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Case Report

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Supernumerary phantom limbs in a patient with neuromyelitis optica spectrum disorder and rehabilitation challenges: A case report

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Abstract

Supernumerary phantom limb (SPL) is a rare and complex neurological phenomenon characterized by the illusory perception of additional limbs without actual anatomical loss. While typically linked to central nervous system disorders such as stroke, spinal cord injury, and demyelinating diseases, its association with neuromyelitis optica spectrum disorder (NMOSD) has not been well-documented. A 61-year-old female patient was diagnosed with aquaporin-4 (AQP4) antibody-positive NMOSD following a varicella zoster virus infection. Her disease course was marked by longitudinally extensive transverse myelitis and retrobulbar optic neuritis. During inpatient rehabilitation, she developed SPLs emerging from both elbows. Her management included multimodal rehabilitation with gabapentinoids, transcutaneous electrical nerve stimulation and task-specific motor training. However, visual tactile feedback techniques were limited by her severe visual impairment. Despite persistent SPLs, upper limb function improved, with gains in motor function and activities of daily living. In conclusion, this unique case highlights a rare association between NMOSD and SPL, expanding the spectrum of body schema disturbances in demyelinating disease.

Keywords: Neuromyelitis optica, perceptual disorders; phantom limb; rehabilitation.

Phantom limb sensation, also known as phantom limb syndrome (PLS), is a complex neurological phenomenon in which individuals perceive the presence of a limb or appendage despite its physical absence. Supernumerary phantom limb (SPL), on the other hand, is a rare neurological phenomenon in which an individual develops the illusory awareness of additional body parts, limbs or appendages, in the absence of any physical anatomical loss of the relevant body part.[1] It is typically associated with diseases affecting the central nervous system. However, its association with neuromyelitis optica spectrum disorder (NMOSD), a distinct autoimmune demyelinating disease of the central nervous system characterized by optic neuritis and longitudinally extensive transverse myelitis (LTEM), has not been previously reported in the medical literature. In this article, we present a case of a patient with NMOSD who developed SPLs in both her upper limbs, which posed unique treatment challenges.

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CASE REPORT

A 61-year-old Chinese female patient with a past medical history of hyperlipidemia and left breast cancer was admitted to the hospital with a sudden onset of difficulty in walking and blurred vision which occurred after she recently recovered from shingles. Additionally, she had numbness over the periumbilical region. Over a few days, her periumbilical numbness then progressed cranially towards her chest and upper limbs, and caudally down her thighs and lower limbs. Subsequently, she developed paresthesia and dysesthesia in both her upper and lower limbs, including the digits.

She had a normal Glasgow Coma Scale (GCS) score and normal mental status without any hallucinations. Her neurological examination revealed a right eye relative afferent pupillary defect, severely reduced bilateral visual acuity; *i.e.,* she was only able to count fingers or discriminate presence of light, athetoid movements in the bilateral fingers, reduced bilateral upper limb strength of Medical Research Council (MRC) Grade 3, loss of proprioception in her upper and lower limbs and a neurological level of injury at C3.

The patient tested positive for serum aquaporin-4 (AQP4) antibodies and Varicella Zoster virus (VZV) immunoglobulin G. She tested negative for serum myelin oligodendrocyte glycoprotein (MOG) antibodies. Cerebrospinal fluid analysis demonstrated pleocytosis without evidence of malignant cells. A magnetic resonance imaging (MRI) scan of her brain and orbits revealed swelling of the right optic nerve without any abnormality detected in the left optic nerve, optic chiasm and bilateral optic tracts (Figure 1). There were no conal or extraconal mass lesions seen and the cavernous sinuses and cortices were normal in appearance. The MRI scan of her cervical spine showed lesions spanning the dorsal medulla to the C7 spinal cord, with no focal lesion detected in the thoracic cord (Figure 2). Finally, she was diagnosed with AQP4 antibody-positive NMOSD, with cervical LTEM and retrobulbar optic neuritis, thought to have been triggered by her antecedent VZV infection. She received a course of valacyclovir, corticosteroids, plasma exchange (PLEX) and rituximab.

