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Possible Relation between Coronavirus Infection or Vaccine and the Occurence of Spontaneous Spondylodiscitis

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Abstract

Background: Coronavirus disease 2019 (COVID-19) infection brought about tremendous changes in the world. It remains a challenge to the medical community, for it has been a constant uphill battle managing the myriad complications that occur as a result of this deadly virus infection. COVID-19 causes damage to the endothelium of vessels in the lung, giving rise to its pulmonary complications.

Coronavirus disease (COVID-19) is an infectious disease. It affects almost all organs of the human body. Spondylodiscitis is an infection of the vertebral body and disc.

The association of COVID-19 or COVID-19 vaccine with Spondylodiscitis has not been reported in the literature so far. The purpose of this study is to describe a series of fifty cases of spontaneous spondylodiscitis and to describe its association with COVID-19 infection or vaccination.

Aim of Study: The paper aims to determine any possible relation between the occurrence of spontaneous spondylodiscitis and previous or current infection by COVID virus or the reception of its vaccine.

Patients and Methods: This study was carried on 36 patients having adult sponteneous spondylodiscitis (proven clinically and radiologically) and we described its possible relation with COVID-19 infection or vaccination. Some patients received antibiotics for management of the infection, non responding patients or those with progressive neurological deficits, spinal instability or deformity where again managed surgically. The possible tissues or discharge where biopsied for culture to know the causative organism.

Results: 22 (61%) patient received COVID-19 vaccine (21 patients received Sinovac and one patient received Pfizer) before symptoms of spondylodiscitis by mean duration of 93 day SD-/+35 day, all of them experienced confirmed COVID-19 infection before symptom development by mean of 255 day SD =/-93 day, showing significant difference of time span between COVID-19 infection and development of spondylodiscitis against time span between Vaccination and development of spondylodiscitis (p 0.03). The remaining 14 patients, 9 of them experienced COVID-19 infection before spondylodiscitis symptoms by mean of 241 day +/-89 day, 5 patients did not experience COVID-19 infection or received vaccination.

Conclusion: COVID-19 infection is a predisposing factor for spondylodiscitis in many studies, yet in our study much shorter temporal relationship between COVID-19 vaccination and development of spondylodiscitis was noted and needs further research work up.

Key Words: *Spondylodiscitis – COVID-19 infection – Vaccination.*

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Introduction

CORONAVIRUS disease 2019 (COVID-19) infection led to enormous changes worldwide. It remains a great concern to the community of medicine, for it has been a constant struggle to manage the various complications that occur as a result of this fatal virus infection. COVID-19 causes a defect in the endothelium of the vessels in the lung, causing pulmonary complications **[1,2].**

Recently, the systemic involvement of the virus is noticed as in the spine and the neurological system. Epidural and retropharyngeal suppuration have been reported together with the COVID-19 infection. The epidural suppuration can cause neurological weakness **[2-4].**

The causative organism is SARS-COV-2, which can cause an injury to the endothelium, increasing the risk of intravascular coagulation, although asymptomatic cases are common **[5,6]**. In a large series of autopsy, all cases presented variable degrees of endothelial injury and 87% of cases had arteriolar thrombosis **[6-8]**. The treatment of COVID-19 has evolved according to the progress of knowledge on the pathogenesis of the disease and the results of

clinical studies involving different pharmaceutical molecules, the following categories of drugs such as: Antiviral, anti-inflammatory and immuno-modulatory, anticoagulants, antibiotics, other anti-infectious medications, drugs supporting vital functions and symptomatic medication. The choice for each therapeutic intervention depend on the severity of the disease and the patient's risk factors **[9].**

Spondylodiscitis (SD) associated with COVID-19 are new perspective of COVID-19, rarely reported in the medical literature **[10]**. Practically, SD is rare, although the diagnosis could be underestimated, mostly in poor countries, with poor hygiene and difficult access to health care facilities **[11]**. The SD can happen at any age, while is most often found mainly in the sixth decade of life **[12]**. In addition, the risk for occurrence of SD increases with advancing age, diabetes mellitus, steroids intake, rheumatologic diseases, after spine surgery, renal failure, liver cirrhosis, malignancies, intravenous drugs use, intravascular devices, HIV or various skin and soft tissue infections, infection of the mouth mucosa, genitourinary or respiratory tract **[13,14]**. The source of infection in SD is often undetected at the time of diagnosis **[14,15].**

The causative organism could be either bacterial or non-bacterial. The major organism causing spondylodiscitis is Staphylococcus aureus (20-40%), followed by Enterobacteriaceae spp. (7%-33%), Streptococci spp., Enterococci spp. (5%-20%), and anaerobic organisms (less than 4%) **[13,16,17].** Before using antibiotics, spondylodiscitis thought to have led to mortality of one to three quarters of cases, but nowadays is decreasing to 2–12% **[13,18].** The mechanism of infection is mainly blood-borne, either in spontaneous or post-operative SD **[19].** The most common site for spondylodiscitis is the lumbar (60%), the thoracic (30%) and the cervical spine (10%) **[18,20].** The diagnosis of SD is based on clinical, laboratory and radiological criteria. In early stages of SD, there are nonspecific symptoms such as mild fever, malaise, weakness, and loss of weight. The main complaint is the back pain, which increases in the night, although the pain may be absent in 15% of patients **[21].**

