JKKI
SCIENTIA EST BASIC VITAE

JKKI: Jurnal Kedokteran dan Kesehatan Indonesia
Indonesian Journal of Medicine and Health
Journal homepage: https://journal.uii.ac.id/JKKI
P-ISSN 2085-4145 | E-ISSN 2527-2950

Cauda equina syndrome successfully treated with microdiscectomy and methylprednisolone therapy: A case report

Alhoi Hendry Henderson*[1], Sabri Ibrahim[2]

[1]Bunda Thamrin General Hospital, Medan, Sumatera Utara, Indonesia
[2]Department of Neurosurgery, Faculty of Medicine, Universitas Sumatera Utara/Haji Adam Malik General Hospital, Medan, Sumatera Utara, Indonesia

Case Report

ARTICLE INFO

Keywords:
Cauda equina syndrome, decompression, microdiscectomy, methylprednisolone.

***Corresponding author:**
hendersonhendry96@gmail.com

DOI: 10.20885/JKKI.Vol15.Iss2.art16

History:
Received: February 18, 2024
Accepted: August 23, 2024
Online: August 27, 2024

ABSTRACT

Cauda equina syndrome (CES) caused by lumbar disc herniation is a rare neurosurgical emergency in young adults that requires immediate decompression. A surgical delay over 48 hours may result in irreversible ramifications, underscoring a crucial timeframe for managing this condition. We presented a 26-year-old man with lower limb weakness persisting for one week, accompanied with exacerbating bowel and bladder issues after massage. The patient had a history of low back pain and tingling sensations radiating to the left leg over the past year. His physical examination showed right motor strength of 5/4 and left motor strength of 5/3, with weakened bowel peristalsis, loose anal sphincter and saddle anaesthesia. A spinal MRI revealed protruded discs extending to the intervertebral levels of L4-L5 and L5-S1, implicating herniated discs as the likely cause of CES. He underwent a microdiscectomy with a posterior approach, and intravenous methylprednisolone was administered before the procedure. The next surgery demonstrated enhancements in motor strength, bowel and bladder function, and it also reduced pain. He was discharged with persistent tingling sensations in his both legs. We concluded that CES represents a critical neurosurgical emergency, which is diagnosed through clinical symptoms and neuroimaging. Rapid decompression and intravenous administration of methylprednisolone are associated with better prognosis.

INTRODUCTION

Cauda equina syndrome (CES) represents a neurosurgical emergency marked by compression of spinal roots within the lower lumbar region of the spine.[1,2] The aetiology of CES encompasses extruded disc herniation, hematoma, tumour lesions, vertebral fractures, canal stenosis, infection, surgical manipulation, spinal anaesthesia, ankylosing spondylitis, and gunshot wounds.[3,4] The incidence of lumbar disc herniation among young adults diagnosed with CES is estimated to range from 1% to 6%.[5]

The incidence of CES varies from 1:33,000 to 1:100,000 individuals.[4] Symptoms of CES may comprise lower back pain, lower motor weakness, sensory dysfunction in the saddle area (also recognized as saddle anaesthesia), and issues with sexual, balder, or rectal function.[6] Magnetic Resonance Imaging (MRI) is a gold standard for confirming its diagnosis and elucidating the topography and aetiology of compression in CES.[4] Surgical decompression is a recommended treatment for CES and can be performed by using approaches like total laminectomy, hemilaminectomy, or endoscopic surgery.6 Early surgical decompression within 24 hours of symptom onset may help to avoid long-term bladder, bowel, and sexual problems.[7] Some evidence suggests that in CES outcomes worsen when surgery is delayed beyond 48 hours.[8]

The efficacy of steroids in treating acute CES remains unclear.[9] Corticosteroids are thought to alleviate pain by diminishing inflammation through the inhibition of phospholipase A2 (PLA2)

activity and by impeding the transmission of nociceptive C-fibre input.[10] In addition to blocking inflammatory mediators, corticosteroids also induce anti-inflammatory agents.[11]

This case presents a 26-year-old man with CES caused by lumbar disc herniation whose symptoms improve, followed by surgical decompression by using posterior approach microdiscectomy method combined with intravenous methylprednisolone administration. It aims to document the aetiology, the treatment progression, and enhanced outcomes after the surgery in patients with CES presented after the critical 48-hour window.

