



CLINICAL FEATURES OF INFECTIOUS SPONDYLITIS IN PATIENTS WITH COVID-19

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Objective. To analyze the clinical features of the course of infectious spondylitis in patients with COVID-19.

Material and Methods. A continuous retrospective study was performed with the analysis of medical records of 52 patients with infectious spondylitis who were treated in 2021–2022. The patients were divided into two groups: the study group ($n = 24$) – with a history of a new coronavirus infection; and the control group ($n = 28$) – without coronavirus infection.

Results. The features of infectious spondylitis in patients with COVID-19 are the predominance of facultative anaerobic gram-negative flora in the focus of infection, a higher frequency of multilevel lesions, a tendency to increase the number of negative results of surgical treatment, and a chronic protracted course. At the same time, the course of infectious spondylitis associated with COVID-19 is accompanied by less destructive changes in the affected segment leading to a violation of the supporting function of the spine. Nevertheless, there is a statistically significant increase in the period of relief of the inflammatory process in the spine in these patients: 18.04 ± 3.84 weeks in the study group and 10.08 ± 2.34 weeks in the control group ($U_{\text{emp}} < 240$; $p = 0.001$).

Conclusion. The secondary infectious lesion of the spine against the background of a new coronavirus infection is caused by gram-negative pathogens in the vast majority of cases, proceeds without severe bone destruction, with a tendency to a chronic protracted course. Surgical treatment of COVID-associated spondylitis is associated with a higher risk of postoperative complications.

Key Words: infectious spondylitis, spondylodiscitis, coronavirus infection, COVID-19.

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Over the past three years, COVID-19 has changed the pattern of morbidity around the world. Coronavirus infection, mainly affecting the respiratory system, has a direct and indirect effect on other organ systems, including the musculoskeletal system [1, 3]. Tissues expressing hACE2, such as the lungs, heart, kidneys, testicles and others become the target organ in COVID infection [2, 3]. Meanwhile, the virus is isolated from blood, urine, and feces, as well as lung, liver, and gallbladder tissues [4]. The results of a series of pathohistological studies of patients with COVID-19 showed vascular lesions, congestive phenomena, necrosis, and hemorrhages, obtained not only in the lung tissue but also in other organs and systems. Detection of cyto-necrotic changes, infiltration of inflammatory cells (mononuclear cells and macrophages), and virions in histological samples is considered to be a consequence of both direct viral infection and hyperactivation of

the immune system [4, 5]. In patients with severe COVID-19, serum levels of cytokines and chemokines coincide with those in sepsis [5]. If sepsis as a bacterial generalized infection develops acutely, then with COVID-19, secondary sepsis of organs and tissues may have a relatively late manifestation and a chronic course [6]. Thromboembolism and hypercoagulopathy are the predictors and criteria of severe COVID-19 [6–9]. In the severe course of coronavirus infection, inflammatory processes in the focus of osteomyelitis contribute to bone resorption and create preconditions for necrosis [3]. Additionally, the formation of infectious foci in other organs and tissues (as well as in spinal tissues) may be influenced by systemic corticosteroids used in patients with severe COVID-19 symptoms. These drugs suppress systemic inflammation and the cytokine storm [10]. Their use significantly increases the risk of hyperglycemia, bacterial secondary infection with spreading and

development of avascular necrosis of the joints [10–12]. It is reasonable to assume that these factors can contribute to the development of infectious processes in the spine.

Nowadays, inflammatory lesions of the spine remain one of the most complicated problems in both social and medical aspects. Infectious spondylitis occurs with a frequency of 1.0–2.5 cases per 100 thousand people, making up 4–8 % of the structure of purulent inflammatory skeletal diseases. Purulent lesions of the spine are complicated by sepsis in 5.8–14.6 % of cases, and they result in mortality in 1.2–8.0 % of cases [13]. As a rule, spondylitis is a consequence of the progression and generalization of the infectious process. Chronic and acute septic lesions of the spine require an integrated approach to treatment. Despite the great attention paid to this pathology, complications and therapeutic failures reach 25 % of cases [14]. The features of the pathogenesis, the progressive course

and severe comorbidities do not provide an unambiguous definition of the treatment strategy for such patients. Undoubtedly, the course of infectious spondylitis associated with COVID-19 has its own peculiarities in view of the pathogenesis of coronavirus infection.

