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DEGENERATIVE HIP DISORDERS IN CHILDREN

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A group of degenerative hip disorders in children is discussed in the current review. The key pathogenic focus of these disturbances is an initial hyaline cartilage alteration or subchondral bone, which provokes damage of the epiphyseal hip zone. Eventually, such events lead to a local inflammatory reaction in the hip joint, cytokine cascade with hypoxia and ischemia, and apoptosis and necrosis in the hip. Developmental hip dysplasia, Legg-Calvé-Perthes disease, and slipped capital femoral epiphysis are analyzed in this review as the spreading forms of degenerative hip disorders in children. The key points of etiology, pathogenesis, diagnostics, and treatment of each disease are characterized. A group of degenerative hip joint diseases remains under the close supervision of pediatric orthopedists and traumatologists because of their high prevalence, severity of clinical manifestations, damage of life quality, and development of complications in the form of arthritis. In addition, the lack of unified approaches to the application of treatment methods for degenerative hip joint diseases is the subject of discussion among surgeons and often causes a decrease in the quality of care in terms of time and volume.

Keywords: degenerative hip disorders in children; children; hyaline cartilage alteration; osteonecrosis.

ДЕГЕНЕРАТИВНЫЕ ЗАБОЛЕВАНИЯ ТАЗОБЕДРЕННЫХ СУСТАВОВ У ДЕТЕЙ

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В представленном обзоре освещена проблема группы дегенеративных заболеваний тазобедренных суставов у детей (ДЗТС). Для данной гетерогенной по этиологии группы болезней характерно общее ключевое звено патогенеза в виде первичной альтерации гиалинового хряща и/или субхондральной кости с последующим вовлечением в процесс метаэпифиза проксимального отдела бедра. Впоследствии данные нарушения приводят к развитию локального воспалительного ответа внутри сустава, каскадной реакции цитокинов с исходом в гипоксико-ишемическое повреждение структур, апоптоз и некроз с точки зрения патофизиологии. Представлены наиболее часто встречаемые варианты ДЗТС, к которым относятся дисплазия тазобедренных суставов, болезнь Легга – Кальве – Пертеса, юношеский эпифизеолиз головки бедра. Освещены основные моменты, касающиеся этиологии, патогенеза, методов диагностики и лечения каждой нозологии. Сделан вывод о том, что группа ДЗТС находится под пристальным наблюдением детских ортопедов-травматологов в связи со значительной распространенностью, тяжестью клинических проявлений, ухудшением качества жизни и развитием осложнений в виде артроза. Кроме того, отсутствие единых подходов к применению методов лечения ДЗТС является предметом дискуссий хирургов и зачастую обусловливает снижение качества оказания помощи по срокам и объемам.

Ключевые слова: дегенеративные заболевания тазобедренных суставов; дети; повреждение гиалинового хряща; остеонекроз.

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The pathology of hip joints remains an urgent problem in the field of pediatric orthopedics in spite of significant advances in the diagnostics and treatment of diseases [1, 2]. The highest frequency of visits to a pediatric orthopedist is associated with degenerative diseases of the hip joints (DDHJ), with primary alteration in the hyaline cartilage and/or subchondral bone with subsequent involvement of the proximal femur into the epiphyseal cartilage process being a key element in the pathogenesis [1, 2]. Subsequently, these disorders lead to the development of a local inflammatory response in the joint, a cascade reaction of cytokine response resulting in hypoxic–ischemic structural damage, apoptosis, and necrosis from the viewpoint of pathophysiology [3]. Clinical and instrumental examination of children with DDHJ reveals impairment in the anatomical relationship in the joint, development of foci of heterotopic ossification (osteophytosis), restriction of the amplitude of movements, and deterioration of the criteria for quality of life given by the World Health Organization (physical, psychological, level of independence, social life, and the environment) [2].

