OPEN ACCESS
World Journal of Applied Medical Sciences
ISSN: 3049-0200 (Online) | Vol. 3, No. 1, 2026
Website: https://wasrpublication.com/index.php/wjams/

INFLUENCE OF SPINE DEGENERATION IN THE CERVICAL DEPARTMENT ON THE NEUROLOGICAL STATUS IN PERSONS WHO HAVE SURVIVED COVID-19

Melnyk Yu.V.[1], Andreeva T.O.[2], Kalashnikov V.Y.[3*], Stoyanov O.M.[4], Stoianov A.O.[5], Kugel Ya.I.[6], Nanish I.I.[7], Pastukhov O.O.[7]

[1]Neurologist, Director of the medical center Neuro-Cardio Lab, Odessa; Ukraine

[2]Ph.D, Associate Professor Department of Therapeutic Disciplines; Petro Mohyla Black Sea National University; Mikolaiv; Ukraine

[3]Doctor of Medical Sciences, Professor of the Department of Ultrasound and Functional Diagnostics; Kharkiv National Medical University; Ukraine

[4]Doctor of Medical Sciences, Professor of the Department of Neurology and Neurosurgery; Odessa National Medical University; Ukraine

[5]Orthopedic-traumatologist; KNP «City Clinical Hospital N11, Odessa; Ukraine

[6]Intern, neurologist, Department of Neurology and Neurosurgery; Odessa National Medical University; Ukraine

[7]V-year student; Odessa National Medical University; Ukraine.

Received: 28/09/2025 | Accepted: 10/12/2025 | Published: 15/01/2026

Abstract: The COVID-19 pandemic has highlighted the problem of long-term consequences of the disease, in particular on the nervous system. More and more clinical observations indicate that patients who have had COVID-19 have persistent or progressive neurological disorders, especially in the presence of degenerative changes in the spine. Myalgias and arthralgias are characteristic manifestations of COVID-19 and are observed in 50–90% of patients in the acute period. During the convalescence period, prerequisites are formed for the chronicity of symptoms with the involvement of the autonomic nervous system, which is associated with degenerative-inflammatory changes in the musculoskeletal system, immune disorders and organic damage to all parts of the nervous system. Decompensation of existing vertebrogenic pathology is of particular importance.

The **aim** of this study is to assess the impact of cervical spine degeneration on the neurological status of people who have had COVID-19.

The study included 65 patients with upper body pain syndrome that arose, persisted, or worsened after the acute period of COVID-19; the control group consisted of 14 practically healthy individuals. Pain intensity was assessed using the visual analog scale (VAS). All patients underwent computed tomography of the cervical spine with determination of the Pavlov–Torg index and morphometric-densometric assessment of vertebral body density. scan.

Pain syndrome was the leading complaint mainly in the form of cervicalgia, often combined with upper back pain and irradiation to the upper extremities. Headache, vestibular and autonomic disorders had reflex-vascular mechanisms of implementation. Signs of acquired cervical stenosing pathology with a maximum in the C6 segment were found in 75.4% of patients, accompanied by pronounced neurological symptoms.

Conclusions. The transferred COVID-19 acts as a trigger for decompensation of degenerative changes in the cervical spine with the formation of persistent pain and neurological syndromes. This necessitates a multi-level assessment of the patient's condition, individualized treatment and rehabilitation tactics, and dynamic neurological monitoring.

Keywords: Long COVID, pain syndromes, cervical spine degeneration, neurological disorders.

Introduction

The virus has been shown to have widespread effects on the central (CNS) and peripheral nervous systems (PNS), both in the acute phase and in the long term after clinical recovery [1,2].

Some patients continue to experience persistent or recurrent neurological symptoms, indicating prolonged or delayed damage to neural regulation (Long COVID). Pain syndrome, myalgia, neuropathic pain, arthralgia and spinal pain, sensorimotor disorders, and autonomic dysfunction, including after 6 months, are particularly common [3].

These manifestations are likely related to neuroinflammation, damage to nociceptive pathways, impaired microcirculation, and immune changes. They are not explained by residual viral load. COVID-19 often acts as a trigger for exacerbations or involves the osteochondral and musculoskeletal systems [4,5]. Thus, COVID-

***Corresponding Author**
Valeriy Yosypovych Kalashnikov*

19 should be considered not only as an acute respiratory disease, but as a factor in systemic and long-term damage to the nervous system, capable of initiating or aggravating chronic neurological and neuromuscular disorders [6].

