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DENTAL STATUS IN CHILDREN AND ADOLESCENTS WITH CONNECTIVE TISSUE DYSPLASIA

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Abstract

It is known that connective tissue (CT) forms the basis of the body, participates in the formation of the structure of all organs and systems. At the same time, CT dysplasia (DCT) is primarily due to the fact that CT dysfunction can affect the course of intercurrent diseases – causing their faster or gradual development. Today, according to classical traditions, DCT is considered as a pathological condition - caused by a genetically determined violation of the development of CT in the embryonic and postnatal periods [12].

There is morphological confirmation of DCT as hereditary changes in the genes encoding the synthesis and spatial organization of collagen, structural protein-carbohydrate complexes, as well as gene defects of enzymes and cofactors to them, leading to changes in the structure of collagen, elastic fibrils, glycoproteins, proteoglycans and fibroblasts. [17] Other authors also claim that the basis of CTD are molecular genetic and ontogenetic, pathogenetic mechanisms that lead to changes in its structure and function. Tissue abnormalities of ontogenesis are closely associated with both disorders of collagen synthesis and fibrillogenesis, and with changes in its biodegradation, fermentopathies, defects of fibronectin, elastin, glycoproteins, proteoglycans, as well as with a deficiency of various enzyme cofactors (copper, zinc, ascorbic acid, oxygen, etc.) involved in the formation of transverse covalent bonds necessary for stabilization of collagen structures [18]. It is also known that the pathology of CT is the basis for the formation of chronic diseases in children and adolescents. However, it has been described that carrying CTD increases the risk of developing acute life-threatening conditions for the body, such as aortic aneurysm, thromboembolism, infectious endocarditis and arrhythmias, which can cause sudden death of patients [4]. This determines the significance and prospects for the development of CT pathology problems for practical medicine.

DCT classifications are often used, which are based on the statement of the phenotype, on the point system and on the allocation of certain syndromes, while the criteria for the severity of DCT include a set of points determined on the basis of the diagnostic significance of individual phenotypic and visceral signs. The classification of DCT is proposed, which distinguishes specific syndromes (asthenic, vertebrogenic, valvular, vascular, thoracodiaphragmatic, muscular, bronchopulmonary, arrhythmic, articular, visceral, hemorrhagic, syndromes of pathology of vision and foot, neurological disorders, small heart abnormalities, etc.), each of which includes a number of symptoms [10]. For example, 94% of young people have single external phenotypic signs, however, when diagnosing DCT for six or more external signs, the frequency of DCT detection decreases to 20-25%, and the clinical significance of the detected anomalies increases, also, according to skeletal pathology – in 57-94% of all dysplasias; of them; kyphoscoliosis (70-80%), flat feet (60-78%), arachnodactyly (36%), hollow foot (16%), hyperkyphosis, hyperlordosis (11-19%), hypermobility of joints (25-33%), polysegmental early osteochondrosis (38%) [4].

Currently, there is information about the role of exogenous factors in the development of DCT, that the variety of clinical manifestations of dystrophy can be explained not only by mutation of various genes or variable expressiveness of one of them, but also by the action of environmental factors [8]. It has been shown that, for example, the variability of the phenotype in Ehlers-Danlos syndrome (SED) is caused by genetic defects only in 11.5% of cases, and in the rest it is associated with the influence of environmental factors [7]. Dysplastic changes in CT can be caused by unfavorable environmental conditions, inadequate nutrition, stresses that affected the body during ontogenesis. Hereditary diseases of CT are divided into differentiated (DDCT) and undifferentiated (UDCT). DDCT is characterized by a certain type of inheritance, a clearly defined clinical picture, and in some cases – established and fairly well-studied genetic or biochemical

defects - osteogenesis imperfecta, SED and Marfan syndrome. NDTS are widespread and are diagnosed when a patient's set of phenotypic signs does not fit into the clinical picture of any of the collagenopathies [7, 10].