After acute treatment, she was transferred to the inpatient rehabilitation unit. During rehabilitation, she reported the presence of an additional phantom limb emerging from each of her elbow joints, giving rise to the perception of having four distinct forearms. These sensations were reported to have started approximately three months after the diagnosis of NMOSD (Figure 3). On physical examination, there was reduced upper limb strength of MRC Grade 3 to 4 throughout the bilateral upper limb myotomes. Due to impaired voluntary control of her actual upper limbs, fine and gross motor control were impaired. Interestingly, she reported having to consciously direct her phantom upper limbs instead, to perform specific motor actions (e.g., grasping an object, flexing the elbow joint) with her actual hands and forearms. She did not report any pain in the additional phantom upper limbs or actual limbs themselves at that juncture. Upon tactile stimulation of her actual upper limbs, she reported perceiving light touch and pin-pricks in the phantom limbs rather than the actual upper limbs, albeit with reduced intensity. The positions of these phantom limbs in relation to the actual limbs were also variable, sometimes being lateral to the actual forearm and at other times, medial to the actual forearm. These perceptions of additional limbs were present regardless of voluntary action of her upper limbs.

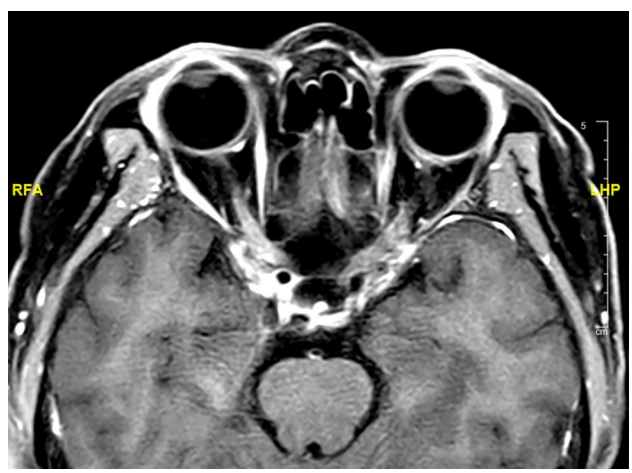

Figure 1. Magnetic resonance imaging brain/orbits with contrast: There is mild swelling in the intracanalicular segment of the right optic nerve without overt hyperintensity on T2/FLAIR. No abnormality is detected in the left optic nerve.
FLAIR, fluid-attenuated inversion recovery.

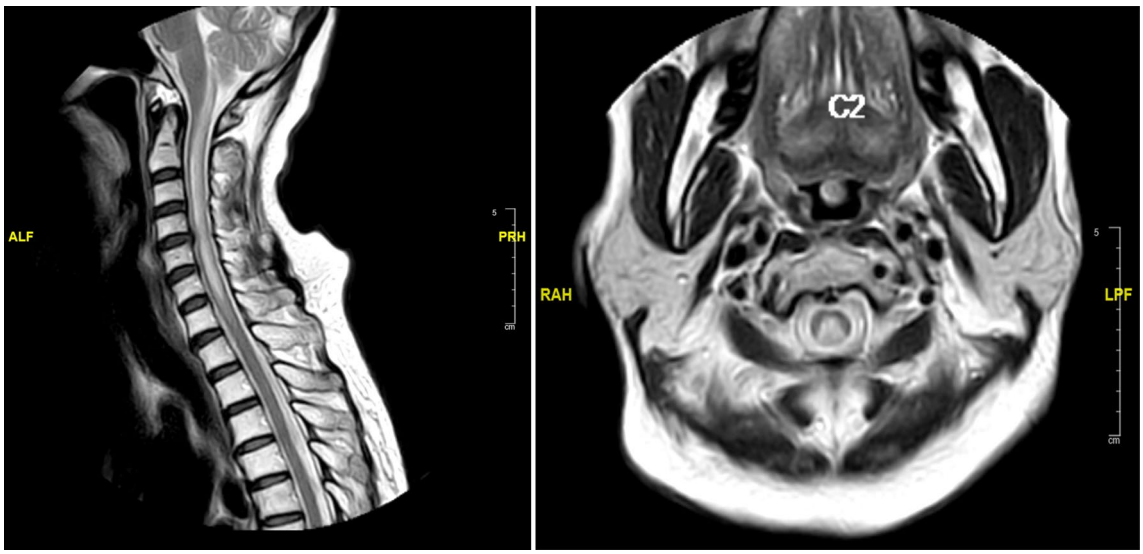

Figure 2. Magnetic resonance imaging spine with contrast: There are T2w-hyperintense lesions from the dorsal medulla to the C7 level with predominant involvement of the central and posterior cord with patchy peripheral enhancement.