The development of peripheral nerve and CNS pathology is also associated with systemic vasculitis, thromboembolic complications at the level of small and large vessels, imbalance of the renin-angiotensin-aldosterone system and systemic accumulation of cytokines. Clinically, this has manifestations of axonal demyelination, focal neuronal necrosis, functional sensory, neuromuscular and CNS disorders [7-9].

Myalgias and arthralgias are characteristic manifestations of viral infection. These symptoms occur in 50-90% of patients in the acute period of COVID-19, while their frequency and severity correlate with the severity of the disease [10].

It is known that for the chronicity of a number of symptoms of the disease with the obligatory involvement of the autonomic nervous system (ANS) with the possibility of developing peripheral and central sensitization, psychoemotional problems, as well as visceral pathology [11-13].

There is data on the clinic of "prolonged COVID", which is noted in approximately 10%-26.5% of patients [8]. Egyptian scientists I. Galal et al. [9] indicate that myalgias were 60%, arthralgias - 56%.

After severe ("hospital") COVID-19, 1-2 months after discharge, musculoskeletal pain was noted in 30% - 50%, and in 15% of patients with a milder course of the disease [16,17].

In meta-analyses conducted on the main symptoms of ACS [18], the frequency of arthralgia was on average 19% (from 7% to 34%), chest pain – 16% (from 10% to 22%), myalgia – (from 5% to 43%) of convalescents.

In addition, it is necessary to take into account the exacerbation of existing chronic musculoskeletal neuropathic pain syndromes [19] with the presence of polyneuropathies, radiculopathies, spinal cord injury. During this period of convalescence, an increase in algic phenomena was noted.

COVID-19 can stimulate the development/exacerbation of fibromyalgia, chronic fatigue syndrome (benign myalgic encephalomyelitis), as well as psychoemotional, trophic, vegetative-vascular, sympathalgic accompaniment. The above additionally contributes to the rapid chronicity of the pathological process [20,21].

It should be noted that, despite the high immunogenicity of the SARS-CoV-2 virus and the significant frequency of joint pain, patients rarely developed true reactive arthritis [22], which shifts the responsibility onto the shoulders of neurologists. Therefore, we focused on musculoskeletal pain, especially against the background of degenerative-dystrophic lesions of the cervical spine (CDS) and the characteristic autonomic dysfunctions [23-25].

The above highlights the need for further research to develop effective diagnostic approaches and treatment strategies in the long-term follow-up of patients after COVID-19.

The aim of this study is to assess the impact of cervical spine degeneration on neurological status in individuals who have had COVID-19.

Materials and research methods. The study included 65 patients with pain syndrome in the upper body, which was preserved, caused, or increased after an acute period of infection with the SARS-CoV-2 virus. In this case, damage to the segmental branches of the nervous system, conductivity disorders, and DD processes in the CDS were recorded.

Criteria for excluding patients from further investigation: the presence of comorbid pathology, such as development, dysplasia and dysraphism of the spinal cords, interspine joints, cystic changes in the bodies of the spine, oncopathology, diagnosis of osteoporosis and systemic diseases of the tissue.

Number of men - 25 (38.5%), women - 40 (61.5%). The second period of patients: men (38.5%) from 29 to 65 years, women (61.5%) – from 20 to 65 years. The average age of patients was 41.5 ± 5.4 years. The control group consisted of 14 apparently healthy people of the positive age group.

Pain syndrome began during the acute period of COVID-19 and persisted in the future in 31 (47.6%) patients, and arose after the acute period in 11 (16.9%) patients, or significantly intensified (zagostrennya) in 23 (35.6%) patients. All patients underwent computed tomography (CT) using the Pavlov-Torg index [26-30] and the morphometric-densometric thickness of the C5-C7 vertebral body CDS degeneration [28].
The intensity of the pain syndrome was recorded using an additional 10-point visual analogue scale (VAS) [31,32].
Statistical analysis was carried out using nonparametric methods. The Wilcoxon T-test, the Krushkal-Wallis H-test (pairs) and Friedman's Z-statistics, as well as the Kolmogorov-Smirnov and Pearson's $\chi 2$ tests were used. [33,34].

Analysis and discussion of results

Degenerative changes in CDS were most often detected in the form of deformable spondyloarthrosis - 78.5%, spinal canal stenosis - 75.4%, swelling of the intervertebral openings - 72.3%, late-onset hypertrophy and yellow ligaments – 6% deformable spondylolisthesis.

