COVID-19 Related Spinal Subdural Hematoma Presented with Acute Compressive Myelopathy

Hasanain A. Al-Khalidi[1], Hayder K Hassoun[1], Zahra Aljid[1], Zuhair Allebban[2]

[1]Faculty of Medicine–Kufa University, Middle Euphrates Neurosciences Center, Al-Najaf Al-Ashraf, Iraq.

[2]Faculty of Medicine-Kufa University, Middle Euphrates Unit for Cancer Research, Al-Najaf Al-Ashraf, Iraq.

[1]Hasanain A. Al-Khalidi, MD, PhD, Clinical Neurologist
E-mail: hasanaina.alkhalidi@uokufa.edu.iq

[1]Hayder K Hassoun, MD, PhD, Clinical Neurologist
E-mail: drhayder67@hotmail.com

[1]Zahra Aljid, MD, PhD, Clinical Radiologist
E-mail: zhraaaayad313@gmail.com

[2]Zuhair Allebban, BVMS, MS, PhD, Clinical Pathology

E-mail: zuhair.allabban@uokufa.edu.iq

Address correspondence to:
Zuhair Allebban, BVMS, MS, PhD, MB(ASCP), M(ASCP)
Professor of Pathology
Middle Euphrates Unit for Cancer Research
Faculty of Medicine, Kufa University, Najaf, Iraq
Phone: 009647804272223
E-mail: zuhair.allabban@uokufa.edu.iq
ORCID ID: https://orcid.org/0000-0001-5150-0097

Abstract

Spinal subdural hematoma (SSDH) was rarely reported in association with COVID-19. A fifty-five-year-old febrile male was presented with sudden onset of areflexic paraparesis, urinary retention, loss of all sensations below the twelve spinal thoracic segment, and severe back pain. COVID-19 was confirmed by positive RT-PCR and spinal MRI showed SSDH.

KEYWORDS: Spinal subdural hematoma, COVID-19, Acute paraparesis.

KEY CLINICAL MESSAGE:

Neurological complication due to COVID-19 is accumulating and compressive myelopathy due to SSDH is rarely reported in association with COVID-19, which necessitate urgent spinal cord MRI followed by an urgent surgery to save the spinal cord.

INTRODUCTION:

A novel case of severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) also known as COVID-19 was first reported in Wuhan, China in December 2019 and was rapidly spread throughout the world as a pandemic[1]. Since COVID-19 causes mild to severe acute respiratory syndrome[2], most studies in this field have only focused on different aspects of pathogenesis in the respiratory system. However, evidence suggests that COVID-19 may also affect the central nervous system (CNS).

In recent COVID-19 study, concerns have been raised that this virus may be neuroinvasive. Neurologic manifestations in up to 84% of hospitalized patients have been described in series from China[3]. Neurological symptoms were reported in 37% (50% in severe infection) of 214 COVID-19 patients reported from Wuhan, including nonspecific symptoms of headache and dizziness (20% of cases), altered mentation (15% of severe infection), sever muscle ache and myalgia (20% of cases) and loss of taste and smell sense, which may give initial clue to COVID-19 infection[3]. Symptoms related to olfactory dysfunction have been interpreted as evidence of CNS involvement and showed olfactory neuropathy within 3 weeks from the onset of illness[3,4].

COVID-19 uses the spike protein to attach to the host angiotensin-converting enzyme 2 (ACE-2) receptor and enter the target cells. These receptors are expressed in various tissues such as the respiratory epithelial cells, glial cells, neurons, lung, kidney, intestine, brain and vascular

endothelium[5]. In the brain, ACE-2 is also expressed in brainstem nuclei, and plays a role in cardio-respiration regulation making them a potential entry point for the virus[6,7]. This virus caused multi-organ dysfunction with median incubation period within 4-5 days and about 97.5% of patients will develop symptoms within 11 days[8].

The world is still dealing with new COVID-19 cases which caused the death of more than one million individuals worldwide including 9948 died in Iraq[9]. Many COVID-19 cases demonstrated an increased risk of thrombotic complications due to hyper-coagulability state and inflammation, resulting in arterial and venous thrombosis[10,11].

Iraq is still facing a rapid increase in new COVID-19 cases and this is possibly due to the deterioration in the health system with ineffective public awareness and prevention measures[12,13].

