CLINICAL AND THERAPEUTIC ASPECTS OF CERVICAL OSTEOCHONDROSIS: PATHOGENESIS, SYNDROMOLOGY, AND MODERN TREATMENT APPROACHES

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Abstract: Degenerative-dystrophic diseases of the spine represent one of the most prevalent pathologies in modern medicine. Among them, cervical osteochondrosis occupies a leading position due to its wide range of neurological and vascular complications. This article presents a comprehensive analysis of the clinical syndromes associated with cervical osteochondrosis, including radicular, reflex, vascular, spinal, and cerebral variants. The pathogenesis of neurological manifestations involves both direct degenerative changes in spinal motion segments and secondary reflex disorders resulting from irritation of nervous and autonomic structures. The main conservative treatment approaches—immobilization, pharmacotherapy (NSAIDs, COX-2 inhibitors, muscle relaxants, vasoactive agents), and physiotherapy—are discussed in detail, with emphasis on complex rehabilitation strategies. Indications for surgical intervention are outlined for severe cases of vertebrobasilar insufficiency and spinal cord compression. The study underscores the importance of timely diagnosis, pathogenetically oriented therapy, and an integrated approach to the management of cervical osteochondrosis.

Keywords: cervical osteochondrosis, radicular syndrome, vertebral artery syndrome, reflex disorders, COX-2 inhibitors, physiotherapy, rehabilitation, surgical treatment.

КЛИНИКО-ТЕРАПЕВТИЧЕСКИЕ АСПЕКТЫ ШЕЙНОГО ОСТЕОХОНДРОЗА: ПАТОГЕНЕЗ, СИНДРОМОЛОГИЯ И СОВРЕМЕННЫЕ ПОДХОДЫ К ЛЕЧЕНИЮ

Аннотация: Дегенеративно-дистрофические заболевания позвоночника являются одной из наиболее распространённых патологий в современной медицине. Среди них особое место занимает шейный остеохондроз, характеризующийся множеством неврологических и сосудистых осложнений. В статье представлен комплексный анализ клинических синдромов шейного остеохондроза, включая корешковые, рефлекторные, сосудистые, спинальные и церебральные формы. Патогенез неврологических проявлений обусловлен как прямыми дегенеративными изменениями позвоночного двигательного сегмента, так и вторичными рефлекторными нарушениями вследствие раздражения нервных и вегетативных структур. Подробно рассмотрены основные направления консервативной терапии — иммобилизация, медикаментозное лечение (НПВС, ингибиторы ЦОГ-2, миорелаксанты, вазоактивные препараты) и физиотерапия, а также показания к хирургическому вмешательству при тяжёлых формах компрессии спинного мозга и вертебробазилярной недостаточности. Отмечена важность своевременной диагностики и комплексного подхода к лечению шейного остеохондроза.

Ключевые слова: шейный остеохондроз, корешковый синдром, синдром позвоночной артерии, рефлекторные расстройства, ингибиторы ЦОГ-2, физиотерапия, реабилитация, хирургическое лечение.

SERVIKAL OSTEOXONDROZNING KLINIK VA TERAPEVTIK JIHATLARI: PATOGENEZI, SINDROMOLOGIYASI VA ZAMONAVIY DAVOLASH USULLARI

Annotatsiya: Umurtqa pog‘onasining degenerativ-distrofik kasalliklari zamonaviy tibbiyotda eng ko‘p uchraydigan patologiyalar sirasiga kiradi. Ular orasida bo‘yin (servikal) osteoxondroz o‘zining keng nevrologik va qon tomir asoratlari bilan ajralib turadi. Ushbu maqolada servikal osteoxondrozning klinik sindromlari — radikulyar, reflektor, qon tomir, orqa miya va miya shakllari — batafsil tahlil qilingan. Nevrologik belgilar patogenezi umurtqa harakat segmentlaridagi to‘g‘ridan-to‘g‘ri degenerativ o‘zgarishlar va nerv hamda vegetativ tuzilmalar ta’sirlanishi natijasidagi ikkilamchi reflektor buzilishlar bilan bog‘liq. Konservativ davolashning asosiy yo‘nalishlari — immobilizatsiya, farmakoterapiya (YQA vositalari, COX-2 ingibitorlari, miorelaksantlar, vazoaktiv preparatlar) va fizioterapiya — keng yoritilgan. Og‘ir hollarda orqa miya siqilishi yoki vertebrobazilyar yetishmovchilik kuzatilganda jarrohlik aralashuvi ko‘rsatmalari ko‘rib chiqilgan. Maqolada erta tashxis, patogenetik yo‘naltirilgan davolash va kompleks yondashuvning ahamiyati ta’kidlanadi.