Everything is listed and the presence of protrusion of the disc dorsally or dorsolaterally (100.0%), cystic swelling of the edges of the ridges and intervertebral angles corresponds to stenotic changes in the spinal canal and intervertebral joints opening with the development of persistent pain syndrome and neurological deficit (Table 1). In the control, single cob DD changes in CDS were recorded. For all the simple parameters of organic changes in the CDS, there are small significant differences in the control group, which is small compared to the zero index according to the Bonferroni multiple t-test.

Table 1. Organic changes in degenerative-dystrophic process of the cervical spine

Surveyed	Hernias/protrusions	Spondylo-arthrosis deformans	Spondylo-listhesis deformas	Spinal stenosis	Narrowing of the interver-tebral foramina	Hypertrophy of the longitudinal and yellow ligament
Examine patients n=65	36/29** 55.4/44.6	51*** 78.5	30** 46.2	49*** 75.4	47*** 72.3	42** 64.6
Control n=14	1/0 7.1/0	1 7.1/0	1 7.1/0	0 0	1 7.1/0	2 14.3

Notes: the numerator indicates absolute numbers, the denominator indicates relative numbers

Determination of the Pavlov-Torg index by CT morphometry in the main and control groups revealed significant differences between the average values of Sag of the C6 vertebral body (decrease by 1.3 mm compared to the control, $p<0.05$); diameter (Sag) of the spinal canal at the C6 level – by 4.2 mm, $p<0.05$). The most pronounced were the differences in the number of stenoses according to the Pavlov-Torg index – more than 10 times ($p<0.01$). Thus, DD changes in the CDS have a direct impact on changes in the size of the vertebra, the diameter of the spinal canal and ultimately, on the development of cervicogenic canal stenosis (Table 2).

Table 2. Average morphometric measurements in humans (M±m)

Surveyed	Average (Sag) size of the vertebral body C6	Diameter (Sag) of the spinal canal at the level C6	Amount of stenosis to Pavlov-Torg index (<0,8)
Examine patients n=65	16.1±0.6 *	10.0±0.7*	49 (75.4%) **
Control n=14	17.4±0.5	14.2±0.8	1 (7.1%)

Notes: * – $p<0.05$, ** – $p<0.01$ – significant differences in the studied indicators compared to those in practically healthy patients (Kruskal-Wallis test)

Naturally, such stenotic changes in the spinal canal are formed with the active intervention of a number of factors in the form of disc protrusion, bone growths, ligament hypertrophy, deforming changes in the joints and displacements of the vertebrae along the axis of the spine. All these organic pathological processes were diagnosed in patients of the main group, and their combination was characteristic of each examined.

In the control group, the factors that form spinal canal stenosis were either registered in significantly smaller quantities (hernias and protrusions 4.8 times, $p<0.05$; spondyloarthrosis - 11 times, $p<0.01$; spinal canal stenosis - 10 times, $p<0.01$; narrowing of the intervertebral foramina - 10 times, $p<0.01$), or completely absent (spondylolisthesis and hypertrophy of the spinal ligaments).

The above-listed DD changes in the CDS can lead to the development of persistent chronic pain syndrome. The latter, according to the pathophysiological classification, is neuropathic, that is, caused by structural changes in the nervous tissue itself at various levels.

Pain syndrome was the main complaint in all patients. Cervical pain was exclusively registered in 23 patients (35.4%), pain in the upper back - in 32 (49.2%), irradiation to the upper extremities was characteristic in 32 (49.2%). The intensity of pain according to VAS ranged from 1 to 5 points, the average score was 3.1±0.4 points. Headache (76.9%), mainly combined with neck pain and was practically absent in the control group of patients (14.3%, $p<0.05$), that is, it is possible that DD triggers the mechanisms of headache realization.

Dizziness (58.5%), which was one of the leading clinical syndromes with DD changes in the CDS in the main group. In the control - 7.1% ($p<0.01$). They were of a non-systemic nature, more typical of manifestations of autonomic dysfunctions. The realization of dizziness, as in the case of cephalgia, was associated with the mechanisms of pathological functioning of the structures of the CSF.

