, as xilitoli for example you add sweets, have a role in inhibiting the development of S. Mutans (Pincaham 1994, Marsh 2000).

Correlation between SM and consumption of sugars

Studies on the correlation between presence of SM in saliva and sweet diet is not entirely clear, even data from the literature are sometimes contradictory. While some studies such as those of Polish authors has shown that children with a SM presence is 94% while 56% LB and daily frequency of sweets consumption exceeds 5 times a day (Wierzbicka, 1987). But, so it does not happen with children in Mozambique where annual consumption of sugar for school children is very low (11 kg), while the presence of SM is 98%, 40% of their high value. Sudan is also similar in that although annual consumption is about 18 kg, SM was identified in 90%, moderate values and higher than 50% (Carlsson 1989).

4. Study report

In our previous study (Begzati et al. 2010), the prevalence of ECC and various caries risk factors such as quantity of cariogenic S mutans colonies, was evaluated.

Methods

In the study there were included 1,008 children of both sexes, from 1 to 6 years of age, from 9 kindergartens of Prishtina, capital city of Kosovo. The sample was random, representing 80% of all kindergarten children. The sample size was calculated with a confidence level of 95% and a confidence interval of 2.

Bacterial sampling — Determination of S.mutans

In our study the presence of S mutans was determined using the CRT bacteria test (Ivoclar Vivadent, Liechtenstein) on the saliva previously stimulated by chewing paraffin. Bacterial counts were recorded as colony-forming units per milliliter (CFU/mL) of saliva. The number of bacterial colonies was graded as follows: Class 0 and Class 1 (CFU < 105/mL saliva), and Class 2 and Class 3 (CFU ≥ 105/mL saliva), according to the manufacturers' scoring-card (Ivoclar-Vivadent, Lichtenstein). In younger subjects, with less saliva collected, the modified spatula method was used.

Dental examination and diagnostic criteria

The children were examined in well-lit premises, using a flashlight as the light source, and a dental mirror and dental probe. Diagnostic criteria were calibrated (Hunt 1986), with inter-examiner reliability resulting in kappa = 0.91, based on the examination of 35 children of different ages. Dental caries was scored as the number of decayed, missing, or filled primary teeth (dmft).

ECC was defined as "initial occurrence of caries in cervical region of at least two maxillary incisors." Using a careful lift-the-lip examination, the presence or absence of ECC was recorded depending on the presence of "noncavity caries/white spot lesions" or "cavity caries."

In order to study the clinical and etiological aspects of ECC, a sub-sample of children with ECC was included for further analysis. The latter part of the examination, which included the clinical study of ECC development (according to ECC stages), determination of bacterial colony sampling, oral hygiene index (OHI), and filling out of the questionnaire, was conducted in the Pediatric Dentistry Clinic of the School of Dentistry.Children with ECC were examined using the light of the dental unit, with dental mirror and probe.

Clinical course of ECC

In order to explain the clinical course of ECC, we propose the following stages in the occurrence and progression of carious lesions in ECC: ECCi (initial stage), ECCc (circular stage), ECC_d (destructive stage) and ECC_r (*radix relicta* stage).

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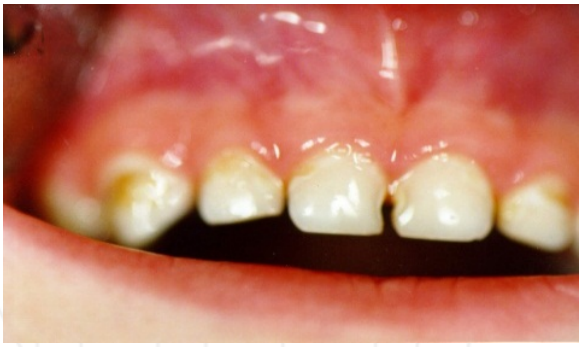

Figure 1. ECCi (initial stage)—white spot lesion or initial defect in enamel of cervix.

