



THE DEGREE OF PROGRESSION OF THE DISEASE AND THE DETECTION OF ENAMEL HYPOPLASIA THE METHOD OF OBSERVATION

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<https://doi.org/10.5281/zenodo.18587346>

Abstract. Hypoplasia is a congenital, or less commonly acquired, pathological condition in which the enamel of permanent or primary teeth in children fails to develop sufficiently. The condition manifests itself as the formation of various plaques and depigmented areas on the enamel surface; in some cases, the condition is accompanied by changes in the shape and size of the teeth.

Keywords. Hypoplasia, enamel, erosive hypoplasia, sulcular hypoplasia, mixed hypoplasia, oral cavity, encephalopathy, nephropathy.

Enamel hypoplasia in children is a fairly common condition associated with metabolic disorders during fetal development. The most severe form of the disease is aplasia—a complete or almost complete absence of tooth enamel. This condition is accompanied by an acute reaction to any irritant, such as cold air, food, or drinks [1,5].

Left untreated, dental hypoplasia leads to the development of dental caries, pulpitis, and the potential loss of affected teeth in children. The condition also directly impacts the development of a proper bite, which can later lead to speech problems, chronic headaches, and frequently recurring ENT problems [5].

There are three types of pathology: systemic, focal, and local.

Systemic enamel hypoplasia in children manifests as extensive damage to a large area of the dentition. It is always a congenital condition and has several forms, including:

macular – accompanied by the formation of rounded, light-colored spots on the enamel without structural damage;

erosive – characterized by the appearance of pits on the tooth surface;

sulcular – manifests as wavy grooves on the enamel;

mixed – combines two or more forms of hypoplasia simultaneously[4].

Focal enamel hypoplasia is localized and affects no more than 2-3 primary or permanent teeth. It manifests as rounded spots, pits, and grooves of varying shapes. It is acquired and develops due to infectious or traumatic injury to the oral cavity [1,7].

Focal enamel hypoplasia in children is distinguished by the fact that it can simultaneously affect both primary and permanent teeth of different ages, with structural changes in all layers of enamel developing on several teeth at once [3,9].

The disease can be associated with intrauterine developmental disorders, exposure to various pathogens, malnutrition, poor environmental conditions, and general illnesses of the mother or child[8].

During pregnancy: poor nutrition and alcohol consumption by the mother during pregnancy; endocrine pathologies; calcium metabolism disorders; infections during pregnancy: toxoplasmosis, rubella, acute respiratory viral infections, influenza; severe pathologies:

encephalopathy, nephropathy, and others; Rhesus incompatibility between mother and child. During and after childbirth: prematurity; asphyxia or trauma during birth; hemolytic disease of the newborn; perinatal infection; blood transfusion in the first days of life[6].

In the first years of life: poor nutrition; artificial feeding; chronic renal failure; severe infections; allergies; gastrointestinal pathologies; congenital diseases (hypothyroidism, metabolic disorders, cardiac and vascular pathologies); iron deficiency anemia. If the causes of the disease lie in intrauterine development, then the manifestation of symptoms is possible already in the first months after the eruption of baby teeth. By the age of 2, enamel underdevelopment can be observed in the cervical area (closer to the gum) of the central and lateral incisors, as well as on the chewing surfaces of the first molars. By the age of 4, signs of the disease become visible on the canines and second molars [8]

If the disease begins in the first years of life, the first symptoms most often appear after 6-7 years of age, during the eruption of permanent teeth. The problem can be identified by whitish spots on the teeth. Most often, the tooth already erupts with these spots. In some cases, longitudinal lines (from the gum to the tip of the tooth) or transverse waves (from the left edge of the tooth to the right) may be present instead of spots. These defects may decrease in size over time, which is associated with the continued formation of enamel [12].

In more severe cases, areas with characteristic depressions resembling erosion are visible on the surface. The tooth enamel in these areas is thin, and sometimes completely absent. The surface of the teeth is rough to the touch. The most severe manifestation of this pathology is the complete absence of enamel (aplasia). In this case, the teeth are extremely sensitive to hot, cold, and acidic substances. The hard tissues are brittle and prone to destruction [1,4].

Since enamel is underdeveloped in problem areas, it does not perform its protective function. Caries-causing bacteria easily attack it, penetrating through it into the dentin. Therefore, caries develops rapidly in young patients. This manifests as spots on the enamel—at first yellowish, then darker. Areas severely affected by caries have a brownish tint and the presence of cavities.

Based on the location of the problem, the following are distinguished:

systemic enamel hypoplasia—observed on all teeth, most often manifesting in the primary dentition;

localized enamel hypoplasia—defects in the form of spots are observed on individual teeth (usually 1-2), a problem typical of the permanent dentition;

focal—significant damage to primary or permanent teeth, their size smaller than normal, and the surface covered with rough spots and erosions.

Based on the defect form, hyperplasia can be of the following types.

Spotted. The simplest form of the disease, characterized by enamel clouding. The spots are smooth to the touch, and their color is most often whitish or light brown. They are clearly defined and easily visible under bright frontal light. They are most often detected by a dentist during a routine examination[7].

Erosive (cup-shaped). Pits with thin or absent enamel can be seen on the surface. Teeth are sensitive to cold and hot. And areas of erosion are most susceptible to caries.

Wavy. The defects take the form of small, wave-like depressions that extend from the roots to the edges of the teeth.

Sulcular. One or more grooves, in which the enamel is thin or absent, run from one edge of the tooth to the other.

Combined. A severe form in which two or three types of defects are present on the tooth surface. Most often, this is a combination of erosions and sulcular depressions.

Aplasia. A complete absence of enamel due to its underdevelopment. Patients suffer from dental hypersensitivity. Exposed dentin becomes easy prey for bacteria. Due to caries, teeth become weak, and multiple chips are observed.

This disease is characterized by symmetrical defects on the left and right sides. We listed the risk factors that can trigger these disorders above. The cause of the disease is a disruption in the child's metabolism responsible for enamel formation [5].

Teeth buds appear in the fetus in the fourth month of pregnancy. Dentin, the main dental tissue, forms first, followed by enamel, the surface layer. Important components of enamel formation are the processes of mineralization and calcification. At the initial stage of development, enamel is soft and resembles cartilage. It contains up to 30% organic matter, which is gradually replaced by mineral salts. Before teeth eruption, enamel goes through two stages of maturation. The third occurs after eruption. Mature enamel is 95% mineral salts. It is the hardest, acellular tissue in the body, and contains no blood vessels or nerves[3,5,9].

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