UDCT - according to terminology; "undifferentiated", "primary", "small forms of dysplasia", "mild generalized pathology", acronym "MASS-phenotype" (Mitral valve, Aorta, Skeleton, Skin) [19,21] acronym CSF-phenotype (Skin, Heart, Skull) [17], widely they are common and are diagnosed when a patient's set of phenotypic signs does not fit into the clinical picture of any of the collagenopathies. In addition, the literature uses terms such as nosologically independent DCT syndrome of polygenic-multifactorial nature, manifested by external phenotypic signs with DCT and clinically significant dysfunction of one or more internal organs [18].

According to clinical signs, the authors divide DCT into three groups corresponding to the primary laying of organs in embryogenesis – meso-, ecto- and endodermal anomalies. When mesodermal anomalies are manifested by changes in the skeleton, skin and soft tissues, internal organs, blood vessels [18].

Changes from the teeth (abnormal location, incorrect formation and earlier prolapse, enamel hypoplasia, multiple caries, gum resorption), eyes (ectopia and mobility of the lens, aphakia, coloboma), central nervous system (CNS) (anisocoria, asymmetry of tendon reflexes, nystagmus, pyramidal disorders) have ectodermal origin, and long and the hypoplastic intestine is endodermal [7]. Skeletal changes are largely associated with a violation of the structure of cartilage. In patients with DCT, a delay in maturation of the epiphyseal cartilage growth zone is determined, which is clinically expressed by elongation of tubular bones [12, 18]. Also, according to the authors, the functional state of CT is closely interrelated with the activity of the immune system; thus, during the clinical and instrumental examination of patients with DCT, it was found that with phenotypically brighter forms of DCT, concomitant diseases of internal organs develop more often. Disorders in the immune system of patients are expressed by immunodeficiency, which at the clinical level is manifested by recurrent and chronic inflammatory processes of the upper respiratory tract, ENT organs, lungs, kidneys and skin [17]. Histocompatibility antigens of classes I and II are not only associated with a predisposition to the development of CTD and with the peculiarities of its clinical manifestations, but also determine the presence of abnormalities in the immune system, which indicates the important role of genetic factors in the pathogenesis of these diseases.

With dysplasia of the renal tissue and with hereditary nephritis, anomalies of the structure of the auricles, epicanthus, bone anomalies are often noted, varus deformity of the little finger is very characteristic [20, 22].

Features of the course of orthopedic diseases against the background of DCT: Dysplastic changes of the cervical spine (DCHCS) - initial manifestations already in 5-7 years, intense temperature, growth, the child complains of frequent headaches, fatigue. Parents note a violation of posture, incorrect setting of the feet. Pedigree analysis reveals segregation of the main phenotypic signs of DCT, osteochondrosis, deforming arthrosis. Chest deformity initial manifestations at the age of 3-5 years, there are no complaints, external cosmetic defects after 7-10 years are accompanied by recurrent bronchitis, changes in the functional state of the cardiovascular and respiratory systems, spinal deformities. Scoliosis is more common in girls aged 12-13 years. In a short time, spinal deformity increases in the thoracic and lumbar regions. During a comprehensive examination, pathology from the cardiovascular, respiratory systems, dental anomalies (PCA), pathology of the feet are revealed. In the absence of stage-by-stage treatment, dysplastic changes in the body turn into a decompensated stage requiring surgical treatment. Flat feet at an early age, a flat-footed foot is detected due to the weakness of the calf muscles, a decrease in their contractile function. Treatment requires a comprehensive approach, as only local exposure to the feet does not give an effect. The results of the study of the cervical spine (DCHCS) and bone mineral density (BMD) radiologically (R) in children with DCT, C1–C2 subluxation and C1 hypoplasia (25.0 and 0%) are detected, almost half of the examined patients are diagnosed with instability of DCHCS (46.0 and 0%) and a violation of statics by the type of kyphosis and /or scoliosis (40.4 and 0%). Hypoplasia of the dentoid process C2 is significantly more often observed (11.5 and 0%). There was no significant relationship between the frequency of headaches, the

nature of R-changes in the DCHCS and the severity of DCT. According to two-energy R-th absorptiometry (osteodensitometry) in two zones (lumbar spine and the entire skeleton), BMD disorders in children with DCT were more often detected in the lumbar vertebrae, reflecting the state of mineral metabolism in spongy bone tissue. This indicator is more sensitive within the framework of the method. The decrease in BMD is more significant - it was noted in 52.8% of children with DCT and 16.7% of children in the comparison group. In one third of the examined children, the decrease in BMD corresponded to osteoporosis. Children with BMD below the age reference standards were 88.9 and 58.3% respectively, which is also significant. The BMD of the entire skeleton reflects the density of tubular bones, fractures of which are more common in children [6, 7].