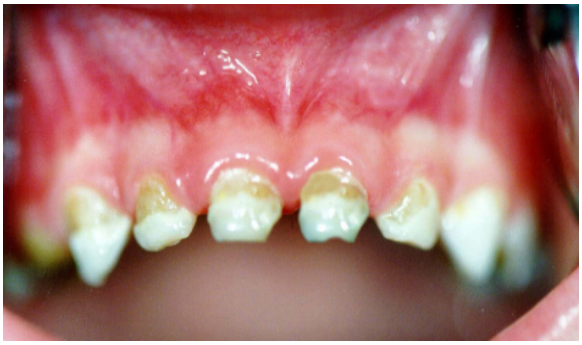

Figure 2. ECCc (circular stage)—lesion in the dentin and circular distribution of this lesion proximally.

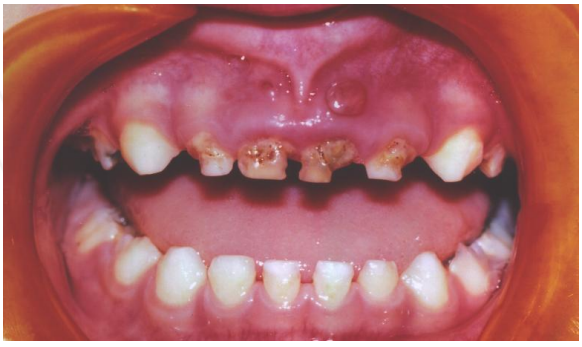

Figure 3. ECC_d (destructive stage)—destruction of more than half the crown without affecting the incisal edge.

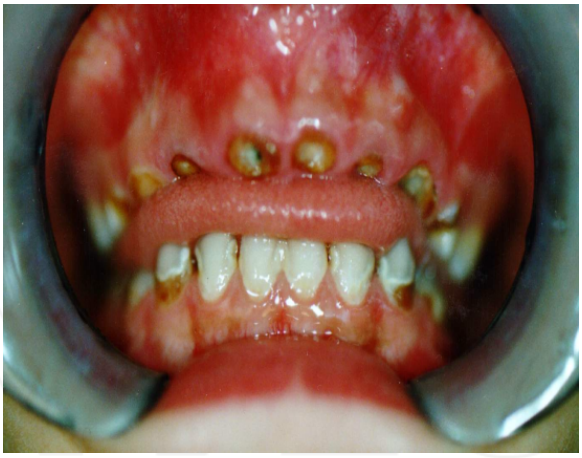

Figure 4. ECC_r (*radix relicta* stage)—total destruction of the crown.

Results of study

From the total 1,008 examined children aged 1-6 years, the caries prevalence expressed in terms of the caries index per person, or dmft > 0, was 86.31%, with a mean dmft of 5.8. The prevalence of ECC was 17.36%, or 175 out of 1,008 examined children (Figure 1). The sub-sample of children with diagnosed ECC consisted of 150 children out of 175 invited for further analysis. Twentyfive children of this group from different kindergartens didn't show up in the Department. The mean age of children with ECC was 3.8 ± 1.2 years. The mean dmft in children with ECC was 11 ± 3.6. There was no statistical difference of ECC prevalence between genders (t test = 1.81, P = 0.07).

As expected, the lowest mean dmft score was found at age 2 (6.47 ± 2.13), with an age-related increase in dmft of 12.8 at age 6 (Table 1). In comparing the mean dmft in ECC children with respect to age, there was a significant statistical difference between age 2 and ages 4, 5, and 6. (One-Way ANOVA test F = 16, P < 0.001).

ECC stages

The ECC stages were not equally distributed. The most common stage present was that of radix relicta (41.7%), while the stage appearing least frequently was the initial stage (15.4%), or 27 out of 150 children with ECC.

There was a significant difference between the stages of ECC (c2 = 211.1, P < 0.0001). Twenty-five of the 27 children with ECC in the initial stage were reexamined 1 year after the baseline examination (2 children did not appear for reexamination dueto address change). The 1-year reexamination showed that the initial stage had advanced to the circular stage in 28% of cases, destructive stage in 20%, radix relicta stage in 36%, and having been extracted due to ECC in 16% of cases (Table 2). Mean age of subjects with initial stage of ECC was 2 ± 0.7. Mean dmft on reexamination showed an increase from 5.1 to 8.8 (P < 0.001).