Kalit so‘zlar: bo‘yin osteoxondrozi, radikulyar sindrom, umurtqa arteriyasi sindromi, reflektor buzilishlar, COX-2 ingibitorlari, fizioterapiya, reabilitatsiya, jarrohlik davolash.

INTRODUCTION

Degenerative-dystrophic lesions of the spine are among the most common human diseases. They begin to develop in adolescence and reach their peak during the most active working period – from 35 to 60 years. Cervical pain syndromes are the second most common clinical manifestation of spinal osteochondrosis after lumbar pain, accounting for up to 35%. These diseases cause suffering to patients and society, and the economic losses associated with them are incalculable [1, 2, 3]. According to the classification of diseases of the peripheral nervous system, a number of main clinical syndromes of cervical osteochondrosis are distinguished: radicular, reflex (muscular-tonic, neurodystrophic, autonomic-vascular, pain), spinal, and cerebral.

In the genesis of neurological manifestations of cervical osteochondrosis, direct pathological changes in the spinal segment (primary pathogenetic factor) are significant, as are irritation of structures innervated by the spinal nerve and involvement of segmental autonomic structures in the pathological process, leading to reflex muscular-tonic, neurodystrophic, and vascular disorders (secondary pathogenetic factor) [1, 4].

MAIN PART

Developing spondyloarthrosis and arthrosis of the uncovertebral joints are largely responsible for microtraumatization of the roots at the cervical level; osteophytes also play a role. The corresponding bone growths narrow the intervertebral foramen; therefore, at the cervical level, the roots are often compressed not due to a disc herniation in the epidural space, but in the intervertebral foramen itself. Cervical disc herniations may occur as a result of hyperextension macrotrauma, occupational microtrauma, or unsuccessful manual therapy at the cervical level. Trauma to the nerve roots may also occur as a result of spinal segment instability. Trauma to a nerve root and its sheath results in swelling of the compressed root and reactive aseptic inflammatory reactions. The seventh cervical root is most commonly affected (70%), while the sixth cervical root is less commonly involved—up to 20% of cases. The remaining 10% are affected by lesions of the fifth and eighth cervical roots. Less frequent lesions of the third and fourth cervical roots are [3, 4, 5].

The main symptoms of radicular lesions are observed in the zone of their innervation: pain and paresthesia, sensory impairment, decreased muscle strength, and hyporeflexia. As a rule, pain intensifies with movement in the cervical spine, especially when tilting the head towards the affected root [3].

Reflex muscle-tonic manifestations of osteochondrosis of the cervical spine are caused by the fact that long-term pathological impulses originate from the receptors of the affected tissues of the spinal motor segment and enter the spinal cord, where they are switched to its anterior and lateral horns,and then travel to the striated muscles, causing their reflex tension. Reflex manifestations of cervical osteochondrosis manifest themselves in the following clinical forms. Cervicalgia, characterized by paroxysmal sharp and constant dull pain deep in the neck, occurring in the morning after sleep, accompanied by tension in the neck muscles and limited range of motion in the cervical spine. Anterior scalene syndrome occurs with irritation of the C5-C7 nerve roots. Pain is localized on the lateral surface of the neck and radiates to the arm along the ulnar surface of the forearm and hand. Sometimes, especially in the morning, paresthesia in the fingers and forearm occurs. Weakness in the hand and atrophy of the thenar and hypothenar muscles may develop.

The anterior scalene muscle is firm and tender upon palpation. Scapulohumeral periarthritis – muscular-tonic and neurodystrophic changes affect the tissues surrounding the shoulder joint. The clinical picture is characterized by quite intense pain in the shoulder joint, which can radiate to the neck, shoulder, and scapula. The pain is accompanied by limited passive and active shoulder motion. Muscle spasms and painful tightness are observed in virtually all muscles that move the shoulder joint. Shoulder-hand syndrome is characterized by scapulohumeral periarthritis combined with edema and other autonomic-dystrophic changes in the hand and wrist, while the elbow joint remains intact. Pectoralis minor syndrome is caused by compression of the neurovascular bundle between the muscle and the humeral head. The bundle is especially often compressed during severe arm abduction, such as during anesthesia or immobilization due to a humeral fracture. Pain and paresthesia are felt in the anterior chest, scapula, and often the arm. Movement disorders in the arm and sensory disturbances in the 4th-5th fingers are possible. The muscle is dense and painful to the touch [3, 4, 6].

Vertebral artery syndrome, or posterior cervical sympathetic syndrome, is caused primarily by the effect of pathological bone and cartilaginous structures on the vertebral artery and its sympathetic plexus. If the lumen of the vessel decreases, vascular compression syndrome occurs. The reflex components of vertebral artery syndrome are based on vasomotor reactions not only to irritation of sympathetic efferents (the sympathetic efferent fiber is the smooth muscle of the vascular wall), but also to afferent irritation (of the sympathetic fibers of the spinal nerve). Two stages can be distinguished in the manifestations of the syndrome: dystonic or functional and organic, with organic stenosis of the artery. If stenosis is not compensated by collateral blood flow, circulatory disorders in the vertebrobasilar system occur.

