The Journal of Spinal Cord Medicine

ISSN: (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/yscm20

Acute cerebellitis, transverse myelitis and polyradiculoneuritis related to post-COVID-19 infection

Ülkü Türk Börü, Cem Bölük, Cansu Köseoğlu Toksoy & Hayri Demirbaş

To cite this article: Ülkü Türk Börü, Cem Bölük, Cansu Köseoğlu Toksoy & Hayri Demirbaş (2022) Acute cerebellitis, transverse myelitis and polyradiculoneuritis related to post-COVID-19 infection, The Journal of Spinal Cord Medicine, 45:5, 765-768, DOI: 10.1080/10790268.2021.1969502

To link to this article: https://doi.org/10.1080/10790268.2021.1969502

Published online: 29 Sep 2022.

Case Report

Acute cerebellitis, transverse myelitis and polyradiculoneuritis related to post-COVID-19 infection

Ülkü Türk Börü [1], Cem Bölük [2], Cansu Köseoğlu Toksoy[1], Hayri Demirbaş [1]

[1]Department of Neurology, Afyonkarahisar University of Health Sciences, Afyonkarahisar, Turkey, [2]Department of Neurology, Cerrahpasa Faculty of Medicine, Istanbul University-Cerrahpasa, Istanbul, Turkey

Context: Guillain–Barré syndrome (GBS), acute cerebellitis and transverse myelitis are rare complications of COVID-19 infection separately. The combination of these three, however, has not yet been reported.
Findings: We present an atypical case (42-year-old man) that developed acute ascending flaccid paraparesis, ataxia and urinary retention two weeks after COVID-19 infection. Neurological examination revealed distal and proximal weakness (4/5) on lower extremities, decreased tendon reflexes, sixth cranial nerve palsy and dysmetria without sensory disturbance. His cranial MRI showed cerebellitis whereas the spinal MRI showed transverse myelitis at the T11/12 level. Albuminocytologic dissociation was present in the cerebrospinal fluid. The nerve conduction study was concordant with early findings of GBS. He recovered well after corticosteroid treatment without needing any immunotherapy. On day seven of hospitalization, the modified Rankin Scale score was 0.
Conclusion: COVID-19 infection may present with a combination of neurological manifestations such as cerebellitis, transverse myelitis and GBS. This patient presented significant functional recovery after treatment with corticosteroid without immunotherapy.

Keywords: COVID-19, Cerebellitis, Transverse myelitis, Guillain–Barré syndrome

Introduction

COVID-19 continues to affect people worldwide and is currently an insurmountable problem. Turkey reports about 30,000 new cases and 200 deaths daily at the time this report was drafted.[1] Although the main symptoms are fever and cough, the disease can course with acute respiratory distress syndrome, pulmonary embolism, cardiac injury, septic shock and renal failure.[2] Increasingly, reports describe many varying neurological manifestations such as stroke, encephalitis, Guillain–Barré syndrome (GBS), acute transverse myelitis, myositis and others.[3–7] However, the combination of peripheral nerve, spinal cord and cerebellar involvement has not yet been reported.

Here, we present a patient with acute cerebellitis, transverse myelitis and GBS related to COVID-19 infection.

Correspondence to: Cem Bölük, Istanbul University-Cerrahpasa, Cerrahpasa Faculty of Medicine, Department of Neurology and Clinical Neurophysiology, 34098 Fatih, Istanbul, Turkey. +905367172960. Email: cem_boluk@hotmail.com

Color versions of one or more of the figures in the article can be found online at www.tandfonline.com/yscm.

Case Report

A 42-year-old man was admitted to our outpatient clinic on 11.05.2020. His complaints were nausea, vomiting, diplopia, ataxia, headache, urinary retention, excessive sweating, weakness in the legs and inability to walk without help. He had a history of COVID-19 infection with myalgia, olfactory loss, fever (>38°C) and excessive sweating that started 13 days ago. His COVID-19 PCR test was positive. He was given oral favipiravir treatment (1200 mg/day) for five days and his presenting complaints resolved within nine days.