The most frequent manifestations of DCT detected in a child with diseases of the ENT organs are high palate, soft, easily collapsing auricles, malocclusion, spinal deformity. The condition of the upper respiratory system depends on the degree of its severity. The overwhelming number of children with grade I severity of DCT have no significant changes in the state of the ENT organs. Features of children with grade II DCT: difficulty in nasal breathing, swelling of the nasal mucosa, curvature of the nasal septum in the cartilaginous region, loosening of the mucous membrane of the oropharyngeal region, from diseases and pathological conditions — vasomotor and allergic rhinitis and hypertrophy of the palatine tonsils. Characteristic changes for children with grade III severity of DCT: irregular shape of the external nose, cyanotic tinge of the nasal mucosa, hypertrophy of adenoids of II–III-degree, increased dryness of the oropharyngeal mucosa, hypertrophy of the palatine tonsils of II–III-degree, polypus growths of the nasal mucosa. Every second child has adenoiditis and chronic tonsillitis [2].

Anomalies of development and deformities of the maxillofacial region in children against the background of DCT - with pathology of the maxillofacial region (MR) segregation of the main phenotypic signs of DCT in families of children with HFA and HHD in MR. History; high incidence of intrauterine infections (20.6%), early (21.9%) and late (24.7%) severe first trimester gestosis in pregnant women, asphyxia in childbirth (17.1%). In 38.5% of cases, rapid and rapid childbirth was observed in children with acquired DS deformities [18]. With acquired deformities of MR - pain of various localization and increased fatigue, the severity of which increases by 9-13 and especially by 14-17 years; pedigree analysis - segregation of signs of DCT in families of children with developmental anomalies and deformities of MR, external: high gothic palate, malocclusion, over-complete teeth, soft auricles, thin and/or moderately stretchy skin and keloid scars. From the visceral side - deformities of the upper and lower extremities; scoliosis; GMS; flat feet; flat-footed foot position; PMK; muscle hypotension; pathology of the visual organs. Features of macro- and microcirculation in CSF in children with anomalies and deformities of CSF and concomitant DCT: a tendency to hypotension and the predominance of parasympathetic autonomic regulation; increased extensibility of the vascular wall; indirect signs of local tissue hypoxia in patients of younger age group [1].

Features of the clinical course of developmental anomalies and deformities of the MR region in children against the background of DCT; absence in the anamnesis of a child with TMJ ankylosis or lower micrognathia of factors contributing to the formation of MR deformity, high gothic palate, malocclusion pathology, over-complete teeth, soft, easily tubulated auricles, thin skin with a tendency to hyperextension and keloid scars; identification of signs of impaired macro- and microcirculation in the MR, manifested by a tendency to hypotension. In 75.3% of children with developmental abnormalities and acquired deformities of MR, concomitant severe and moderate DCT is observed [9], which dictates the expediency of its timely diagnosis and assessment of severity.

Dental manifestations accompanying DCT in adolescents; craniofacial and ocular (dysmorphia of the cerebral and facial skeleton; distal, deep, orthognathic, rectus occlusion, combined malocclusion; tremors, diastema, crowding of teeth; anomalies of attachment of the mucous membrane of the mouth and tongue; small vestibule of the mouth; desquamative gingivitis; geographical tongue; flat, high, gothic palate), characteristic of periodontal disease; defects of dentition due to adentia / hypodontia or oligodontia of the third molars, premolars, less often incisors, canines. Often there is dysfunction of the TMJ and masticatory muscles

(hypotension, parafunctions); non-carious lesions (primary, secondary), teething disorders, retention, milk, supercomplete teeth, caries, violation of oral hygiene (OC) and microcirculation. There are defects of eruption, dystopia, retention of teeth. There are overcomplicated teeth, baby teeth in constant bite, changes in the size, shape, structure of teeth. Clinical variants of latent course or previously suffered TMJ dysfunction are possible when auscultative noises in the joint are detected. TMJ dysfunction syndrome can be observed with a decrease in the interalveolar distance, secondary occlusion deformities, parafunctions [17].

