Rehabilitation Practice and Science

Volume 2024 | Issue 1 Article 5

6-1-2024

Pseudomeningocele Following Traumatic Brachial Plexus Injury: A Case Report

Shih-Ting Huang
Department of Physical Medicine and Rehabilitation Lotung Poh-Ai Hospital, Lo-Hsu Foundation, Yilan County, Taiwan (R.O.C.), p801003@gmail.com

Ming-Hsun Lee
Department of Radiology, Lotung Poh-Ai Hospital, Lo-Hsu Foundation, Yilan County, Taiwan (R.O.C.)

Simon-Fuk Tan Tang
Department of Physical Medicine and Rehabilitation Lotung Poh-Ai Hospital, Lo-Hsu Foundation, Yilan County, Taiwan (R.O.C.)

Ching-Yu Wang
Department of Physical Medicine and Rehabilitation Lotung Poh-Ai Hospital, Lo-Hsu Foundation, Yilan County, Taiwan (R.O.C.)

Yu-Ting Hsieh
Department of Physical Medicine and Rehabilitation Lotung Poh-Ai Hospital, Lo-Hsu Foundation, Yilan County, Taiwan (R.O.C.)

Recommended Citation

Huang, Shih-Ting; Lee, Ming-Hsun; Tang, Simon-Fuk Tan; Wang, Ching-Yu; and Hsieh, Yu-Ting (2024) "Pseudomeningocele Following Traumatic Brachial Plexus Injury: A Case Report," *Rehabilitation Practice and Science*: Vol. 2024: Iss. 1, Article 5.
DOI: https://doi.org/10.6315/3005-3846.2234
Available at: https://rps.researchcommons.org/journal/vol2024/iss1/5

CASE REPORT

Pseudomeningocele Following Traumatic Brachial Plexus Injury: A Case Report

Shih-Ting Huang [a], Ming-Hsun Lee [b], Simon-Fuk T. Tang [a], Ching-Yu Wang [a], Yu-Ting Hsieh [a,*]

[a] Department of Physical Medicine and Rehabilitation, Lotung Poh-Ai Hospital, Lo-Hsu Foundation, Yilan County, Taiwan, ROC
[b] Department of Radiology, Lotung Poh-Ai Hospital, Lo-Hsu Foundation, Yilan County, Taiwan, ROC

Abstract

An injury to the brachial plexus can lead to the development of a pseudomeningocele, a buildup of cerebrospinal fluid (CSF) in the subarachnoid space caused by tears in the affected root sleeves. External forces acting on the brachial plexus represent the main mechanism of injury. The resulting stretching and tearing of the meninges outside the nerve root led to the pseudomeningocele. Although often initially asymptomatic, a pseudomeningocele can eventually cause pain, paresthesia, muscle weakness, and paralysis. The nerve conduction studies (NCS), electromyography (EMG), and magnetic resonance imaging (MRI) can be used to identify the injury, and treatment can involve either conservative management or surgical intervention if necessary.

Here, we present the case of a 27-year-old male patient who suffered brain damage and multiple fractures in a motorcycle accident. After 2 months of rehabilitation, he began experiencing progressive numbness in his right forearm and hand, and clawing of the fourth and fifth fingers. NCS and EMG revealed the radiculopathy of the eighth cervical and first thoracic nerve roots, and a preganglionic nerve root injury. Cervical MRI showed a pseudomeningocele extending from the seventh cervical to the first thoracic vertebra, accompanied by nerve root avulsion − findings that confirmed the need for surgical intervention.

This case highlights the possibility that symptoms in patients with the brachial plexus and traumatic brain injuries might be overlooked, resulting in opportunities for treatment being missed. This report will hopefully serve as a valuable reference for differential diagnosis and management in such cases.