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4.1. Clinical specificities and progress

Even before the child is 2 years, in the gingival third of the labial surface of the upper front teeth, as a result of the enamel decalcification process a chalk colored stain ("white spot lesions") appears, which expands in the enamel of the cervical region of the tooth and for a short time it covers the entire tooth, destroying the whole hard tooth tissue. During this process, initially the enamel on the incisal region of the frontal tooth is resistant, especially canine, that shows that those parts of tooth enamel which are mineralized before birth, are more resistant to caries than the parts that are mineralized after birth (Thomas et al. 1999).

In the initial stage(Fig.1) there is a small loss of minerals from the hydroxylapatite crystals of enamel. As a result of tooth's hard tissue demineralization micropores start forming, which refract the light, and as a result it comes to the formation of so-called *white spots lesions*. Such spots are localized where the concentration of dental plaque is higher. If the destruction continues as a result of the demineralization effect of acids on enamel and apatite removal, the cavity starts to form. (Reisine & Douglass 1998).

This quick progress, helps the caries to quickly affect even the dentin layer, so for a short time the entire tooth crown is destroyed and all that remain are the roots (radix relicta)(Fig.4). Often it happens in 3 year old children, in the upper frontal region, where they have only roots remaining that resemble stumps.

Iritative formation of dentin, which makes the carious lesion get a brown color is a result of permanent irritation in the revealed dentin, while sclerotic tissue can make a full obliteration of tooth canal. The formation of iritative – sclerotic dentin can have an effect in this disease without symptoms, but with difficulties in feeding, speech and aesthetics. Also as a result of reflexive reaction (gums, tongue, lips injury etc.), a number of general symptoms is provoked such as digestive disorders, raised body temperature, increased saliva production, etc. The dental pain starts when the tooth pulp is revealed, in gangrenous teeth or when the infectious pathological process appears in periodoncium.

4.2. Complications and consequences

Early crown destruction – root remaining (radix relicta)

Sometimes, in the upper fornix we can see several changes that, in a quick glance, can lead us to the wrong diagnosis. Since the permanent teeth have palatine position, during their eruption process they put internal pressure in the apical part of the deciduous teeth, so that the deciduous tooth root tip can penetrate the bone and mucosa and erupt in the upper level of vestibular fornix (Fig.5). The erupted roots can make deep and painful decubitus at the upper lip (Fig. 8)

If in the root canal a purulent or gangrenous inflammatory process is present, then in the upper fornix we may encounter isolated purulent process (encapsulated) - abscess. (Fig. 6)

Extension of the inflammatory process - sometimes purulent inflammatory process involves gingiva, on all remaining roots, where the clinical symptoms become much more difficult.

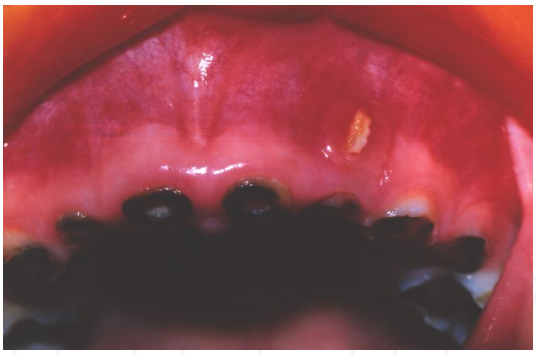

Figure 5. Radix relicta and bone penetration

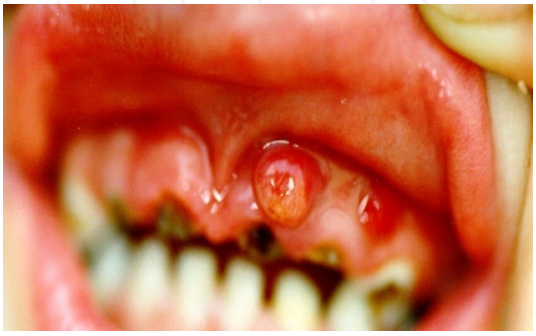

Figure 6. Abscess and fistula

Local situation – the gingiva is edematic, hyperemic and under pressure it is painful. Also while applying a slightly harder pressure from the gingival pocket purulent secret will come out. The tooth is extremely sensitive to palpation and percussion. (Fig.7)