The array of symptoms of infection with COVID-19 is dependent on the patient's age and underlying medical illness, and also on the condition of the immune system of the infected individual[14].

In general, SSDH is a rare condition that may lead to a significant neurologic deficit. Usually, the risk factors for SSDH include anticoagulants drugs, coagulopathy, vascular malformations, infection of the spine, and iatrogenic cause[15]. Moreover, in some cases, no identified risk factor, so it is named "idiopathic" SSDH[16], and this is considered very rare. Most of the SSDH presented at the age of 45-60 years old and were mostly located in the thoracic region[17].

In this report, we present a COVID-19 related spinal subdural hematoma with acute compressive myelopathy which is rarely reported in COVID-19 infections.

CASE PRESENTATION

A 55-year old, Iraqi pediatrician male patient presented to the Middle Euphrates Neurosciences Center in Al-Najaf City in Iraq with sudden onset of bilateral lower limb weakness, urinary retention with severe lower back pain two hours before admission. He had a history of hypertension and diabetes mellitus with antihypertensive and oral hypoglycemic medication. He had a one-week history of low-grade fever following contact with a COVID-19 patient in his family, he had no history of trauma, no history of bleeding diathesis, and not on anticoagulation drugs. The vital signs on admission demonstrated a blood pressure 150/90 mmHg, heart rate

90beat/minute and regular, body temperature 37.8ºC, respiratory rate 18 breath/minute and SPO_2 was 97 % on room oxygen. On neurological examination, the patient was awake, oriented, GCS was 15, normal cranial nerves, no meningeal signs. Upper limbs had normal tone, power, reflexes, and sensation. He has flaccid areflexic paralysis of the lower limbs (power grade 0/5), with a total loss of all sensory modalities below the 12th spinal thoracic segment (T12) and bilateral negative Babinski sign. All Cardiac, abdominal, and locomotor system examinations were normal with mild crackles at the lung bases.

After admission, he was subjected to blood tests, chest CT and spinal MRI. His bladder was evacuated by folly's catheter. The results of the blood test demonstrated classical blood film findings (mainly lymphopenia) in COVID-19 patients, as shown in Table 1, with high readings of serum ferritin, CRP, and D-Dimer usually were seen in the active stage of infection.

A Chest CT scan of the patient showed multiple bilateral peripheral ground-glass opacities of the lungs as shown in Figure 1, which is highly suggestive of COVID-19 infection.

MRI scan for the dorsal and lumbosacral spine with pre- and post-contrast read by radiology specialist showed a long extra-dural lesion at level D12 - D11 - D10 - D9 (hyperintense on T1 and T2) with homogenous post-contrast enhancement, and diagnosed as SSDH. The lesion caused a significant pressure on the spinal cord as illustrated in Figure 2.

Real-time reverse-transcription-polymerase chain reaction (RT-PCR) test using nasopharyngeal swab was positive for COVID-19. Immediately after admission, we administered steroids in the form of methylprednisolone intravenous infusion, proton pump inhibitor, fluid therapy with pain killer drugs in the form of paracetamol injection. On the 2nd day after admission, the patient was transferred to the operation room and three stages of hematoma evacuation were performed by the neurosurgeon with the restoration of spinal cord pulsation. Three weeks later, the sensation and power of the lower limbs improved (power grade 3). Histopathology examination and sensitivity test of the obtained sample revealed Acinetobacter microorganism and was sensitive to cefepime antibiotic, which was administered immediately.

DISCUSSION

This is one of the rare cases reported globally, SSDH is a very rare presentation related to COVID-19 infection that warrants great concern because it results in spinal cord compression with severe neurological deficit. As reported in the literature, there is a wide range of neurological symptoms in COVID-19 cases, extending from specific symptoms (loss of smell or taste, myopathy, total paralysis, and stroke)[18] to more nonspecific symptoms (headache, decreased level of consciousness, vertigo, or seizure). Cerebrovascular accidents are reported more in diabetic and hypertensive patients. Other reported neurological complications including encephalitis, viral meningitis, post-infectious brainstem encephalitis, myositis, postinfections acute disseminated encephalomyelitis, Gillian Barre syndrome, and cerebrovascular accident[19]. One of the rare cases of acute necrotizing encephalitis was also reported in 59-year old patient with COVID-19 presented with fever, dry cough and change in mentality[20]. Recently, the association between COVID-19 and coagulopathy has gained an increased interest especially for severe cases and its association with the fulminant activation of coagulation and consumption of coagulation factors[12, 21].