She was diagnosed with SPLs secondary to the LTEM, as a result of her NMOSD. Treatment of her SPLs was multimodal, involving both pharmacological and non-pharmacological measures. Due to the development of upper limb dysesthesia during the initial period of her admission, gabapentin 400 mg twice daily was started for approximately 1.5 months, before being switched to pregabalin. Pregabalin was, then, uptitrated to 100 mg twice daily until the time of discharge from the inpatient rehabilitation unit. She also underwent a trial of transcutaneous electrical nerve stimulation that was applied to her upper limbs during her inpatient rehabilitation to provide sensory feedback of the spatial position of her actual limbs. In view of her severe visual impairment, incorporating visually dependent tactile feedback therapies was not feasible. She also underwent task-specific training using tactile and verbal cues, strength training of the upper limb, mobility, ambulation and caregiver training. Although no reduction in SPLs or dysesthesia was noted during her inpatient rehabilitation admission, overall functional improvement was observed, with the motor Functional Independence Measure (FIM) subscore increasing from 22 to 31. Transfers, which require hand and upper limb placement, improved from requiring maximal assistance to requiring moderate assistance.

Upon discharge, home-based rehabilitation services were initiated for continued rehabilitation therapy upon return to the community. Therapy consisted of mental imagery, hand-over-hand guidance and assistance for upper limb movements, verbal cues and feedback to guide upper limb movement patterns and continued task-specific training for seated activities of daily living, such

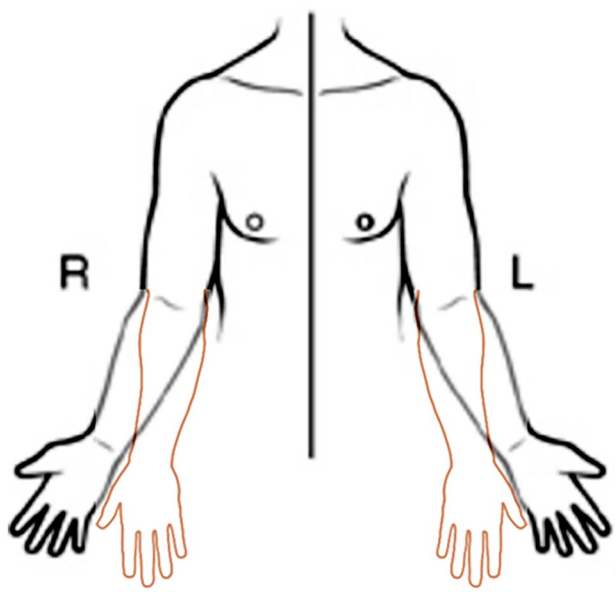

Figure 3. Diagram depicting the patient's perception of where the additional phantom limbs were felt to emerge.

Table 1. Examples of cases of SPL

Author	Year of publication	Type of article	Aetiology of SPL	Location of SPL(s)
Bakheit et al.[2]	2005	Case report	Stroke	Upper limb
Halligan et al.[3]	1993	Case report	Stroke	Upper limb
Halligan and Marshall[4]	1995	Case report	Stroke	Upper limb
Matsuyama et al.[5]	2024	Case report	Stroke	Upper limb
Roger and Franzen[6]	1992	Case report	Traumatic brain injury	Upper limb
Diaz-Segarra et al.[7]	2020	Case report	Traumatic spinal cord injury	Upper limb
Choi et al.[8]	2013	Case report	Traumatic spinal cord injury	Lower limb
Curt et al.[9]	2011	Case report	Traumatic spinal cord injury	Upper limb
Lu et al.[10]	2019	Case report	Traumatic spinal cord injury	Upper limb
Frank and Elliott[11]	1989	Case report	Traumatic spinal cord injury	Upper and lower limbs
Millonig et al.[12]	2011	Case report	Epileptic seizures	Upper and lower limbs
Lamb[13]	2017	Case report	Polytrauma (brain & spinal cord injury)	Upper and lower limbs
Reichert et al.[14]	2002	Case report	Intramedullary ependymoma	Upper limb
Bakheit[15]	2000	Case report	Transverse myelitis	Lower limb
Mayeux and Benson[16]	1979	Case report	Multiple sclerosis	Upper limb
Melinyshyn et al[17]	2016	Case series	Acute inflammatory demyelinating polyneuropathy	Upper and lower limbs

SPL, supernumerary phantom limb.

as feeding and oral hygiene. The patient was reviewed in the outpatient rehabilitation clinic three months after discharge. Despite continued outpatient rehabilitation, her SPLs were still present, remained unchanged in position, and were still perceived as emerging from her bilateral elbow joints. A written informed consent was obtained from the patient for publication of this case report.