During the neurological examination of the subjects of the main group, the following were recorded: weakness in one (21.5%) or two upper limbs (52.3%), i.e. 48 patients (73.8%). In addition, the following were recorded: muscle atrophy of varying severity (21.5%), gait changes (18.5%), weakness in one or both legs (20.0%), fascicular twitching (3.1%). Compared with the control group, in the DD pathology of the CDS, weakness in one arm was observed 3 times more often ($p<0.01$), myalgic manifestations also occurred 2 times more often ($p<0.01$), weakness in the legs - 1.9 times more often ($p<0.05$). Cases of bilateral weakness in the hands, numbness, muscle atrophy, gait changes, and fasciculations were completely absent in the control group (Table 3).

Table 3. Neurological symptoms in degenerative-dystrophic process of the cervical spine

Surveyed	Steno-sis	Weakness in the hands		Numb-ness	Myal-gia	Muscle atrophy	Chan-ge of move	Weak-ness in the legs	Fasci-culations
		One	Two						
Examine patients n=65	57*** 87.7	14** 21.5	34** 52.3	30** 46.1	15** 23.1	14** 21.5	12** 18.5	13* 20.0	2* 3.1
Control n=14	0 0	1 7.1	0 0	0 0	1 7.1	0 0	0 0	1 7.1	0 0

Примітки: у чисельнику позначають абсолютні числа, у знаменнику – відносні;

* – p<0.05, ** – p<0.01, *** – p<0.001 – достовірні відмінності досліджуванихпоказників порівняно з такими у практично здорових пацієнтів (тест Крускала-Уолліса)

Thus, the results obtained indicate the reliable presence of neurological symptoms in the DD process in the CDS.

In the main group, motor deficiency in the form of paresis, mainly of the distal parts of the hands, was registered in 48 patients (73.8%), pyramidal symptoms in the legs - in 32 (49.2%) patients, dysfunction of the pelvic reservoirs (7.0–10.8%), as well as sensory disorders (24.0–36.9%). Regarding the control group, only mild sensory changes were noted in one observation (p<0.01), the rest of the above-listed symptoms characteristic of the clinic of cervical myelopathy were completely absent. Thus, according to the data obtained, myelopathy is one of the leading slowly progressive manifestations of the clinical picture of the degenerative-dystrophic process of the cervical spine. (Table 4).

Table 4. Pyramidal and segmental disorders in stenotic changes of the cervical spine

Surveyed	Paresis of the upper extremities	Pyramidal symptoms	Pelvic tank disorders	Sensory disorders
Examine patients n=65	48*** 73.8	32** 49.2	7* 10.8	24** 36.9
Control n=14	0 0	0 0	0 0	1 7.1

Notes: the numerator indicates absolute numbers, the denominator indicates relative numbers;
* – p<0.05, ** – p<0.01, *** – p<0.001 – significant differences in the studied indicators compared to those in practically healthy patients (Kruskal-Wallis test)

Both local and segmental symptoms, pelvic reservoir dysfunctions and sensory disorders were diagnosed. Symptoms of pyramidal insufficiency were recorded in patients of the main group.

According to CT, degenerative changes in the spinal cord were maximal at the level of the C6 vertebra with the maximum clinical correlation (neurological deficit, pain syndrome, etc.). Morphometric analysis of this level according to the Pavlov-Torg index was < 0.8.

Our studies confirmed the assumption that spinal canal stenosis can be confirmed by manifestations of neurological symptoms in patients. Clinical manifestations in the form of paresis in the hands come to the fore, which can be interpreted as a leading syndrome of myelopathy in spinal canal stenosis in humans (73.8%). Motor deficit was less pronounced in the lower extremities (20.0%), statolocomotor disorders and other symptoms characteristic of secondary cervical myelopathy were also recorded in the main group.

The presence of a large number of joints, muscles, major vessels, spinal cord, nerve conductors and roots, which are located in the cervical region, cause dysfunction of almost all body systems, a variety of pathological conditions due to bone and cartilage deformations in the cervical spine [35-38]. First of all, these are marginal osteophytes of the vertebral bodies, degenerative-inflammatory processes in the intervertebral joints, hypertrophic manifestations in the posterior longitudinal and yellow ligaments, congenital and acquired stenosis of the spinal canal.

It is known that the anatomical structures that form the cervical vertebrae are regularly subjected to various combinations of loads. In addition, several articular joints and places of attachment of ligaments and tendons on each vertebra lead to variations in the type and magnitude of loads on different anatomical areas within each vertebra. Due to the natural curvature of the cervical spine, at each of its levels, the load on the vertebrae is likely to be different.