CASE DESCRIPTION

A 26-year-old man visited our emergency department with lower limb weakness, lower back pain, and problems with bowel and bladder function persisting for one week. He reported a one-year history of low back pain radiating to his left leg, along with recent tingling sensations in the same leg. One week earlier, he had undergone a massage; after that, his lower back pain intensified and extended to both legs, accompanied by tingling sensations and weakness. He noted greater weakness in his left legs, impeding his mobility. The patient also experienced numbness in the perianal area and encountered difficulties with defecation and urination. Living in a different province, he endured several hours of travel to reach the emergency department.

On the physical examination, he was awake and alert with a Glasgow Coma Scale (GCS) E4M6V5. The recorded vital signs were as follows: blood pressure of 120/80 mmHg, heart rate of 87 beats per minute, respiratory rate of 20 breaths per minute, and temperature of 36.6^0C. The examination of his head, chest, and cardiac was normal. He exhibited a right motor strength of 5/4 and left motor strength of 5/3, along with weakened bowel peristalsis, loose anal sphincter, and saddle anaesthesia. Additionally, the examination revealed the absence of pathological reflexes, decreased physiological reflexes, and absence of tone and clonus. He was reported to have pain levels of 8/10 on the visual analog scale without provocation and 9/10 with provocation.

The spinal MRI revealed the presence of herniated discs at intervertebral levels of L4-L5 and L5-S1, exerting pressure on the spinal cord and compressing both the spinal canal and roots, consequently causing spinal canal stenosis (Figure 1).

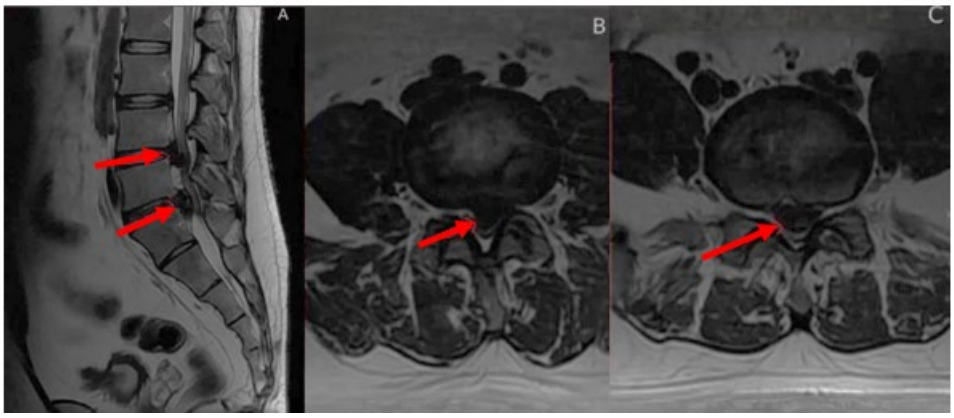

Figure 1. Sagittal view (A) and axial view (B)(C) of spinal magnetic resonance imaging of the herniated discs as high as intervertebral L4-L5 and L5-S1 (red arrow)

He underwent a microdiscectomy by using a posterior approach. Under general anaesthesia, he was positioned prone, and his lower back was disinfected with povidone-iodine of 10% and draped with sterile covers. A midline incision was made from L4 to S1, followed by a dissection of the paraspinal muscles. Laminectomy was performed at L4/L5/S1. The disc at the L4/L5 and L5/S1 levels was excised through a discectomy under a microscope (Figure 2).

Intravenous methylprednisolone, 125 mg every 8 hours, was administered before the procedure and continued for one day afterward. Starting from the second day after surgery, the intravenous methylprednisolone dosage was adjusted to 125 mg every 12 hours until five days following the surgery.