On 11.02.2020 he developed ascending symmetrical leg weakness. Confusion, diplopia and ataxia developed two days later. He was taken to our hospital due to worsening of symptoms. Neurological examination at arrival revealed lethargy, right sixth cranial nerve involvement, symmetrical distal and proximal weakness (4/5) of the lower extremities, mildly decreased deep tendon reflexes, dysmetria, orthostatic hypotension and urinary retention. No sensory deficit was present. His medical history was unremarkable.

DOI 10.1080/10790268.2021.1969502

Laboratory findings showed leucocytosis and lymphopenia without any serological findings. Anti NMO and Anti MOG antibodies were negative. Cerebrospinal fluid (CSF) appearance and opening pressure were normal. The analysis demonstrated elevated protein (53 mg/dl), normal glucose and lactate dehydrogenase, as well as negative cytology, Gram stain, and culture (albuminocytologic dissociation). Oligoclonal band testing was negative. Laboratory findings are shown in Table 1.

Brain MRI showed hyperintensities on T2-weighted flair sequences in the bilateral middle cerebellar peduncles without any gadolinium enhancement (Figure 1). These findings were concordant with cerebellitis. In addition, there was a short segment patchy transverse myelitis seen on spinal MRI at the T11/T12 level with mild diffuse gadolinium enhancement, appearing hypointense on T1-weighted series and hyperintense on T2-weighted series (Figure 2). The cervical spine MRI demonstrated the incidental finding of canal stenosis due to a disc bulging at the C5/6 level, inconsistent with clinical findings.

Table 1 Laboratory findings of the patient.

White blood cell, 10^3/μL	13.98 (4.8–10.8)
Lymphocyte, 10^3/μL	1.11 (1.0–4.8)
Neutrophil, 10^3/μL	12.14 (1.8–7.7)
Thrombocyte, 10^3/μL	313 (130–400)
Erythrocyte, 10^3/μL	6.62 (4.7–6.1)
Hemoglobin, g/dL	16.3 (14–18)
Hematocrit, %	48.9 (42–52)
Mean corpuscular volume, fL	73.9 (80–100)
AST, IU/L	12 (0–35)
ALT, IU/L	24 (0–45)
CK, IU/L	55 (0–170)
TSH, mU/L	3.72 (0.3–5.2)
Urea, mg/dL	46.8 (15–46)
Creatinine, mg/dL	0.89 (0.66–1.09)
CRP, mg/L	1.1 (0–3)
B-12, pg/mL	495 (250–1100)
Folate, ng/mL	8.56 (3–17)
CSF Findings	
Cell count, mm^3	3 (0–10)
Protein, mg/dl	53 (15–40)
Glucose, mg/dl	57.9 (40–70)
LDH	46 (10–45)
Cell culture	Negative

Nerve conduction studies on the third day of hospitalization revealed low amplitude sensory nerve action potentials (SNAP) in the upper extremity, prolonged F wave latencies and decreased F persistence suggested early findings of acute inflammatory demyelinating polyneuropathy (AIDP). Findings are summarized in Table 2.

On day two of hospitalization, he began to improve prior to initiation of treatment. Intravenous methylprednisolone was administered at a dosage of 500 mg daily for five days. Immunotherapy was not administered as the patient recovered rapidly. The neurologic examination was almost normal on the 7th day of hospitalization. No complication was seen related to corticosteroids.

Discussion

Our case showed a combination of post-COVID-19 infection-related cerebellitis, transverse myelitis and acute inflammatory demyelinating polyneuropathy. This case indicated that COVID-19 infection can result in both central and peripheral nervous system involvement at the same time. To our knowledge, this is the first report of simultaneous involvement of the brain, spinal cord and peripheral nervous system following COVID-19 infection, presumably on an autoimmune basis.