Our studies revealed the preservation of physiological lordosis in only 15.4% of all examined patients in the post-covid period and with pain syndrome with neurovegetative accompaniment. Pathological deviations of the spinal configuration (straightening of lordosis - 43.1% and angular kyphosis - 41.5%)

were 84.6% (p<0.05), see Table 5. These indicators significantly and reliably differed from the control group (by 2.4 times).

Pathological deviations of the spinal configuration in the group with DD pathology of the cervical spine (84.6%) mainly manifested themselves in the form of angular kyphosis and straightened lordosis. These organic changes were recorded with the same frequency (41.5% and 43.1%, respectively). The clinical course had the characteristic clinical symptoms described above.

Table 5. Changes in spine configuration in the main and control groups

Surveyed	Number of people surveyed (n)	forms of lordosis SHVH		
		Angular kyphosis	Lordosis straightened	Norm
Examine patients	65	27 (41,5%)*	28 (43,1%)*	10 (15,4%)
Control	14	3 (21,4%)	2 (14,3%)	9 (64,3%)*

*Вірогідність: між групами людей p<0,05

In the control group, pathological changes were recorded significantly less often (35.7%), i.e. 2.4 times in comparison with the main group (p<0.05). In contrast to the latter group, angular kyphosis prevailed 1.5 times more often than straightened lordosis.

Since at the C3 level in the people we examined with the DD process, there were no changes in the vertebral body and intervertebral disc, we accepted these data as a relative norm for this patient. In addition, the HU units of CT of the spinal cord provide reliable information regardless of the measurement plane, age or gender, and the degree of degeneration [39].

Comparing the average density of the C3 vertebral body with the density of the cranial vertebral body at the level of the lesion and the average density of the caudal vertebral body at the level of the lesion (in most cases, the C6 body, the level of the C5-C7 bodies), we obtained a pattern in which the average density of the cranial vertebrae increases, and the distal vertebrae decreases, which is likely due to the characteristic application of force and mechanical energy. Similar patterns were observed in both patients with DD of the CVD and relatively healthy people in the control group. However, the average density of the C3 vertebra practically did not differ in the examined groups, which indicates the reference data of this level regardless of the factors and features of functioning both in pathology and in relatively normal conditions. At the same time, the average density of the cranial vertebrae in the control group is 8.6% higher and the caudal vertebrae are 7.8% higher than in the group with DD changes in the CDS. Thus conditions are created for possible damage to the skeletal system of the spine.

The average bone density of the C3 vertebra and at the level of the DD process correlates with the literature data on the study ± SD comparing the indicators of healthy and patients with radiculopathy, which is likely a consequence of dystrophic processes in the spine, which are stimulated by the ANS as a result of COVID-19.

Adequate and effective is the proposed measurement of bone density in certain anatomical locations in the SVC, which is performed almost exclusively on the central body of the vertebra, either by measuring trabecular bone density in a single CT slice [40-42], or by measuring a large volume of trabecular bone in several CT slices [43-44]. Such a technique can be used to assess the risk of spinal fracture, measure bone loss, including age-related, and monitor the dynamics of osteoporosis and other metabolic bone diseases. Determination of the bone density of the spinal cord can be used for further treatment, surgical fixation of the vertebrae, and its better quality.

Conclusions.

1. Pain syndrome was the main complaint in all examined patients in the post-covid period. Pain syndrome prevailed in the neck against the background of detected dystrophic changes in the cervical spine.

2. Pain syndrome occurred in the acute period of SARS-CoV-2 infection and persisted in 31 (47.6%) patients, occurred after the acute period in 11 (16.9%) patients, or significantly increased (exacerbation) in 23 (35.6%) patients. Intensity according to the VAS scale 3.1±0.4 points.

3. Cervical pain prevailed in combination with pain in the upper back (49.2%), and irradiation to the upper extremities (49.2%). Myalgia also occurred 2 times more often than in the control group.

4. Headache (76.9%), mainly combined with neck pain in the presence of vertebrogenic changes, which trigger a number of nociceptive, neuroautonom mechanisms that support sensitization and realization of pain phenomena.

