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Comparative Characteristics of the Results of Drug Therapy of Synovitis in Gonatrosis

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Relevance. Epidemiological studies conducted in recent years indicate that gonarthrosis (GA) continues to be one of the most common diseases, the progression of which worsens the quality of life of patients and causes disability. Gonarthrosis is known to develop when the balance between anabolic and catabolic processes in the articular cartilage is disturbed. Proteoglycans, degradation products of chondrocytes, and collagen, altered during degenerative processes, can become autoantigens and become involved in the immunopathological process that promotes the development of synovitis. Reactive inflammation of the synovial membrane, which occurs with activation of metalloproteinases and overexpression of proinflammatory cytokines against the background of a deficiency of anti-inflammatory cytokines, exacerbates dystrophic and destructive processes in cartilage tissue. Thus, synovitis, which is secondary in nature, contributes to the progression of osteoarthritis (OA), and with a recurrent torpid course, it can determine a violation of joint function. It has been established that the only energy substrate for chondrocytes is glucose. It has been proven that in patients with diabetes mellitus (DM), synthetic processes in cartilage tissue are disrupted, and chronic hyperglycemia increases damage to muscles and periarticular tissues in OA. In GA, uncompensated chronic hyperglycemia with a glycated hemoglobin content of 10.5% or more contributes to the development of synovitis with intense arthralgia, pronounced exudation, proliferation of the synovial membrane and edema of the collateral ligaments. Synovitis also activates receptors for interleukin-1 (IL-1), fibronectin, and leptin. Despite significant progress in understanding the pathogenetic mechanisms of synovitis development, the importance of metabolic factors for the clinical manifestations, maintenance and severity of the inflammatory response of the knee joint synovium in a combination of initial and compensated disorders of carbohydrate metabolism and obesity, which can affect the composition and effectiveness of treatment, has not yet been fully studied. The main directions of treatment of patients with GA are "pain reduction, correction of joint functional insufficiency, limitation of disease progression and improvement of the quality of life of patients." Today, as many centuries ago, most patients view the effectiveness of treatment through the lens of pain relief. Therefore, during the period of synovitis, NSAIDs are widely used to reduce pain and inflammation in the joints. These drugs remain a key element in the treatment of pain syndrome. However, due to the unsatisfactory safety profile, experts recommend prescribing these drugs in minimally short courses, but before pain relief, and in minimally effective dosages. At the same time, the appointment of chondroitin sulfate and glucosamine has received unconditional recognition from specialists. It has been shown that these drugs have an analgesic effect by suppressing the activity of lysosomal enzymes and inhibiting superoxide radicals, which makes it possible to significantly reduce doses. "In addition, the stabilizing effect of drugs on the width of the articular gap and metabolic processes in the subchondral bone and cartilage were revealed. At the same time, the authors of modern national and international guidelines on treatment with OA indicates the need for long-term medication, and adherence to long-term outpatient therapy with chondroprotectors in non-working people of older age groups significantly reduces the price of drugs. Osteoarthritis of the knee joint requires patient education, lifestyle changes, physical exercises, weight loss that reduces the load on the joints, and the use of non-pharmacological treatment methods. Laser therapy, according to the results of a meta-analysis conducted on the evidence base of the effectiveness of treatment of knee OA, was assessed as effective, with an RCT quality of 100% and an evidence level of 1a. This is above balneotherapy, SPA/sauna, electrical stimulation, ultrasound, radiotherapy, thermotherapy, massage. Laser therapy

