ZULFAQAR Journal of Defence Science, Engineering & Technology
e-ISSN: 2773-5281
Vol. 8, Issue 1 (2025)
DOI: https://doi.org/10.58247/jdset-2025-0801-06
Journal homepage: https://zulfaqarjdset.upnm.edu.my

PROLONGED NEUROLOGICAL DEFICIT AFTER SPINAL ANAESTHESIA: A DIAGNOSTIC CHALLENGE IN UNDIAGNOSED SPINAL PATHOLOGY PATIENT

Siti Hajar Haryati Fauzi[a], Nadiawati Abdul Razak[a*]

[a] Department of Clinical, Faculty of Medicine and Defence Health, National Defence University of Malaysia, Sg. Besi Camp, 57000 Kuala Lumpur, Malaysia

ARTICLE INFO

ARTICLE HISTORY
Received : 10-09-2024
Revised : 01-12-2024
Accepted : 21-04-2025
Published : 31-05-2025

KEYWORDS
Spinal Anaesthesia
Neurological Deficit
Postoperative Complication
Lumbar Spondylosis

ABSTRACT

Spinal anaesthesia is a widely used regional anaesthesia technique known for its effectiveness and safety. However, post-operative neurological deficits, though rare, can pose diagnostic challenges. This case report discusses a 53-year-old male with hypertension and type 2 diabetes mellitus who underwent spinal anaesthesia for an emergency incision and drainage of a gluteal abscess. Despite an uneventful perioperative course, the patient developed persistent bilateral lower limb weakness and numbness post-operatively. MRI findings revealed an L4/L5 posterior disc bulge with impingement of the left L4 exiting nerve root, but no evidence of cauda equina syndrome or epidural hematoma. This case underscores the importance of thorough preoperative neurological assessment and the need for differentiating between anaesthesia-related complications and pre-existing spinal pathology. Early multidisciplinary evaluation is crucial in managing prolonged post-spinal anaesthesia neurological deficits.

1.0 INTRODUCTION

Spinal anaesthesia is a commonly employed regional anaesthesia technique, particularly for lower limb and lower abdominal surgeries, due to its efficacy, rapid onset, and minimal systemic effects [1]. However, despite its generally favourable safety profile, rare complications such as prolonged neurological deficits can occur, leading to diagnostic challenges in postoperative care [2]. This case report describes a 53-year-old male with underlying hypertension and type 2 diabetes mellitus who underwent spinal anaesthesia for an emergency incision and drainage of a gluteal abscess. While the perioperative course was uneventful, the patient developed postoperative bilateral lower limb weakness and numbness, raising concerns for a possible spinal anaesthesia-related complication.

The differential diagnosis for postoperative neurological deficits following spinal anaesthesia includes transient neurological symptoms, spinal hematoma, arachnoiditis, and cauda equina syndrome [3]. Given the patient's pre-existing chronic back pain, left thigh numbness, and history of prior neurological episodes, it was essential to distinguish between anaesthesia-related complications and underlying chronic spinal pathology. Postoperative magnetic resonance imaging (MRI) of the lumbosacral spine revealed a diffuse posterior disc bulge at the L4/L5 level with impingement of the left L4 exiting nerve root. However, there was no radiological evidence of cauda equina syndrome or epidural hematoma.

This case highlights the importance of thorough preoperative neurological history-taking, the potential for confounding pre-existing spinal pathology, and the need for a multidisciplinary approach in evaluating prolonged post-spinal anaesthesia neurological deficits. By presenting this case, we aim to emphasise the diagnostic considerations in post-spinal anaesthesia neurological deficits and discuss best practices in anaesthetic management and post-operative monitoring for patients with pre-existing spinal conditions.

*Corresponding Author | Abdul Razak, N. | nadiawati@upnm.edu.my

2.0 CASE PRESENTATION

A 53-year-old gentleman with underlying hypertension and type 2 diabetes mellitus was admitted to the surgical ward for a gluteal abscess. He was posted under emergency operation for incision and drainage. The patient was seen by an anaesthetist medical officer pre-operatively. The assessment was done before the operation, and the findings were unremarkable. The neurological history and assessment were not thoroughly documented on the pre-operative assessment chart. Detailed blood laboratory analyses are summarised in Table 1.