There are single case histories of spinal cord injury associated with the development of COVID-19 [15, 16] and with proven bacterial nature of the lesion are described in the literature. According to the literature sources, there is no data for cohort observations on the treatment of infectious diseases of the musculoskeletal system, in particular infectious spondylitis, in patients with coronavirus infection.

The objective is to analyze the clinical features of the course of infectious spondylitis in patients with COVID-19.

Materials and Methods

A continuous retrospective single-center study was performed with the analysis of the medical records of 52 patients with infectious spondylitis who were treated in 2021–2022. The patients were divided into two groups with the presence of new coronavirus infection (the study group, $n = 24$) and without it (the control group, $n = 28$).

Patients

The patients of the study group suffered new coronavirus of moderate and severe course and received medical care in the specialized infectious in-patient units of third-party hospitals for 21.2 ± 7.6 days. Information about the severity of COVID-19 is received from medical records provided by patients. The level of antibodies to SARS-CoV2 was determined by an enzyme immunoassay system (SARS-CoV-2-IgG-ELISA-BEST) for panel testing of immunoglobulins of classes M and G, calculated quantitatively in units of BAU/ml [17].

The IgM level was 4.521 ± 2.312 BAU/ml and the IgG level was 13.562 ± 4.281 BAU/ml, which indicated a recent acute infection. In the control group, patients had no history of new coronavirus, and the levels of antibodies to SARS-CoV2 (IgM and IgG) had negative values. The age of patients in the study

group was 52.4 ± 16.4 years; in the control group – 51.2 ± 11.6 years ($p > 0.05$).

Techniques

Patients of both groups underwent general clinical studies, including MRI, CT of the spine, and trepanobiopsy of the affected region with microbiological (cultural) testing. Microscopy with Ziehl – Neelsen staining was used to identify acid-resistant microorganisms; Gram staining was used to define gram-positive and gram-negative microorganisms. Molecular genetic testing was used to detect *M. tuberculosis* DNA, which was not revealed in our study. If surgery was performed, a microbiological examination of the removed tissues in the affected region was done. Trepanobiopsy was performed within 1–2 days after a patient's admission to the hospital. In all cases, a picture of nonspecific spondylitis was obtained in pathomorphological study without differences between groups in the cellular response and the nature of inflammation.

Radiological changes in the spine were reported under the Pola classification [18]. According to radiological findings, patients of both groups had signs of inflammation in the spine as evidenced by changes on MRI, with the spread to adjacent vertebrae in the form of bone marrow edema, destruction of end plates, the presence of paravertebral abscesses of soft tissues, and epidural leakage. CT examination showed destruction of the endplates and bodies of adjacent vertebrae and thickening of paravertebral tissues at the level of the affected region. Indications for surgical treatment were determined according to clinical guidelines in both groups for spino-neural conflict, impairment of the support function of the spine, and the need to debride the focus of infection [14].

According to the clinical and radiological symptoms and the results of laboratory diagnostics, microbiological, and pathomorphological examinations conducted at the clinic of the National Medical Research Center of Phthisiopulmonology and Infectious Diseases (Moscow), infectious spondylitis was diagnosed with verification of the causative factor.

The follow-up after discharge from the hospital was performed for 8–12 months. The treatment outcomes of patients were evaluated by the pain severity according to VAS, improvement of lab test values (white blood cells, ESR, C-reactive protein) and restoration of the support function of the spine. A positive outcome was considered to be a decrease in pain syndrome by VAS to 1–2 points, regression of neurological signs and the involution of inflammatory changes during control radiological examinations of the affected spine. After discharge from the hospital, patients were observed by remote telemedicine monitoring technology using a medical messenger (Medsenger).

The following outcomes of treatment were considered unsatisfactory: mortality, spreading of the infectious process, progression of destruction of the affected segment of the spine, spread of inflammation to other segments and increase in neurological impairment.