The diagnostic of SD is often delayed, due to the low specificity of clinical presentation **[19,22]**. The occurrence of complications, as sepsis, multiorgan failure or neurological symptoms could be related to para or intraspinal abscess or destruction of vertebral bone, raising the mortality to 17% **[22]**. High titers of C-reactive protein (CRP) and increased white blood cell count are usually markers of infections, although their significance is limited for the detection of SD **[19,21]**. The accurate diagnosis requires histological analysis or direct detection of aetiologic organism from the blood cultures or cultures of biopsy samples **[12]**. Blood culture is the easiest, cheapest, and most effective method for confirming the etiology, if is obtained before the antibiotic treatment. X-ray images of the spinal segments have occasionally associated osteolysis and shadowing of the paravertebral soft tissue, suggesting a spinal abscess.

Magnetic resonance imaging (MRI) is more sensitive and specific for the diagnosis of SD and therefore is the method of choice **[12]**. Paravertebral abscess could be a complication of spondylodiscitis **[23].** A half of cases has normal white blood cell and no fever, while bacteremia is detected in 60% and Staphylococcus aureus is responsible for 70% of cases **[24]**. The antibiotic conservative treatment is the standard of care for SD, but the failure rate range between 25% to 56%. There are no guidelines for the duration of therapy, but patients typically require 4–8 weeks of therapy **[12,18,19]**. The surgical treatment is recommended for patients with cauda equina syndrome, extensive bone destruction or antibiotic treatment Coronavirus disease 2019 (COVID-19) infection led to enormous changes worldwide. It remains a great concern to the community of medicine, for it has been a constant struggle to manage the various complications that occur as a result of this fatal virus infection. COVID-19 causes a defect in the endothelium of the vessels in the lung, causing pulmonary complications **[1,2].** Recently, the systemic involvement of the virus is noticed as in the spine and the neurological system. Epidural and retropharyngeal suppuration have been reported together with the COVID-19 infection. The epidural suppuration can cause neurological weakness **[2-4].**

The causative organism is SARS-COV-2, which can cause an injury to the endothelium, increasing the risk of intravascular coagulation, although asymptomatic cases are common **[5,6]**. In a large series of autopsy, all cases presented variable degrees of endothelial injury and 87% of cases had arteriolar thrombosis **[6-8]**. The treatment of COVID-19 has evolved according to the progress of knowledge on the pathogenesis of the disease and the results of clinical studies envolving different pharma ceutical molecules, the following categories of drugs such as: Antiviral, anti-inflammatory and immuno-modulatory, anticoagulants, antibiotics, other anti-infectious medications, drugs supporting vital functions and symptomatic medication. The choice for each therapeutic intervention depend on the severity of the disease and the patient's risk factors **[9]**.

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tologic diseases, after spine surgery, renal failure, liver cirrhosis, malignancies, intravenous drugs use, intravascular devices, HIV or various skin and soft tissue infections, infection of the mouth mucosa, genitourinary or respiratory tract **[13,14].**

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Patients and Methods

This retrospective analytical study was carried out on thirty six patients diagnosed as having spontaneous spondylodiscitis.

The study was conducted at Alexandria Main University Hospitals in Egypt from from Oct. 2021 till Oct. 2022.

Ethical approval and consent to participate:

All procedures performed in the study involving human participants were in accordance with the ethical standards of the institution and approved by the Ethics Committee of Alexandria University.

Statistical analysis:

- Microsoft excel 2013 will be used for data entry and the statistical package for social science (SPSS version 24) will be used for data analysis.
- Arithmetic mean and standard deviation will be used for summary of normal quantitative data, median and interquartile range will be used for summary of abnormal quantitative data, and frequencies will be used for qualitative data.
- Bivariate relationship will be displayed in cross tabulations and comparison of proportions will be performed using the chi-square and Fisher's exact tests where appropriate.
- *t*-independent will be used to compare normally distributed quantitative data and Mann Whitney for skewed data.
- *p*-value will be calculated to assess statistical significance, a value less than 0.05 will be considered statistically significant.

Methodology in details:

Patients presented to Alexandria Main University hospital during the period from Oct. 2021 till Oct. 2022 suffering from suspected spondylodiscitis were evaluated by history taking, history of COVID-19 infection or recent COVID-19 vaccination, including the duration between each and development of symptoms. Patients are evaluated clinically and laboratory to confirm the diagnosis and exclude other causes of immune deficiency, localizing factors (recent surgical intervention at or near by levels) and to exclude specific infections (for example TB) and sources of bacteraemia (such as bacterial endocarditis).

Imaging evaluation included radiography in at least two perpendicular planes at the sites of pain and tenderness, CT and MRI examination including axial and sagittal T1 WI axial and sagittal T2, sagittal T2 fat sat, In and Out-of-phase T1 WI, sagittal T1 fat sat post Gadolinium contrast.

Antibiotic treatment and its duration before and after presentation, clinical picture, presence of neurological signs, surgical intervention, blood, fluid aspiration culture, antibiogram results and medical treatment were evaluated.