According to the most acceptable proposed classification provided by Zucker et al. [4], DDHJ in children includes the following:

1) Diseases with a genetic/congenital predisposition, namely hip dysplasia (subluxation, dislocation)
2) Diseases acquired during the growth period, namely Legg–Calve–Perthes disease (LCPD), varus deformity of the femoral neck (*coxa vara*)
3) Diseases of a traumatic nature, namely slipped capital femoral epiphysis (SCFE)

Dysplasia of hip joints (DHJ) is a pathology of childhood associated with anatomical and biomechanical as well as biochemical and histological changes in the structural tissues of the hip joint [3, 5, 6]. There are both mild forms of the disease (minimal changes in the acetabular head and hood) and moderate and severe (marked disorders), with options for stability and instability of the joint as a whole [7]. The actual prevalence of DHJ exceeds the official data for different countries of the world, and even official figures vary widely. Thus, a detailed systematic review of data from more than 44 studies on dysplasia in Europe, America, and Australia reported a prevalence of 1.6–28.5 per 1000 people for the unstable forms and 1–3 per 1000 people for the stable forms [7]. The problem of specifying epidemiological data for the disease is directly related to the lack of official universal criteria and the classification concept of DHJ.

The effect of breech presentation, swaddling, and burdened family history are proven risk factors for DHJ. However, most researchers believe that risk factors worsen the prognosis for the development of the pathology slightly, with the exception of female sex, a factor that increases the disease risk by 75% [7]. Embryologically, the following periods of DHJ risk can be distinguished: *the period of 12 weeks* is a period of medial rotation of the lower limb around the center point of the hip joint axis, and this variant of dysplasia is attributed to intrauterine developmental defects; *the period of 18 weeks* is a critical period for the maturation of the muscle tissue and its nervous environment that stimulates the development of DHJ of a neuromuscular nature with predominant damage to the head, meta-, and epiphysis of the femur; *the perinatal period and the week 1 after birth* is a period of enhanced growth of the femoral head relative to the acetabulum and its insufficient coverage and leads to DHJ with impaired joint biomechanics, oligoamnios, and breech presentation; and *the postnatal period* is a period of rapid growth of the acetabular labrum and ligamentous apparatus with their extensibility, instability, and softening under the influence of estrogens, which causes functional insufficiency and dysplasia [8, 9].

The primary pathogenic mechanisms for the development of dystrophic changes in the hip joint with dysplasia include the following:

1) disorder of the structure of the hyaline cartilage (the loss of proper orientation of the microfibrils, appearance of the spindled chondrocytes, loss of the surface cell-free membrane, erosion of the cartilage, and disintegration of proteoglycan conjugates),
2) activation of cellular immune mechanisms and cytokines in the joint cavity (synovial macrophages and tissue antibodies) in response to the presence of degraded components of the hyaline cartilage, and
3) destruction of the bone rod system of the subchondral bone (osteolysis) and neoformation of the fibrous tissue instead of osteons [10, 11].

Physical, ultrasonography (US), and traditional radiography examinations are distinguished diagnostic methods for DHJ; recently, computed

tomography (CT) and magnetic resonance imaging (MRI) methods and arthrography are also used [7, 12, 13]. Upon examination by the orthopedic surgeon, in children aged 1–12 months, symptoms associated with a high risk of dysplasia were identified; however, their informational value is not absolute. Among the most frequent criteria, the asymmetry of the gluteal and femoral folds, Galeazzi test (different length of the limbs), limitation or redundancy of movements in the hip joints, and the Ortolani and Barlow maneuvers (negative after the age of 3 years) are emphasized. When a child reaches the age of 3 years, dysplasia symptoms, such as gait disturbance, changes in the amplitude of hip joint movement, and complaints of fatigue and exhaustion by parents and the child while walking, become apparent [7]. US is widely used as a screening method for diagnosing the condition of the bone and cartilage tissue of the joint because it is a safe, cost-effective, and versatile method. However, US of the hip joints does not enable the study of the developmental disorders of the joints in children older than 12 months because the shadow of the developing ossification nucleus closes the structures of the acetabulum [14]. Radiography of the hip joint is a traditional diagnostic method. Using this method, it is impossible to assess the condition of the cartilage component of the joint and to understand the nature of the blood supply; however, this method enables us to see the changes in the subchondral bone typical of the late stages of the disease [13].

The use of CT is limited; however, this method is highly informative and accurate for diagnosing changes in the bone tissue. The restriction is associated with a large radiation load on the child and the inability to provide detailed information on the state of the cartilage and the vascular bed. MRI provides important information regarding the state of the cartilage, paraarticular tissues, and blood vessels; however, it is associated with labor intensity, difficulty in administering to young children, and the frequent requirement for medication sleep in children <4 years of age [12, 13]. Thus, most of the diagnostic methods in DHJ diagnostics focus on determining the state of the bone components of the joint that worsens their diagnostic value. Currently, the active development of possible early and minimally invasive methods for DHJ diagnostics in pediatric practice is ongoing, particularly for the use of specific biomarkers associated with the state of bone and cartilage tissue [3].

