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THE INFLAMMATORY-DEGENERATIVE PROCESS OF CONNECTIVE TISSUE IN PATIENTS DEPENDING ON THE DEGREE OF SPINE DEFORMATION

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Resume

A survey of 67 patients with axial spinal deformities was performed. It was revealed that with an increase in the degree of axial deformities of the spine, inflammation, total oxidative activity, serum glycosaminoglycan and total proteolytic activity increase; the concentration of carbohydrate-protein compounds decreases, which indicates the decay of connective tissue. The pathogenetic mechanism of the development of the "vicious circle" of the progression of axial deformities of the spine is presented.

Keywords: oxidative stress, inflammatory and degenerative markers, glycosaminoglycans, proteolytic activity, axial spinal deformities.

ВОСПАЛИТЕЛЬНО-ДЕГЕНЕРАТИВНЫЙ ПРОЦЕСС СОЕДИНИТЕЛЬНОЙ ТКАНИ У БОЛЬНЫХ В ЗАВИСИМОСТИ ОТ СТЕПЕНИ ДЕФОРМАЦИИ ПОЗВОНОЧНИКА

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Резюме

Проведено обследование 67 больных с осевыми деформациями позвоночника. Выявлено, что с увеличением степени осевых деформациях позвоночника увеличиваются показатели воспаления, общая оксидантная активность, уровень сывороточного гликозаминогликана и суммарная протеолитическая активность; уменьшается концентрация углевод-белковых соединений, что свидетельствует о процессах распада соединительной ткани. Представлен патогенетический механизм развития «порочного круга» прогрессирования осевых деформаций позвоночника.

Ключевые слова: *окислительный стресс, воспалительно-дегенеративные маркеры, гликозаминогликаны, протеолитическая активность, осевые деформации позвоночника.*

БЕМОРЛАРДАГИ УМУРТҚА ДЕФОРМАЦИЯЛАРИ ДАРАЖАСИГА НИСБАТАН БИРИКТИРУВЧИ ТЎКИМАСИНИ ЯЛЛИҒЛАНИШ-ДЕГЕНЕРАТИВ КЎРИНИШ ИФОДАЛАНИШИ

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Резюме

Умуртқа ўқи деформациялари бўлган 67 беморда текширув ўтказилди. Беморларда умуртқа деформациялари даражаси ошганда: яллиғланиш кўрсатгичлари, умумий оксидант активлиги, қондаги гликозаминогликан даражаси ва протеолитик активлигини йиғиндисини ошиши; оқсил-углевод моддаларнинг концентрациясини камайиши аниқланди. Бу бириктирувчи тўқима парчаланиш

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процессини исботлади. Мақолада зўрайиб борувчи умуртқа деформацияларини «жадаллаштирувчи доира» ривожланиш патогенетик механизми кўрсатилган.

***Калит сўзлар**: оксидатив стресс, яллиғланиш-дегенератив маркерлар, гликозаминогликанлар, протеолитик активлик, умуртқа ўқи деформациялари.*

Relevance

Progressive with axial spinal deformities (ASD) lead to early disability and social maladaptation of patients and impaired functions of the musculoskeletal, cardio-respiratory, neuromuscular and other body systems. Deformation in the frontal plane takes the first place among diseases of the spine in children and adolescents, their frequency among population exceeds 15%, including gross scoliosis with severe cosmetic and functional disorders that make up 1.5-2%. The social significance of scoliotic disease is determined by a decrease in the quality of life of patients with severe forms. The progression of II and III degrees of scoliosis in children during the period of incomplete growth is of particular difficulty [1, 5, 6, 9, 10].

It is known that during the manifestation of osteoarthritis, inflammation occurs. Damage to the cartilage and other tissues of the joints is facilitated by weakening of the ligaments with an increase in stability, a decrease in muscle strength, a slowdown in peripheral nerve conduction, an improper distribution of pressure on the cartilage, and changes in the subchondral bone [11]. The essence of inflammation is the body's reaction to local damage, which is aimed at isolating and eliminating the damaging factor, and then tissue regeneration [2, 3].

The aim of the research was to study the indicators of connective tissue inflammation, oxidative stress, matrix proteins, glycosaminoglycan levels and total proteolytic activity, depending on the severity of spinal deformity.