Results

We recruited 58 adult patients presented to Alexandria Main University Hospital during the period from Oct. 2021 till Oct. 2022 suffering from clinically diagnosed, radiologically and laboratory confirmed to have spondylodiscitis, excluded 22 patients of them due to association with uncontrolled diabetes (5 patients 3 of them suffering from concomitant or history of diabetic foot), other immune compromised state (2 patients due to recently managed malignancy by chemotherapy during the past 6 month and one patient HIV positive suffering from skin lesions), low BMI (7 patients below 18.5 MBI), tuberculous spondylodiscitis patients (two are giving history of old TB infection, the third with no previously confirmed TB infection but positive for serum quantiferon showing adequate response to anti tuberculous drugs), injectable drug addiction (2 patients) and recent surgical intervention at the same or nearby level (2 patients with lumbar laminectomy discectomy and internal fixation by plates and screws). None of the patients suffered from bacterial endocarditis, Rheumatic heart disease or prosthetic valve replacement.

Age ranged from 22 to 67 year old, with mild male predominance 22 males (61%) and 14 females (39%), 24 suffering from lumbar spine spondylodiscitis, 11 suffering from dorsal spondylodiscitis and a patient suffered from cervical spondylodiscitis.

All patients suffered from pain, low grade fever (30 patients), high grade fever (6 patients) 14 patients suffered from localizing neurological signs (10 of the dorsal, two of the lumbar and the patient who suffered from cervical spondylodiscitis), showing bilateral lower limb weakness (motor power ranging from grade 4 to 1 according to MMT scale), sensory level at or below nipple line in two patients, both suffered from dorsal spondylodiscitis, the patient suffered from cervical spondylodiscitis showed severe quadri-paresis. All patients showed elevated inflammatory markers (Serum CRP ranging from 4.5 to 11.4, elevated WBC ranging from 5.9 to 8.2), 33 patients have received anti-inflammatory drugs (paracetamol/NSAID) and antibiotics for duration ranging from 2 to 19 day before presentation.

22 (61%) patient received COVID-19 vaccine (21 patients received Sinovac and one patient received Pfizer) before symptoms of spondylodiscitis by mean duration of 93 day SD-/+35 day, all of them experienced confirmed COVID-19 infection before symptom development by mean of 255 day SD =/- 93 day, showing significant difference of time span between COVID-19 infection and development of spondylodiscitis against time span between Vaccination and development of spondylodiscitis (*p* 0.03). The remaining 14 patients, 9 of them experienced COVID-19 infection before spondylodiscitis symptoms by mean of 241 day +/-89 day, 5 patients did not experience COVID-19 infection or received vaccination.

All patients were subject to medical examination, laboratory evaluation for base line inflammatory markers, underwent radiographic evaluation, CT and MRI examination (31 underwent contrast enhanced MRI and 5 refused contrast enhanced study). Radiographic evaluation failed to help in showing signs of spondylodiscitis in 12 patients (33.3% 10 of them were suffering from dorsal spondylodiscitis, two (5%) are suffering from lumber spondylodiscitis), 24 patients showed evidence of endplate erosive changes.

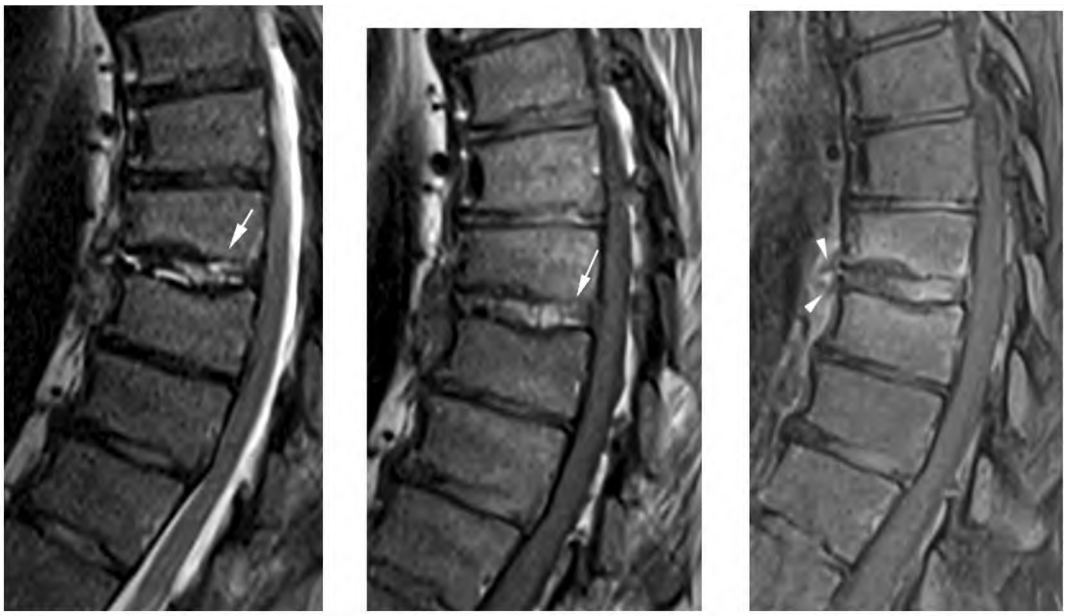
The examined 36 patient's MRI revealed variable degrees of T1 hypo T2 hyper intense signal marrow edema at the adjacent vertebral body segments related to the disc space affected, less noted in patients received Antibiotics before presenting to our institute, lost normal marked hypo intense endplate signal at the levels affected with intervertebral T1 hypo T2 hyper intense signal edema, except for a patient showed initial T1, T2 and T2 fat sat hyper intense disc signal, all showed significant signal suppression on out-of-phase sequence compared to in-phase sequence (mean signal suppression 35% SD+/-20), lost normal disc nuclear cleft signal (in 100% of the patients) with marginal post contrast enhancement (in all who received Gadolinium contrast).