DISCUSSION

Supernumerary phantom limbs are neurological phenomena characterized by the vivid perception of additional limbs in the absence of any actual limb loss.[1] Unlike classical phantom limb sensations, they occur without prior loss of limb secondary to amputation or peripheral deafferentation of the affected limb. Supernumerary phantom limbs can either affect entire limbs or be confined to localized areas of the limb, such as the hands or forearms. Cases of SPL in the medical literature report associations with a myriad of central nervous system pathologies (Table 1). These include stroke,[2-5] traumatic brain injury,[6] spinal cord injury (SCI),[7-11] polytrauma,[12] epilepsy,[13] space occupying lesions[14] and demyelinating diseases such as transverse myelitis,[15] multiple sclerosis[16] and acute inflammatory demyelinating polyneuropathy.[17] To the best of our knowledge, no prior case of SPL associated with NMOSD has been reported in the literature.

The exact pathophysiological mechanisms underpinning SPLs still remain poorly understood. One hypothesis suggests that SPLs arise from the brain's inability to integrate visual, tactile and proprioceptive afferent inputs as a result of acute deafferentation. This sudden disruption of communication between the ascending sensory tracts and brain results in an altered body schema.[18] In the event of sudden deafferentation by any lesion along the afferent pathways, internal representations may be altered, leading to a failure to generate a normal experience of self and body location, ultimately resulting in phantom limbs.[8] This occurrence of maladaptive cortical reorganization after deafferentation, in which aberrant neuroplasticity occurs, has also been implicated in the genesis of phantom sensations, particularly in the context of spinal cord lesions.[19] Collectively, the development of SPLs is attributed to distorted

central body representation secondary to maladaptive responses to neural injury.

Management of SPLs remains largely empirical, with no clear consensus on the optimal rehabilitation approach. This is probably due to the rarity of SPLs and heterogeneity of underlying etiologies associated with them in the literature, thereby limiting the development of treatment protocols and conduct of robust studies. Nonetheless, treatments have mostly been adapted from approaches used in phantom limb sensations and pain, based on the premise that correcting maladaptive neural representations may reduce the symptom severity.

Visual tactile feedback therapy forms the primary non-pharmacological strategy used in the treatment of SPLs, though with variable outcomes. It is based on the premise of reconciling the incongruence between sensory modalities and normalizing internal body schema representation.[7] Other non-pharmacological strategies include mirror therapy and virtual reality systems, which are employed to visually recreate an image of the affected extremity, allowing patients to visualize and attempt to move the phantom limb in synchrony with actual intact limb movements.[20] In addition, task-specific motor training, tailored to the patient's specific cognitive, motor and sensory impairments, remains vital in optimizing the function in such patients. However, therapeutic applications of these active rehabilitation strategies are often not suitable in patients with NMOSD, in whom visual impairment is typically severe. In case of our patient with NMOSD, her visual impairment severely limited the feasibility and effectiveness of the aforementioned vision-dependent rehabilitation strategies.

Passive rehabilitative therapies, including pharmacological agents and non-invasive brain stimulation, are supported by existing evidence. Pharmacological management typically involves gabapentinoids such as gabapentin and pregabalin, particularly in cases where SPL is accompanied by dysesthesia or neuropathic pain. Of note, there is limited evidence suggesting that gabapentin may have a synergistic effect when combined with visual feedback therapy in the treatment of SPLs.[7] High-frequency repetitive transcranial magnetic stimulation has also been explored with some success in a case of SPL resulting from a SCI.[10]

In conclusion, SPL is a rare neuropsychiatric manifestation associated with central nervous system pathology and may arise in the context of extensive sensory pathway disruption. This unique case highlights a rare association between NMOSD and SPL, expanding the spectrum of body schema disturbances in demyelinating disease. Severe visual impairment and sensorimotor deficits limited the effectiveness of conventional rehabilitation approaches. Although functional improvement was achieved, SPL symptoms persisted. Further studies are needed to explore long-term outcomes and optimized therapies for SPL, particularly in visually impaired populations.

Declaration of Conflicting Interests

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Author Contributions

L.Y.C.T., Z.Y.V.N., M.R.J.T.: Contributed equally to this manuscript and had full access to the above data; L.Y.C.T., Z.Y.V.N.: Participated in the writing of the paper; M.R.J.T.: Participated in the critical revision of the paper. All authors above take full responsibility for the integrity of the data and the accuracy of the data analysis.

Data Availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

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AI Disclosure

The authors declare that artificial intelligence (AI) tools were not used, or were used solely for language editing, and had no role in data analysis, interpretation, or the formulation of conclusions. All scientific content, data interpretation, and conclusions are the sole responsibility of the authors. The authors further confirm that AI tools were not used to generate, fabricate, or 'hallucinate' references, and that all references have been carefully verified for accuracy.