improves blood microcirculation by reducing platelet and erythrocyte aggregation, normalizes arterial inflow, venous and lymphatic outflow of the lower extremities, has anti-inflammatory, analgesic and immunocorrective effects, improves the course of metabolic processes in the body. However, the beneficial effect of laser radiation is manifested only under certain irradiation conditions. The strict dose dependence of the effects requires the development and implementation in medical practice of a dose-standardized and laser-energy-minimized laser treatment technique. Thus, a clinical study of scientific and practical importance is important, aimed at clarifying the significance of carbohydrate metabolism disorders in the nature of the inflammatory reaction of the synovial membrane of the knee joint in obese patients, and evaluating the effectiveness of complex therapy using a non—medicinal method, low-dose laser therapy, on the course of synovitis. The severity of arthralgia in synovitis in women with abdominal obesity and early disorders of carbohydrate metabolism has been established and its dependence on the presence of fasting hyperglycemia. The preservation of rehabilitation potential during synovitis with gonatrosis of stages I—III in patients without metabolic disorders and its decrease in obese women and impaired carbohydrate metabolism were revealed. The low effectiveness of drug therapy of synovitis in stage I—III gonarthrosis in obese patients with impaired carbohydrate metabolism has been shown, and its predictor has been identified: chronic hyperglycemia with a glycated hemoglobin level of 5.7% or higher. The positive effect of personalized laser therapy included in the complex treatment of synovitis in patients with stage I—III gonatrosis on the severity of the inflammatory process in the joint has been proven. An algorithm is proposed to identify a risk group for low—efficacy drug therapy of synovitis among patients with stage I-III gonarthrosis. A differentiated approach to the treatment of synovitis in patients with stage I—III gonarthrosis has been developed, including the use of low-dose laser therapy in combination with pharmacotherapy. The clinical and functional characteristics of synovitis in gonarthrosis, which are significant for the differentiated appointment of low-dose laser therapy, are highlighted. The expediency of using indicators of carbohydrate metabolism, waist circumference, body mass index, and rehabilitation index for this is shown.

The advantages and safety of personalized laser therapy over unified laser therapy used in combination with pharmacotherapy for synovitis in patients with osteoarthritis of the knee joints are shown. The features of the course of synovitis in stage I—III gonarthrosis in patients with obesity and impaired carbohydrate metabolism are a high intensity of arthralgia at rest, associated with fasting glycemia levels, a large thickness of the synovial membrane, a high frequency of formation of a significant volume of effusion in the knee joint, a decrease in the ability to perform daily activities and the rehabilitation potential of the body against the background of emotional disorders, and There is also a lower effectiveness of pharmacotherapy due to chronic hyperglycemia. The inclusion of low-dose laser therapy in the complex therapy of synovitis, which takes into account the clinical and functional features of the inflammatory process in the joint, the presence of obesity, carbohydrate metabolism disorders and the level of rehabilitation potential of the patient, contributes to more effective relief of inflammation in the joint in stage I—III gonarthrosis, reduces the dose of nonsteroidal anti-inflammatory drugs and improves the quality of rehabilitation of patients. Cartilage consists of cells (chondrocytes, chondroblasts) and an intercellular substance (matrix) [32, 54, 211]. Chondrocytes provide synthesis and degradation of cartilage matrix components. The main components of the cartilage matrix are collagen macromolecules and proteoglycans (PG). PG are proteins combined with hyaluronic acid (GK), chondroitin sulfate (HT) and keratan sulfate (KS). This structure has high hydrophobicity, low viscosity and is an ideal molecule capable of resisting stress on the joint. In OA, the synthesis of proteoglycans by chondrocytes is disrupted, and catabolic processes in cartilage begin to prevail over anabolic ones. The cartilage matrix loses CT, CS, GC, and the PG molecule decreases. Small GHGs can absorb water, but they are not able to hold it firmly. Collagen binds excess water, although it swells and loosens. Cartilage resistance is decreasing. Thus, proteoglycan deficiency becomes the main cause of cartilage degeneration and the loss of its unique adaptive properties. There are many factors contributing to the development of OA. Among them: old age, overweight, hereditary predisposition, metabolic and circulatory disorders, endocrine imbalance, congenital and acquired diseases, joint injuries. In general, the discrepancy between the mechanical load on the cartilage

surface and the ability to withstand this load is the main reason that provokes the development of OA. However, the question of the effect of hyperglycemia on the development of secondary inflammation in the joint cavity and diabetic peripheral polyneuropathy on the course of gonatrosis remained open. In 2004, K. D. Brand and T. Yamasaki hypothesized that diabetic peripheral polyneuropathy of the lower extremities may have an effect on damage to the musculoskeletal system. Based on the above, it can be assumed that the question of the effect of glycemia, as well as its dynamics on the course of knee joint OA, has been little studied. In addition, the problem of the effect of neuropathy progression on the prognosis of OA is not covered at all in the literature. As suggested by A. Morandas (1979), cartilage undergoes excessive mechanical stress, leading to rupture of the collagen network in its surface layer, deep cracks form in the cartilage, through which proteoglycans are leached out. Thus deprived of its components, the cartilage loses its integrity, strength, and pathological changes develop in it. However, the link between obesity and the development of OA is not limited to mechanical overload of the joints. There are three types of mechanical receptors on the surface of HC: alpha-5 beta-1 integrans, channels activated by stretching, and SD 44. Pressure and stretching activate integrans and channels that trigger signaling pathways and further stimulate the release of secondary mediators (calcium, adenosine monophosphate, inositol).