Enhancing peri vertebral phlegmon was noted (in 100% of the patients), associated with non-enhancing necrotic fluid signal foci (in 30 patient 83.3%, ranging from 3 to 8mm in thickness, noted at the anterior peri vertebral region (in 30 patients) and anterior epidural space (in 20 patients, 7 of them at dorsal levels and one at cervical level ranging from 2 to 4mm). Abnormal T1 hypo T2 hyper intense signal of the cord was noted (in 7 patients, all having epidural non enhancing necrotic fluid signal). T2 fat sat WI did not show significant signal difference between phlegmon and necrotic fluid (small abscesses).

All patients were subject to blood culture (only 16 patients had positive blood culture 44.4%) received empirical antibiotic treatment for a duration ranging from 5 to 7 days then shifted to antibiotics courses according to the antibiograms for duration ranging from 21 to 28 day, surgical intervention with posterior laminectomy, epidural fluid aspiration, laboratory evaluation and culture sensitivity done for 19 patients, 9 of them with lower limb power less than 3 and 10 failed medical treatment with persistent pain and MRI evidence of residu-

al peri vertebral abscesses. A patient suffered from cervical spondylodiscitis underwent anterior laminectomy with Pyra mesh placement, CV3 through CV7 fixation by plate and screws, followed by plate upward migration and anterior displacement with obvious evidence of deep cervical abscess. Managing abscess by drainage followed by fluid leakage from the surgical wound specially after drinking, suspected plate erosion into the pharyngeal wall was documented, followed by anterior and posterior reconstruction. 16 patients cured by medical treatment until resolution of symptoms and normalization of CRP levels. All the 20 patients needed surgical intervention were from the group received COVID-19 vaccine (with significant difference p 0.001).

Culture sensitivity proved sterile in 8 patients (40%), Pseudomonas in 2 patients (10%), Klebsiella and Streptococcus aureus also in 2 patients each, MRSA and Staphylococcus in 1 patient each of them (5%).



(A) (B) (C)

Fig. (1): Showing hyper intense intervertebral disc signal on sagittal T2 WI (A), sagittal T1 WI (B) with lost normal signal void appearance of the superior end-plate (arrows) associated with mild marrow edema, diffuse disc, sub-endplate enhancement is noted with enhancing anterior epidural phlegmon noted on sagittal post contrast T1 fat sat WI (C). Small non enhancing foci are noted, representing small osteophytes (small arrow heads in image C).

(A) (B) (C) (D)

Fig. (2): Significant signal suppression on out-of-phase sequence (A), compared to in-phase sequence (B), confirming marrow edema on sagittal T1 WI (C). Diffuse disc space and related vertebral body enhancement noted on sagittal T1 fat sat WI (D).