Treatment of DHJ involves the use of both conservative and surgical methods based on the child's age, severity of the pathological process, and surgeon's preferences [15]. Conservative treatment is performed for children aged 0 months–2 years; however, in case of severe disease (pronounced underdevelopment of the acetabulum and high dislocation), surgical treatment is recommended from the age of 6 months [15, 16]. The use of the surgical method in children aged ≥2 years is considered typical [15, 16].

Conservative treatment options include a functional method using the Pavlik harness and abduction splints of various modifications that enable the achievement of good results with unstable forms of DHJ [16, 17]. Functional devices create optimal conditions for repositioning of the head with gradual correction; good elasticity of the capsular and ligament apparatus in children aged <1 year also provides a positive contribution. The average period for which a child uses these devices is 6–12 weeks with mandatory dynamic control 1–2 times every 2 weeks. Simultaneously, physiotherapy sessions that involve strengthening massages for the muscles of the lower extremities are conducted [15].

In the absence of successful results of conservative treatment, various surgical procedures are performed. The previously popular method of manual correction of dysplasia (closed reduction) with fixation using a plaster cast in the biomechanically advantageous position of the extremities ("remodeling" of the hip joint) is currently considered only in the historical aspect [18].

Reconstructive interventions in the joint cavity (pelvic osteotomy) and reconstructive interventions on the femoral component (intertrochanteric osteotomy) are common methods of surgical correction [19]. Open reduction is used in the case of severe pathology (dislocation) for head transposition into the natural cavity, often complemented by cavity reconstruction and in some instances, by hip reconstruction [15, 20].

Among degenerative diseases acquired during the growth period, LCPD is the most common [20]. LCPD is an idiopathic osteonecrosis of the femoral head with subsequent complications, such as deformity and osteoarthritis [20]. LCPD commonly

occurs in children aged 2–12 years, boys are 3–4 times more likely to experience it than girls, and bilateral lesions are noted in 10%–15% of all cases [21, 22]. An indispensable condition for LCPD progression is the development of phase impairments on parts of the microvasculature; persistent ischemia and stasis in the arterioles, leading to the necrosis of the head and hip osteons; deformity of the metaphysical zones; and cystic degeneration of the acetabulum [21, 22]. The incidence of LCPD varies, with an average incidence of 0.4–20.9 per 100,000 population [23].

Etiological factors for the occurrence are also discussed, in particular, mutations (gene defects of the *COL2A1* of 12q13 chromosome) [24]; chronic microtrauma of the hip joint tissues; dysfunction of the vascular endothelial microcirculation of the hip joint [25]; clinically latent coagulopathy, in particular, thrombophilia and mutations of the V factor of Leyding [26, 27]; hormonal disorders of lipolysis; and the predominance of lipogenesis processes with increased leptin concentration [28]. The concept of idiopathic vascular ischemia of the microcirculation of the femoral head with an outcome in the form of a heart attack and necrosis forms the core of the LCPD pathogenesis [29]. The drop in intravascular pressure leads to adhesion of the walls of the microcirculatory vessels, resulting in ischemic stasis, impaired tissue metabolism, hypoxia aggravation, and predominance of the glycolytic process of cellular respiration. In addition, the accumulation of insufficiently oxidized metabolic products (pyruvate, lactate, and ketone bodies) activates metabolic acidosis and cell death [30]. Various classifications of LCPD based on radiological criteria or MRI data have been developed. Waldenstrom founded the diagnostic signs of the disease and described four stages of osteonecrosis, including initial changes, fragmentation, restoration, and residual effects [31]. Subsequently, the modified criteria of Catterall (1971), and Salter–Thompson were created based on the degree of involvement in the pathological process of the femoral head and epiphysis [31]. Thus, according to Catterall, four groups of disorders are distinguished: group I corresponds to the focal necrosis of the anterior medial part of the head, group II corresponds to the lesion of the anterior and central segment, group III represents the partial fragmentation of the epiphysis, and group IV represents complete fragmentation [32]. According to Herring, damage to the femoral head is classified as follows: group A represents focal necrosis of the lateral segment of the head with no loss of shape, group B corresponds to destruction of < 50% of the lateral segment and disorder in the central segment, and group C corresponds to necrosis of > 50% of the head [33].