The most frequent manifestations of DCT accompanying dental diseases in a teenager: External: asthenic physique, posture disorder, foot pathology; dolichocephalic, mesocephalic types of the cerebral skeleton, convex, straight, rarely concave types of the face profile. Features of aesthetic parameters are a relative increase in the width of the nose in the area of the wings and nasal shells, a relative increase in the morphological height of the face due to a relative decrease in the width of the face in the areas of the zygomatic arches, angles of the lower jaw, a relative decrease in the nasal angle (lowering of the tip of the nose). At the same time, visceral features of aesthetic parameters are a relative increase in the width of the nose in the area of the wings and nasal shells, a relative increase in the morphological height of the face due to a relative decrease in the width of the face in the areas of the zygomatic arches, angles of the lower jaw, a relative decrease under the nasal angle [3, 17, 18].

It is commonly believed that the disease (syndrome) Marfan was described by the French pediatrician A. Marfan in 1896. Subsequently, it was shown that the patient presented by him had a different pathology. The true clinical picture of Marfan's disease, apparently, was first described by E.S. Ashar in 1902. Marfan disease (OMIM1 No.154700) is a hereditary CT disease with an autosomal dominant type of inheritance [3] according to the authors, the disease is based on a violation of the synthesis of one of the main CT proteins - fibrillin, which leads to a disturbed structure of the type I collagen and elastin chains that are part of the structure of heart valves, myocardium, vascular walls, the organ of vision and the musculoskeletal system. Marfan disease has a pronounced genetic heterogeneity: to date, more than 550 mutations of the FBN1 gene have been identified in various families. Among them, missensmutations account for 57%, mutations with a reading frame shift - 18%, mutations in splicing sites - 16%, nonsense mutations - 8%. In recent years, Marfan syndrome type 2 has been isolated due to mutation of the gene of the transforming growth factor receptor 2. Skeletal deformities and cardiovascular disorders prevail in the clinical status, but there is no eye damage. Apparently, in some cases, Marfan syndrome is associated with mutations in the FBN2, FBN3 genes. Therefore, it is advisable to use not only clinical, but also molecular genetic methods to verify the diagnosis.

The organ specificity of the cellular elements of CT is expressed in the number, shape and ratio of different types of cells, in their metabolism and functions optimally adapted to the function of the organ. The specificity of cellular elements is also manifested in their interaction with each other, in the features of their internal structure. The specificity of CT is also found in the ratio of cells and extracellular structures in various parts of the body. Violation of the structure and metabolism of CT is currently considered as DCT, which in most cases has a systemic character. According to the authors, DCT is a violation of the development of CT in the embryonic and postnatal periods due to genetically altered fibrillogenesis of the non—cellular matrix, leading to a disorder of homeostasis at the tissue, organ and organismal levels and characterized by a progressive course [7].

According to the authors, one of the types of differentiated DCT is osteogenesis imperfecta (OI). Osteogenesis imperfecta is a group of hereditary diseases characterized by generalized osteopenia and bone fragility. With all types of osteogeneses imperfecta, the risk of fractures is increased even with minor loads and injuries. The population frequency of OI is 7.2:10,000 [18]. Osteogenesis imperfecta is caused by mutations that disrupt the primary structure, synthesis and assembly of type I collagen. This type of collagen is part of loose and dense connective tissue, but predominates in bone. Therefore, the most severe lesions are bone lesions.