5. Vestibular disorders (58.5%) - the second most frequent syndrome, which also indicates the presence of autonom dysfunctions in the post-covid period. They mainly had vegetative-vascular and reflex mechanisms of symptomatology. Vertigo syndrome was non-systemic in nature, more typical for manifestations of disorders of ANS regulation, without involvement of the peripheral department of the vestibular analyzer

6. In patients who had undergone SARS-CoV-2 infection, compared with controls, paresis was detected in the hands (73.8%, p<0.001), in the legs (20.0%, p<0.05), and gait changes (18.5%, p<0.01); increased tendon reflexes (49.2%, p<0.01); numbness (46.1%, p<0.01); myalgia (23.1%, p<0.01); muscle atrophy (21.5%, p<0.01. They are the leading clinical manifestations of damage to all parts of the nervous system as a result of a combination of spinal pathology and other nearby structures.

7. Acquired cervical stenosis in patients who have had COVID-19, according to the Pavlov-Torg index, is reliable for manifestations of DD neck damage (75.4%) with a maximum at the C6 level and bright clinical and neurological support.

8. The detected pathological curvature of the physiological lordosis in patients who have had COVID-19 additionally increases the likelihood of stenosis and compression of the spinal cord against the background of DD processes in the cervical spine. Such deviations amounted to 84.6%. In the control group, they were observed 2.4 times less often ($p<0.05$) with a predominance of angular kyphosis without clinical symptoms.

9. In patients after COVID-19, often observed: pain syndromes mainly in the neck and upper half of the trunk with a pronounced autonom component, impaired muscle tone, asthenic syndrome and decreased activity of the cervical spine, which contributes to the disruption of the formation of the correct configuration of the spine, increasing the risk. Given the above, a multi-level assessment and careful rehabilitation strategy aimed at preventing spinal cord compression are required. Dynamic supervision with neurological examination is necessary. Treatment tactics should be formed individually by a neurologist, neurosurgeon.

References

1. Wood H. Exploring the long-term neurological consequences of COVID-19. Nat Rev Neurol. 2025; 21:65. doi:10.1038/s41582-025-01056-z

2. Ellul MA, Benjamin L, Singh B, et al. Neurological associations of COVID-19. Lancet Neurol. 2020; 19(9):767-783. doi:10.1016/S1474-4422(20)30221-0

3. Taquet M, Geddes JR, Husain M, Luciano S, Harrison PJ. 6-month neurological and psychiatric outcomes in 236 379 survivors of COVID-19: a retrospective cohort study. Lancet Psychiatry. 2021; 8(5):416-427. doi:10.1016/S2215-0366(21)00084-5

4. Nalbandian A, Sehgal K, Gupta A, et al. Post-acute COVID-19 syndrome. Nat Med. 2021; 27(4):601-615. doi:10.1038/s41591-021-01283-z

5. Ceban F, Ling S, Lui LMW, et al. Fatigue and cognitive impairment in post-COVID-19 syndrome: a systematic review and meta-analysis. Brain Behav Immun. 2022; 101:93-135. doi:10.1016/j.bbi.2021.12.020

6. Mina Yu. et al. Deep phenotyping of neurological post-covid complications caused by SARS-CoV-2 infection. Neurology-neuroimmunology-neuroinflammation. May 5, 2023. DOI: 10.1212/NXI.0000000000200097

7. Lahiri D, Ardila A. COVID-19 pandemic: A neurological perspective. Cureus. 2020; 29;12(4):e7889. doi: 10.7759/cureus.7889;

8. Javed A. Neurological associations of SARS-CoV-2 infection: A systematic review. CNS Neurol Disord Drug Targets. 2021; 16. doi: 10.2174/1871527320666210216121211;

9. Bandeira IP, Schlindwein MAM, Breis LC, Peron JPS, Gonçalves MVM. Neurological complications of the COVID-19 pandemic: What have we got so far? Adv Exp Med Biol. 2021; 1321:21-31. doi: 10.1007/978-3-030-59261-5_2

10. Weng LM, Su X, Wang XQ. Pain symptoms in patients with coronavirus disease (COVID-19): A literature review. J Pain Res. 2021; 26;14:147-159. doi: 10.2147/JPR.S269206

11. Walitt B, Bartrum E. A clinical primer for the expected and potential post-COVID-19 syndromes. Pain Rep. 2021; 16;6(1):e887. doi: 10.1097/PR9.0000000000000887

12. Oronsky B, Larson C, Hammond TC, Oronsky A, Kesari S, Lybeck M, et al. A review of persistent post-COVID syndrome (PPCS). Clin Rev Allergy Immunol. 2021; 20:1-9. doi: 10.1007/s12016-021-08848-3