The clinical picture of the initial LCPD changes in most cases has a latent course. The primary clinical symptoms (lameness, post-load pain, and limb shortening) appear at the stage of massive head injury. Patient history commonly includes episodes of prolonged intermittent pain in the region of the rectus muscle of the thigh or in the region of the knee joint as well as episodes of prolonged physical exertion (sports and/or injuries) [34]. US of the hip joint in combination with Doppler velocimetry that indirectly reveals a disorder of the structure and density of the femoral head, inflammatory changes in the synovial membrane, and microcirculation aspects have become an increasingly common screening method [34]. The methods of LCPD diagnostics include radiography (standard, special setup), which is the so-called "gold standard". This method enables the assessment of the severity of damage to the bone components of the joint, possible risk factors, and therapeutic tactics [35]. However, the method does not enable the diagnosis of the disorders at the stage of initial changes. At the same time, MRI with Burgener assessment facilitates the assessment of early changes in osteonecrosis of the femoral head and enables the determination of the detailed state of the intraarticular structures, epiphysis, and hyaline cartilage [36]. Some authors have proposed the use of hip scintigraphy that offers the advantage of detailed topography of the lateral segment of the femoral head [37]. As a rule, arthrography is considered to be an additional diagnostic method in the operating room that allows the surgeon to adjust the planned surgical procedures [38].

Surgical treatment of LCPD includes several areas based on the stage of the disease and the severity of pathogenetic mechanisms. The so-called decompression surgeries (different in the performance of osteoperforation) are important with a retrospective viewpoint because they allow the elimination of the venous plethora in the femoral head [39], and osteoplastic interventions (auto- and allografting of the defect) make it

possible to preserve and remodel the femoral head and eliminate the focus of necrosis. The exclusion of these surgeries from the primary treatment methods for LCPD is associated with several factors as follows: low efficiency in relation to bone tissue remodeling, inability to restore the congruence of the articular surfaces, lengthy postoperative recovery period, and delayed onset of habilitation (by 15%–20% compared to that with the acetabuloplasty and osteotomy) [40, 41].

The most advanced group of surgeries comprises several types of acetabuloplasty, supplemented, if necessary, with intertrochanteric osteotomy [42–45]. In most cases, orthopedists prefer using pelvic osteotomies according to Salter–Thompson and modified versions of the triple osteotomy of the pelvis, such as Steel, Tonnis, Chiari, rotary acetabular, Ganz, and Bernese osteotomies [43]. This group of surgical interventions offers several advantages, such as the removal of the necrosis source from the load with reliable fixation of bone fragments and restoration of the biomechanically beneficial relationship of the epiphysis. Moreover, there is significant reduction in the postoperative rehabilitation time [42, 43]. Moreover, these surgeries are the treatment of choice for the treatment of severe stages of LCPD [43].

As per the classification by Zucker et al. [4], among the diseases that are predominantly traumatic in nature, SCFE requires special attention. SCFE occupies a leading position in the structure of hip joint diseases among children aged 9–15 years [46]. Currently, majority of traumatologists–orthopedists have adopted the polyetiological nature of the disease, and the main risk factors include biomechanical disorders (increased femoral retroversion and epiphyseal plate skewness with stress syndrome) in the joint itself and obesity [45]. SCFE is also associated with several endocrinopathies, particularly hypothyroidism, hypogonadism, and hypopituitarism [46, 47]. Children with chronic kidney disease who undergo radiation therapy in the pelvic area are also at risk [47]. Thus far, few large-scale national studies have been performed on SCFE [47]. The epidemiological aspects of the disease include ethnicity (predominance in countries of the northern latitudes), male sex, and weather (autumn–winter period) [47, 48]. The average incidence of SCFE is 4.8 per 100,000 cases, boys are 1.7 times more likely to develop it, and the peak incidence is during the age range of 12–13 years [47, 48]. A change in the shape and structure of the epiphyseal cartilage plate, accompanied by varying degrees of its displacement from the physiological position towards the femoral neck, with primary disorders occurring in the intermediate calcification zone is a pathognomonic symptom of SCFE [49, 50]. With respect to disease pathogenesis, a primary role is played by the general biomechanical failure of all hip joint structures, including epiphyseal cartilage instability and tilt syndrome, neck deformity, and primary disorders of the cartilage structure in the growth plate [49, 50]. Subsequently, prominence in the epiphyseal cartilage, retrotorsion of the proximal femur (in rare cases, antetorsion of the proximal femur), damage to the anterior edge of the acetabular labrum, and ring in the joint and femur acetabular impingement syndrome [49] develop.

