Modern Views on the Prevalence, Etiology and Pathogenesis of Dental Fluorosis in Children

Akhmedov Alibek Bahodirovich[1], Rakhmatullayeva Dilnoza Utkirovna[2]
[1]Bukhara State Medical Institute, [2]Tashkent State Dental Institute

ABSTRACT

Fluorosis is an endemic disease caused by fluoride intoxication and resulting from the consumption of drinking water with a high fluoride content. One of the earliest signs of fluorosis is tooth damage. The concentration of fluoride in water exceeding 6 mg / l can cause changes in already formed teeth. The percentage of the prevalence of fluorosis by countries and continents has been established - in African countries 46% - 84%, in North America - 22% - 78%, in South America - 53% - 89%, in Australia - 17% - 32%, in Europe - from 15% to 100%. Analyzing the information on the pathology of fluorosis in the available literature, it can be concluded that to date, no unified provisions on the causes and mechanisms of the formation of dental fluorosis have been formulated. This problem is an urgent problem and the need for further research.

KEYWORDS: *fluorosis, prevalence, etiology, pathogenesis, dental hard tissues*

In dentistry, fluorosis is commonly understood as a systemic disorder of the development of hard tissues caused by the intake of a large amount of fluoride into the body at the stages of tooth formation, accompanied by the formation of chalky and pigmented spots, the destruction of enamel. Fluorosis is an endemic disease caused by fluoride intoxication and resulting from the consumption of drinking water with an increased content of fluoride. One of the earliest signs of fluorosis is tooth damage [42, 43].

Etiology and pathogenesis. Fluorine is the most active element of the halogen series and is widely distributed in nature. An adult receives on average 0.5— 1.1 mg of fluoride per day with food and 2.2—2.5 mg with water. It is characteristic that fluoride of food products is absorbed worse [4] than fluorides soluble in water. Based on clinical observations, it was found that the optimal fluoride content in drinking water is 1 mg/l. At such a concentration, fluorosis is rarely observed (or manifests itself as a mild form) and there is a pronounced cariesostatic effect [25]. The concentration of fluoride in water exceeding 6 mg/l can cause changes in already formed teeth [19, 42]. In places with a hot climate, pronounced dental fluorosis can be observed with a moderate content of fluoride in drinking water (0.5-0.7 mg / l). This is due to the increased introduction of water into the body. The severity of dental fluorosis is also determined by the degree of sensitivity of the body to fluoride intoxication and its ability to resist this effect. The exact mechanism of fluorosis has not yet been fully studied. The idea of the hematogenic toxic effect of fluoride on the enamel oblasts during the development of the dental epithelial organ, leading to the incorrect formation of enamel, should be considered more reasonable. It is assumed that fluorine, being an enzymatic poison, reduces the activity of phosphatase and thereby disrupts the mineralization of enamel. Fluorosis mainly affects the permanent teeth of children (milk teeth are rare) who live from birth in a focus of endemic fluorosis or settled there at the age of 3-4 years. The authors believe that the intensity of the signs of this pathology directly depends on the duration of the children's stay in the endemic zone. Localization of fluoric defects is caused by the timing of mineralization of dental lesions during the

period of receipt of excessive amounts of fluoride compounds in the child's body [12, 27, 35, and 36]. This judgment is confirmed by the fact that if the fluoride content in the body is increased in infants under the age of 12 months, then fluorosis develops on the first permanent molars and central upper incisors. Further exposure of fluorine compounds to children under the age of two, three years leads to damage to the premolars and second permanent molars [15, 34].

Endemic dental fluorosis is widespread everywhere. According to the latest data from the World Health Organization (WHO), there are more than 300 foci of endemic fluorosis worldwide. The percentage ratio of the degree of fluorosis spread across countries and continents has been established - in Africa 46% - 84%, in North America - 22% - 78%, in South America - 53% - 89%, in Australia - 17% - 32%, in Europe - from 15% to 100%. Among the population of a number of African countries, such as Uganda, Nigeria, Morocco and South Africa, the incidence of fluorosis reaches 50%, in Tanzania - 74%, in Ethiopia - 84%. This can be explained by a significant overabundance of fluorides in water intake sources, soil and rocks. Individual foci of endemic of this disease can be found in the countries of the Middle East, as well as Asia: Kuwait, Kazakhstan, Iran, etc., in Azerbaijan, in some areas there is a high level of - 64% [28, 37, and 38]. According to the data obtained, Maude Ya. - In the Republic of Yemen, fluorosis affects 19.7% - 83.6% of the population of all age categories. [5]. In India, this pathology is detected in 23% - 30% of children [30, 40]. In Mexico, there is an increased level of the degree of fluorosis in children. According to the results of dental epidemiological examination, fluorosis was detected, to one degree or another, in all examined children under 12 years of age, more than 30% of cases with severe damage to the hard tissues of the teeth [3, 41]. Significantly increase the risk of developing the disease by eating fluorinated salt, uncontrolled fluoridation of water. It is noted that the severity of the disease is much more pronounced in the population of the highlands than in the highlands [39]. Based on the results of a comparative analysis conducted in areas of the United States where the permissible content of fluorides in tap water is exceeded, I concluded that the number of children affected by this pathology increased from 15% in 1940 to 75% in 2004. In most European countries (Germany, England, Italy, Bulgaria) there are also some areas with foci of endemic fluorosis. On the territory of the Russian Federation there are areas endemic to excess fluorine saturation of drinking water. Epidemiological studies conducted in Russia have revealed that in children under 12 years of age, on average, the disease with fluorosis is 6.9% [14]. But there are some endemic areas where this figure reaches 100%.