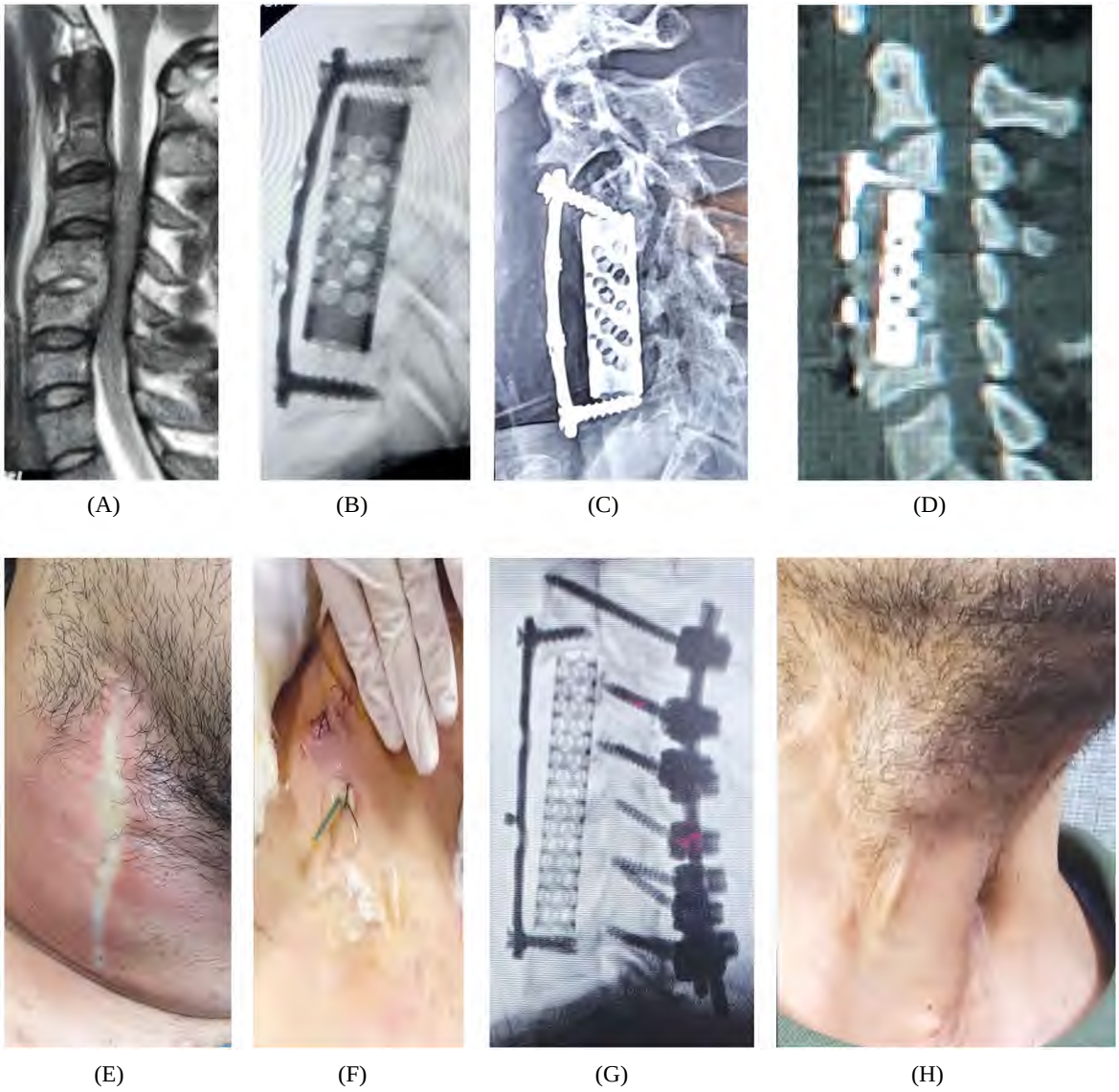


Fig. (3): (A) Sagittal T2 MRI cervical spine: Initial sagittal T2 MRI on presentation, showing CV4/5 inter-vertebral disc hyper-intense signal, vertebral sub-end-plate marrow edema and significant stenosis of the cervical canal, resulting in hyper intense signal cord compression myelopathy. (B) Lateral radiographic image after step one operative management by CV4 through CV6 corpectomy, Pira mesh and anterior fixation. Patient at this point refused posterior fixation. (C) Lateral radiography of the cervical spine a month after fixation developed plate and screw loosening, superior migration indenting CV2 anterior body inspite of being on antibiotic course, showing reduced CV3 body height representing erosive changes. (D) Sagittal reformatted image of CT done at the same duration proving upper screw loosening, plate superior migration is less obvious due to supine CT patient's position. (E) Patient at second presentation with neck abscess and systemic inflammatory signs. (F) Surgical drainage of the abscess with drain placement to improve patient's medical condition before definitive management, showing clear fluid oozing around the drain, raising the possibility of pharyngeal erosion by the plate. (G) lateral radiography after definitive management by CV3 through CV6 corpectomy, Pira mesh placement with anterior and posterior fixation. (H) The patient after completing another 60 day antibiotic course.

Discussion

Spondylodiscitis usually induced by a variety of microorganisms, with S. aureus being the most common bacteria, responsible for 40-65% of cases, then E. coli, Proteus sp, lastly Pseudomonas. Presenting initially by non-specific symptoms, subacute course, usually associated with back pain, high grade fever is not usual and can be associated with neurological deficits [25,26]. Commonly presenting by elevated acute phase reactants (ESR, CRP, procalcitonin), together with or without leukocytosis [27]. MRI is considered most diagnostic sensitive and specific method, detecting vertebral body edema as well as intervertebral disc fluid signal represented by T1 hypo T2 hyper intense signal, both showing enhancement together with lost signal void appearance of the vertebral end-plate of affected vertebral bodies being most sensitive and specific for diagnosis. Enhancing peri vertebral phlegmon and non-enhancing necrotic fluid representing abscess loculations are very valuable to put a plan for

treatment **[28]**. In and Out-of-phase sequence proved valuable to document benignity of marrow sub-endplate changes as marrow edema showed significant signal suppression on out of phase sequence usually more than the proposed cut off value (signal suppression more than 10%) **[29].** All patients with suspected spondylodiscitis should be evaluated by blood culture, yet only 25 to 70% of them are being positive **[30]**. Blood culture negative cases undergo percutaneous aspiration of the peri-vertebral tissue, in case of no indication for open biopsy (for example surgical decompression of the spinal cord) **[31]**. Respecting the pathophysiology of osteomyelitis (inflammation with superadded ischemia, necrosis and bone sequestration), so antimicrobials show limited penetration. Hence high doses of broad-spectrum antibiotics are recommended, initially empirical then changed according to anti-biogram results **[32]**. The recommended antibiotic course duration is from 4 to 6 weeks, and can be extended up to 8 weeks in cases of associated abscesses or bone necrosis **[33,34]**. Surgical management is recommended to debride soft tissues, to enhance tissue perfusion as well as to ensure spine stability **[35].**