Table 1. Summary of investigations

Tests	Results	Normal range
Full blood count		
Haemoglobin (Hb)	10.3 g/dL	11 – 16 g/dL
Total white cell	27 x 10^9/L	4.5 – 13.5 x 10^9/L
Platelet	333 x 10^9/L	150 – 400 x 10^9/L
Renal profile		
Sodium	128 mmol/L	135- 145 mmol/L
Potassium	4.2 mmol/L	3.5 – 5.0 mmol/L
Creatinine	104 mmol/L	62 – 115 mmol/L
Creatinine clearance	62 mL/min	more than 50 mL/min
Urea	11 mmol/L	7 -20 mmol/L
Random blood sugar		
Sugar level (sc insulatard 12 units 8 hourly was given to the patient)	between 6-10 mmol/L	

The surgery proceeded, and the patient underwent spinal anaesthesia in a single attempt between the third lumbar (L3) and fourth lumbar (L4) intervertebral space with good cerebrospinal fluid (CSF) flow. Hyperbaric bupivacaine 0.5% with fentanyl 20 mcg, a total of 3.0 ml, was given to the patient. Level of anaesthesia achieved from T8 dermatome level extended caudally. The duration of the surgery was 55 minutes and was uneventful. Post-operatively, the patient was monitored in the recovery room for 30 minutes and was discharged to the general surgical ward with normal vital signs and a Bromage score of 2.

At 24 hours post-operatively, the patient continued to complain of numbness and weakness in both lower limbs, with an inability to ambulate due to bilateral lower limb motor impairment. Notably, there was no evidence of bowel or bladder dysfunction. Considering the persistent neurological symptoms, the anaesthesia team was consulted to evaluate the possibility of an anaesthesia-related complication. Upon further evaluation, the patient disclosed a prior history of chronic back pain associated with numbness in the left thigh and intermittent weakness of the left lower limb, consistent with sciatica radiating to the same limb. His past medical history revealed that he underwent a computed tomography (CT) scan of the brain and lumbar puncture procedure because of his severe headache five years earlier. Both investigations yielded unremarkable findings, and he was subsequently discharged after a few days of observation.

In the ward, serial postoperative blood investigations demonstrated a significant improvement in the total white cell count, decreasing from 27 x 10^9/L to 16 x 10^9/L. Physical examination revealed mild tenderness over the lower lumbar region at the injection site. Additionally, there was reduced sensation to touch in both lower limbs, corresponding to the L5 dermatome and below. The muscle strength assessment indicated significant weakness, with a power rating of 1/5.

The patient was referred to a neurologist for assessment, and a magnetic resonance imaging (MRI) was ordered. An MRI spinal arachnoiditis protocol was performed on postoperative day two and revealed a herniated posterior disc at the fourth and fifth lumbar (L4–L5) level, more prominent in the left foraminal zone, resulting in narrowing of the left neural foramen. Additionally, there was impingement of the left L4 exiting nerve root. There was no spinal canal stenosis, crowding of cauda equina, ligamentum flavum hypertrophy or facet arthropathy. The report by the radiologist gave the impression that there was no magnetic resonance (MR) evidence to suggest cauda equina syndrome, epidural hematoma or inflammation. However, there was present mild lumbar spondylosis with impingement of the left L4 exiting nerve root and no spinal canal narrowing. There were no nerve conduction studies done on this patient.

*Corresponding Author | Abdul Razak, N. | nadiawati@upnm.edu.my

Upon discharge from the surgical ward, the patient was scheduled for a neurology outpatient follow-up after two weeks. At the three-month postoperative assessment, muscle weakness persisted, with only a slight improvement in muscle strength to 2/5. The patient remained unable to ambulate independently, and due to his disability, he was unable to return to work as a security guard.

3.0 DISCUSSION

Neurological complications after spinal anaesthesia have been well-documented since the 1950s [4]. Fortunately, over time, the incidence of major neurological complications has reduced [5]. However, anaesthesiologists up to this day still face challenges as complications still occur. In our case, even though the MRI showed there is no evidence of direct injury from spinal anaesthesia (no evidence of hematoma or cauda equina syndrome), the worsening of the symptoms and serious consequences occurred to the patient as the patient was unable to ambulate freely.

This patient had a history of bilateral lower limb weakness and sciatica, which pointed out that he might have an undiagnosed prolapsed intervertebral disc. According to the New York Society of Regional Anaesthesiologists (NYSORA), a relative contraindication to neuraxial blockade is indeterminate neurological disease [6]. However, no clinical study has clearly stated that spinal anaesthesia worsens such neurologic diseases [7]. In this case, our findings underscore the critical importance of a thorough history and physical examination in identifying potential complications following anaesthetic procedures. Given the urgency of the situation, there was no opportunity for further preoperative investigations. We acknowledge the importance of comprehensive neurological assessment in cases of prolonged neurological deficits. We also recognise the limitation that a nerve conduction study was not performed at the time of reporting, which may have provided additional insight into the nature and extent of the neurological involvement.

