Turkish Journal of Neurology

Turk J Neurol 2024;30(1):62-64
Doi: 10.55697/tnd.2024.70

IMAGES IN CLINICAL NEUROLOGY

Acute transverse myelitis related to decompression sickness

Dekompresyon hastalığı ilişkili akut transvers miyelit

Selen Üçem[1], **Arda Duman**[2], **Gökhan Erkol**[2]

[1]Department of Neurology, Koç University, Koç University Hospital, Istanbul, Türkiye
[2]Department of Neurology, Koç University Hospital, Istanbul, Türkiye

A 51-year-old male patient presented with sensory changes in the left arm that slowly progressed to the left leg and right arm within 24 h. These were accompanied by Lhermitte's sign and an abdominal pain that extended to the back. The symptoms started 10 h after recreational scuba diving; hence, spinal cord decompression sickness (DCS) was added into differential diagnosis. The personal and family history were unremarkable. On neurological examination, there was mild pronator drift bilaterally, dysesthesia of right-sided extremities, Babinski reflex was noted bilaterally, and deep tendon reflexes were globally increased. The patient was admitted to our neurology ward for further investigation.

The laboratory results were unremarkable except for mild elevation of liver enzymes. The complement levels, anti-dsDNA (double-stranded deoxyribonucleic acid), anti-nuclear antibody, lupus anticoagulant, anticardiolipin, beta-2 glycoprotein, angiotensin-converting enzyme, and calcium levels were all in normal limits. Anti-MOG (myelin oligodendrocyte glycoprotein) and anti-aquaporin 4 antibodies were also negative. There was no recent history of vaccination, infection, or any recent or previous history of oral aphthous or genital ulcers. The cerebrospinal fluid examination revealed no erythrocytes, very few lymphocytes (<5), glucose of 70.5 mg/dL, and a total protein of 43.9 mg/dL. Cerebrospinal fluid oligoclonal bands, immunoglobulin G index, and bacterial studies were also negative. Magnetic resonance imaging (MRI) showed high signal intensity at T2-weighted images and mild edema on the cervical spinal cord extending from C2 to C5 (Figure 1a). Bilateral heterogeneous posterior cord enhancement was observed after intravenous contrast administration (Figure 1b). The MRI of the brain was normal.

Subacute sensory dysfunction and mild motor weakness of upper extremities with edema and gadolinium enhancement on MRI that correlated with the level of the symptoms, combined with the exclusion of etiologies, fit the diagnosis of idiopathic transverse myelitis (ITM). However, with the symptoms starting hours after a diving incident, a question of spinal cord DCS that could trigger the myelitis attack remained.

Diagnosis of ITM includes bilateral symptoms of sensory (with clearly defined sensory level), motor, or autonomic dysfunction that progresses to nadir between 4 h and 21 days, with evidence of inflammation of the spinal cord by neuroimaging or lumbar puncture. It also requires the exclusion of radiation exposure, ischemic etiology, compressive lesion, any bacterial, parasitic, or viral cause, evidence of connective tissue disease, or diagnosis of other demyelinating disorder.[1] Hence, ITM is a diagnosis of exclusion, and it was recently discovered that 70% of patients with suspected diagnosis of ITM were found to have an alternative diagnosis for their myelopathies.[2] Fifteen to 30%

Correspondence: Selen Üçem, MD. Koç Üniversitesi, Koç Üniversitesi Hastanesi, Nöroloji Anabilim Dalı, 34010 Zeytinburnu, İstanbul, Türkiye.
E-mail: selen.ucem@gmail.com
Received: January 18, 2023 **Accepted:** April 24, 2023 **Published online:** March 26, 2024
Cite this article as: Üçem S, Duman A, Erkol G. Acute transverse myelitis related to decompression sickness. Turk J Neurol 2024;30(1):62-64. doi: 10.55697/tnd.2024.70.

of transverse myelitis cases are ultimately defined as idiopathic,[3] and with the discovery of new antibodies and better radiological accuracy, specific causes for patients' previously classified ITM have started to be identified[4]

Type 1 DCS is usually mild and occurs in skin, joints, and muscles, while type 2 may be severe and involve the central nervous system[5] Although the exact mechanism of how nitrogen bubbles cause brain and spinal cord damage is not fully determined, the nerves can be damaged by mechanical disruption, compression, vascular stenosis or obstruction, and activation of inflammatory pathways, leading to myelopathy.[6] In spinal cord DCS, the MRI may be normal, or it might show findings of necrosis and edema; however, it typically would not reveal any contrast enhancement.[7] We excluded the diagnosis of pure spinal cord DCS-related myelopathy due to the MRI findings and the onset of the patient's symptoms, as the symptoms of DCS normally appear within 6 h after surfacing in 99% of the cases.[8] After five days of 1 g/day pulse methylprednisolone treatment, there was complete remission of the symptoms and the radiographic lesions (Figure 1c, d), confirming the diagnosis of acute transverse myelitis with the etiology of DCS as a trigger.

In conclusion, the outcome of this case shows that the potential etiologies of myelitis should be in the differential diagnosis of ITM as the term idiopathic slowly loses its meaning with new advances in imaging techniques, autoimmune neurology, and our understanding of the etiologies of various neurological disorders.