REFERENCES

1. Cipriani G, Picchi L, Vedovello M, Nuti A, Fiorino MD. The phantom and the supernumerary phantom limb: Historical review and new case. Neurosci Bull 2011;27359-65. doi: 10.1007/s12264-011-1737-6.
2. Bakheit AM, Roundhill S. Supernumerary phantom limb after stroke. Postgrad Med J 2005;81:e2. doi: 10.1136/pgmj.2004.027086.
3. Halligan PW, Marshall JC, Wade DT. Three arms: A case study of supernumerary phantom limb after right hemisphere stroke. J Neurol Neurosurg Psychiatry 1993;56:159-66. doi: 10.1136/jnnp.56.2.159.
4. Halligan PW, Marshall JC. Supernumerary phantom limb after right hemispheric stroke. J Neurol Neurosurg Psychiatry 1995;59:341-2. doi: 10.1136/jnnp.59.3.341-a.
5. Matsuyama T, Hayashi K, Nakaya Y, Suzuki A, Kobayashi Y, Sato M. Supernumerary phantom limb after stroke in the left hemisphere: A case report. Cureus 2024;16:e66681. doi: 10.7759/cureus.66681.
6. Rogers MJ, Franzen MD. Delusional reduplication following closed-head injury. Brain Inj 1992;6:469-76. doi: 10.3109/02699059209008142.
7. Diaz-Segarra N, McKay O, Kirshblum S, Yonclas P. Management of nonpainful supernumerary phantom limbs after incomplete spinal cord injury with visual-tactile feedback therapy: A case report. Spinal Cord Ser Cases 2020;6:62. doi: 10.1038/s41394-020-0312-7.
8. Choi JY, Kim HI, Lee KC, Han ZA. Atypical supernumerary phantom limb and phantom limb pain in a patient with spinal cord injury: Case report. Ann Rehabil Med 2013;37:901-6. doi: 10.5535/arm.2013.37.6.901.
9. Curt A, Yengue CN, Hilti LM, Brugger P. Supernumerary phantom limbs in spinal cord injury. Spinal Cord 2011;49:588-95. doi: 10.1038/sc.2010.143.
10. Lu YS, Tong P, Guo TC, Ding XH, Zhang S, Zhang XJ. Effects of combined rTMS and visual feedback on the rehabilitation of supernumerary phantom limbs in a patient with spinal cord injury: A case report. World J Clin Cases 2019;7:3120-5. doi: 10.12998/wjcc.v7.i19.3120.
11. Frank RG, Elliott TR. Spinal cord injury and health locus of control beliefs. Paraplegia 1989;27:250-6. doi: 10.1038/sc.1989.38.
12. Millonig A, Bodner T, Donnemiller E, Wolf E, Unterberger I. Supernumerary phantom limb as a rare symptom of epileptic seizures--case report and literature review. Epilepsia 2011;52:e97-e100. doi: 10.1111/j.1528-1167.2011.03156.x.
13. Lamb SC. Post-traumatic visualized supernumerary phantom limbs: A case presentation. PM R 2017;9:943-5. doi: 10.1016/j.pmrj.2017.01.002.
14. Reichert K, Sewekow K. A man with 3 arms. Phantom limb perception in intramedullary ependymoma. Nervenarzt 2002;73:548-51. German. doi: 10.1007/s00115-002-1286-3.
15. Bakheit AM. Phantom limb sensations after complete thoracic transverse myelitis. J Neurol Neurosurg Psychiatry 2000;69:275-6. doi: 10.1136/jnnp.69.2.275.
16. Mayeux R, Benson DF. Phantom limb and multiple sclerosis. Neurology 1979;29:724-6. doi: 10.1212/wnl.29.5.724.
17. Melinyshyn AN, Gofton TE, Schulz V. Supernumerary phantom limbs in ICU patients with acute inflammatory demyelinating polyneuropathy. Neurology 2016;86:1726-8. doi: 10.1212/WNL.0000000000002541.
18. Khateb A, Simon SR, Dieguez S, Lazeyras F, Momjian-Mayor I, Blanke O, et al. Seeing the phantom: A functional magnetic resonance imaging study of a supernumerary phantom limb. Ann Neurol 2009;65:698-705. doi: 10.1002/ana.21647.
19. Moxon KA, Oliviero A, Aguilar J, Foffani G. Cortical reorganization after spinal cord injury: Always for good? Neuroscience 2014;283:78-94. doi: 10.1016/j.neuroscience.2014.06.056.
20. Katayama O, Iki H, Sawa S, Osumi M, Morioka S. The effect of virtual visual feedback on supernumerary phantom limb pain in a patient with high cervical cord injury: A single-case design study. Neurocase 2015;21:786-92. doi: 10.1080/13554794.2015.1011664.