Keywords: Pseudomeningocele, Brachial plexus injury, Traumatic brain injury

1. Introduction

Pseudomeningocele is a complication resulting from traumatic arm damage, leading to the tearing of brachial plexus nerve roots.[1] This complication is frequently observed in young men between 20 and 30 years of age who have experienced motorcycle accidents. The stretching and pulling of the brachial plexus caused by external forces, leading to the tearing of the dura mater that envelops the nerve roots, is the main pathophysiologic mechanism behind the pseudomeningocele.

Although most cases of the pseudomeningocele are initially asymptomatic, the subsequent accumulation of cerebrospinal fluid (CSF) leaking into the extradural or soft tissues can compress the nerves over time,[2] leading to the emergence or worsening of clinical symptoms. These

Received 16 April 2023; revised 4 May 2023; accepted 15 May 2023.
Available online 1 June 2024

* Corresponding author at: Department of Physical Medicine and Rehabilitation, Lotung Poh-Ai Hospital, Lo-Hsu Foundation, No. 83 Nanchang St., Yilan County, Taiwan, ROC.
E-mail address: 82B014@mail.pohai.org.tw (Y.-T. Hsieh).

https://doi.org/10.6315/TJPMR.2234

symptoms can include neck pain, sensory abnormalities or loss, muscle weakness, limb paralysis, and even posture-related hypotension.[3] Notably, symptoms might not manifest until several months or years after the injury[4,5] and might be overlooked in patients with combined traumatic arm and brain injuries.

2. Case report

A 27-year-old male with no considerable medical history experienced a motorcycle accident resulting in brain damage, fractures of the right scaphoid bone, right rib, the transverse process of the seventh cervical vertebra, and left acetabulum, left hip dislocation, pneumorrhachis, and lung contusion and pneumothorax. At the time of admission, the patient had a Glasgow Coma Scale score of E1V2M1 and he was directly taken to the intensive care unit for surgical intervention. As a result of a traumatic brain injury, the patient is demonstrating cognitive dysfunction at Level IV on the Rancho Los Amigos scale (RLAS), as well as bilateral limb weakness with a manual muscle testing (MMT) grade of 3, mild increased deep tendon reflexes in all four limbs, and complete dependence on others for activities of daily living (ADL). After two months of rehabilitation, which included muscle strength training, balance training, ambulation training, and cognitive function training, the patient's cognitive function has improved to RLAS level VII, and muscle strength of upper limbs have also recovered to MMT grade of 4. Furthermore, the patient was able to ambulate independently with the use of the walker. The patient has reported an aggravation of numbness in his right forearm, palm, fourth and fifth fingers, despite the progress he has made in his rehabilitation. Along with this, he has been experiencing weakness in his right wrist extension, finger flexion, and palmar opposition of the thumb, with MMT grades of 3 and 2, respectively. Additionally, the patient has exhibited clawing in his fourth and fifth fingers on the right hand, as well as atrophy of the intrinsic muscles in the right hand. The deep tendon reflexes of the biceps brachii and triceps brachii muscles were within normal limits to mildly increased, and the Hoffman sign was negative. Initially, a compressive peripheral neuropathy in the ulnar nerve at elbow level was suspected, which prompted the performance of nerve conduction studies (NCS) and electromyography (EMG).

The nerve conduction results (Table 1) showed prolonged distal motor latency of the median and ulnar nerves, a decrease in the amplitude of the compound motor action potential, and a reduction in nerve conduction velocity, but normal sensory nerve conduction velocity for both the sensory nerves. The abductor pollicis brevis, first dorsal interosseous, flexor carpi ulnaris, and flexor digitorum superficialis showed abnormal spontaneous activity, with recruitment decrement (Table 2). Only a slight recruitment decrement was observed in the extensor digitorum communis, but the triceps was normal. These findings suggested that the probable cause of the patient's symptoms was preganglionic injuries to the eighth cervical and first thoracic nerve roots.

Cervical magnetic resonance imaging (MRI) revealed a pseudomeningocele from

Table 1. Summary of the nerve conduction study of the patient's upper limb.