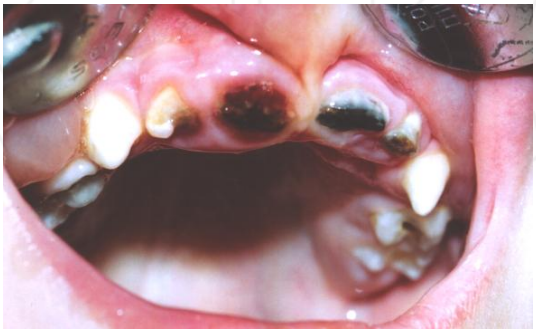

Figure 7. Edematic and hyperemic gingiva

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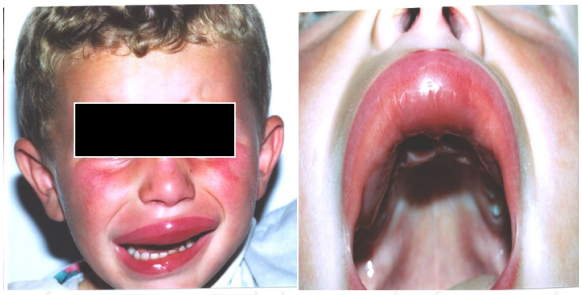

Figure 8. Spread of infection- result of ECC complications

General condition - pain, elevated temperature, fever, loss of appetite, the patient is pale and frightened. The patient cannot be fed as a result of edema of the lip and the gum inflammation. The food intake is affected due to the great sensitivity of the gangrenous roots in the upper front. (Fig.8)

Dental eruption disorders as a result of the remaining roots

As a result of root persistence, among others, it may have an effect in the eruption disorders of permanent teeth causing orthodontic abnormality (Fig. 9, 10.)

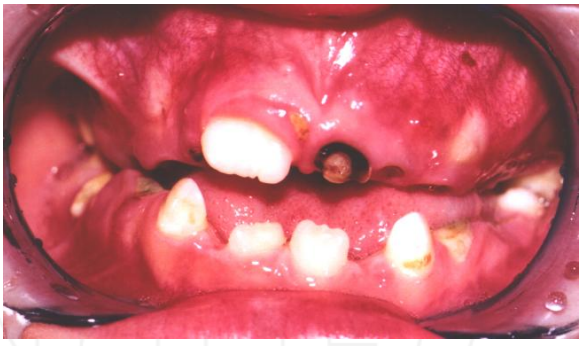

Figure 9. Persistence of radix relicta and disorders of permanent tooth eruption

Early Extraction

Consequence of an early childhood caries is the "loss" or extraction of teeth (Figure 11 & 12). Extraction of the teeth is approved when the clinical conditions become more serious, as a result of complications. But also: extraction may be due to unprofessional interference from the insisting parent, and the acceptance by the physician to do the extraction. The extraction, for example may be serial if it is decided by the therapist.

Consequences of Early extraction can be:

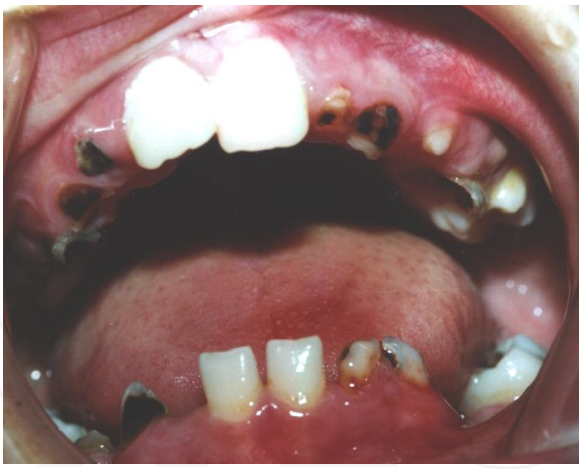

Figure 10. Orthodontic abnormality-result of radix relicta persistence

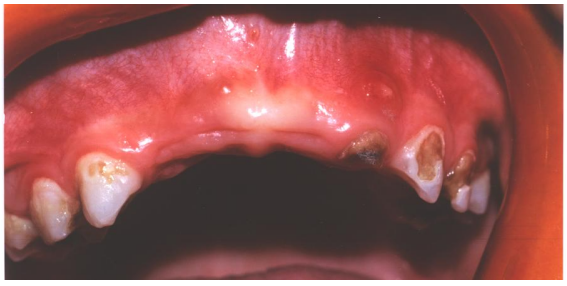

Figure 11. Early extraction of teeth

- abnormalities in the tooth eruption,
- speech impediment (incorrect pronunciation of letters),
- barriers in eating, poor aesthetics, etc.