Material and methods

The study involved 67 children aged 11-15 (average age 12.5 ± 1.1) years with clinically established (by measuring the Cobb angle) and radiologically confirmed ASD of I-IV degree according to the classification of V.D.Chaklin [8, 10]. Children with III-IV degree of deformations, the diagnosis of idiopathic scoliosis has been established. To analyze the results of all examined, they were divided into groups depending on the degree of spinal deformity: I degree – 19 children, II degree – 14, III-IV degree – 34. The control group (14 children) was formed from adolescents without severe skeletal deformity. Total oxidative activity (TOA) and total antioxidant activity (TAA) were calculated by the colorimetric method A.P.Golikova (1997), the level of carbohydrate-protein compounds in blood serum was studied using the method of V.G.Kolba and V.S.Kamyshnikov (1982), the content of glycosaminoglycans – according to the method of V.S. Kamyshnikov (2000). The total proteolytic activity of blood serum was determined by the method of K.N.Veremeenko and O.P.Goloborodko (1988) [4]. This clinical trial was approved by the Regional Independent Ethics Committee under the Ministry of Health of the Republic of Uzbekistan (RIEC meeting dated November 6, 2018, protocol No. 11). Mathematical processing of the results was undertaken using the integrated package for statistical analysis "Statistica 6,0".

Research results and discussion

According to many authors, the degenerative process in the vertebral-motor segment with ASD is of a polyetiological nature, and their occurrence and development is associated with primary and secondary circulatory disorders, resulting in progression of spinal deformity, which leads to a change in the shape of the chest and pelvis, and also desynchronization of anabolic and catabolic processes.

In the pathogenesis of degenerative changes in the spinal-motor segment during spinal deformities, the mechanical load on the cartilage in the joints of the spine causes regional vascular reactions and inflammation. The latter is confirmed by indicators of sedimentation rate of erythrocytes (SRE) and C-reactive protein (CRP) (Table 1), the concentration of which in the blood serum is increased and is directly dependent on the severity of the disease.

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Table 1. ***Indicators of inflammatory and degenerative markers in patients depending on the severity of spinal deformity***

Indicators	I degree ASD n=19	II degree ASD n=14	III-IV degree ASD n=34	Control n=14
SRE, mm/h	11,54±1,04*	14,78±1,52*	20,34±2,04*	6,89±0,53
CRP ng/ml	14,23±1,34*	26,13±2,41*	36,48±3,52*	8,79±0,68
Total oxidative activity (TOA) %	11,78±1,26	16,54±1,33*	18,47±1,21*	9,72±0,87
Total antioxidant activity (TAA) %	128,54±10,05	109,27±8,91	92,76±7,43*	116,12±8,19
TOA/TAA	0,092	0,151	0,199	0,083
The concentration of carbohydrate-protein compounds (g/l)	0,72±0,06	0,61±0,05*	0,54±0,04*	0,97±0,06
Serum Glycosaminoglycan Level (g/l)	0,06±0,01	0,083±0,01*	0,109±0,02*	0,023±0,01
Total proteolytic activity mmol / hour /l.	1,52±0,14	1,71±0,16*	2,44±0,18*	1,24±0,11

*Note: * - significance of differences P<0.05*

As can be seen from the research results in the table, the intensity of lipid peroxidation processes in the presence of ASD has a peculiar character. In the first degree of disease severity in patients, a significant increase in the content of lipoperoxidation products in plasma was revealed. At the same time, TOA indices increased by 21% compared to healthy individuals. Along with this, there was an increase in total antioxidant activity (TAA), compared with baseline data. In the second severity degree of the disease, activation of lipid peroxidation processes led to a decrease in the antioxidant defense of the body. So, with an increase in TAA by 1.7 times, a decrease in TAA activity by 6% was noted. With the III degree of ASD, a significant average 2 times increase in the activity of TOA was observed, with a decrease in the activity of TAA by 20%.

When studying the ratio of TOA / TAA, it was revealed that this indicator tends to increase with an increase in the degree of spinal deformation (Table 1).

The study of blood serum samples for the content of carbohydrate-protein compounds showed that their concentration in patients with ASD is lower than under physiological conditions (Table 1). Moreover, the more severe the degree of the disease, the lower the level of carbohydrate-protein compounds of blood serum when compared with the control group.

Due to the fact that almost all blood plasma proteins (with the exception of albumin and lipoproteins) are carbohydrate-protein compounds that provide regulatory, homeostatic, adaptive and reparative functions (that is, they are biologically active compounds), a detected decrease in their concentration during ASD can explain the decrease in the processes of their synthesis due to depletion of the adaptive capabilities of the body in the chronic course of the disease.

When examining blood serum glycosaminoglycans during degeneration of articular cartilage in patients with ASD, it was found that their content was statistically significantly increased in patients with I degree of ASD. In patients with II and III degree of ASD, the studied parameter was higher than the initial values by 3.6 and 4.7 times, respectively. The obtained research results are probably associated with increased catabolic processes in the articular cartilage of the spine. High values of serum glycosaminoglycans in patients with ASD are probably associated with accelerated hydrolysis of proteoglycans in the affected articular cartilage of the spine and the release of degradation products into the bloodstream.