Nerve-Site	Latency (ms)		Amplitude (mV)		Velocity (m/s)	
	Right	Left	Right	Left	Right	Left
Motor NCS						
Median-Wrist	4.6	3.7	0.3	7.3		
Median-Elbow	9.5	7.9	0.1	7.2	43	50
Ulnar-Wrist	4.4	2.4	0.8	9.5		
Ulnar-Below elbow	10.3	6.1	0.8	9.2	34	54
Ulnar-Above elbow	13.4	8.1	0.4	9.0	34	53
Sensory NCS						
Median-Plam	1.1	1.1	70	63	64	64
Median-Wrist	3.0	2.6	40	48	54	53
Median-Elbow	6.3	6.0	20	26	63	62
Ulnar-Wrist	2.1	2.1	34	59	62	62

Table 2. Summary of the electromyography of the patient's right upper limb.

Needle EMG					
Right upper limb muscle	Insert Activity	Resting		Voluntary motor units	
	Normal	Fibrillation	Positive sharp wave	Polyphasic motor unit potentials	Recruitment
Triceps brachii (C6 7 8)	Normal	−	−	−	−1
Extensor digitorum communis (C7 8)	Normal	+−	+−	2+	−2
Abductor pollicis brevis (C8 T1)	Increased	2+	2+	Scanty MUAP	
Flexor carpi ulnaris (C8 T1)	Normal	1+	1+	2+	−3
First dorsal interosseous (C8 T1)	Increased	2+	2+	Scanty MUAP	
Flexor digitorum sublimis (C7 8)	Normal	1+	1+	2+	−3

Underline indicates predominant root innervation. MUAP = motor unit action potential.

the seventh cervical to the first thoracic vertebra, with an accompanying nerve root avulsion (Figs. 1 and 2). The patient was subsequently referred for microscopic surgery and repair of the ruptured dura mater. In our patient's case, fibrin sealant was used to repair the ruptured dura mater along the suture line. Despite a two-year follow-up after surgery, there has been no improvement in the patient's right upper limb muscle strength. The manual muscle testing (MMT) scores for wrist extension, finger flexion, and thumb opposition are grade 3, 2, and 2, respectively. Additionally, the patient still presents with clawing in the fourth and fifth fingers of his right hand. The reason for this patient's poor prognosis may be due to delayed surgery or severe damage to the brachial plexus.

3. Discussion

The pseudomeningocele is a serious potential complication of traumatic brachial plexus injuries, frequently associated with nerve root avulsion.[1] Unlike a meningocele, where the CSF is confined by the dura mater, a pseudomeningocele is a swelling that may form over time due to CSF leakage. This swelling has mass-like characteristics and is not surrounded by a membrane but contained within a cavity in the soft tissues or extradural space.[2,6] The formation of the pseudomeningocele often occurs without symptoms, and the dura tear can resolve on its own. However, if CSF continues to leak and accumulates in considerable quantities, it can exert pressure on the nerves, leading to the various clinical manifestations already