COVID-19 infection was a possible predisposing factor that raised the risk for Spondylodiscitis in many studies **[26,34-38]**, respecting the temporal relation between both infections, however at our study COVID-19 vaccination was documented in the larger proportion of the patients with a better temporal relationship between vaccination and spondylodiscitis than COVID-19 infection itself. Also, it was noticed that the number of cases suffering from spondylodiscitis raised significantly following implementation of vaccination strategies. Adverse reactions have occasionally been reported for all types of COVID-19 vaccines, especially thromboembolic adverse effects **[39,40]** as well as inflammatory rheumatic diseases **[41].** The causative relationship between COVID-19 infection and spondylodiscitis is less understood. They supposed linkage between COVID-19 and spondylodiscitis due to disseminated bacterial or other hospital related infections during COVID-19 as a cause of spondylodiscitis. Viral infection induced immunosuppression, corticosteroid therapy or immunomodulator agents in general increasing the susceptibility for infections and associated increased intrathoracic tension during management of respiratory dysfunction **[42]**. But different studies reported maximum duration between COVID-19 infection and spondylodiscitis of about 4 to 6 weeks.

In our study development of spondylodiscitis was much remote than COVID-19 infection, yet shortly after completing vaccination doses for COVID-19. Exclusion of any other predisposing factors including immune compromised state, diabetes mellitus, chronic granulomatous infections, local factors predisposing for development of spondylodiscitis such as previous operative interference at or nearby spondylodiscitis level, injectable drug addiction, sources of bacteremia such as rheumatic heart disease, bacterial endocarditis and even malnutrition form patients suffering from spondylodiscitis, puts COVID-19 vaccine into consideration as a predisposing factor specially when documenting obvious overall increase in number of cases suffering from spondylodiscitis referred to our university hospitals and needs further research work up. Post vaccine infections in general can be explained by IgG4 and IgG2 class switches. Increased IgG4 levels after booster vaccination is associated with an increased risk of infections. Also, the increased non-cytophilic to cytophilic antibody ratio correlates with reduced functionality **[43]**. The underlying mechanism is still not clear but needs further research.

Conclusion: Spondylodiscitis is rare disease presenting initially by non-specific symptoms, MRI is considered most diagnostic sensitive and specific method. COVID-19 infection is a culprit predisposing factor for spondylodiscitis in many studies, yet in our study much shorter temporal relationship between COVID-19 vaccination and development of spondylodiscitis was noted and needs further research work up.

References

1- TALAMONTI G., COLISTRA D., CRISÀ F., CENZATO M., GIORGI P. and D'ALIBERTI G.: Spinal epidural abscess in COVID-19 patients. J. Neurol., 268: 2320-6, 2021.

2- CHOUDHURY I., HAN H., MANTHANI K., GANDHI S. and DABHI R.: COVID-19 as a possible cause of functional exhaustion of CD4 and CD8 T-cells and persistent cause of methicillin-sensitive Staphylococcus aureus bacteremia. Cureus, 12: e9000, 2020.

3- ZHANG Y., GENG X., TAN Y., et al.: New understanding of the damage of SARS-CoV-2 infection outside the respiratory system. Biomed Pharmacother., 127: 110195, 2020.

4- SOH P., DOAN N., MANNING B. and DOAN H.: Spinal cord injury from an epidural abscess as a serious complication of COVID-19 infection. Cureus, 12: e11327, 2020.

5- VAKILI M. and CRUM-CIANFONE N.F.: Spinal epidural abscess: A series of 101 cases. Am. J. Med., 130: 1458–1463, 2017.

6- CARSANA L., SONZOGNI A., NASR A., et al.: Pulmonary postmortem findings in a series of COVID-19 cases from northern Italy: A two-centre descriptive study. Lancet Infect Dis., 2020.

7- ZHANG Y., GENG X., TAN Y., LI Q., XU C., et al.: New understanding of the damage of SARS-CoV-2 infection outside the respiratory system. Biomed Pharmacother., 127: 110195, 2020.

8- LODIGIANI C., IAPICHINO G., CARENZO L., CECCONI M., et al.: Humanitas COVID-19 Task Force. Venous and arterial thromboembolic complications in COVID-19

patients admitted to an academic hospital in Milan, Italy. Thromb. Res., 191: 9-14, 2020.

9- World Health Organization. Clinical Management of COVID-19: Living Guideline, 15 September Geneva: World Health Organization, 2022 (WHO/2019-nCoV/Clinical/2022.2).

10- USTENKO I.I., KUSHNIR Y.B., AMELIN A.V., GOTOVCHIKOV A.A., GORANCHUK D.V., et al.: Ase reports: Spondylodiscitis and epiduritis after suffering COVID-19. Journal of Clinical Practice, 13: 107–117, 2022.

11- SAI KIRAN N.A., VAISHYA S., KALE S.S., SHARMA B.S. and MAHAPATRA A.K.: Surgical results in patients with tuberculosis of the spine and severe lower-extremity motor deficits: A retrospective study of 48 patients. J. Neurosurg. Spine, 6: 320-326, 2007.

12- LENER S., HARTMANN S., BARBAGALLO G.M.V., et al.: Management of spinal infection: A review of the literature. Acta. Neurochir., 160: 487–496, 2018.