Statistical analysis

Statistical data processing was performed using the StatTeh v2.8.0 software, Pearson's chi-squared test, the Mann-Whitney U test, and the Chi-square test. The differences were considered significant with a confidence interval equal to 95 %, which did not contain 1 ($p = 0.05$).

Results

In the course of the study, it was found that the onset of a spinal lesion in patients of the study group began with axial pain syndrome 14.3 ± 3.8 days after the manifestation of clinical symptoms of COVID-19, that is, during admission to an infectious inpatient facility. Nevertheless, the detection of infectious spondylitis in all patients was performed 34.2 ± 9.4 days after the onset of the first symptoms and after discharge for outpatient treatment. In the control group, the preliminary diagnosis of infectious spondylitis was established after 13.1 ± 4.0 days ($p < 0.01$; $\chi^2 = 4.527$), after the onset of pain and an increase in the inflammatory syndrome. It means that the presence of COVID-19 significantly prolonged

the diagnostic stage associated with a pronounced inflammatory syndrome, which does not allow differentiating the cause of axial pain in the spine. Moreover, isolated pathogens are often resistant to the antibiotic therapy prescribed in the infectious unit.

According to Table 1, there was a significant increase in C-reactive protein and ESR characterizing the systemic inflammatory response in the study group. This is likely due to coming through a new coronavirus in a moderate-to-severe and severe degrees and the duration of the period of involution (recovery) of inflammatory markers, during which the development of infectious spondylitis occurred. It is remarkable that in the study group, despite a significant increase in CRP and ESR, insignificant leukocytosis was noted (Table 1), which may also be a consequence of a viral infection.

According to the results of microbiological examination in the study group, the pathogenic agents of infectious spondylitis were identified in 16 (66.8 %) cases, while in the control group they were identified in 20 (71.5 %; OR = 0.800). Moreover, in blood cultures for sterility, in four cases in the study group and in two cases in the control group, a pathogen was also detected that coincided with the results of the microbiological examination of the surgical specimen.

The distribution of infectious spondylitis pathogens in compared groups is shown in Table 2.

According to Table 2, there is a significant predominance of gram-negative microorganisms in the study group, while the main causative agent in the control group is *Staphylococcus aureus* (57.1 %). In one case, the pathogen *Candida albicans* was isolated from the blood of a patient of the study group and during the examination of the trepanobiopsy specimen.

The distribution of patients into groups depending on the presence of complications and the prevalence of the pathological process is given in Table 3.

Analyzing the data from Table 3, it was revealed that in the study group, despite the longer diagnostic stage and

the prolonged course of the disease, the involvement of neurological structures in the infectious process and the destruction of supporting structures with the formation of unstable lesions are observed in fewer cases.

In COVID-associated spondylitis, according to radiological findings, the destructive process in the vertebral bodies was less pronounced and was most often limited to the lesion of intervertebral discs with endplates without involving supporting structures (Table 4).

In the study group, changes with smaller destructive processes prevailed (A, B1, C1, C3): 70.8 %; in the control group – 35.7 % ($p = 0.014$). In the control group, more pronounced destructive lesions (types B3, C4) were noted (39.3 %); in the study group – 12.5 % ($p = 0.057$).

Disorders of the spinal support function (B3, C2, C4 according to Pola): 3 (12.5 %) patients in the study group, and 13 (46.4 %) in the control group ($p = 0.015$).

Spinal lesions with neurological disorders in the study group were associated with the presence of an epidural abscess with the formation of spino-neural conflict (type C3 according to Pola). Fig. 1 illustrates a clinical case of the development of an epidural abscess of the thoracolumbar junction in a patient with COVID-19.

In the control group, neurological impairment was associated, as a rule, with a pathological fracture and progressive kyphotic deformity at the lesion level (type C4, according to Pola).

Multilevel lesion of the spine was more common in the patients of the study group (Table 5) than in patients from the control group ($p = 0.039$). Fig. 2

illustrates a clinical case of multilevel spinal lesion in a patient who has suffered new coronavirus.