However, in the context of elective procedures, advanced imaging modalities such as spinal MRI may be considered to identify any underlying pathology. This would facilitate a correlation between radiological findings and the patient's clinical presentation, thereby enabling the selection of the most appropriate anaesthetic approach. If the patient is deemed at high risk for general anaesthesia, neuraxial blockade remains a preferable alternative. In such cases, ultrasound guidance can be employed to enhance precision in needle placement, thereby mitigating the risk of complications and avoiding pathological sites [7].

Currently, there are no established guidelines for the evaluation and management of suspected neuraxial injury following spinal-epidural anaesthesia. In this case, the aetiology of persistent muscle weakness remains uncertain. However, there have been several case reports documenting occurrences of transverse myelitis following both regional and general anaesthesia, highlighting the need for further investigation into potential anaesthesia-related neurological complications. Kitazaki et al. (2020) reported a case of longitudinally extensive transverse myelitis (LETM) with an isolated pontine lesion that developed following epidural and spinal anaesthesia; MRI findings resembled those of autoimmune diseases [8]. The causal relationship between anaesthesia and transverse myelitis remains a subject of debate due to the limited number of published studies on this topic.

Acute transverse myelitis (ATM) is a rare clinical syndrome with an incidence of one to four patients per million per year [9]. ATM is an acquired focal inflammatory disorder or demyelinating disease of the spinal cord, often presenting with rapid onset motor weakness, a decrease in deep tendon reflex spasticity of the extremities, sensory deficits which cause bilateral hypoesthesia, and bowel/bladder dysfunction in the areas lower than affected spinal segment, without any observation of spinal cord injury, spinal lesion, or tumour, and no observation of pressure on the spinal cord. The histopathophysiology of ATM varies and is related to the underlying aetiology. Classically, most cases are characterised by perivascular infiltration, demyelination, and axonal injury by monocytes and lymphocytes at the lesion site [10]. Heterogeneity, along with both grey and white matter involvement, gives evidence that this is not a pure demyelinating disorder. ATM may be a mixed inflammatory disorder involving neurons, axons, oligodendrocytes, and myelin. Alternative histopathologic causes of ATM have been reported to include molecular mimicry and superantigen-mediated disease associated with autoimmune causes [11].

Generally occurring independently, often as a complication of infection, it may also exist as part of a continuum of other neuro-inflammatory disorders [12]. ATM generally occurs in the spinal cord at any level, but most commonly affects the thoracic region. The disorder traverses the spinal cord, causing

*Corresponding Author | Abdul Razak, N. | nadiawati@upnm.edu.my

bilateral deficiencies. However, there may only be partial or asymmetric involvement. The duration of this disease may be as little as three to six months or may become permanently debilitating. At peak deficit, 50% of patients are completely paraplegic, with virtually all the patients having a degree of bladder/bowel dysfunction. Approximately 33% of patients recover with little to no lasting deficits, 33% have a moderate degree of permanent disability, and 33% are permanently disabled [10]. To diagnose ATM, a compressive cord lesion must be excluded first.

Exclusion is usually performed by MRI. This is followed by confirmation of inflammation by a gadolinium-enhanced MRI or lumbar puncture (LP). The diagnostic criteria of ATM, as shown in Table 2, were developed but are generally reserved for research purposes, as not all features must be diagnosed in a clinical setting [13].

Table 2. Diagnostic criteria

1.	Sensory, motor, or autonomic dysfunction originating from the spinal cord	*** most important
2.	T2 hyperintense signal changes on MRI	*** most important
3.	No evidence of a compressive lesion	*** most important
4.	Bilateral signs/symptoms.	
5.	Clearly defined sensory level	
6.	Gadolinium enhancement on MRI and cerebrospinal fluid (CSF) analysis demonstrates evidence of an inflammatory process with pleocytosis or an elevated immunoglobulin G (IgG) index	
7.	Progression to nadir between 4 hours and 21 days	

Although this patient did not exhibit urinary or bowel incontinence, the presence of persistent muscle weakness in the absence of radiological evidence of spinal cord compression necessitates further investigation. Paraesthesia resulting from spinal cord injury may arise not only from direct needle trauma but also from potential neurotoxicity of the local anaesthetic agent, secondary inflammatory responses and oedema following injection, epidural or spinal hematoma, vascular compromise due to spinal artery occlusion, or states of hypoperfusion.