13- MAVROGENIS A.F., MEGALOIKONOMOS P.D., IGOUMENOU V.G., PANAGOPOULOS G.N., GIANNITSIOTI E., PAPADOPOULOS A. and PAPAGELOPOULOS P.J.: Spondylodiscitis revisited. EFORT Open Rev., 2: 447-461, 2017.

14- HERREN C., JUNG N., PISHNAMAZ M., BREUNINGER M., SIEWE J., et al.: Spondylodiscitis: Diagnosis and Treatment Options. Dtsch Arztebl Int., 114: 875-882, 2017.

15- SAI KIRAN N.A., VAISHYA S., KALE S.S., SHARMA B.S. and MAHAPATRA A.K.: Surgical results in patients with tuberculosis of the spine and severe lower-extremity motor deficits: A retrospective study of 48 patients. J. Neurosurg Spine, 6: 320-326, 2007.

16- PETKOVA A.S., ZHELYAZKOV C.B. and KITOV B.D.: Spontaneous spondylodiscitis - epidemiology, clinical features, diagnosis and treatment. Folia Medica., 59: 254-260, 2017.

17- PJSKIĆ M., CARL B., SCHMÖCKEL V., VÖLLGER B., NIMSKY C. and SAß B.: Neurosurgical Management and Outcome Parameters in 237 Patients with Spondylodiscitis. Brain Sciences, 11: 1019, 2021.

18- HOMAGK L., MARMELSTEIN D., HOMAGK N. and HOFMANN G.O.: Spon DT (Spondylodiscitis Diagnosis and Treatment): Spondylodiscitis scoring system. J. Orthop. Surg. Res., 14: 100, 2019.

19- TSANTES A.G, PAPADOPOULOS D.V., VRIONI G., SIOUTIS S., SAPKAS G., BENZAKOUR A., et al.: Mavrogenis AF on behalf of the World Association against Infection in Orthopedics and Trauma (W.A.I.O.T.) Study Group on Bone and Joint Infection Definitions. Spinal Infections: An Update. Microorganisms., 8: 476, 2020.

20- BAŞAK A.T., ÇAKıCı N. and ÖZBEK M.: A Combined Diagnosis and Treatment Algorithm for Spine Infection Management: A Single-Center Experience. Cureus., 14: e28251, 2022.

21- SUFWAN ELBASHER ALSADIQ ALGRMI MAHMOUD MUSTAFA MOHAMED TAHA, IBRAHIM METWALY ABDEL FATTAH and ESSAM MOHAMED ELSAYED YOUSSEF: Management of Patients with Spondylodiscitis: An Overview. European Journal of Molecular & Clinical Medicine, 8: 3023-3034, 2021.

22- LAZZERI E., BOZZAO A., CATALDO M.A., PETROSILLO N., MANFRÈ L., TRAMPUZ A., SIGNORE A. and MUTO M.: Joint EANM/ESNR and ESCMID-endorsed consensus document for the diagnosis of spine infection (spondylodiscitis) in adults. Eur. J. Nucl. Med., 46: 2464–2487, 2019.

23- SHARFMAN Z.T., GELFAND Y., SHAH P., HOLTZMAN A.J., MENDELIS J.R., KINON M.D., KRYSTAL J.D., BROOK A., YASSARI R. and KRAMER D.C.: Spinal Epidural Abscess: A Review of Presentation, Management, and Medicolegal Implications. Asian Spine J., 14: 742–759, 2020.

24- KOLINSKY D.C. and LIANG S.Y.: Musculoskeletal Infections in the Emergency Department. Emerg. Med. Clin. North Am., 36: 751–766, 2018.

25- CARFì A., BERNABEI R., LANDI F., et al.: Persistent symptoms in patients after acute COVID-19. JAMA, 324: 603-605, 2020. Available in: https://doi.org.10.1001/jama.2020.12603.

26- NADERI S., OSTADRAHIMI N., SHEIKHREZAEI A.R., et al.: COVID-19 Infection Leading to Lethal Spondylodiscitis with Spinal Abscess. Iran J. Neurosurg., 6: 151-154, 2020. Available in: https:/irjns.org/article-1-219-en.html

27- MAHMOUD I., AOUINI M., BEN TEKAYA A., et al.: AB1220 Characteristics of infectious spondylodiscitis in elderly. Annals of the Rheumatic Diseases, 81: 1723, 2022.

28- MOUNA G., KHALIFA D., SAGHNI H., et al.: AB1177 spinal epidural abscess associated with infectious spondylodiscitis. Annals of the Rheumatic Diseases, 82: 1818, 2023. Available in: https://doi.org/10.1007/ s00402-009-0928-3.

29- DALIA Z. ZIDAN, LOBNA A. HABIB and NEVINE A.: Chalabi Quantitative chemical-shift MR imaging cutoff value: Benign versus malignant vertebral compression – Initial experience.The Egyptian Journal of Radiology and Nuclear Medicine, 45: 779-786, 2014.

30- BOUDABBOUS S., NICODÈME P.E., DELATTRE B.M. A., et al.: Spinal disorders mimicking infection, 12: 176, 2021.

31- CZUCZMAN G.J., MARRERO D.E., HUANG A.J., et al.: Diagnostic yield of repeat CT-guided biopsy for suspected infectious spondylodiscitis. Skeletal Radiol., 47: 1403-1410, 2018. Available in: https://doi.org/10.1007/s00256-018-2972-y.