Treatment of patients was started, as a rule, at the prehospital stage with the use of broad-spectrum antibiotic test therapy. During admission to the hospital, treatment was not changed or canceled until the results of the microbiological examination of blood, urine, feces and biopsy material were obtained. After verification of the pathogen, antimicrobial therapy was adjusted in accordance with individual sensitivity. Antimicrobial therapy was continued until the improvement of lab test values for the inflammatory syndrome

According to the set of criteria described in the medical records, the mean duration of relief of the inflammatory syndrome in the study group was 18.04 ± 3.84 (Me = 17.5; m = 0.96) weeks; in the control group, it was 10.08 ± 2.34 (Me = 10; m = 0.58) weeks ($U_{\text{emp}} < 240$; $p = 0.001$). Therefore, COVID-associated infectious spondylitis progressed for a longer time and required an increase in the duration of antibacterial therapy.

If there were signs of spino-neural conflict, disorders of the spinal support function, debridement and stabilization were performed according to standard techniques [13, 14].

In the study group, surgery was performed in five patients in the volume of combined fusion with the debridement of the focus of infection. In two patients of the study group, after surgical treatment performed for urgent indications (increased compression of the spinal cord), death occurred in 14 and 21 days. The cause of the lethal outcome was the spread of bacterial infection with throm-

Table 1
Values of laboratory inflammatory markers in groups (Mann – Whitney U test)

Laboratory parameters	Study group (n = 24)	Control group (n = 28)	$U_{\text{emp}} < 240$; $p < 0.05$
ESR, mm/h	42.50 ± 15.09	39.25 ± 16.09	285 (insignificant)
Leukocytosis, $10^9/\text{L}$	9.82 ± 4.12	16.73 ± 6.07	127 (significant)
C-reactive protein, mg/L	73.40 ± 37.20	24.60 ± 13.10	73 (significant)

Table 2

Distribution of pathogens isolated from the lesion focus in compared groups, n (%)

Pathogen		Study group (n = 24)	Control group (n = 28)	Fisher's f-test, p
Gram-positive	<i>Stph. aureus</i>	2 (8.3)	16 (57.1)	0.001
	<i>Stph. Aureus MRSA</i>	2 (8.3)	—	
	<i>Stph. Epyderm.</i>	—	2 (7.2)	
	Total:	4 (16.6)	18 (64.3)	
Gram-negative	<i>Ent. faecalis</i>	2 (8.3)	1 (3.6)	0.001
	<i>Klebs.pneumonia</i>	4 (16.6)	—	
	<i>E.coli</i>	4 (16.6)	1 (3.6)	
	<i>Proteus mirabilis</i>	2 (8.3)	—	
	Total:	12 (50.0)	2 (7.2)	
<i>Candida</i>	<i>Candida albicans</i>	1 (4.2)	—	—
No pathogens		7 (29.2)	8 (28.5)	—
Total:		24 (100.0)	28 (100.0)	—

Table 3

Volume of lesions and complications of infectious spondylitis in the compared groups, n (%)

Complications	Study group (n = 24)	Control group (n = 28)	Fisher's f-test, p
Epidural abscesses	3 (12.5)	12 (42.9)	0.030
Disorders of the supporting function of the spine	3 (12.5)	13 (46.4)	0.015
Neurological disorders	2 (8.3)	7 (28.6)	0.152
Polysegmental lesions	0 (0.0)	4 (14.3)	0.756
Multilevel non-adjacent lesions	3 (12.5)	0 (0.0)	1.000

Table 4

Distribution of patients into groups depending on the types of lesions according to the Pola classification [18], n (%)

Pola classification (2017)	Study group (n = 24)	Control group (n = 28)	Fisher's f-test, p
<i>Type A</i>	8 (33.3)	3 (10.7)	0.086
A1	0	0	
A2	1	0	
A3	3	3	
A4	4	0	0.593
<i>Type B</i>	13 (54.2)	13 (46.4)	
B1	6	2	
B2	4	3	
B3	3	6	0.031
<i>Type C</i>	3 (12.5)	12 (42.9)	
C1	1	3	
C2	0	2	
C3	2	2	
C4	0	5	

boembolic complications associated with acute inflammatory syndrome.