ATM was not initially suspected; therefore, no further diagnostic tests were performed. In this case, no association was identified between the spinal anaesthesia procedure and the progression of neurological symptoms. Neurological monitoring was conducted through regular clinical assessments. Advanced imaging studies were not routinely performed, as there was no clinical evidence of worsening neurological deficits. While the overall prognosis is generally favourable, severe cases may result in permanent disability. Early diagnosis and prompt treatment are crucial in optimising recovery outcomes for suspected cases. This patient requires ongoing monitoring for as long as neurological deficits persist, along with continuous medical management. Additionally, the postoperative onset of disability likely placed the patient in a challenging situation. As a holistic approach to the management of the patient, referral to social welfare services for physiotherapy and social support is essential to facilitate rehabilitation and improve quality of life.

4.0 CONCLUSIONS

This case highlights the complexities of diagnosing prolonged neurological deficits following spinal anaesthesia, particularly in patients with pre-existing spinal pathology. Although spinal anaesthesia is generally safe, thorough preoperative evaluation and risk assessment are crucial in minimising complications. The presence of chronic spinal conditions may obscure the differentiation between anaesthesia-related neurological complications and underlying pathology, necessitating a multidisciplinary approach. Early postoperative monitoring, timely imaging, and collaborative management with neurologists and orthopaedic specialists are essential for optimal patient outcomes. This case underscores the importance of comprehensive history-taking and individualised anaesthetic planning to enhance patient safety and improve postoperative recovery.

5.0 CONFLICT OF INTEREST

The authors declare no conflict of interest.

*Corresponding Author | Abdul Razak, N. | nadiawati@upnm.edu.my

6.0 AUTHORS' CONTRIBUTION

Fauzi, S. H. H. (Conceptualization; Literature review; Clinical data collection; Writing - original draft)
Abdul Razak, N. (Literature review; Writing - critical revision of the article for important intellectual content, Resources; Supervision)

7.0 ACKNOWLEDGEMENTS

The authors would like to extend special gratitude to the National Defence University of Malaysia (NDUM) for having access to cases for this case report.

List of Reference

[1] Brown, D. L. (2010). Spinal, epidural, and caudal anesthesia. *Miller's anesthesia*, 1611-1638.
[2] Hadzic, A. (Ed.). (2017). *Hadzic's textbook of regional anaesthesia and acute pain management* (pp. 568-573). New York: McGraw-Hill Education.
[3] Aldrete, J. A. (2003). Neurologic deficits and arachnoiditis following neuroaxial anesthesia. *Acta anaesthesiologica scandinavica*, *47*(1), 3-12.
[4] Faccenda, K. A., & Finucane, B. (2001). Complications of regional anaesthesia: incidence and prevention. *Drug safety*, *24*, 413-442.
[5] Liu, L. L., & Larson, M. D. (2022). Patient safety during anaesthesia: 100 years of progress documented in Anaesthesia & Analgesia. *Anesthesia & Analgesia*, *135*(2S), S37-S47.
[6] https://www.nysora.com/ (last accessed on 10 March 2025)
[7] Hewson, D. W., Bedforth, N. M., & Hardman, J. G. (2018). Spinal cord injury arising in anaesthesia practice. *Anaesthesia*, *73*, 43-50.
[8] Kitazaki, Y., Ueno, A., Maeda, K., Asano, R., Satomi, H., Nishio, T., ... & Hamano, T. (2020). A case of longitudinally extensive transverse myelitis with an isolated pontine lesion following epidural and spinal anaesthesia for cesarean section. *Eneurologicalsci*, *21*, 100264.
[9] Berman, M., Feldman, S., Alter, M., Zilber, N., & Kahana, E. (1981). Acute transverse myelitis: incidence and etiologic considerations. *Neurology*, *31*(8), 966-966.
[10] Krishnan, C., Kaplin, A. I., Pardo, C. A., Kerr, D. A., & Keswani, S. C. (2006). Demyelinating disorders: update on transverse myelitis. *Current neurology and neuroscience reports*, *6*(3), 236-243.
[11] Kaplin, A. I., Krishnan, C., Deshpande, D. M., Pardo, C. A., & Kerr, D. A. (2005). Diagnosis and management of acute myelopathies. *The neurologist*, *11*(1), 2-18.
[12] West, T. W., Hess, C., & Cree, B. A. (2012, April). Acute transverse myelitis: demyelinating, inflammatory, and infectious myelopathies. In *Seminars in Neurology* (Vol. 32, No. 02, pp. 097-113). Thieme Medical Publishers.
[13] Transverse Myelitis Consortium Working Group*. (2002). Proposed diagnostic criteria and nosology of acute transverse myelitis. *Neurology*, *59*(4), 499-505.

*Corresponding Author | Abdul Razak, N. | nadiawati@upnm.edu.my