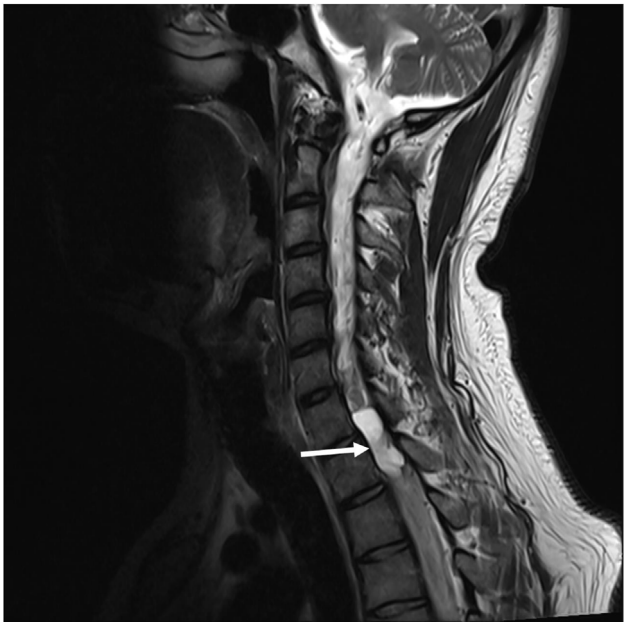

Fig. 1. Sagittal view from T2-weighted magnetic resonance imaging of the patient's cervical spine shows cystic lesions with high signal intensity, extending from the seventh cervical vertebra to the first thoracic vertebra.

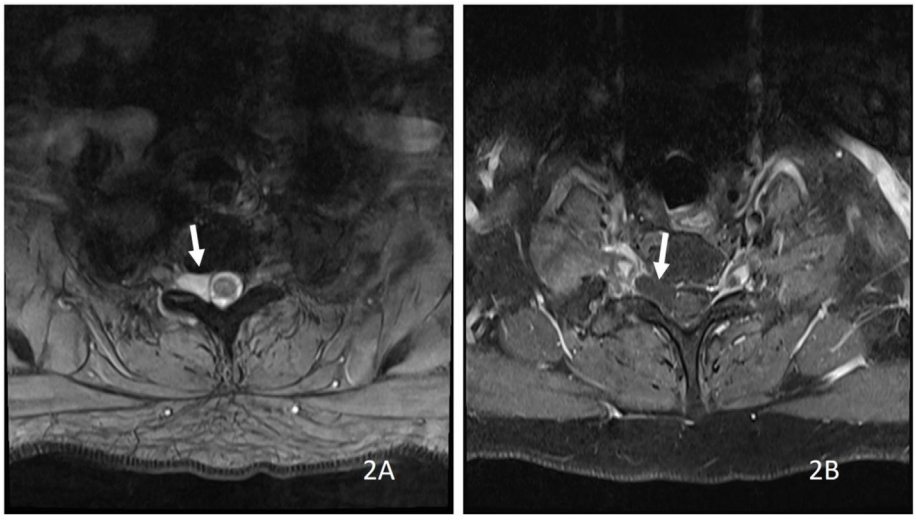

Fig. 2. Axial view from T2-weighted magnetic resonance imaging of the patient's cervical spine (Fig. 2A) shows cystic lesions with high signal intensity in the neural foramen of the seventh cervical vertebra. Axial view from T1-weighted magnetic resonance imaging of the patient's cervical spine (Fig. 2B) shows cystic lesions with low signal intensity, extending from the seventh cervical vertebra to the first thoracic vertebra.

described. The onset of the symptoms in the pseudomeningocele typically varies in a range from 6 months to 20 years after the initial nerve injury,[5] but case reports indicate that the symptoms can develop within a few days.[7]

In patients with traumatic brain injury, progressive limb weakness and sensory numbness may be overlooked because of cognitive impairments. In a patient with a history of brain injury, worsening sensory numbness, and increasing weakness or even paralysis of the upper limbs, additional potential causes such as the brachial plexus injury with the pseudomeningocele, cervical syringomyelia, cervical spinal cord tumors, parietal lobe tumors, brain or spinal cord infections, and monomelic amyotrophy should therefore be considered in the differential diagnosis.[8] A thorough medical history and physical examination are critical for early detection and accurate diagnosis, which in turn facilitates timely and appropriate treatment. Tests such as NCSs and EMG can be used to differentiate preganglionic from postganglionic nerve injuries to accurately identify nerve damage and to assess the severity of the nerve injury. In preganglionic nerve injuries, motor nerve conduction is affected, whereas sensory nerve conduction remains normal. Conversely, the motor and sensory nerve conductions are affected in postganglionic nerve damage.