The progression of infectious spondylitis at an adjacent level was revealed in one patient 16 weeks after the initial surgery (Fig. 3). *Candida albicans* was seeded in the blood and in the surgical specimen during trepanobiopsy of the L2–L3 disc. Subsequently, fungal meningitis developed in the patient (640/3, 40 % neutrophils, 60 % white blood cells, culture of cerebrospinal fluid: *Candida albicans* $\times 10^3$).

In the control group, surgery was performed in 13 (46.3 %) cases associated with a disturbance of the spinal support function and spino-neural conflict. There were no negative surgical outcomes in this group.

Discussion

The study analyzed a group of patients with new coronavirus infection complicated by a secondary bacterial lesion of the spine. The pathogenesis of destruction of the spinal column structures in new coronavirus infection remains controversial. According to the results of the microbiological examination, the pathogen was detected in 66.8 % of patients in the study group. Nevertheless, in the case of a purulent spinal lesion associated with COVID-19, there is a significant predominance of facultative anaerobes in the focus of destruction (2 = 15.8; $p = 0.001$), which is more frequent in hospital-acquired infections.

All patients in the study group underwent inpatient treatment for new coronavirus infection and received long-term intensive hormone therapy using dexamethasone in large doses, which results in immunosuppression and activation of conditionally pathogenic flora [10]. Conditions for secondary bacterial infection are created due to formation of local necrosis in tissues, including in the spine, associated with a viral infection. As for the systemic hyperreaction of the body to the coronavirus due to the cytokine storm, it results in immunological exhaustion [5]. Treatment of patients with immunosuppressive drugs and steroids reduces the body's defense against

bacterial infection. Therefore, the local lesion of the spine in combination with COVID-19 is a consequence of a viral lesion with a defensive circle of bacterial infection and the possible development of sepsis. The viral nature of the focal lesion of the spine has not been proven due to the admission of patients already in the phase of bacterial spondylodiscitis. Infectious spondylitis in COVID-19 develops according to the type of septic condition with secondary damage to the spinal structures and an increase in the proportion of multilevel lesions.

Differential diagnosis is complicated due to the severity of the condition and the stratification of the symptoms of viral infection, which causes late diagnosis, and requires greater attention to patients' complaints of back pain associated with new coronavirus infection. The tendency for the process to become chronic due to local thrombosis and systemic coagulopathy, associated with a reduced immune response and conditionally pathogenic and hospital-acquired flora results in a significant prolongation of the treatment period and, as a consequence, the

need for long-term antibacterial therapy. Adverse outcomes in the study group were 60 % and were absent in the control group. The spread of infection after surgery is due to the mutual worsening of the two diseases, which makes the application of surgical treatment for this pathology as carefully as possible.

Conclusion

1. An acute course of COVID-19 may be complicated by secondary bacterial lesions of the spine.
2. The infectious process associated with COVID-19 is susceptible to spreading with the destruction of several levels and is distinguished by a prolonged chronic course, which requires longer antibiotic therapy.
3. The specific features of infectious spondylitis in COVID-19 patients are the significant predominance of facultative anaerobes as the pathogenic agent, which requires mandatory microbiological verification of infection for the selection of appropriate antibacterial therapy.
4. In the case of new coronavirus infection, the secondary infectious lesion of the spine proceeds in the vast majority of cases without pronounced bone destruction.
5. Surgical treatment of COVID-associated spondylitis is associated with the higher risks of postoperative complications.



Fig. 1

Patient A, male, born in 1963, with the onset of new coronavirus infection, pain in the lumbar spine: on admission to the infectious disease ward, a CT-3 lesion was verified on by CT of lungs dated January 20, 2022 (a); MRI of the spine dated February 13, 2022 revealed spondylodiscitis of L1–L2 with the formation of an epidural abscess of type C1 according to Pola (b)

Table 5

Distribution of patients by levels of spinal lesion, n (%)

Spine department	Study group	Control group	Fisher's f-test, p
Cervical	2 (8.3)	0 (0.0)	0.208
Thoracic	8 (33.3)	12 (42.9)	0.773
Lumbosacral	10 (41.7)	16 (57.1)	0.404
Multilevel lesion	4 (16.7)	0 (0.0)	0.039
Total:	24 (100.0)	28 (100.0)	—

The study had no sponsors.