Various neurological manifestations have been reported since the very beginning of this pandemic.[8] Different variants of GBS including AIDP, acute motor axonal neuropathy (AMAN), acute motor sensory axonal neuropathy (AMSAN), and Miller Fisher syndrome (MFS) can be seen 1–4 weeks after the first COVID-19 symptoms. The demyelinating form is more prevalent and has a better prognosis than axonal forms as was the case for our patient.[9,10] We also have experience with two additional GBS cases with rapid recovery following COVID-19.[11]

COVID-19 associated acute transverse myelitis cases were also reported. The lesions were generally described

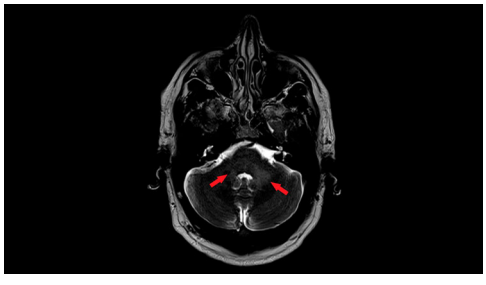

Figure 1 Hyperintensities on T2-weighted sequences in the bilateral middle cerebellar peduncles without any gadolinium enhancement (arrows).

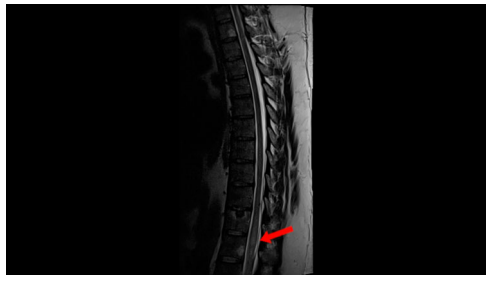

Figure 2 Short segment, patchy hyperintensities at the T11/T12 level on T2-weighted series (arrow).

Table 2 The nerve conduction study of patient.

	Latency, ms, (ref)	Amplitude, mV, (ref)	Conduction velocity, m/s, (ref)
Median motor, APB, R			
Wrist	3.41 (<4.4)	6.7 (>4)	
Elbow	8.00	6.3	49.1 (>49)
Ulnar motor, ADM, R			
Wrist	2.69(<3.3)	5.2 (>5)	
Elbow	5.67	4.3	55.2 (>49)
Above elbow	7.56	4.2	50.1 (>49)
Peroneal motor, EDB, R			
Ankle	4.10 (<6.5)	4.5 (>2)	
Fib. Head	10.5	3.7	46.9 (>44)
Above knee	12.3	3.1	48.0 (>44)
Tibial motor, AHB, R			
Ankle	4.15 (<5.8)	6.5 (>3)	
Knee	15.3	3.1	36.2 (>41)
	Latency, ms	Amplitude, μV	Conduction velocity, m/s
Median sensory, R			
2nd digit	4.27 (<3.5)	4.2 (>10)	38.1 (>50)
Ulnar sensory, R			
5th digit	5.63 (<3.1)	2.6 (>10)	34.7 (>50)
Sural sensory, R			
Lat malleolus	3.19 (<4.4)	15.9 (>6)	55.6 (>40)
F response			
Median, R	44.3/40% (<31)		
Tibial, R	68.1/30% (<56)		

as hyperintensities in T2-weighted images mostly at the thoracic level.[12, 13] In one case, myelitis was observed at the level of T7-T10 whereas another case had multiple lesions at the cervical and thoracic levels. The spinal involvement of our case is at the T11/T12 level. Acute hemorrhagic transverse myelitis is also described.[14] Regardless of etiology, the prognosis of transverse myelitis is variable. The known predictors of poor prognosis include spinal shock, back pain and rapid progression of symptoms.[15,16] However, the reported prognosis of COVID-19 related transverse myelitis was generally good unless hemorrhagic or other complications were seen.[13,14,17–19] COVID-19 related transverse myelitis cases are summarized in Table 3.

Table 3 Summary of reported COVID-19 related transverse myelitis cases.