Avoidance of these effects is done by prosthetic work, whose role would be: space maintenance, the normal pronunciation of letters, aesthetic improvement, etc.

5. Discussion

- *Risk factors of ECC*

Considering the data from the literature, the role of S mutans in the etiology of ECC, especially in the initial phase, is very crucial. These data also demonstrate the high prevalence of this

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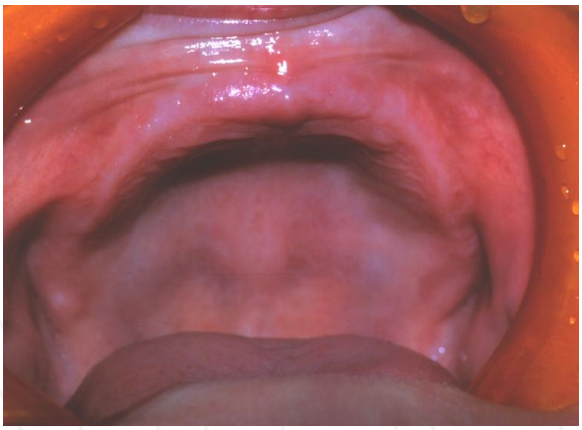

Figure 12. Total extraction of teeth-result of ECC complications

bacterium in preschool children. S mutans is found at the earliest ages, with the prevalence of 53% in 6- to 12-month-old children (Milgrom 2000), 60% in 15-month-olds (Karn 1998), 67% in 18-month-old Swedes (Hallonsten et al. 1995), and 94.7% in 3- to 4-year-old Chinese (Li et al. 1994). Almost all preschool urban Icelandic children were found to carry S mutans (Holbrook 1993). According to the studies of Ge and Caufield, all S-ECC children were S mutans–positive (Ge 2008). Borutta 2002, found that in 80% of children (3 years old) diagnosed with caries, the presence of S mutans was demonstrated, while higher counts of this bacterium were found in children with ECC.

The high prevalence of S mutans was also demonstrated in our study: 98% of preschool children. Expressed in colony-forming units (CFU/mL saliva), 93% of the ECC children in our study had a high S. mutans counts (CFU > 10^5). Higher salivary counts of *S. mutans* have been correlated with high dmft values (11.5) in our study. This significant correlation between high dmft or caries experience and high S mutans counts has been demonstrated in other studies (Köhler et al. 1995, Twetman & Frostner 1991, Maciel 2001).

In our study, the sweets consumption of children with ECC was very high. Almost 4/5 of ECC children have sweet snacks more than twice a day. It is of great concern that kindergartens as educational institutions do not have a more serious approach to a healthy diet and reduction of sugary food. On the contrary, at least once a day, sweet food (jam, chocolate, cream, biscuits, or cake) is served to children. Also, serving of this food is very common between meals. The literature also shows a high consumption of sweets between meals (Ölmez 2003) and high caries values in children who have frequent sweets (Holbrook 1989).

Another important factor in the etiology of ECC is bottle feeding, which is accompanied by high salivary counts of *S mutans.* The relationship between bottle usage and salivary counts of S mutans (Mohan 1998) has been reported. In the children that were in the study, the duration of bottle feeding with sweetened milk or juice was very long, wherein nearly 4/5 of children were bottle fed from 1 to 3 and more years.

Another harmful practice is putting children to sleep with a juice-filled bottle, which is practiced in 2/3 of children with ECC, although Johnsen has reported that 78% of parents of children with ECC had attempted to substitute water for a cariogenic liquid (e.g., apple juice, formula) in the bedtime nursing bottle [Johnsen]. A review of the literature from the etiological point of view of ECC shows that "the use of a bottle at night" is not the only cause of ECC (Plat 2000).

Oral hygiene habits established at the age of 1 can be maintained throughout early childhood (Wendt 1995). There is a high level of negligence in the oral hygiene of our children. More than half do not brush their teeth at all, exhibiting a very high oral hygiene index-OHI (1.52). The importance of the primary dentition of oral health promotion must be focused on the education of mothers to motivate their children for oral hygiene. Unfortunately, we found "bad conviction" of mothers regarding primary teeth that they will be replaced, thus neglecting the care for children's teeth. Data from the literature show that cooperation of mothers is very important in overcoming the belief that the deciduous dentition can be neglected (Rosamund 2003).