There are domestic and foreign classifications of fluorosis. According to the International Classification of Diseases ICD-10, the subsection of oral diseases is represented by the cipher K00.3 "Speckled teeth", which includes "Endemic enamel fluorosis" (K00.30); Foreign authors widely use the systematization of hard tissue lesions in fluorosis Dean H. (1942), where 5 forms are distinguished:

I form – doubtful (interrogative) fluorosis – faintly distinguishable whitish inclusions, insignificant spots are fixed on the enamel;

II form – very weak fluorosis - the appearance of chalky whitish spots covering less than a quarter of the crown surface; form

III form – weak fluorosis - similar large matte white spots are characteristic, while most of the surface layer of enamel remains intact;

IV form – moderate fluorosis - the appearance of brown spots is observed, erasability and changes in the structure of the enamel occur;

V form – severe fluorosis - lesions are fixed on absolutely all surfaces of the teeth and differ in the formation of significant areas with characteristic brown staining, the appearance of sources of

enamel destruction.

In accordance with the classification of Novice I.O., three stages of the development of dental fluorosis have been identified. Maksimenko P.T. and Nikolishin A.K. identified four degrees of fluorosis development, introduced the concepts of limited and widespread fluorosis (generalized). In our country, the greatest preference is given to the classification of V.K. Patrikeeva. He proposed to consider fluorosis in the following forms:

- I form - dashed;
- II form - spotted;
- III form - chalky-speckled;
- IV form - erosive;
- V form - destructive.

Different forms of fluorosis have their own characteristic features and signs. Both with dashed and spotted forms, the enamel surface remains smooth and shiny. With the transition to a chalky-speckled form, the gloss is lost, the enamel becomes opaque, separate chalky spots are observed on the vestibular surface of the central teeth. Moderate and severe forms of the disease are characterized by the presence of pigmented spots with different color intensity, differing in size and shape [7, 10, 24, and 26]. Erosive and destructive forms are referred to as heavy forms. Teeth affected by erosive and destructive forms of fluorosis are characterized by increased fragility and the appearance of defects in the hard tissues of the teeth, which contributes to the rapid abrasion of enamel and even dentin, leading to chipping of hard tissues [11, 16, and 20]. The patient may have not one, but a set of signs that simultaneously correspond to different forms of fluorosis: spot and merged erosion, erasability and chipping of enamel, chalky and pigmented spots [15, 19].

When diagnosing fluorosis, it is necessary to carefully analyze the visible clinical signs, they may also be characteristic of other diseases [31]. Dashed and spotted forms have similar manifestations with enamel pigmentation, caries in the spot stage and hypoplasia. The chalky-speckled form is differentiated with superficial caries, acid necrosis, hypoplasia and marble disease, Stanton-Capdepon syndrome. Erosive and destructive forms should be distinguished from superficial and medium caries, erosions, wedge-shaped defects, and imperfect amelogenesis [1, 4, 13, 14, 16, and 29]. The concept of the pathogenesis of dental fluorosis is based on the data that during the maturation phase, an increased content of fluorides can have a toxic effect on tooth enamel [6, 8]. The effect of fluoride on unformed enamel is to change enzymatic processes, which contributes to the disruption of the enamel protein matrix and the connection of protein and mineral components. Ovrutsky G.D. He claims that as a result of the direct toxic effect of fluorides on ameloblast cells, their degeneration occurs. This, in turn, stops the formation of enamel prisms and disrupts the development of enamel. Fluorides affect ameloblasts in a negative way, interfering with enzymatic processes, deprive ameloblasts of the ability to produce proteolysis enzymes that promote the breakdown of amelogenin, disrupt the ability to remove protein and water from the maturing enamel [32]. In 1981 , Fejerskov O. a scheme of fluorosis pathogenesis was proposed, according to which in the secretory phase there is a pronounced cytotoxic effect on ameloblasts, the amount and (or) composition of synthesized enamel proteins; thus, a direct effect on crystal growth is realized. During the maturation phase, there is a cytotoxic effect against ameloblasts with an effect on the enzymatic system and the enamel organ.