Vasodystonic disorders manifest as: 1) cranialgia and sensory disturbances in the facial area,which is characterized by typical paroxysmal pulsating, burning pain, which usually spreads from the back of the head to the temple, parietal region and often the superciliary region, the so-called symptom of removing a helmet. Headache is usually unilateral. Blood pressure is often asymmetrical, changing during periods of increased pain; 2) cochleovestibular disorders on the side of the vestibular nerve lesion - noise, crackling, buzzing in the ears, dizziness; 3) visual disturbances - pain in the eyes, fog before the eyes, sensation of sand in the eyes [3, 4, 7].

The basis of vascular spinal syndrome is chronic vascular cervical myelopathy, developing as a result of chronic trauma to the spinal cord vessels by osteophytes or a herniated disc, compression of the radiculomedullary arteries and the anterior spinal artery. Myelopathy is manifested by movement disorders, mainly in the form of spastic lower paraparesis. Flaccid upper paraparesis develops gradually. Conduction sensory disturbances are mild and manifest as hypoesthesia, the border of which is located 5-6 segments below the level of the lesion. Pelvic organ function is often impaired [1, 4, 8, 9, 10, 11].

In the pathogenesis of cerebral vascular syndrome, both direct compression of the vertebral arteries with limited blood flow in the vertebrobasilar system and irritation of the sympathetic plexus of the vertebral artery with subsequent vasoconstriction play a role. The clinical picture is represented by transient or persistent disturbances in the vertebrobasilar system (visual impairment syndromes, cerebellar, bulbar, pseudobulbar and other brainstem syndromes) [1, 4].

Before determining the therapeutic tactics, it is necessary to decide what type of treatment will be undertaken: conservative or neurosurgical. Currently, priority is given to conservative treatment methods. Conservative therapy during the acute phase of radicular syndrome requires immobilization of the cervical spine (wearing a soft Schantz collar) and sleeping with a bolster under the head of the bed. In the acute stage of radicular syndrome, a pain-relieving cocktail is prescribed (analgin, vitamin B12, or no-shpa solution). In severe cases of radicular syndrome, especially at onset, dehydrating agents are prescribed to reduce nerve root swelling: furosemide, hypothiazide, or diacarb orally for 3-5 days. For intense pain, intravenous mannitol solution is recommended (at a rate of 1 g/kg of body weight). Also, in the acute phase, novocaine (paravertebral) blocks are performed at points C2-C8, as well as blocks of the most tense and painful muscles. For the blockade, 0.5%, 2% solution of novocaine or cocktails with the addition of hydrocortisone, voltaren can be used [4, 5, 10]. Applications with a 33% solution of dimexide to the cervical area for 30-60 minutes or applications of dimexide with the addition of non-steroidal anti-inflammatory drugs, analgesics are effective,nicotinic acid [4, 5]. Local anesthetics and analgesics are used - Dolgit cream, menovazine, etc. Non-steroidal anti-inflammatory drugs (NSAIDs) in the acute period are recommended to be prescribed as injections. For these purposes, the following drugs are used: Voltaren, Xefocam, Melbek and other drugs in appropriate dosages. Recently, specific cyclooxygenase 2 (COX-2) inhibitors have been widely used. COX-2 is induced in response to the inflammatory process. This leads to the synthesis and accumulation of inflammatory prostanoids, in particular, prostaglandin E2, which causes inflammation, swelling and pain. The anti-inflammatory effect of COX-2 inhibitors is achieved by blocking the production of inflammatory prostanoids. In most cases, COX-2 inhibitors are as effective as standard NSAIDs for both acute and chronic pain. Also, COX-2 inhibitors are less likely to cause severe gastrointestinal side effects [12].

One of the specific COX-2 inhibitors is the drug "Celcox" (celecoxib). At therapeutic concentrations in humans, "Celcox" does not inhibit cyclooxygenase-1 (COX-1), therefore, it does not affect the prostanoids synthesized due to the activation of COX-1, and therefore does not interfere with the normal physiological processes associated with COX-1 in tissues, especially in the stomach, intestines, and platelets. Criteria for determining specificity in relation to COX-2 include the absence of side effects caused by the action of COX-1, in particular ulcerative lesions of the stomach and duodenum, gastrointestinal complications (bleeding, perforation) and the absence of inhibition of platelet aggregation activity. Celcox has no effect on platelet aggregation and bleeding time when used in therapeutic doses and doses six times higher than therapeutic ones

[12, 13]. The use of muscle relaxants, such as sirdalud or mydocalm, is justified. Sirdalud is prescribed throughout the acute period. Mydocalm is used initially as intramuscular or intravenous injections, then in tablet form [5, 14, 15, 16].