32- ALINE G., DESVAUX M., DELEPINE C., et al.: POS1186 Study of spondylodiscitis without bacteriological documentation from a cohort of 142 patients with suspected infectious spondylodiscitis on imaging. Annals of the Rheumatic Diseases, 81: 920-921, 2022. Available in: http://doi.org/10.1136/annrheumdis-2022-eular.365.

33- SUJAL RAJ, VISHNU L.A.L., ARJUN KUMAR TULI, et al.: Management and outcome of infectious spondyl-

odiscitis in patients with and without neurological deficit: A prospective study, 18, 2023. Available in: DOI. 10.37532/1897- 2276.2022.18(2).81.

34- TALAMONTI G., COLISTRA D., CRISÀ F., et al.: Spinal epidural abscess in COVID-19 patients. J. Neurol., 268: 2320-2326, 2021. Available in: https://doi.org/10.1007/s00415- 020-10211-z.

35- KHALIL M.O., AYASA L.A., ODEH A., et al.: Unveiling the culprit: Candida-induced spondylodiscitis following SARSCoV-2 infection. Cureus., 15: e42079, 2023. Available in: https://doi.org.107759/cureus.42079.

36- NADERI S., OSTADRAHIMI N., SHEIKHREZAEI A.R., HÄNGGI D. and MUHAMMAD S.: COVID-19 Infection Leading to Lethal Spondylodiscitis with Spinal Abscess. Iran J. Neurosurg., 6 (3): 151-154, 2020.

37- MOHAMED RAMLEE F., HARUN M., NAGARETNAM V., et al.: A Case Series of Spinal Infections Following COVID-19: A Delayed Complication. Cureus, September 17; 14 (9): e29272, 2022. doi:10.7759/cureus.29272.

38- POPOVICI G.-C., GEORGESCU C.-V., ARBUNE A.-A., VASILE M.-C., OLTEANU I. and ARBUNE M.: Post-COVID-19 Spondylodiscitis: A Case Study and Review of the Literature. Medicina, 59: 616, 2023. HYPERLINK "https://doi.org/10.3390/medicina59030616" https://doi.org/10.3390/medicina59030616.

39- MITCHELL O.R., DAVE R., BEKKER J. and BRENNAN P.A.: Supraclavicular lymphadenopathy following COVID-19 vaccination: an increasing presentation to the two-week wait neck lump clinic?. Br. J. Oral Maxillofac. Surg., 59: pp. 384-385, 2021.

40- GOSS A.L., SAMUDRALWAR R.D., DAS R.R. and NATH A.: ANA investigates: Neurological complications of COVID-19 vaccines. Ann. Neurol., 89: pp. 856-857, 2021.

41- AKKUZU G., BES C., ÖZGÜR D.S., KARAALIOĞLU B., MUTLU M.Y., YıLDıRıM F., ATAGÜNDÜZ P., GÜNDÜZ A. and SOY M.: Inflammatory rheumatic diseases developed after COVID-19 vaccination: Presentation of a case series and review of the literature. Eur. Rev. Med. Pharmacol. Sci., 27 (5): 2143-2151, 2023.

42- SCALIA G., UMANA G.E., MARRONE S., GRAZIANO F., GIUFFRIDA A., PONZO G., GIUFFRIDA M., FURNARI M., GALVANO G., NICOLETTI G.F., et al.: Spontaneous anterior cervicothoracic spinal epidural hematoma extending to clivus in SARS-CoV-2 infection. Surg. Neurol. Int., 12: 181, 2021. doi: 10.25259/SNI_40_2021.

43- MARTÍN PÉREZ, et al.: Post-vaccination IgG4 and IgG2 class switch associates with increased risk of SARS-CoV-2 infections. Journal of infection, Volume 90. Issue 4, 106473, 2025.

العلاقة المحتملة بين الإصابة بفيروس كورونا المستجد أو تلقى التطعيم وحدوث التهابات تلقائية بالغضروف الفقارى

يعتبر فيروس كورونا المستجد هو احدى جائحات العصر الحديث الذى يصيب الجهاز التنفسى مسبباً قصور فى وظائف التنفس.

فى هذه الدراسة تمت مناقشة العلاقة المحتملة بين الاصابة بفيروس الكورونا المستجد او تلقى التطعيم وحدوث الالتهابات التلقائية لغضاريف العمود الفقرى نظرا لنقص مناعة الجسم وحدوث خلل فى بعض وظائف الاعضاء المختلفة.

تم اخذ بيانات من المرضى حول اصابتهم بالفيروس او تلقيهم التطعيم. وتم التعامل مع المرضى دوائياً بمضادات الفيروسات والمضادات الحيوية او جراحياً عن طريق تفريغ الخراج الناتج عن الالتهابات داخل الغضروف ومتابعة دلالات الالتهابات بعد العلاج الدوائي او الجراحى.

تبين بالنتائج ان هناك علاقة بين الاصابة بفيروس الكورونا المستجد او تلقى التطعيم وزيادة نسبة حدوث الاصابة بالتهابات الغضاريف بعدها وتمت مراجعة النتائج مع باقى المنشورات العلمية لتأكيد نتائج البحث.