The result is the production of a number of substances, including pro-inflammatory cytokines. It should also be noted that adipose tissue is a source of adipokines, which have pleiotropic effects and have an effect on the formation of bone tissue and blood vessels. One of the adipokines, leptin, is involved in maintaining HC homeostasis. Thus, OA is not a "mechanical disease", but a systemic pathology associated with metabolic disorders in obesity. The risk factors for developing OA include defects of the musculoskeletal system: scoliosis, kyphosis, flat feet, genu valgum, genu varum, and varicose veins. With them, the probability of developing OA increases by 7 times. Inflammation plays an essential role in the pathogenesis of OA. Inflammation is mainly caused by macrophage-type synoviocytes that synthesize proteases and proinflammatory cytokines. When anti-inflammatory cytokines are deficient from the synovial membrane through the synovial fluid, interleukins IL-1, IL-3, tumor necrosis factor a (TNF-a), IL-6, IL-17, and leukemic inhibitory factor diffuse into the articular cartilage, where they activate HC for the production of inflammatory mediators. Observation of animals with OA has shown that IL-1 blockade is an effective tool capable of preventing the destruction of articular cartilage, and TNF-a blockade provides a slight reduction in inflammation in the articular tissues. Many researchers believe that chronic inflammation of the synovial membrane of the joint in osteoarthritis, associated with the activation of matrix metalloproteinases and overexpression of proinflammatory cytokines, contributes to the intensification of dystrophic and destructive changes in articular cartilage. The condition of the subchondral bone is important in the pathogenesis of OA. Simultaneously with the destruction of articular cartilage, the pathological process also affects the underlying bone tissue. It is suggested that the thickening of the subchondral plate significantly contributes to the development of OA. Subchondral sclerosis increases the stiffness of bone tissue and thus contributes to the destruction of articular cartilage. The question of whether the changes in the subchondral bone are primary or secondary is still open. Microcrystals may play a role in the pathogenesis of OA. They are found in the synovial fluid of 30-60% of patients with OA. There is an assumption that the secondary deposition of crystals contributes to the acceleration of cartilage degeneration (the theory of the "amplification loop"). It is also possible that synoviocytes first phagocytize crystals, after which they secrete proteolytic enzymes or secrete cytokines. In stage I—III gonarthrosis in patients without metabolic disorders, the course of the inflammatory process in the joint is characterized by severe arthralgia only during movement and moderate pain syndrome at rest, a slight decrease in the ability to perform normal daily activities, minimal thickening of the synovial membrane and a slight or moderate volume of effusion in the joint; the recurrent course of synovitis is observed at against the background of subclinical depression and high rehabilitation potential.

The course of synovitis in stage I—III gonarthrosis in patients with abdominal obesity and early disorders of carbohydrate metabolism, compared with people without metabolic disorders, is characterized by an increase in the severity of arthralgia at rest, a greater thickness of the synovial

membrane, moderate or significant volume of effusion in the joint; synovitis develops against the background of a decrease in rehabilitation potential. body conditions, subclinical depression, as well as subclinical anxiety. conclusions In stage I—III gonarthrosis in patients with mixed obesity and type II diabetes mellitus, compared with patients without metabolic disorders, women with abdominal obesity and early disorders of carbohydrate metabolism, the synovitis clinic is characterized by the most severe arthralgias at rest and during movement, maximum thickening of the synovial membrane, a high frequency of formation of a significant volume of effusion in the knee joint damage, up to Baker's cyst, marked decrease in the ability to perform daily activities; The torpid course of the inflammatory process is observed against the background of increasing emotional disorders and low rehabilitation potential.