Electron microscopic examination has shown that most osteoblasts contain a reduced number of elements of granular endoplasmic reticulin, while in osteoblasts of normal bone, the granular endoplasmic network is well developed. The latter is responsible for the synthetic

processes of proteins and enzymes. In addition, the mitochondria of osteoblasts of bone tissue contain osmiophilic inclusions in the form of individual crystals of hydroxyapatite or their clusters. Mitochondria have a swollen matrix and disintegration of cysts. At the same time, the ultrastructure of the osteocyte has its own characteristics. The absence of partitions of mineralized bone base material between individual osteocytes indicates delayed bone formation, possibly associated with a decrease in the function of bone cells. When studying the ultrastructure of the mineralized matrix of bone beams, it was found that the mineralization intensity of collagen fibrils is not the same everywhere. A sharp decrease in intraperiod transverse lines, as well as changes in the diameter of fibrils and their disintegration explain the fragile connection between hydroxyapatite crystals and collagen fibrils [17]. The clinical picture, BUT depends on the type of disease, the age of its manifestation, the severity of the lesion of the skeleton and other organs. If the disease is detected in a newborn, then it is most likely BUT type II (the traditional name is congenital osteogenesis imperfecta). When, BUT type II, a child is born with multiple fractures of long tubular bones; after the fractures heal, the bones shorten, thicken and deform. The skull is soft, resembles a rubber bag, on which individual bone plates are felt. Fontanelles and seams are wide. The birth of a dead fetus or the early death of a newborn is usually caused by respiratory failure or damage to the central nervous system [7].

According to the literature data, the results of the study of the frequency and structure of dental diseases in children with DCT in the Sakha Republic indicate an unfavorable clinical and epidemiological situation. At the same time, in the key age group of 12-year-olds, a high level of intensity of dental caries damage is determined, the dynamic analysis carried out indicates an increase in the trend line over the last period. The structure of periodontal diseases in school-age children is gingivitis, mainly a chronic catarrhal form. Frequent phenotypic signs of DCT, manifested in PTSD, are TMJ dysfunctions, anomalies of the position of teeth and attachment of the bridles of the tongue and lips, PTSD. In conclusion, the authors establish a high level of prevalence of dental diseases and phenotypic signs manifested in the ESR. At the same time, the intensity of tooth decay in the key age group of 12-year-olds is characterized as a high level, and the examined children have an unsatisfactory hygienic condition of the teeth, which indicates a low level of sanitary culture of children and their parents. In the structure of phenotypic signs of DCT, TMJ and HFA dysfunctions were most often detected. The revealed clinical features of DCT manifestations in organs and tissues of OC, as well as MR require an integrated approach. According to the authors, the facts dictate the need for further research aimed at preventing DCT, as well as improving the provision of dental care to children in the region [14].

A number of authors claim that the total frequency of hereditary DCT is a fraction of a percent, while NDCT are much more widespread, reaching in some populations from 10% to 30%. Hereditary DCT is a heterogeneous group of monogenic diseases caused by genetic defects in the synthesis and breakdown of extracellular matrix proteins, as well as proteins involved in the morphogenesis of CT [10]. According to the authors, NDCT with a marfanoid phenotype is characterized by skeletal anomalies, small anomalies of the heart and blood vessels, visual organ disorders, etc. With NDT with an ehler-like phenotype, patients show a tendency to hyperextension of the skin, hypermobility of the joints, bluish sclera and other signs of CT of varying severity. Diagnosis of the severity of undifferentiated DCT in children should be comprehensive and include a thorough clinical examination of a sick child and his family members according to a single diagnostic program, targeted instrumental diagnostics taking into account the "target organ" and a special laboratory examination [9].

According to other authors [13], congenital inferiority of CT can be diagnosed if a child has a combination of main and secondary signs. In total, the classification contains 15 signs related mainly to the main ones – hyper-mobility of the joints, hyperelasticity of the skin, deformation of the chest and spine, pathology of the organ of vision, high palate, flat feet and pronounced venous network on the skin. The authors attribute anomalies of the auricles, teeth, hernias, etc. to secondary signs. A mild degree of DCT is diagnosed when 2 mains; the average – 3-4 main and 2 secondaries, and pronounced – 5 or more main and 3-4 secondary signs. The disadvantage of this classification, in our opinion, is a limited number of main and secondary signs, which undoubtedly plays an important role in the diagnosis of connective tissue dysplasia.