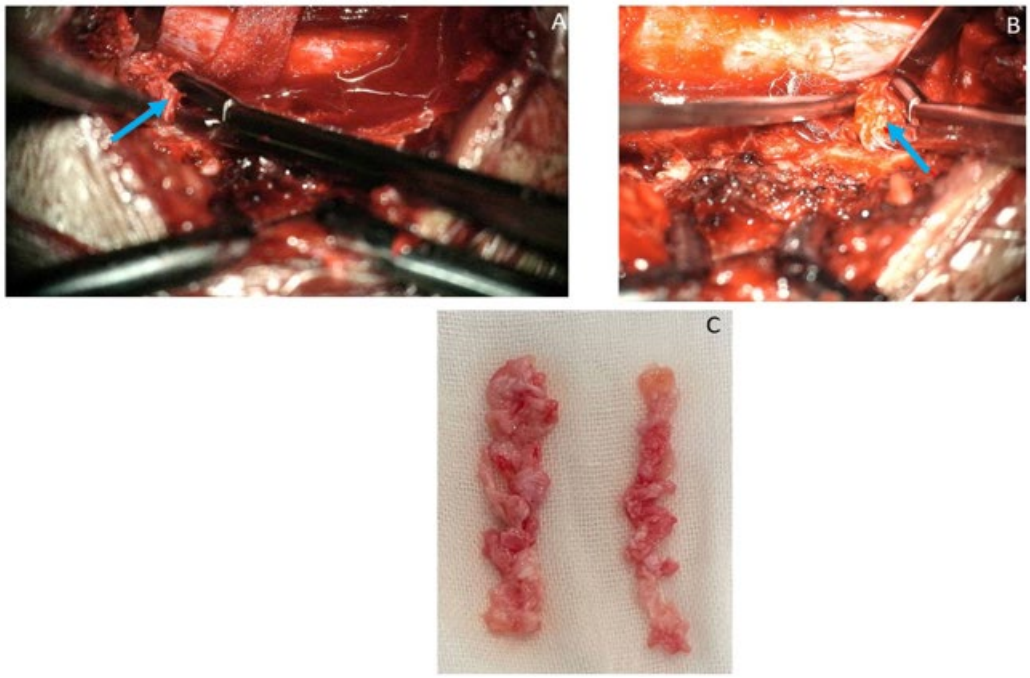

Figure 2. Excision of the L4/L5 disc during microdiscectomy (blue arrow) (A) Excision of the L5/S1 disc during microdiscectomy (blue arrow) (B) The protruded discs after microdiscectomy (C)

Additionally, he was administered by intravenous ketorolac at a dosage of 30 mg every 8 hours, intravenous ranitidine at a dosage of 50 mg every 12 hours, intravenous ceftriaxone at a dosage of 1 g every 12 hours, and intravenous gentamicin at a dosage of 80 mg every 8 hours. A day after surgery, the patient's lower back pain had reduced to 5/10 on the visual analog scale, and their lower limb strength had improved to 5/4 on both sides. However, he still reported tingling sensations in both legs. On the following day, his pain further decreased to 3/10 on the visual analog scale. He could sit comfortably without the need to strain during defecation or urination. By the fourth day after surgery, he reported complete pain relief, and his motor strength improved to 5/5 in both legs. Although able to walk, tingling sensations persisted in his both legs. He was discharged from the hospital five days after the surgery.

DISCUSSION

Cauda equina syndrome represents a neurosurgical emergency arising from significant compression of the lower sacral nerve root due to complete or near-complete occlusion of the spinal canal.[1,12] The CES can result from various factors occurring at the L4/L5 or L5/S1 level.[3,4] Herniated discs at the L4-L5 level are the most common cause of CES, contributing to only 2% of cases.[13,14] The incidence of CES is higher in women and people aged over 30 years old.[2,4] In this study, the patient was a man aged under 30 years, with a herniated disc identified as the underlying cause of his CES.

The pathophysiology of CES involves the compression of various components of the cauda equina.[13] Cauda equina syndrome typically arises from nerve root compression due to lumbar disc herniation. Compression-induced functional changes involve mechanical nerve fibre deformation, microcirculation restriction, local ischemia, intraneural oedema, and subsequent Wallerian degeneration.[15] Ischemic injury is an important factor in the pathophysiology of CES. The main vascular supply to the spinal cord comes from the anterior spinal artery and the dorsolateral arteries. The arteries supplying the nerve roots are less well-defined. A U-shaped hypovascular area below the level of the conus medullaris correlates with the vascular anastomosis in the cauda equina. This hypovascular area forms an anatomical basis for neuro-ischemic manifestations in CES. The nerve roots of the cauda equina are prone to injury from compression or traction. These nerve roots are autonomic and lack a Schwann cell sheath.[16]

Based on the speed of symptom onset, CES is classified into three groups: rapid onset without a prior history of back issues, acute bladder dysfunction with preceding low back pain and sciatica, and chronic backache and sciatica with progressively developing CES, frequently

associated with canal stenosis.[17] According to clinical presentation, CES is classified into two types: CES characterized by urinary retention and incomplete CES with difficulty in urination or loss of urinary urge without retention.[18] In incomplete CES, some patients experience urinary difficulties of neurogenic factors, including altered urinary sensation, reduced desire to void, weak urinary stream, and the need to strain during micturate. Partial or unilateral saddle and genital sensory deficits occur with intact trigone sensation. Complete syndrome (CES-retention) is marked by extensive or complete saddle and genital sensory deficits along with impaired trigone sensation.[17]

Symptoms suggestive of CES diagnosis include severe low back pain, sensory deficits, motor weakness, diminished lower limb reflexes, saddle anaesthesia, recent onset of bladder dysfunction, faecal incontinence, and sexual dysfunction.[7,13,19] The MRI is the gold standard for diagnosing CES and effectively differentiates between CES caused by disc pathology and by other conditions such as infection or malignancy.[19] In this study, the patient had several red flag symptoms indicative of CES, and the MRI demonstrated compression of the cauda equina root by a herniated disc.