	Age	Sex	Day after first COVID-19 symptoms	Segment	Findings	Treatment	Outcome
Chakraborty *et al.*[3]	59	F	4	T6–T7	Paraplegia (0/5), urinary retention	Steroid	Initially recovered, exitus then
AlKetbi *et al.*[9]	32	M	3	Multiple segments starting from C2	Paraplegia (0/5), urinary retention	Steroid	Slightly recovered
Chow *et al.*[10]	60	M	16	T7-T10	Paraparesis, hyperreflexia, reduced proprioception	Steroid	Rapid and almost complete recovery
Sotoca *et al.*[11]	69	F	7	C7-T1	Left hand paresis and hypoesthesia, hyperreflexia	Steroid and plasmapheresis	Slightly recovered
Sarma *et al.*[15]	28	F	9	Multiple segments starting from T5	Hypoesthesia, urinary retention, Lhermitte's sign	Steroid and plasmapheresis	Rapid and almost complete recovery
Zachariadis *et al.*[16]	63	M	12	T10	Paraparesis, hyperreflexia, hypoesthesia	IVIG and Steroid	Slightly recovered
Our case	42	M	10	T11–T12	Paraparesis, urinary retention, hyporeflexia	Steroid	Rapid and almost complete recovery

The combination of myelitis and GBS is a very rare condition. Recently a patient was reported who developed acute necrotizing myelitis and acute motor axonal polyneuropathy together one week after COVID-19 infection and he received plasmapheresis.[20]

On the other hand, cerebellar involvement is a known condition after viral infection such as influenza A.[21] However, up to now, only one case with an acute cerebellitis related to COVID-19 has been described with radiologic findings similar to our case.[22] As in that case, our patient also responded well to treatment and recovered very rapidly. The combined involvement of the brain, spinal cord and peripheral nerves two weeks after COVID-19 infection suggests an immune-mediated response.

In conclusion, the neurological manifestations of COVID-19 may vary widely and the combination of central and peripheral nervous system involvement may be seen at the same time. In our opinion, patients are responding well to treatments and the prognosis is generally good unless longitudinally extensive myelitis or hemorrhagic complications are present.

Disclaimer statements

Contributors None.

Funding None.

Conflicts of interest Authors have no conflict of interests to declare.

ORCID

Ülkü Türk Börü http://orcid.org/0000-0002-0094-5624
Cem Bölük http://orcid.org/0000-0003-0125-7660
Hayri Demirbaş http://orcid.org/0000-0002-8339-7765