An analysis of the results of the studies presented in the table showed an increase in the activity of proteolytic enzymes in patients with ASD. So, at the first degree of ASD in the examined children, an increase in proteolysis by 23% was observed when compared with healthy ones. Subsequently, an increase in the severity of the disease was accompanied by activation of the proteolysis process by an average of 38% and 98%, respectively.

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Increased pressure on cartilage changes the architectonics of the matrix. Models of mechanically induced osteoarthrosis show an increase in the joint concentration of inflammatory cytokines and mediators. Chondrocytes respond to mechanical stress by activating synthetic activity or inflammatory cytokines (they are also synthesized by other tissues of the joints) [12]. Mechanical stress, cartilage degradation products stimulate signaling pathways (kinase cascades) that activating interleukin (AI) -1, tumor necrosis factor (TNF) -α. Proinflammatory cytokines produced and delivered into the synovial fluid enhance catabolic processes in articular cartilage. Inflammatory changes in the synovial vagina cause damage to the tendons and ligaments. Inflammation can spread to neighboring muscles as well. Chondrocytes synthesize AI-1, which induces matrix cleavage. Then secondary mediators are activated – derivatives of prostaglandins, TNF-α. They cause the release of lysing enzymes from chondrocytes. Under the influence of mechanical force, the production of AI-1, -6, -8, -17, -18 through chondrocytes increases, and the catabolic activity of these cells increases. Under the influence of AI-1, synthesis of proteolytic enzymes (aggrecanases, matrix metalloproteinases), degradation of collagen and cartilage prostaglandin are enhanced. Catabolic processes in the tissues of joints initiated by inflammation are superimposed on already existing involutive articular changes and further exacerbate them [2, 4, 7, 13].

In general, the pathological process can be represented as a chain: a deformed spine has a mechanical damaging effect on the cartilage of the vertebra and facet joints → damage to the cartilage → induction of inflammation of the synovia and other tissues → increases the level of serum glycosaminoglycan → increases the overall oxidative activity along with a decrease in the overall antioxidant activity→ the total proteolytic activity increases → the concentration of carbohydrate-protein compounds decreases (connective tissue decomposition processes) → all this helps to weaken the ligaments, impair stability and decrease muscle strength → the degree of deformation of the spinal column increases → it has a mechanical damaging effect on the cartilage of the vertebra and facet joints = formed "vicious circle". Thus, as a result of the study, the features of inflammatory, oxidative, carbohydrate-protein metabolism, the level of serum glycosaminoglycan and total proteolytic activity during degenerative-dystrophic changes in the articular cartilages in patients with ASD were revealed, which made it possible to clarify some aspects of the pathogenetic process in them.

Conclusion

1. With an increase in the degree of axial deformations of the spine, inflammation, total oxidative activity, serum glycosaminoglycan and total proteolytic activity increase; the concentration of carbohydrate-protein compounds decreases, which indicates the decay of connective tissue.

2. The pathogenetic mechanism of the development of the "vicious circle" of the progression of axial deformities of the spine is described.

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ФАКТОРЫ РИСКА ФОРМИРОВАНИЯ ПЕРИНАТАЛЬНЫХ ПОРАЖЕНИЙ У НОВОРОЖДЕННЫХ

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Резюме

Цель исследования: изучить особенности клинических показателей и безусловных рефлексов у маловесных новорожденных, оценить уровень содержания микроэлементов у новорожденных детей, при осложнении периода беременности анемией.

Материалы и методы: обследованы 20 доношенные новорожденные, родившиеся с малым весом и 35 новорожденные с нормальным весом и их матери.

Результаты: Неудовлетворительные условия развития плода у матерей с анемией проявляется рождением их с малым весом, поражением нервной системы, снижением функциональных характеристик организма новорожденных во время родов и раннем неонатальном периоде.

Ключевые слова: *новорожденные, микроэлементы, рождение с малым весом.*

ЧАҚАЛОҚЛАРДА ПЕРИНАТАЛ ЖАРОХАТЛАРНИ ШАКЛЛАНТИРУВЧИ ҲАВФЛИ ОМИЛЛАР

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Резюме

Тадқиқот мақсади - камвазнлик билан туғилган чақалоқларда клиник белгиларни ва шартсиз рефлексларни хусусиятларини ўрганиш, ҳамда оналарда ҳомиладорлик анемия билан асоратланганда,

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