Surgery is a primary treatment for CES, typically involving a wide laminectomy and extensive decompression of the nerves. Following decompression, the removal of disc space material is commonly performed. This surgical approach aims to alleviate compression on the nerves, addressing the underlying cause of CES and potentially improving the patient outcomes.[7] Surgical decompression, encompassing both minimally invasive and open surgical decompression, is employed in the treatment of CES.6 The standard treatment for CES caused by a herniated disc includes lumbar decompression along with the removal of either the disc fragments (sequestrectomy) or the entire disc (discectomy).[18]

A study by Deniz and colleagues reported that earlier intervention (within 24 hours of symptom onset) could result in improved outcomes.[13] Delayed surgery beyond 48 hours may increase risks of permanent bladder damage, sexual dysfunction, chronic pain, and bowel dysfunction.[7] According to a study by Lai et al.,[20] patients with complete CES who underwent delayed decompression indicated notable improvement after surgery, whereas those with incomplete CES were able to resume their regular activities. Lim et al. stated that patients who underwent delayed decompression for CES achieved complete recovery seven weeks after surgery.[21] Surgical intervention should be recommended for all patients with CES, whether it is complete or incomplete, to reduce morbidity and to prevent complications. Early surgery is crucial to alleviate nerve pressure, improve recovery outcomes, and minimize the risk of permanent neurological damage.[22]

However, some authors argue that emergency decompression surgery may not significantly improve outcomes in CES compared to a delayed approach and may even cause more harm when performed under suboptimal emergency conditions.[21] Sulaiman mentioned that more than 80% of patients with delayed surgery did not experience significant recovery of bladder symptoms, indicating that delayed decompression should not be postponed.[23] Nonetheless, early surgery is still recommended to prevent incomplete CES from progressing to complete CES, which involves total loss of sphincter control, motor function, and sensory function.[21]

Several factors could elucidate the functional improvement observed in patients after delayed decompression for CES. Firstly, in cases of progressing CES, the compressed nerve roots may progressively adapt to hypoxic conditions, potentially preserving the ability for functional recovery despite the delayed decompression. Secondly, being peripheral nerves, the cauda equina nerves have ability for regeneration and may recover from reversible injuries induced by compression. Consequently, cauda equina root damage may not be inherently irreversible when the decompression is delayed.[20] Neurological deficits become irreversible when intraspinal pressure exceeds a critical level for a sustained period. Therefore, the intraspinal pressure exceeds with a long period may cause significant impais recovery. Timely intervention is essential to prevent permanent damage and optimize outcomes in patients with CES.[24] In this study, the patient with late decompression experienced reduced of low back pain, enhanced motor strength, and ameliorated bladder and bowel dysfunction, although with a persistent tingling sensation in both legs.

In our case, the patient received intravenous methylprednisolone both before and after surgery,

demonstrating its efficacy in improving clinical outcomes. A study by Waqas et al. reported that intravenous methylprednisolone administration could alleviate postoperative back and leg pain.[25] Corticosteroids alleviate inflammation by inhibiting the synthesis or release of diverse proinflammatory substances, inducing reversible local anaesthetic effects and diluting chemical irritants. Corticosteroids accomplish this by inhibiting the synthesis of neural peptides, blocking PLA2 activity, prolonging the suppression of ongoing neuronal discharge, and suppressing of sensitization of dorsal horn neurons.[26] Wu et al. pointed out that patients who received intravenous methylprednisolone perioperatively showed reduced length of hospital stay.[27] Xie et al. administered intravenous methylprednisolone at a dosage of 80 mg every 12 hours before surgery. This dosage was reduced to 40 mg every 12 hours three days after surgery and further decreased to 40 mg on the fifth day after surgery. Methylprednisolone was employed to mitigate postoperative oedema of the spinal nerve root and for its anti-inflammatory properties.[28]

CONCLUSION

CES is a neurosurgical emergency resulting from compression of the cauda equina nerve roots, often due to a herniated disc in the lower back. Early decompression surgery is recommended to achieve optimal outcomes as it alleviates nerve pressure and enhances the post surgery outcome. However, even delayed decompression can provide significant benefits, reduce the risk of permanent nerve damage and improving patient symptoms. Additionally, intravenous methylprednisolone can be administered to help reduce inflammation, thereby shortening the duration of hospitalization and alleviating pain after surgery. This anti-inflammatory treatment help to minimize swelling around the compressed nerves, promoting a quicker recovery.

CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest.

ACKNOWLEDGEMENTS

The authors thank the Director of Bunda Thamrin General Hospital for their support and permission during this case report. The authors also extend gratitude to the medical staffs of Bunda Thamrin Hospital for their assistance with the patient treatment. Then the patient provided written informed consent for the therapy and this publication.

AUTHOR CONTRIBUTIONS

AHH wrote the manuscript. SI wrote the manuscript, reviewed it, and gave his final approval.

LIST OF ABBREVIATIONS

CES: Cauda equina syndrome; MRI: Magnetic Resonance Imaging, GCS: Glasgow Coma Scale, E: Eye, M: Movement, V: Verbal, PLA2: Phospholipase A2.

REFERENCES

1. Lavy C, Marks P, Dangas K, Todd N. Cauda equina syndrome—A practical guide to definition and classification. Int Orthop. 2022;46(2):165–9. DOI:10.1007/s00264-021-05273-1.
2. Buell KG, Sivasubramaniyam S, Sykes M, Zafar K, Bingham L, Mitra A. Expediting the management of cauda equina syndrome in the emergency department through clinical pathway design. BMJ Open Qual. 2019;8(4):8–13. DOI:10.1136/bmjoq-2018-000597.
3. Barraclough K. Cauda equina syndrome. BMJ. 2021;372:6–10. DOI:10.1136/bmj.n32.
4. Dias ALN, Araújo FF de, Cristante AF, Marcon RM, Barros Filho TEP de, Letaif OB. Epidemiology of cauda equina syndrome. What changed until 2015. Rev Bras Ortop. 2018;53(1):107–12. DOI:10.1016/j.rboe.2017.11.006.
5. Basol N, Deniz FE, Gokce E, Sahin F. A young patient with acute cauda equina syndrome due to traumatic disc herniation. JAEMCR. 2015;6(4):81–3. DOI:10.5152/jaemcr.2015.1156.
6. Yankang L, Leiming Z, Lewandrowski KU, Xiangyu T, Zexing Z, Jianbiao X, et al. Full endoscopic lumbar discectomy versus laminectomy for cauda equina syndrome. Int J Spine Surg. 2021;15(1):105–12. DOI:10.14444/8014.
7. Kapetanakis S, Chaniotakis C, Kazakos C, Papathanasiou J V. Cauda equina syndrome due to lumbar disc herniation: A review of literature. Folia Med. 2017;59(4):377–86. DOI:10.1515/folmed-2017-0038.