References

1 Ministry of Health, Republic of Turkey (MoH-TR). COVID-19 web page of the Republic of Turkey, Ministry of Health. Available at: https://covid19.saglik.gov.tr [Internet].
2 Jiang F, Deng L, Zhang L, Cai Y, Cheung CW, Xia Z. Review of the clinical characteristics of coronavirus disease 2019 (COVID-19). J Gen Intern Med. 2020 May;35(5):1545–9. doi:10.1007/s11606-020-05762-w.
3 Ahmad I, Rathore FA. Neurological manifestations and complications of COVID-19: a literature review. J Clin Neurosci. 2020;77:8–12. doi:10.1016/j.jocn.2020.05.017.
4 Avula A, Nalleballe K, Narula N, Sapozhnikov S, Dandu V, Toom S, *et al.* COVID-19 presenting as stroke. Brain Behav Immun. 2020 Jul;87:115–19. doi:10.1016/j.bbi.2020.04.077.
5 Chakraborty U, Chandra A, Ray AK, Biswas P. COVID-19-associated acute transverse myelitis: a rare entity. BMJ Case Rep. 2020 Aug 25;13(8):e238668. doi:10.1136/bcr-2020-238668.
6 Sedaghat Z, Karimi N. Guillain Barre Syndrome associated with COVID-19 infection: a case report. J Clin Neurosci. 2020 Jun;76:233–5. doi:10.1016/j.jocn.2020.04.062.
7 Zhang H, Charmchi Z, Seidman RJ, Anziska Y, Velayudhan V, Perk J. COVID-19-associated myositis with severe proximal and bulbar weakness. Muscle Nerve 2020 Sep;62(3):E57–E60. doi:10.1002/mus.27003.
8 Mao L, Jin H, Wang M, *et al.* Neurologic manifestations of hospitalized patients with coronavirus disease 2019 in Wuhan, China [published online ahead of print, 2020 Apr 10]. JAMA Neurol. 2020;77(6):1–9. doi:10.1001/jamaneurol.2020.1127.
9 Bracaglia M, Naldi I, Govoni A, Brillanti Ventura D, De Massis P. Acute inflammatory demyelinating polyneuritis in association with an asymptomatic infection by SARS-CoV-2. J Neurol. 2020 Nov;267(11):3166–8. doi:10.1007/s00415-020-10014-2.
10 Rahimi K. Guillain-Barre syndrome during COVID-19 pandemic: an overview of the reports. Neurol Sci. 2020 Nov;41(11):3149–56. doi:10.1007/s10072-020-04693-y.
11 Türk Börü Ü, Köseoğlu Toksoy C, Bölük C, Demirbaş H, Yılmaz AÇ. A case of Guillain-Barré syndrome related to COVID-19 infection. Int J Neurosci 2021: 1–3. doi:10.1080/00207454.2021.1886097.
12 AlKetbi R, AlNuaimi D, AlMulla M, AlTalai N, Samir M, Kumar N, AlBastaki U. Acute myelitis as a neurological complication of Covid-19: a case report and MRI findings. Radiol Case Rep. 2020 Jun 6;15(9):1591–5. doi:10.1016/j.radcr.2020.06.001.
13 Chow CCN, Magnussen J, Ip J, Su Y. Acute transverse myelitis in COVID-19 infection. BMJ Case Rep 2020 Aug 11;13(8):e236720. doi:10.1136/bcr-2020-236720.
14 Sotoca J, Rodríguez-Álvarez Y. COVID-19-associated acute necrotizing myelitis. Neurol Neuroimmunol Neuroinflamm. 2020 Jun 10;7(5):e803. doi:10.1212/NXI.0000000000000803.
15 Kalita J, Misra UK, Mandal SK. Prognostic predictors of acute transverse myelitis. Acta Neurol Scand. 1998;98:60–3.
16 Ford B, Tampieri D, Francis G. Long-term follow-up of acute partial transverse myelopathy. Neurology 1992;42:250–2
17 Kaur H, Mason JA, Bajracharya M, McGee J, Gunderson MD, Hart BL, Dehority W, Link N, Moore B, Phillips JP, Rogers D. Transverse myelitis in a child With COVID-19. Pediatr Neurol. 2020 Nov;112:5–6. doi:10.1016/j.pediatrneurol.2020.07.017.
18 Sarma D, Bilello LA. A case report of acute transverse myelitis following novel coronavirus infection. Clin Pract Cases Emerg Med. 2020 Aug;4(3):321–3. doi:10.5811/cpcem.2020.5.47937.
19 Zachariadis A, Tulbu A, Strambo D, Dumoulin A, Di Virgilio G. Transverse myelitis related to COVID-19 infection. J Neurol. 2020 Dec;267(12):3459–61. doi:10.1007/s00415-020-09997-9.
20 Valiuddin H, Skwirsk B, Paz-Arabo P. Acute transverse myelitis associated with SARS-CoV-2: A Case-Report. Brain Behav Immun Health. 2020 May;5:100091. doi:10.1016/j.bbih.2020.100091.
21 Sfeir MM, Najem CE. Cerebellitis associated with influenza A (H1N1)pdm09, United States, 2013. Emerg Infect Dis 2014 Sep;20(9):1578–80. doi:10.3201/eid2009.140160.
22 Fadakar N, Ghaemmaghami S, Masoompour SM, Shirazi Yeganeh B, Akbari A, Hooshmandi S, Ostovan VR. A first case of acute cerebellitis associated with coronavirus disease (COVID-19): a case report and literature review. Cerebellum 2020 Dec;19(6):911–14. doi:10.1007/s12311-020-01177-9.