Mothers' knowledge and behaviours of oral hygiene are the key components for children's oral health care. The child imitates parental behaviours, including oral hygiene habits; thus, tooth brushing at an early age depends on maternal knowledge and behaviours. In our study, 38% of the mothers stated that their children did not brush their teeth at all. Only 11% of the interviewed mothers demonstrated proper techniques of tooth brushing. Unfortunately, a relatively low percentage of mothers (24%) stated that tooth brushing should last at least 2 to 3 minutes. The interviewed mothers rarely assisted their children during tooth brushing (5%).Even though fluoride and antimicrobial agents have a beneficial role in preventing caries, an insignificant number of interviewed mothers stated that they had knowledge regarding fluoride and they did not practice these preventive methods with their children (Begzati et al. 2014).

Besides fluoride treatments, an antimicrobial treatment option has become a serious consideration for many dental professionals. The data from the literature have confirmed the positive antibacterial role of chlorhexidine in the destruction of S. mutans colonies and inhibiting caries (Featherstone 2004, Zhang et al. 2006).

From the answers of mothers concerning fluoride use, we ascertained a marked lack of knowledge about the benefits of this agent in maintaining healthy tooth structure. This information gap can be inferred from their answers. When asked, "Do you give fluoride tablets to your child?" their answers were stated as if they have been asked about some medication: "I give those tablets to my child as needed." The absence of fluoride in Kosovo's municipal drinking water may highly influence caries prevalence rates in children.

Nutritional counseling, fluoride therapy, and oral hygiene may be required to prevent development of carious lesions in children. In the case of high-risk patients such as ECC children with a predominance of high salivary counts of S mutans, the use of either the antibacterial rinse chlorhexidine gluconate or the oral health care gel chlorhexidine has been suggested (Featherstone 2004).

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The oral health promotion and preventive measures are also influenced by social and economic factors. Statistical data from Kosovo are as follows: large families (with average size of 6.5 members), high unemployment rate (in 2008 it marked 45.4%, for female 56.4%), high birth rate (16%) and the lowest economic growth in the region [56], represent some of the aggravating factors when dealing with the health issues of the population, including oral health issues (Ministry of Public Administration. Statistical Office of Kosovo 2010).

Given the complexity of factors associated with ECC, it is unfortunate that most of the interest has only been from dental organizations. The critical change needed to accomplish the necessary research related to prevention of ECC is to expand our network through inclusion other health professionals, community leaders, national organizations serving children, and political leaders (Ismaili 1998).

- *Consequences of ECC*

Scientific research suggests that the development of ECC occurs in 3 stages. The first stage is characterized by a primary infection of the oral cavity with ECC. The second stage is the proliferation of these organisms to pathogenic levels as a consequence of frequent and prolonged exposure to cariogenic substrates. Finally, a rapid demineralization and cavitation of the enamel occurs, resulting in rampant dental caries (Wyne 1998). A 1-year follow-up of ECC development from the initial stage, representing decay at the enamel level and its progression to more destructive stages, shows even development in all affected teeth. It is quite an acute development, because in 2/3 of the children, the ECC has progressed to more complicated stages (destructive and radix relicta stages). Within 1 year, the dmft values have increased to 3.7. Consecutively, these children commonly experience pain from pulpitis, gangrene, and apical periodontitis. Also, these conditions are often followed by abscesses and cellulitis, sometimes with phlegmona, seriously endangering the child's general health. De Grauwe, in describing the progression of ECC, has noticed that the development of caries from the enamel to the dentin level can occur within 6 months (De Grauwe et al. 2004). The rapid development of ECC and its clinical appearance, especially in primary incisors, identifies it in its initial stages as a risk factor for future caries in the primary and permanent dentitions (Al-Shalan et al. 1997).

Children with congenital heart anomalies are frequent patients in our departments, some of them exhibiting severe ECC. There is strong evidence that untreated dental disease is an important etiological factor in the pathogenesis of infective endocarditis, a condition that still carries a high risk of mortality (Child 1996).