The author [15] studied the relationship of a significant number of major and minor clinical manifestations of connective tissue dysplasia in groups of sick and healthy children. The association of joint hypermobility with hyper elasticity and severity of the venous network of the skin, chest deformity, flat feet, weakness of the muscles of the anterior abdominal wall and transverse striation of the feet was revealed. A less pronounced correlation was noted between hypertelorism of the eyes and epicanthus, sandal-shaped cleft of the foot and wrinkled skin, i.e., with secondary signs. It was found that only 39 out of 65 phenotypic traits studied significantly correlate with the severity of DCT.

According to the results of the study, the authors found that the lesion of the hard tissues of the teeth and periodontal tissue lesions with the pathologies of DDCT and NDCT, at all ages with the pathologies of DDCT is more marked than in patients with NDCT; at the same time, the incidence of caries, non-carious lesions and periodontal tissue lesions is 20% to 2 times less among patients, which means that the analysis of these data confirms that carious and non-carious lesions of hard dental tissues and periodontal tissue diseases increases directly with the age of patients in each group, while the indicators of patients' C/G were lower than in patients of the 1st and 2nd groups. Also, in patients with DDCT and NDCT, there is a deterioration in the indicators of the hygienic condition of the OC directly related to the increase in age and the depth of the periodontal pocket in patients with DDCT (syn. Marfan) aged 18-20 years -5.4 ± 0.1; 21-29 years -6.4± 0.4; 30-37 years -8.8±0.6 or in patients with pathologies of NDCT 3.2± 0.1; -4.4±0.8; -5.8±0.8 respectively [5]. They observed in the R-th picture that periodontitis tissues in DCT are specific, that is, progressive disease and bone resorption do not always correspond to reality. With the observance of hygienic rules, and sometimes without them, in most patients, bone changes remain stable for years both in quantitative and qualitative characteristics. Analysis of the results of parathyroid hormones and electrolytes studies claim that the average values of Ca, P and Mg in men and women with Marfan syndrome and NDCT did not have significant differences in comparative evaluation ($p>0.05$). In relation to/ G, the Mg content in the blood serum was significantly reduced ($p <0.05$), and P - increased, moreover, in patients with Marfan syndrome, the level of these indicators was more pronounced than in NDCT. In men with Marfan syndrome, serum osteocalcin was significantly reduced not only in relation to the data K/G ($p <0.05$), but also in relation to the indicators of women suffering from this disease ($p <0.05$). The same changes are observed in the assessment of the content of alkaline phosphatase: its level in men significantly exceeded that in women and C/G ($p <0.05$). However, it should be noted that in women with Marfan syndrome, the level of alkaline phosphatase in the blood serum was significantly higher than in those with C/G ($p <0.05$). In conclusion, the authors cite data that the condition of the hard tissues of the teeth against the background of reduced BMD is characterized by a high intensity of the number of teeth removed; also, in patients with DCT, there are specific changes in the periodontal tissue's characteristic of severe periodontal pathology. At the same time, the course of severe CGP in young and middle-aged people has gender differences. In addition, these changes are noted in young and middle-aged patients without inflammatory periodontal pathology, especially in patients with Marfan syndromes, which can serve as a diagnostic criterion for a decrease in BMD. An imbalance in the system of Ca-regulating hormones in middle-aged patients with DCT and NDCT of both sexes contributes to the development of an aggressive course of the disease, which is determined by a significantly significant ($p <0.05$) deterioration in all indicators of periodontal indices, an increase in attachment loss and a greater degree of bone resorption. Thus, the analysis of the literature available to us has shown that the study of the role of disorders of mineral and bone metabolism in the occurrence and development of aggressive forms of periodontitis has a broad scientific perspective. Obtaining new data on the state of systemic regulatory factors and their influence on the local mechanisms of periodontal tissue damage will not only improve the diagnosis of various forms, but also increase the effectiveness of treatment of this pathology.

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