The authors declare that they have no conflict of interest.

The study was approved by the local ethical committee of the institution.

All authors contributed significantly to the research and preparation of the article, read and approved the final version before publication.



Fig. 2

Patient G., male, born in 1983, with multilevel spondylitis of C6–C7 **(a)** and L1–L2 **(b)** of type C1 according to Pola

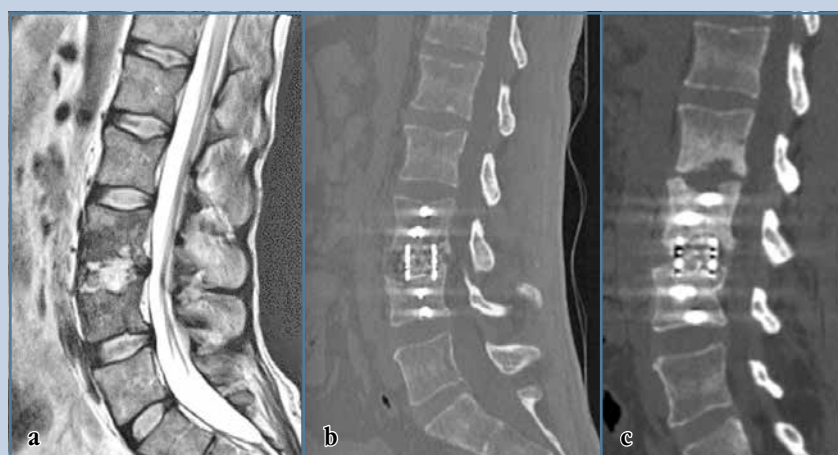


Fig. 3

Patient B., male, born in 1985, with coronavirus infection COVID-19 (confirmed), severe form of bilateral polysegmental interstitial pneumonia, respiratory failure of type 2 (Non-Invasive Ventilation from September 15, 2021 to October 01, 2021); in-patient stay in the infectious diseases ward from September 14, 2022 to October 06, 2022 (21 days); has been experiencing pain in the spine since October 2021; body temperature returned to normal by November 2021; spondylodiscitis of L3–L4 (B1 according to Pola) was diagnosed on January 15, 2022 by MRI T2 **(a)**; surgical treatment, considering the lack of effect of conservative therapy on January 18, 2022; CT scan of the lumbar spine dated February 1, 2022 **(b)**; progression of spondylitis of L2–L3 (B1 according to Pola) according to CT scan of the lumbar spine on June 5, 2022 **(c)**; on August 11, 2022, fungal meningitis was diagnosed (640/3, 40 % – neutrophils, 60 % – lymphocytes, *Candida albicans* $\times 10^3$ in cerebrospinal fluid culture)