The symptoms caused by SSDH vary greatly from only back pain to complete paralysis[22]. The cause of SSDH includes trauma, coagulopathy, vascular lesions, malignancy, and idiopathic. A high degree of clinical suspicion is very important for early diagnosis especially in patients with unknown causes for sudden severe back pain and neurologic deficit. In our case presentation with the presence of urinary retention, sensory level, and sudden onset, raised high clinical suspicion of acute vascular myelopathy. Moreover, the loss of all modality of sensation with the presence of severe back pain makes ischemic myelopathy unlikely, that led to an urgent MRI-dorsal spine that was done immediately. SSDH was diagnosed as shown in figure 2 and surgery was arranged quickly within 12 hours of admission. Per operatively, the hematoma was evacuated and sent for culture and histopathology which indicated the presence of SSDH. In subsequent days the patient sensory level descends with improvement in the power 7 days post-operation reaching grade 3 with the help of physiotherapy.

CONCLUSIONS

Spinal subdural hematoma should be suspected in any patient presented with acute onset of severe back pain and myelopathy without a history of trauma. Coagulopathy associated with

COVID-19 infection should increase the suspicion for SSDH which needs immediate surgical treatment to save the spinal cord and prevent a devastating neurological sequel.

ACKNOWLEDGMENT

None

CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

AUTHOR CONTRIBUTIONS

HAA: performed the clinical evaluation and data collection and revised the final draft of the manuscript. **HKH**: Supervised and performed the clinical evaluation and data collection, drafted the initial version of the manuscript and revised the final draft of the manuscript. **ZA**: performed the CT scan and MRI and interpreted the data and revised the final draft of the manuscript. **ZuA**: performed the laboratory testing, collected the laboratory data, reviewed the literature and revised the final draft of the manuscript.

ETHICAL APPROVAL

An informed consent from the patient, including the approval to publish this case report and any accompanying images were obtained. Additionally, there is no information revealing the patient' identity. An approval letter from the Institutional Review Board at Kufa University were obtained.

FUNDING

No funding was received for this study

ORCID ID

Zuhair Allebban: https://orcid.org/0000-0001-5150-0097

REFERENCES

1. Lam TT-Y, Shum MH-H, Zhu H-C, Tong Y-G, Ni X-B, Liao Y-S et al. Identification of 2019-nCoV related coronaviruses in Malayan pangolins in southern China. Nature 2020; 583:282–285.

2. Fahad AM, Mohammad AA, Al−Khalidi HA, Alshewered AS. Spontaneous pneumothorax as a complication in COVID−19 male patient: A case report. Clin. Case Rep. 2020; 00: 1–4.

3. Mao L, Jin H, Wang M, Hu Y, Chen S, He Q, Chang J, Hong C, Zhou Y, Wang D, Miao X, Li Y, Hu B. Neurologic Manifestations of Hospitalized Patients with Coronavirus Disease 2019 in Wuhan, China:*JAMA Neurol*. 2020; 77(6):683-690.

4. Eliezer M, Hautefort C, Hamel AL, Verillaud B, Herman P, Houdart E, Eloit C. Sudden and complete olfactory loss function as a possible symptom of COVID-19. JAMA Otolaryngol. 2020; 11:806-810.

5. Khatoon F, Prasad K, Kumar V. Neurological manifestations of COVID-19: available evidences and a new paradigm J NeuroVirol. 2020; 26:619–630.

6. Morgello, S. Coronaviruses and the central nervous system. J NeuroVirol. 2020; 26:459–473.

7. Baig AM, Khaleeq A, Ali U, and Syeda H. Evidence of the COVID-19 virus targeting the CNS: tissue distribution, host-virus interaction, and proposed neurotropic mechanisms. ACS Chem Neurosci. 2020; 11(7):995-998.

8. Kevin J. Clerkin, Justin A. Fried, Jayant Raikhelkar, et al. COVID-19 and cardiovascular disease. Circulation. 2020; 141:1648-1655.