In the past, computed tomography myelography was considered the "gold standard" for diagnosing the traumatic brachial plexus injuries. It was also useful in evaluating adjacent fractures and preganglionic nerve injuries.[9] However, the procedure has some drawbacks, including invasiveness, the side effects of contrast agents, and radiation exposure. More recently, MRI has emerged as a noninvasive and radiation-free alternative. MRI can differentiate between various soft-tissue injuries such as hematoma, edema or fibrosis of peripheral tissue, pseudomeningocele, and nerve damage. Studies[10−14] have demonstrated that spinal cord MRI has a sensitivity ranging from 60 % to 88 % and specificity ranging from 89 % to 100 % for diagnosing the brachial plexus injuries. Furthermore, compared with the computed tomography myelography, nerve MRI can more accurately differentiate among preganglionic, postganglionic, and mixed-nerve injuries, making it an increasingly popular diagnostic tool.[15]

The pseudomeningocele typically appears as a hypointense lesion on T1-weighted images and as a hyperintense lesion on T2-weighted images. Although nerve root avulsion frequently accompanies the pseudomeningocele in cases of the brachial plexus injury, the presence of the pseudomeningocele does not always indicate nerve

root avulsion. A comprehensive evaluation − including physical examination, electrophysiologic testing, and imaging − is therefore necessary to accurately assess the presence of nerve root damage.

The brachial plexus injury can be managed either conservatively or with surgery. Conservative management aims to maintain the range of motion of the affected limb, strengthen the remaining functional muscles, and alleviate pain. Nonsteroidal anti-inflammatory drugs and opioids are typically the first-line treatments for nociceptive pain, whereas anticonvulsant or antidepressant drugs may be prescribed for neuropathic pain.[16] Surgical intervention depends on the severity and location of the nerve damage. If symptomatic pseudomeningoceles occur, treatment options may include needle aspiration, CSF diversion, and surgical removal of the pseudomeningocele with repair of the underlying dura mater. In addition, autologous tissue, homologous allografts, or fibrin glue can be used to repair dural defects.[17] Nerve transfer or nerve grafting are the only possibilities for preganglionic injuries, but the nerve transfer, nerve grafting, end-to-end nerve anastomosis, and palliative reconstruction are all potential repairs for postganglionic injuries. Ideally, surgery for postganglionic injuries is performed between 3 months and 2 years after the injury because spontaneous repair within the first 3 months after the nerve injury is a possibility.[18] For preganglionic injuries, early surgical intervention is essential, typically within 4 weeks.[19] The prognosis with surgical intervention is positive. More than 80 % of patients who undergo the nerve transfer can regain elbow flexion, and most patients who undergo surgical reconstruction experience an improvement in their quality of life, with only approximately one-fifth experiencing permanent disability.[20]

4. Conclusion

In patients with traumatic brachial plexus injury and coexisting traumatic brain injury, any deterioration in upper limb sensory or motor function must prompt a careful consideration of the possibility of an associated pseudomeningocele. Such vigilance is crucial because the early detection of the pseudomeningocele is vital to prevent potentially irreversible nerve root damage.

Funding/support statement

None.

Conflict of interest

The authors declare that there is no conflict of interest.

Acknowledgement

We would like to thank the Department of Radiology, Lotung Poh-Ai Hospital and Department of Physical Medicine and Rehabilitation, Lotung Poh-Ai Hospital.