Thus, the direct effect of fusion on mineral metabolism is realized. In 1995, A.K. Nikolishin identified 3 stages of dental fluorosis formation:

1. The initial stage of fluorosis formation - fluorides, at the stage of enamel genesis, enter the enamel oblasts through the blood vessels of the dental sac. At the same time, fluorine ions, combining with the calcium-binding protein of the formed enamel, form hydroxyphlorapatite.

2. The period of intra-maxillary formation of fluorosis - fluoride compounds, deposited on the enamel surface in the form of calcium, form layers on hydroxyapatite crystals. The peak activity of these phenomena occurs at the age of two to four years after the completion of enamel calcification.

3. The stage of extra-maxillary formation of fluorosis is characterized by prolongation of calcium fluoride synthesis after the end of tooth eruption. Due to the insufficient strength of the bond between the calcium fluoride of the enamel surface layer and Fluor apatite of the tooth enamel, defects and destruction of enamel are formed under the influence of mechanical influences. Similar phenomena are observed for 3 years after teething. The greater the thickness of the calcium fluoride layer on the enamel surface, the greater the severity of fluorosis [17].

Many authors associate the pathogenesis of fluorosis with the fact that with a large amount of fluorides, fluorine can directly interact with structural proteins and enzymes, negatively affecting the metabolism of proteins and amino acids. Prolonged intake of excess fluoride into the body suppresses the activity of phosphatase, which negatively affects the process of enamel mineralization [16, 22, and 23].

The probability of fluorosis is not excluded at any stage of enamel development, from its maturation, secretion to final formation. Having analyzed the stages of formation of various groups of teeth, the periods of the greatest danger of the development of this pathology are established: - from 0 to 4 years - the maturation of the enamel of incisors and premolars; - from 4 to 6 years - development of the first and second molars; - older than 6 years - development of the third molars [18].

In the works of some foreign authors, it is noted that excessive fluoride content disrupts the normal expression of the gene that promotes the synthesis of the protein matrix of tooth enamel. This affects the change in the entire process of protein synthesis [9]. The high content of fluorides affects the cytoplasmic network of ameloblasts, causes the occurrence of "stress" of growing cells [44]. Anokhina A.S., based on the results of the study, notes that excessive fluorine saturation for a long period of time causes an imbalance of phosphorus-calcium metabolism, manifested by metabolic shifts in bone tissue and restructuring of the hormonal system: parathyroid hormone - calcitonin. Subsequently, metabolic changes lead to cytochemical disorders, they can affect kidney function and water-salt metabolism at the systemic level [2]. Borovsky E.V. revealed a pattern between the shape and structural disorders of the hard tissues of teeth with fluoric lesions. In the initial form of the disease, there are zones of changes in the subsurface layer that differ from each other in size and shape. The Gunter-Schrager bands are strongly outlined, the Retzius lines are clearly defined [4]. Unlike the mineralized surface layer, the subsurface layer of enamel is hypo mineralized. The following changes are observed in the chalklike degenerated enamel: the interprism spaces increase, the density of the enamel decreases, and its permeability increases. These processes cause enamel pigmentation due to the diffusion of coloring substances. In severe forms, the structures of hydroxyapatite crystals undergo changes, enamel decay zones appear. Dentinoemal connection is characterized by a toothed shape. The structure of the main substance of dentin is compacted, a hyper calcination area is detected around the dentine tubules, and the micro hardness index of dentin is increased [16]. The calcium content in enamel and dentin in teeth with a severe form of the disease is noticeably reduced in relation to the same indicators in healthy teeth. The enamel of teeth affected by fluorosis, due to the saturation of the inner layer with fluorides, is more susceptible to abrasion and chipping than normal tooth enamel [14].

How to explain the fact that there are different forms of fluorosis in one endemic area – both severe

and mild? In addition, there are absolutely healthy children in such areas [21, 33]. It can be concluded that the body's reaction to the perception of fluoride is individual. Despite the identical amount of fluoride intake from the same source of water supply, the perception of the body is not the same. In addition, there is no reliable information in the available literature about what features of dental fluorosis are observed in people who have left the endemic focus of the disease at one time or another. The identification of these features, as well as the comparative analysis with the manifestations of fluorosis in persons permanently residing in the endemic zone, in our opinion, is of great scientific and practical interest.

Thus, analyzing the information on the pathology of fluorosis in the available literature, it can be concluded that to date there are no uniform provisions on the causes and mechanisms of the formation of dental fluorosis. This problem is urgent and there is a need for further research.

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