Preventive measures for ECC (Begzati et al. 2012)

Early childhood caries (ECC) is a health problem with biological, social and behavioral determinants. Intervention treatment does not resolve this problem. It is difficult, sometimes impossible and expensive.

The only safest way is prevention of this complex pathology. European Academy of Pediatric Dentistry (2008) has recommended general strategies for ECC prevention:

- Oral health assessments with counseling at regularly scheduled visits during the first year of life are an important strategy to prevent ECC
- Children's teeth should be brushed daily with a smear of fluoride toothpaste as soon as they erupt
- Professional applications of fluoride varnish are recommended at least twice yearly in groups or individuals at risk.
- Parents of infants and toddlers should be encouraged to reduce behaviours that promote the early transmission of mutans streptococci.

Based on these recommendations, we will describe detailed preventive measures: primary prevention – prenatal and postnatal care; and secondary prevention – parents' and dental professionals' role.

- **Primary prevention**

It should begin during prenatal period and it consists of pregnant woman's needs' fulfillment with necessary and healthy products;

Proper quality of food for the newborn during the enamel maturation phase;

Fluoridation of newly-erupted teeth;

Antimicrobial therapy with chlorhexidine.

- **Secondary prevention**

Mothers' education on recognizing the first signs of ECC using "lift-the-lip" technique. The aim of this measure is early detection of the so-called "white spot".

Parents should be encouraged to avoid bad feeding habits of their children and give effort for proper feeding:

- breast-feeding of the baby;
- the use of cup instead of the bottle as early as possible;
- not sleeping with bottle in mouth;
- avoid the use of fabricated juices or soda;
- the use of natural, a little sweetened, juice or tea, or just water;
- avoid the discontinuation of bottle use by the method "bottle is gone";
- reduce the liquid in the bottle, gradually by night,
- reduce sweets as much as possible;
- no sweets between meals;
- daily tooth brushing, at least twice a day, obligatory before going to bed.

Necessary consultations with the dentist -

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Professional education activity targeting primary care health providers (pediatricians, internists, family physicians, obstetricians, mid-level medical practitioners):

- early identification of disease,
- fluoride supplements as appropriate,
- healthful feeding practices,
- snacking behaviors that promote good oral health, and
- referral to the dentist by 12 months of age.

6. Conclusions

Oral health is integral to general health and should not be considered in isolation. Oral diseases have detrimental effects on an individual's physical and psychological well-being and reduce quality of life. The commonest disease is dental caries. Caries progression or reversal is determined by the balance between protective and pathological factors in the mouth. The most important component in the treatment of the caries disease is prevention. Understanding the balance between pathological factors and protective factors is the key to successful prevention of caries. Analyzing the etiology, prevalence, clinical specifics, consequences and complications, caries in general and ECC in particular are estimated as serious diseases, which represent not only health problem, but also a great serious social and economic problem.

Consequence of an early childhood caries, especially in underdeveloped countries, can be very severe, spanning from tooth loss to general health disorders. One of the complications of untreated ECC is the "loss" or extraction of teeth. Consequences of early extraction can be: abnormalities in the tooth eruption, speech impediment (incorrect pronunciation of letters), barriers in eating, poor aesthetics, etc.

The rapid development of ECC, especially in primary incisors, identifies it in its initial stages as a risk factor for future caries in the primary and permanent dentitions. There is strong evidence that untreated dental disease is an important etiological factor in the pathogenesis of infective endocarditis, a condition that still carries a high risk of mortality.

The risk factors for early childhood caries include a number of social and behavioural determinants.

Primary prevention must start in the prenatal stage to fulfill the needs of pregnancy. Parents should be encouraged to avoid bad feeding habits and to instruct and supervise their children in tooth brushing. Mothers should be instructed to use the lift-the-lip technique to spot the white-spot lesions as first signs of dental caries. Newly erupted teeth must be treated with fluoride agents, and, as needed, antimicrobial agents containing chlorhexidine and thymol. Further investigation is needed to assess the effectiveness of new intervention strategies beyond traditional measures that are not strictly dependent on access to dental professional providers.

Permanent and sustained oral health promotion organized with the participation of the entire civil society, with the mandatory presence of key stakeholders in the areas of education and healthcare, represent one of the highest priorities. The WHO strategies and objectives implementation regarding oral health promotion should be understood in the right manner and should be implemented continuously.

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