8. Attabib N, Kurban D, Cheng CL, Rivers CS, Bailey CS, Christie S, et al. Factors associated with recovery in motor strength, walking ability, and bowel and bladder function after traumatic cauda equina injury. J Neurotrauma. 2021;38(3):322–9. DOI:10.1089/neu.2020.7303.
9. Vaishya S, Pojskic M, Bedi MS, Oertel J, Sippl C, Robertson S, et al. Cauda equina, conus medullaris and syndromes mimicking sciatic pain: WFNS spine committee recommendations. World Neurosurg X. 2024;22:100274. DOI:10.1016/j.wnsx.2024.100274.
10. Rajkumari K, Singh AJ, Singh LN, Chabungbam M, Sreejith C, Janet M, et al. Transforaminal epidural injection of dexamethasone vs methylprednisolone in reducing low back pain and disability in prolapsed lumbar intervertebral disc in Manipur, India: A RCT. JCDR. 2021;15(9):6–9. DOI:10.7860/jcdr/2021/48704.15370.
11. Tihista M, Gu A, Wei C, Weinreb JH, Rao RD. The impact of long-term corticosteroid use on acute postoperative complications following lumbar decompression surgery. J Clin Orthop Trauma. 2020;11(5):921–7. DOI:10.1016/j.jcot.2020.04.010.
12. Zusman NL, Radoslovich SS, Smith SJ, Tanski M, Gundle KR, Yoo JU. Physical examination is predictive of cauda equina syndrome: MRI to rule out diagnosis is unnecessary. Global Spine J. 2022;12(2):209–14. DOI:10.1177/2192568220948804.
13. Deniz K, Pararajasingham J. Cauda equina syndrome. InnovAiT. 2011;4(10):551–5. DOI:10.1093/innovait/inq205.
14. Greenberg MS. Handbook of neurosurgery. 9th ed. Handbook of neurosurgery. Rio de Janeiro: Thieme Medical Publisher; 2020. eISBN 978-1-68420-138-9.
15. Aziz S, Newton-ede M. Cauda equina syndrome - An overview. Journal of Trauma & Orthopedics. 2023;11(1):50–2. https://www.boa.ac.uk/static/3a39535d-8d31-4eb7-9c6b369672da8c0e/Cauda-equina-syndrome-an-overview.pdf
16. Airlangga PA, Arifin. Thoracic spine canal stenosis with cauda equine syndrome: Case report. Journal Orthopaedi and Traumatology Surabaya. 2019;8(2):86–92. https://doi.org/10.20473/joints.v8i2.2019.86-92.
17. Wani A, Thoker A. Cauda equina syndrome: A simple entity with lot of enigma! Indian Journal of Neurosurgery. 2018;07(03):173–5. DOI:10.1055/s-0038-1676902.
18. Shivji F, Tsegaye M. Cauda equina syndrome: The importance of complete multidisciplinary team management. BMJ Case Reports. 2013;1–4. https://doi.org/10.1136/bcr-2012-007806.
19. Polsky Z, Greidanus M, Pandya A, Bradley Jacobs W. Just the facts: Risk stratifying nontraumatic back pain for cauda equina syndrome in the emergency department. CJEM. 2020;22(5):652–4. DOI:10.1017/cem.2020.382.
20. Lai XW, Li W, Wang JX, Zhang HJ, Peng HM, Yang DH. Delayed decompression for cauda equina syndrome secondary to lumbar disc herniation: long-term follow-up results. Nan Fang Yi Ke Da Xue Xue Bao (Journal of Southern Medical University). 2017;37(9):1143–8. DOI:10.3969/j.issn.1673-4254.2017.09.01.
21. Lim SM, Johari J, Yusof MI. Outcome of delayed decompression surgery for cauda equina syndrome secondary to lumbar disc herniation: A case report. IMJM. 2019;18(2):113–6. DOI: https://doi.org/10.31436/imjm.v18i2.88
22. Miller JK, West J, Khawar H, Middleton R. Cauda equina syndrome. Br J Hosp Med. 2023;84(11):1–7. DOI:10.12968/hmed.2023.0012.
23. Sath S. Does surgical decompression alleviate neglected cauda equina syndromes attributed to lumbar disc herniation and/or degenerative canal stenosis? Surg Neurol Int. 2020;11(278). DOI: 10.25259/SNI_512_2020
24. Viswanathan VK, Shanmuganathan R, Aiyer SN, Kanna R, Shetty AP. Factors affecting early and 1-year motor recovery following lumbar microdiscectomy in patients with lumbar disc herniation: A prospective cohort review. Asian Spine J. 2018;13(1):135–45. DOI:10.31616/asj.2018.0111.
25. Waqas M, Shallwani H, Shamim M, Ahmad K. Perioperative steroids for lumbar disc surgery: A meta-analysis of randomized controlled trials. Surg Neurol Int. 2017;8:42 DOI:10.4103/sni.sni_478_16.
26. Shahien R, Wiegler KB, Dekel L, Sharabi-Nov A, Saleh SA. Retrospective study assessing the efficacy of i.v. dexamethasone, SNRB, and

nonsteroidal treatment for radiculopathy. Medicine (Baltimore). 2022;101(28):e29272. DOI:10.1097/MD.0000000000029272.
27. Wu H, Wang H, Liu Y, Wu Z. Can preoperative intravenous corticosteroids administration reduce postoperative pain scores following spinal fusion?: A meta-analysis. J Invest Surg. 2020;33(4):307–16. DOI:10.1080/08941939.2018.1505983.
28. Xie LI, Zhu J, Chen XD, Yang CY, Yan ZI, Dan P. Potential risk of impertinent administration of methylprednisolone in lumbar spine surgery: A case report. Int J Surg Case Rep. 2020;75:246–51. DOI:10.1016/j.ijscr.2020.09.061.