13. Marinangeli F, Giarratano A, Petrini F. Chronic pain and COVID-19: Pathophysiological, clinical and organizational issues. Minerva Anestesiol. 2020. doi: 10.23736/S0375-9393.20.15029-6

14. Kemp H, Corner E, Colvin L. Chronic pain after COVID-19: Implications for rehabilitation. Br J Anaesth. 2020; 125(4):436-440. doi: 10.1016/j.bja.2020.05.021

15. Galal I, Mohamed Hussein A, Amin M, Saad MM, Zayan HEE, Abdelsayed MZ, et al. Determinants of persistent post-COVID-19 symptoms: Value of a novel COVID-19 symptom score. The Egyptian Journal of Bronchology. 2021; 15:10. doi: 10.1186/s43168-020-00049-4

16. Halpin S, McIvor C, Whyatt G, Adams A, Harvey O, McLean L, et al. Postdischarge symptoms and rehabilitation needs in survivors of COVID-19 infection: A cross-sectional evaluation. J Med Virol. 2021; 93(2):1013-1022. doi: 10.1002/jmv.26368

17. Jacobs L, Paleoudis E, Bari D, Nyirenda T, Friedman T, Gupta A, et al. Persistence of symptoms and quality of life at 35 days after hospitalization for COVID-19 infection. PLoS One. 2020;11;15(12):e0243882. doi: 10.1371/journal.pone.0243882

18. Lopez-Leon S, Wegman-Ostrosky T, Perelman C, Sepulveda R, Rebolledo P, Cuapio A, et al. More than 50 long-term effects of COVID-19: A systematic review and meta-analysis. Res Sq. 2021:rs.3.rs-266574. doi: 10.21203/rs.3.rs-266574/v1

19. Attal N, Martinez V, Bouhassira D. Potential for increased prevalence of neuropathic pain after the COVID-19 pandemic. Pain Rep. 2021; 6(1):e884. doi: 10.1097/PR9.0000000000000884

20. Li Y, Scherer N, Felix L, Kuper H. Prevalence of depression, anxiety and post-traumatic stress disorder in health care workers during the COVID-19 pandemic: A systematic review and meta-analysis. PLoS One. 2021; 16(3):e0246454. doi: 10.1371/journal.pone.0246454

21. Stoyanov ON. Neurovegetology of neuropathic pain, Textbook: Kyiv, 2015, 40 p (In Ukrainian).

22. Parisi S, Borrelli R, Bianchi S, Fusaro E. Viral arthritis and COVID-19. Lancet Rheumatol. 2020; 2(11):e655-e657. doi: 10.1016/S2665-9913(20)30348-9

23. Hypocalcemia in COVID-19: Prevalence, clinical significance and therapeutic implications / L. di Filippo, M. Doga, S. Frara, A. Giustina // Reviews in Endocrine & Metabolic Disorders. - 2022. - Vol. 23 (2). - P. 299–308. - DOI: 10.1007/s11154-021-09655-z.

25. Vitamin D Deficiency and Outcome of COVID-19 Patients /A. Radujkovic, T. Hippchen, S. Tiwari-Heckler [et al.] // Nutrients. - 2020. - Vol. 12 (9). - Article ID: 2757. - DOI: 10.3390/nu12092757.

24. Serum calcium and Vitamin D levels: correlation with severity of COVID-19 in hospitalized patients in Royal Hospital, Oman / W. Osman, F. Al Fahdi, I. Al Salmi [et al.] // International Journal of Infectious Diseases. - 2021. - Vol. 107. - P. 153–163. - DOI: 10.1016/j.ijid.2021.04.050.

25. COVID-19 and Vitamin D- a Systematic Review / T. Jordan, D. Siuka, N. K. Rotovnik, M. Pfeifer // ZdravstvenoVarstvo. - 2022. - Vol. 61(2). - P. 124–132. - DOI: 10.2478/sjph-2022-0017.