LIST OF LITERATURE

1. Агеева В.В., Красильникова Е.И., Зубина И.М., Шляхто Е.В. Взаимосвязь инсулинорезистентиости и нарушений липидного обмена у больных с ожирением. Тер. архив 2002; 10: 12-15.
2. Байкова О.А., Люсов В.А., Евсиков Е.М. и др. Характер изменения уровня половых гормонов у женщин, больных артериальной гипертензией, сожирением в разные периоды генеративной активности. Кардиология 2004; 3: 52-57.
3. Волкова Э.Р., Салихов И.Г., Тухбатуллин М.Г. Клинико-ультразвуковая характеристика поражения периартикулярных тканей коленного сустава при остеоартрозе. Науч.-практ. ревматол. 2003; 2: 21.
4. Голованова Е.Д., Осипова Т.В., Белоусова Ю.А., Завадкин А.В. Распространенность и особенности течения остеоартроза у гериатричесикх больных с артериальной гипертензией. Науч.-практ. ревматол. 2004; 2: 100.
5. Дворяшина И.В., Рогозина И.А., Коробицын А.А. Особенности метаболического синдрома при впервые выявленном нарушении толерантности к глюкозе на фоне ишемической болезни сердца. Проблемы эндокринологии 2001; 1: 3-7.
6. Ивлева А.Я., Полуянова Н.В., Клюева Т.В. и др. Повышение эффективности антигипертензивной фармакотерапии при уменьшении массы тела у молодых пациентов с алиментарно-конституциональным (первичным) ожирением. Артериальная гипертензия 2003; 2: 42-46.
7. Кайсаров Г.А., Багирова В.В. Дегенеративно-дистрофические заболевания опорно-двигательного аппарата у лиц, работающих на металлургическом комбинате. Тер. архив 2004; 2: 57-63.
8. Лаврухина А.А., Красивина И.Г., Долгова Л.Н. и др. Метаболический синдром и периартикулярное поражение при гонартрозе. В кн.: Актуальные вопросы клинической эндокринологии. Ярославль. 2004; 181-185.
9. Мамедов М.Н. Артериальная гипертония в рамках метаболическогосиндрома: особенности течения и принципы медикаментозной коррекции. Кардиология 2004; 4: 95-100.
10. Назарова О.А. Клинико-патогенетические подходы к диагностике и коррекции нарушений микроциркуляции при некоторых ревматических заболеваниях. Автореф. .дисс. докт. Мед. наук. Ярославль. 2000; 60.
11. Оганов Р.Г., Масленникова Г.Я. Вклад сердечно-сосудистых заболеваний в здоровье населения России. Сердце 2003; 2: 58-61.
12. Позин А.А. Значение периферического кровообращения в формировании структурных и функциональных нарушений коленных и тазобедренных суставов у больных ревматоидным артритом и остеоартрозом. Автореф. .дисс. докт. мед. наук. Ярославль; 2000:43.

13. Романенко И.А., Шамолина Е.А. Состояние микроциркуляции у пациентов с метаболическим синдромом. Тезисы Конгресса ассоциации кардиологов стран СНГ, Санкт-Петербург, приложение к научно-практическому журналу «Кардиология СНГ». 2003; 1: 243.
14. Савельева JT.B. Современный взгляд на лечение ожирения. Качество жизни. Медицина. Сахарный диабет 2003; 54-57.
15. Хадипаш Л.А., Перова Н.В., Мамедов М.Н. Кластеры компонентов метаболического синдрома у больных сахарным диабетом типа 2. Проблемы эндокринологии 2001; 4: 30-32.
16. Aliabadi P., Nikpoor N., Alparslan L. Imaging of neuropathic arthropathy. Semin. Musculoskelet. Radiol. 2003; 7(3): 217-225.
17. Bailor D., Poehman E. Exircise-training enhanses fat-free mass pre-serva-tion during diet-induced weight loss: a meta-analytical finding. Int. J. Obes. Relat. Metab. Disord. 1994; 18: 35-40.
18. Campbell M., Mathys M. Pharmacologic optinons for the treatment of obesity. Am. J. health Syst. Pharm. 2001; 14: 1301-1308.
19. Doucet E., Umbeault P., Almeras N. et al. Phusical activity and lowfat diet: is it enough to maintain weight stability in the reduced-obease individual following weight loss by drug therapy and energy restruction? Obes. Res. 1999; 7(4): 323-333.
20. Hardie L., Rayner D., Holmes S. et al. Circulating leptin levels are modulated by fasting, cold exposure and insulin administration in lean but not Zucker (fa/fa) rats as measured by ELISA. Biochem. Biophys. Res. Commun. 1996; 223(3): 660-665.
21. Kopf D., Muhlen I., Kroning G.et al. Insulin sensitivity and sodium excretion in normotensive offspring and hypertensive patients. Metabolism 2001; 50(8): 929-935.
22. Schlundt D., Hill J., Pope-Cordle J. et al. Randomized evaluation of low fat ad libitum carbohydrate diet for weight reduction. Int. J. Obes. 1993; 17: 623198
23. Spector T.D., Cicuttini F., Baker J. et al. Genetic influences on osteoar-tritis in women: a twin study. BMJ 1996; 312: 940-944.