References

1. Haider AS, Watson IT, Sulhan S, Leonard D, Arrey EN, Khan U, et al. Traumatic cervical nerve root avulsion with pseudomeningocele formation. *Cureus* 2017;9:e1028.
2. Knight S. Pseudomeningocele. In: Kreutzer J, DeLuca J, Caplan B, eds. *Encyclopedia of Clinical Neuropsychology*. Cham: Springer; 2018.
3. Schievink WI, Chu RM, Maya MM, Johnson JP, Cohen HC. Spinal manifestations of spontaneous intracranial hypotension. *J Neurosurg Spine* 2013;18:96−101.
4. Rahimizadeh A, Javadi SA. Symptomatic intraspinal lumbosacral pseudomeningocele, a late consequence of root avulsion injury secondary to a gunshot wound. *N Am Spine Soc J* 2020;3:100025.
5. Hader WJ, Fairholm D. Giant intraspinal pseudomeningoceles cause delayed neurological dysfunction after brachial plexus injury: report of three cases. *Neurosurgery* 2000;46:1245−9.
6. Trapp B, de Andrade Lourencao Freddi T, de Oliveira Morais Hans M, Fonseca Teixeira Lemos Calixto I, Fujino E, Alves Rojas LC, et al. A practical approach to diagnosis of spinal dysraphism. *Radiographics* 2021;41:559−75.
7. Yoon J, Redmond M. Pseudomeningocele from brachial plexus root avulsion. *Surg Case Rep* 2019;2:1−3.
8. Kumar S, Kaul S. Approach to a patient with hemiplegia and monoplegia. *Prog Med* 2017;1:141−6. Published by Association of Physicians of India (API).
9. Tse R, Nixon JN, Iyer RS, Kuhlman-Wood KA, Ishak GE, et al. The diagnostic value of CT myelography, MR myelography, and both in neonatal brachial plexus palsy. *AJNR Am J Neuroradiol* 2014;35(7):1425−32.
10. Grahn P, Pöyhiä T, Sommarhem A, Nietosvaara Y. Clinical significance of cervical MRI in brachial plexus birth injury. *Acta Orthop* 2019;90:111−8.
11. Gad DM, Hussein MT, Omar NNM, Kotb MM, Abdel-Tawab M, Yousef HAZ. Role of MRI in the diagnosis of adult traumatic and obstetric brachial plexus injury compared to intraoperative findings. *Egypt J Radiol Nucl Med* 2020;51:195.
12. Gunes A, Bulut E, Uzumcugil A, Oguz KK. Brachial plexus ultrasound and MRI in children with

brachial plexus birth injury. *AJNR Am J Neuroradiol* 2018;39:1745−50.
13. Somashekar D, Yang LJ, Ibrahim M, Parmar HA. High-resolution MRI evaluation of neonatal brachial plexus palsy: a promising alternative to traditional CT myelography. *AJNR Am J Neuroradiol* 2014;35:1209−13.
14. Medina LS, Yaylali I, Zurakowski D, Ruiz J, Altman NR, Grossman JA. Diagnostic performance of MRI and MR myelography in infants with a brachial plexus birth injury. *Pediatr Radiol* 2006;36(12):1295−9.
15. Fuzari HKB, Dornelas de Andrade A, Vilar CF, Sayão LB, Diniz PRB, Souza FH. Diagnostic accuracy of magnetic resonance imaging in post-traumatic brachial plexus injuries: a systematic review. *Clin Neurol Neurosurg* 2018;164:5−10.
16. Sakellariou VI, Badilas NK, Stavropoulos NA, Mazis G, Kotoulas HK, Kyriakopoulos S, et al. Treatment options for brachial plexus injuries. *ISRN Orthop* 2014;2014:314137.
17. Weng YJ, Cheng CC, Li YY, Huang TJ, Hsu RWW. Management of giant pseudomeningoceles after spinal surgery. *BMC Muscoskel Disord* 2010;11:53.
18. Bertelli JA, Ghizoni MF. Results and current approach for brachial plexus reconstruction. *J Brachial Plexus Peripher Nerve Inj* 2011;6:2.
19. Sinha S, Khani M, Mansoori N, Midha R. Adult brachial plexus injuries: surgical strategies and approaches. *Neurol India* 2016;64:289−96.
20. Park HR, Lee GS, Kim IS, Chang JC. Brachial plexus injury in adults. *Nerve* 2017;3:1−11.