9. WHO, Director-General's opening remarks at the media briefing on COVID-19-23 July 2020? https://www.who.int/dg/speeches/detail/who-director-generals-opening-remarks-at-the-media-briefing-on-covid-19---23-July-2020.

10. Bikdeli B, Madhavan, MV, Jimenez D, et al. COVID-19 and thrombotic or thromboembolic disease: implications for prevention, antithrombotic therapy, and follow up. JACC. 2020; 75(23): 2950-2973.

11. Fahad AM, Mohammad AA, Al-Khalidi HA, Lazim QJ, Ibrahim F, Alshewered AS. Case Report: COVID-19 in a female patient who presented with acute lower limb ischemia. F1000Res. 2020; 9:778-783.

12. Alyssa M, Giada Z. COVID-19: What went wrong in Italy and Spain? Anadolu Agency, https://www.aa.com.tr/en/europe/covid-19-what-went-wrong-in-italy-and-spain/1797461(2020).

13. UNICEF Iraq. Covid-19 Situation Iraq.Reportno.4. June 2020. https://www.unicef.org/iraq/reports/unicef-iraq-covid-19-situation-0).

14. Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, Zhang L, Fan G, Xu J, Gu X, Cheng Z, Yu T, Xia J, Wei Y, Wu W, Xie X, Yin W, Li H, Liu M, Xiao Y, Gao H, Guo L, Xie J, Wang G, Jiang R, Gao Z, Jin Q, Wang J, Cao B. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 2020; 395(10223):497–506.

15. Jimbo H, Asamoto S, Mitsuyama T, Hatayama K, Iwasaki Y, Fukui Y. Spinal chronic subdural hematoma in association with anticoagulant therapy: a case report and literature review. Spine (Phila Pa 1976). 2006; 3: E184-E187.

16. Park J, Ahn R, Son D, Kang B, Yang D. Acute spinal subdural hematoma with hemiplegia after acupuncture: a case report and review of the literature. Spine J. 2013;13(10): e59–e63.

17. Michael Payer, Reto Agosti. Spontaneous acute spinal subdural hematoma: spontaneous recovery from severe paraparesis—case report and review. Acta Neurochir. 2020;152(11):1981–1984.

18. Li Z, Liu T, Yang N, Han D, Mi X , Li Y, Liu K, Vuylsteke A, Xiang H , Guo X. Neurological manifestations of patients with COVID-19: potential routes of SARS-CoV-2 neuroinvasion from the periphery to the brain. Frontiers of med. May 2020.DOI: 10.1007/s11684-020-0786-5 (in press).

19. Nath A. Neurologic complications of coronavirus Infections. Neurologic complications of coronavirus Infections; Neurology®. 2020; 94:809-810.

20. Poyiadji N, Shahin G, Noujaim D, Stone M, Patel S, Griffith B. COVID-19–associated Acute Hemorrhagic Necrotizing Encephalopathy: CT and MRI Features. Radiology. 2020; 296: E119–E120.

21. Bellosta R, Luzzani L, Piffaaretti G, et al. Acute limb ischemia in patient with COVID-19 pneumonia. J vascul surg. 2020; 395:1546.

22. Longatti PL, Freschi P, Moro M, Trincia G, Carteri A. Spontaneous spinal subdural hematoma. J Neurosurg Sci. 1994; 38:197-199.

Figure and Table legends

TABLE 1. Results of the laboratory blood test

FIGURE 1. CT scan of the chest (non-contrast-lung window). (A) sagittal, (B) axial, (C) coronal, showing multiple bilateral peripheral ground glass opacities involved (less than 25%) of lung volume, which is highly suggestive COVID-19 infection.

FIGURE 2. Multiple sections MRI scan for dorsal and lumbosacral spine. (A) T2 axial, (B) T1 axil pre-contrast, (C) sagittal T1 pre-contrast, (D) sagittal T1 post-contrast, (E) sagittal T2, showing a long extra-dural lesion at level D9, D10, D11, and D12 (hyperintense on T1 and T2) with homogenous post-contrast enhancement, and diagnosed as (SSDH). The lesion caused a significant pressure effect on the spinal cord.