26. Pavlov, H., Torg, J.S., Robie, B., & Jahre, C. (1987). Cervical spinal stenosis. Determination with vertebral body ratio method. Radiology, 164(3), 771–775

27. Andreeva, T., Stoyanov, O., Mirdjuraev, E., Chebotareva, G., Kalashnikov, V., Vastyanov, R., Dariy, V. Clinical and morphometric features in cervical spine pathology in humans and animals. Pain, joints, spine. 2024; 14(3): 147–154. https://doi.org/10.22141/pjs.14.3.2024.430

28 Andreyeva, T.O., Stoyanov, OM., Chebotaryova, G.M., Kalashnikov, V.I., Vastyanov, R.S., & Mashchenko, S.S. (2023). Densitometric correlates of degenerative-dystrophic processes in cervical vertebrae of humans and domestic animals. Regulatory Mechanisms in Biosystems, 14(3), 386–392. doi:10.15421/022357

29. Andreyeva, T.O., Stoyanov, O.M., Chebotaryova, G.M., Vastyanov, R.S., Kalashnikov, V. I., & Stoyanov, A. O. (2022). Comparative clinical and morphometric investigations of cervical stenosis of the spinal canal in humans and dogs. Regulatory Mechanisms in Biosystems, 13(3), 301–307. doi:10.15421/022239

30. Andreyeva TO., Stoyanov OM., Chebotaryova HM., Manicheva NV., Kokidko LA. Comparison of morphometric, densitometrical data in the examination of the cervical spine of mammals. The 6th International scientific and practical conference “Science and technology: problems, prospects and innovations” (March 16-18, 2023) CPN Publishing Group, Osaka, Japan. 2023. 13-15

31. Kryvenko V.I., Pakhomova S.P., Fedorova O.P., et al. Formalized assessment of the patient's condition using scales for major internal diseases: Manual. Zaporizhzhia: Zaporizhzhia State Medical University, 2015. 97 p. (In Ukrainian).

32. Savchenko, V. M., Kharchenko, H. D., Kerestei, V. V., Buriak, O. Y., Pohrebniak, Y. M. Methodological peculiarities of domain assessment of international classification of functioning, disability and health that is used in physical therapy, ergotherapy. Rehabilitation and Recreation. 2023; (14), 98–112. https://doi.org/10.32782/2522-1795.2023.14.11 (In Ukrainian).

33. Ivchenko G.I., Medvedev Yu.I., Mathematical Statistics: Textbook.: Book House. "LIBROCOM", 2020. 219 p.

34. Narkevich I.A., Zubov N.N., Kuvakin V.I. Statistics in biomedicine, pharmacy and pharmaceuticals. Manual.: KnoPus, 2019. 300 p.

35. Holck P. Cervikalcolumnas anatomi [Anatomy of the cervical spine]. Tidsskr Nor Laegeforen. 2010 Jan 14;130(1):29-32. Norwegian. doi: 10.4045/tidsskr.09.0296. PMID: 20094120

36. Hautier L., Weisbecker V., Sánchez-Villagra M.R. et.all. Skeletal development in sloths and the evolution of mammalian vertebral patterning // Proc. Nat. Acad. Sci. USA. 2010; 107 (44):18903-18908. doi:10.1073/pnas.1010335107

37. Kaiser, J.T., Reddy, V., & Lugo-Pico, J.G. Anatomy, Head and Neck: Cervical Vertebrae. [Updated 2022 Oct 6]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK539734/

38. Bland, J.H.,& Boushey, D.R. Anatomy and physiology of the cervical spine. Semin Arthritis Rheum. 1990; 20(1):1-20. doi: 10.1016/0049-0172(90)90090-3

39. Kyungmin Han, Soon Tae You, Ho Jin Lee, Il Sup Kim, Jae Taek Hong & Jae Hoon Sung Hounsfield unit measurement method and related factors that most appropriately reflect bone mineral density on cervical spine computed tomography. Skeletal Radiology. 2022; 51:1987–1993.

40. Ordway NR, Lu YM, Zhang X, et al. Correlation of cervical endplate strength with CT measured subchondral bone density. Eur Spine J. 2007; 16(12):2104–2109

41. Kandziora F, Pflugmacher R, Scholz M, et al. Comparison between sheep and human cervical spines: an anatomic, radiographic, bone mineral density, and biomechanical study. Spine (Phila Pa 1976) 2001; 26(9):1028–1037

42. Weishaupt D, Schweitzer ME, DiCuccio MN, et al. Relationships of cervical, thoracic, and lumbar bone mineral density by quantitative CT. J Comput Assist Tomogr. 2001; 25(1):146–150

43. Yoganandan N, Pintar FA, Stemper BD, et al. Trabecular bone density of male human cervical and lumbar vertebrae. Bone. 2006; 39(2):336–344

44. Yoganandan N, Pintar FA, Stemper BD, et al. Bone mineral density of human female cervical and lumbar spines from quantitative computed tomography. Spine (Phila Pa 1976) 2006; 31(1):73–76