However, in most countries, SCFE diagnosis is established at late and clinically advanced stages of the disease when significant cartilage lesions and deformity of the epiphyseal cartilage are noted [51]. This is attributable to the absence of specific complaints during the early stages (pain and lameness) and to the presence of excess weight that limits the normal range of motion in the hip joint and modifies gait [51]. Clinical examination reveals a limitation of the amplitude of movements in the joint, such as internal rotation (most often) and flexion; in some cases, lameness or waddling gait are observed [49, 50]. Radiography of the hip joint in standard and special projections serves as the "gold standard" for diagnostics. CT is used in some cases and enables the determination of the degree of displacement of the epiphysis of the femur. MRI provides an opportunity to observe detailed changes in the epiphyseal cartilage plate and in other, mainly cartilaginous, structures [47, 48]. All the modern classifications of SCFE are based on the degree of mechanical stability in the epiphysial cartilage zone [52], that is, the anatomical stage classification that is as follows: the pre-dislocation stage (extension of the epiphyseal cartilage plate and change in its structure without displacement), mild degree (shift of the growth plate to $^1/_3$ and tilt of the head by 30°), medium degree (displacement by $^1/_3$–$^1/_2$ and tilt of the head by 30°–60°), and severe degree (displacement > $^1/_2$ and tilt of the head by over 60°). Several authors have proposed clinical classifications of stability in SCFE [53, 54] based

on the time of symptom onset; in the acute form, the onset of clinical symptoms is within 2 weeks, whereas in the chronic form, clinical symptoms are apparent after 2 weeks, the possibility of full-fledged walking [54].

Surgical treatment is the primary treatment method for SCFE because it enables pain relief, reconstruction of the epiphyseal cartilage zone, and fixation of the femoral neck area to prevent slipping and deformity of the head and possibly impingement syndrome and depends on the stability of the form [54]. In the long term, it delays the development of coxarthrosis. The method of choice for the treatment of a stable form of SCFE with displacement of no more than $^1/_3$–$^1/_2$ is the central epiphysiodesis *in situ*. Some surgeons also prefer it in case of unstable forms of SCFE [55, 56]. At the second stage, osteochondroplasty in the epiphyseal cartilage zone is recommended. However, some surgeons do not prefer epiphysiodesis because it does not allow femoral head remodeling and leads to frequent development of early coxarthrosis during its implementation. In this regard, they prefer active osteotomies by type of the Dunn procedure that enables the reconstruction of the proximal femur (it is possible to use them in unstable forms) [56, 57]. However, an important disadvantage of Dunn-osteotomy is increased risk of avascular necrosis of the femoral head. Several researchers have reported successful results of open repositioning at the SCFE. Osteotomy advocates debate regarding their level of performance, namely intracapsular/extracapsular, intertrochanteric, or subtrochanteric nature [56–58].

Additionally, several orthopedists, such as Loder at al., when analyzing a large cohort of patients with SCFE, reveal the lack of evidence regarding the advantages of one or another method of surgical treatment [58–61]. The use of preventive epiphysiodesis of the contralateral joint in SCFE is also debatable. Secondary coxarthrosis in young and middle age becomes a mandatory outcome of this disease, and the only treatment method is total endoprosthesis replacement of the hip joints [45]. Thus, in the United Kingdom, up to 8% of all annual endoprosthesis replacements of the hip joints in the working-age population were performed for coxarthrosis resulting from SCFE [45].

Thus, the DDHJ group is under close supervision by pediatric orthopedists and traumatologists owing to the significant prevalence of the disorder; severity of clinical manifestations; deterioration of the quality of life; and development of complications, such as arthrosis. Moreover, the lack of common approaches for the use of DDHJ treatment methods is a subject of discussion among surgeons and frequently affects the timing and amount of assistance provided.

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Contribution of the authors

S.A. Rubashkin — collection and processing of materials;

A.V. Sertakova — analysis of the materials received, writing the text;

M.M. Dokhov — collection and processing of materials;

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