References

1. Polyakova YuV, Papichev EV, Akhverdyan YR, Sivordova LE, Zavodovskiy BV. New coronavirus infection – direct and indirect impact on patients with diseases of the musculoskeletal system and connective tissue. *Modern Problems of Science and Education*. 2021;(6):164. DOI: 10.17513/spno.31342.
2. Cai Q, Huang D, Yu H, Zhu Z, Xia Z, Su Y, Li Z, Zhou G, Gou J, Qu J, Sun Y, Liu Y, He Q, Chen J, Liu L, Xu L. COVID-19: Abnormal liver function tests. *J Hepatol*. 2020;73:566–574. DOI: 10.1016/j.jhep.2020.04.006.
3. Disser NP, De Micheli AJ, Schonk MM, Konnaris MA, Piacentini AN, Edon DL, Toresdahl BG, Rodeo SA, Casey EK, Mendias CL. Musculoskeletal Consequences of COVID-19. *J Bone Joint Surg Am*. 2020;102:1197–1204. DOI: 10.2106/JBJS.20.00847.
4. Remmelink M, De Mendonca R, D'Haene N, De Clercq S, Verocq C, Lebrun L, Lavis P, Racu ML, Tr pant AL, Maris C, Rorive S, Goffard JC, De Witte O, Peluso L, Vincent JL, Decaestecker C, Taccone FS, Salmon I. Unspecific post-mortem findings despite multiorgan viral spread in COVID-19 patients. *Crit Care*. 2020;24:495. DOI: 10.1186/s13054-020-03218-5.
5. Kocak Tufan Z, Kayaaslan B, Mer M. COVID-19 and sepsis. *Turk J Med Sci*. 2021;51(SI-1):3301–3311. DOI: 10.3906/sag-2108-239.
6. Hu B, Huang S, Yin L. The cytokine storm and COVID-19. *J Med Virol*. 2021;93:250–256. DOI: 10.1002/jmv.26232.
7. Li H, Liu L, Zhang D, Xu J, Dai H, Tang N, Su X, Cao B. SARS-CoV-2 and viral sepsis: observations and hypotheses. *Lancet*. 2020;395:1517–1520. DOI: 10.1016/S0140-6736(20)30920-X.
8. Lowenstein CJ, Solomon SD. Severe COVID-19 is a microvascular disease. *Circulation*. 2020;142:1609–1611. DOI: 10.1161/CIRCULATIONAHA.120.050354.
9. Panigada M, Bottino N, Tagliabue P, Grasselli G, Novembrino C, Chantarangkul V, Pesenti A, Peyvandi F, Tripodi A. Hypercoagulability of COVID-19 patients in intensive care unit: A report of thromboelastography findings and other parameters of hemostasis. *J Throm Haemost*. 2020;18:1738–1742. DOI: 10.1111/jth.14850.
10. Mammen MJ, Aryal K, Alhazzani W, Alexander PE. Corticosteroids for patients with acute respiratory distress syndrome: a systematic review and meta-analysis of randomized trials. *Pol Arch Intern Med*. 2020;130:276–286. DOI: 10.20452/pamw.15239.
11. Pavli A, Theodoridou M, Maltezou HC. Post-COVID syndrome: incidence, clinical spectrum, and challenges for primary healthcare professionals. *Arch Med Res*. 2021;52:575–581. DOI: 10.1016/j.arcmed.2021.03.010.
12. Tao H, Ge G, Li W, Liang X, Wang H, Li N, Sun H, Zhang W, Geng D. Dysimmunity and inflammatory storm: Watch out for bone lesions in COVID-19 infection. *Med Hypotheses*. 2020;145:110332. DOI:10.1016/j.mehy.2020.110332.
13. Herren C, Jung N, Pishnamaz M, Breuninger M, Siewe J, Sobottke R. Spondylodiscitis: diagnosis and treatment options. *Dtsch Arztebl Int*. 2017;114:875–882. DOI: 10.3238/arztebl.2017.0875.
14. Mushkin AYU, Vishnevsky AA, Peretsmanas EO, Bazarov AYU, Basankin IV. Infectious lesions of the spine: draft national clinical guidelines. *Hir. Pozvonoc*. 2019;16(4): 63–75. DOI: 0.14531/ss2019.4.63-76.
15. Tanaka M, Takahashi S, Ishibe T, Masuda N. COVID-19 masked by pyogenic lumbar discitis and bacteremia: a case report. *JBJS Case Connect*. 2021;11(4). DOI: 10.2106/JBJS.CC.21.00059.
16. Qiu M, Jayasekara D, Jayasekara A. Post-COVID-19 infection with methicillin-sensitive *Staphylococcus aureus* (MSSA) bacteremia, discitis/osteomyelitis, and diffuse abscesses: a case report. *Cureus*. 2022;14:e25824. DOI: 10.7759/cureus.25824.
17. Tataeva ZM, Ataeva AU, Zhiburt EB. Quantitative assessment of the content of antibodies to SARS-CoV-2 in the plasma of donors. *Handbook of the head of the CDL*. 2021;(9):7–11.
18. Pola E, Autore G, Formica VM, Pambianco V, Colangelo D, Cauda R, Fantoni M. New classification for the treatment of pyogenic spondylodiscitis: validation study on a population of 250 patients with a follow-up of 2 years. *Eur Spine J*. 2017;26(Suppl 4): 479–488. DOI: 10.1007/s00586-017-5043-5.

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