It is recommended to prescribe vascular drugs that improve microcirculation - trental, cavinton, stugeron and promote venous outflow - euphyllin, escusan [4]. Physiotherapeutic methods that can be used include diadynamic currents, sinusoidal modulated currents (especially for the vegetative component of pain syndrome), electrophoresis of novocaine solutions, mydocalm, and magnetic therapy [4, 15, 16].

Acupressure and acupuncture play an important role in the treatment of back pain. Massage, therapeutic exercise, and physiotherapy also play a significant role. In the subacute period, therapy is continued with non-steroidal anti-inflammatory drugs and vasoactive drugs, to which vitamin therapy is added: B vitamins, milgamma, neurorubin [5, 17].

In the chronic stage of pain syndrome, it is advisable to prescribe antidepressants to relieve the psychovegetative component of long-term pain syndrome. For radicular hernia, anticholinesterase drugs are used: proserin, nivalin, neuromedin [18].

Physiotherapy, massage, and acupuncture are also prescribed. Of the physiotherapeutic methods, diadynamic currents, electrophoresis with novocaine, UHF, and in the subacute and chronic periods - hydrocortisone phonophoresis, ultrasound, and a pulsating magnetic field are recommended. Physiotherapy is also effective. For reflex muscle-tonic manifestations (pectoralis minor syndrome, scalene muscle syndrome) in the acute period, hydrocortisone blockades of the anterior scalene muscle and the tendon of the pectoralis minor muscle are used. The prescription of muscle relaxants is also advisable. In acute cases of shoulder-hand and scapulohumeral periarthritis syndromes, rest with temporary immobilization of the arm in a physiological position is prescribed. Local applications of dimethyl sulfoxide with novocaine and hydrocortisone are used, while physiotherapy includes initially applying a pulsating magnetic field, followed by phonophoresis with anesthesin. Injections of hydrocortisone and novocaine into the periarticular tissues of the shoulder joint are effective. Electrophoresis with euphyllin is effective in the subacute stage with pronounced vascular and autonomic components. In the chronic stage, radon baths, segmental mud applications to the cervical-collar zone and arm (at a temperature no higher than 35-37°C), therapeutic exercise, and massage are prescribed [4].

Conservative therapy for vascular, cerebral, and spinal syndromes of cervical osteochondrosis includes vasoactive, metabolic, noorotropic drugs, muscle relaxants, and antispasmodics. Acupuncture and electrophoresis with euphyllin are prescribed [4, 10].

Indications for surgical treatment of cervical spine lesions are as follows: in cerebral syndrome—severe recurrent vertebrobasilar insufficiency; in vascular myelopathy—progressive development of the process, signs of spinal cord compression, the presence of a subarachnoid space block; operations include laminectomy, removal of osteophytes, immobilization of vertebrae in case of instability, etc. [1, 4].

In the acute stage of scapulohumeral periarthritis, rest is prescribed with temporary immobilization of the arm in a physiological position. Local applications of dimethyl sulfoxide with novocaine and hydrocortisone are used, and physiotherapy includes initially applying a pulsating magnetic field, followed by phonophoresis with anesthesin. Injections of hydrocortisone and novocaine into the periarticular tissues of the shoulder joint are effective. With pronounced vascular and vegetative components in the subacute stage, electrophoresis with euphyllin is effective. In the chronic stage, radon baths, segmental mud applications to the cervical-collar zone

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CONCLUSION

Cervical osteochondrosis remains one of the most significant and socially relevant problems in modern neurology and vertebrology. The complex interplay of degenerative, vascular, and reflex mechanisms underlies the diversity of its clinical manifestations, from radicular pain to vertebrobasilar insufficiency and myelopathy. The disease not only leads to severe neurological and autonomic disorders but also substantially reduces the quality of life and work capacity of patients during their most productive years.

Comprehensive treatment must be individualized and pathogenetically grounded, combining immobilization, pharmacotherapy, physiotherapy, and rehabilitation. The use of modern nonsteroidal anti-inflammatory drugs, particularly COX-2 inhibitors, as well as muscle relaxants and vasoactive agents, allows effective control of pain and inflammation while minimizing side effects. Physiotherapy, massage, acupuncture, and therapeutic exercise remain essential components of recovery and long-term stabilization.

Surgical intervention is indicated only in cases of persistent compression of the spinal cord or vertebrobasilar insufficiency resistant to conservative measures. Early diagnosis, timely initiation of therapy, and adherence to a multidisciplinary approach are key to preventing chronic pain syndromes, disability, and recurrent exacerbations. The integration of modern pharmacological and rehabilitative methods ensures a more favorable prognosis and improved functional outcomes in patients with cervical osteochondrosis.

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