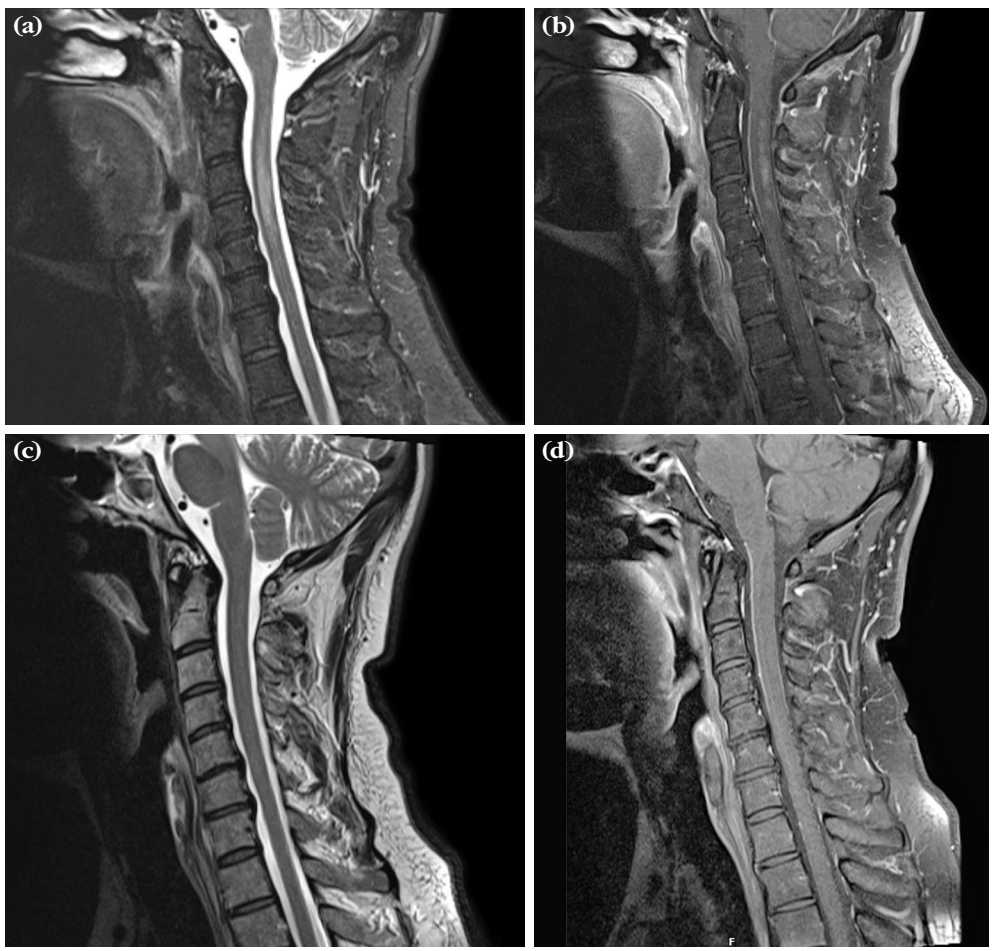

Figure 1. T2-weighted magnetic resonance imaging assessment of the cervical spinal with intravenous contrast administration before and after intravenous methylprednisolone treatment. **(a)** shows increased signal intensity and mild edema on the cervical spinal cord extending from C2 to C5. **(b)** demonstrates patchy contrast enhancement of posterior spinal cord. **(c, d)** show resolution of the lesions after treatment.

Patient Consent for Publication: A written informed consent was obtained from the patient.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reason able request.

Author Contributions: Was the lead clinician responsible for the patient's care, and supervised the project and is responsible from the integrity of the project: G.E.; Were also responsible from the patient's care, and wrote the majortiy of the manuscript: A.D., S.Ü.

Conflict of Interest: The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding: The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Transverse Myelitis Consortium Working Group. Proposed diagnostic criteria and nosology of acute transverse myelitis. Neurology 2002;59:499-505. doi: 10.1212/wnl.59.4.499.
2. Zalewski NL, Flanagan EP, Keegan BM. Evaluation of idiopathic transverse myelitis revealing specific myelopathy diagnoses. Neurology 2018;90:e96-102. doi: 10.1212/WNL.0000000000004796.
3. de Seze J, Stojkovic T, Breteau G, Lucas C, Michon-Pasturel U, Gauvrit JY, et al. Acute myelopathies: Clinical, laboratory and outcome profiles in 79 cases. Brain 2001;124:1509-21. doi: 10.1093/brain/124.8.1509.
4. Lopez Chiriboga S, Flanagan EP. Myelitis and other autoimmune myelopathies. Continuum (Minneap Minn) 2021;27:62-92. doi: 10.1212/CON.0000000000000900.
5. Melamed Y, Shupak A, Bitterman H. Medical problems associated with underwater diving. N Engl J Med 1992;326:30-5. doi: 10.1056/NEJM199201023260105.
6. Newton HB. Neurologic complications of scuba diving. Am Fam Physician 2001;63:2211-8.
7. Kamtchum Tatuene J, Pignel R, Pollak P, Lovblad KO, Kleinschmidt A, Vargas MI. Neuroimaging of diving-related decompression illness: Current knowledge and perspectives. AJNR Am J Neuroradiol 2014;35:2039-44. doi: 10.3174/ajnr.A4005.
8. Xu W, Liu W, Huang G, Zou Z, Cai Z, Xu W. Decompression illness: Clinical aspects of 5278 consecutive cases treated in a single hyperbaric unit. PLoS One 2012;7:e50079. doi: 10.1371/journal.pone.0050079.