Rehabilitation Practice and Science

Volume 2023 | Issue 1 Article 3

6-1-2023

Caudal Epidural Block Relieves Low Back Pain in Guillain-Barré Syndrome: A Case Report

Yu-Shan Fu
Department of Physical Medicine and Rehabilitation, Shin Kong Wu Ho-Su Memorial Hospital, Taipei 11101, Taiwan

Lin-Fen Hsieh
School of Medicine, Fu Jen Catholic University, New Taipei City 24205, Taiwan, M001026@ms.skh.org.tw

Recommended Citation
Fu, Yu-Shan and Hsieh, Lin-Fen (2023) "Caudal Epidural Block Relieves Low Back Pain in Guillain-Barré Syndrome: A Case Report," *Rehabilitation Practice and Science*: Vol. 2023: Iss. 1, Article 3.
DOI: 10.6315/TJPMR.202306_2023(1).0003
Available at: https://rps.researchcommons.org/journal/vol2023/iss1/3

Case Report

Caudal Epidural Block Relieves Low Back Pain in Guillain-Barré Syndrome: A Case Report

Yu-Shan Fu[1], Lin-Fen Hsieh[1,2]

[1]Department of Physical Medicine and Rehabilitation, Shin Kong Wu Ho-Su Memorial Hospital, Taipei 11101, Taiwan;
[2]School of Medicine, Fu Jen Catholic University, New Taipei City 24205, Taiwan.

A 65-year-old man reported severe low back pain in Guillain–Barré syndrome. Although adequate doses of oral medications (75 mg of pregabalin three times a day, 300 mg of oxcarbazepine before sleep, and 300 mg of oxcarbazepine twice a day) and physical modalities (electrical nerve stimulation and a hot pack) were administered, his low back pain improved slightly, with his Numerical Rating Scale score decreasing from 10 to 8. In response, an ultrasound-guided caudal epidural block with 40 mg of methylprednisolone, 2 mL of 1% xylocaine, and 18 mL of normal saline was performed on day 78 after onset, resulting in a considerable improvement in the low back pain, with his score of Numerical Rating Scale decreasing from 8 to 3. No adverse effects were noted after the intervention, and his pain was controlled successfully without the requirement of further injection. This study presented the first documented case of caudal epidural block considerably relieving low back pain in Guillain–Barré syndrome. Ultrasound-guided caudal epidural block was noted to be an excellent choice for treating low back pain in Guillain–Barré syndrome if oral medications fail to relieve the symptom. (Rehabil Pract Sci 2023; 2023(1): 21 - 27)

Key Words: Guillain–Barré syndrome, low back pain, caudal epidural block, ultrasound

INTRODUCTION

Guillain-Barré syndrome (GBS), an inflammatory disease of the peripheral nervous system, is the most common cause of acute flaccid paralysis. At least one-third of patients with GBS suffer from severe pain 1 year after disease onset, and the pain can persist for >10 years.[1] Several treatments exist for GBS-associated pain, including oral medication,[2] peripheral nerve stimulation with implanted electrodes,[3] and transcutaneous electric nerve simulation (TENS).[4] The frequency of low back pain in GBS ranges from 13% to 62%.[5] The pathophysiology of low back pain in GBS is likely multifactorial.[5] Affected nerve roots may explain the occurrence of radicular nociceptive nerve pain in the low back with radiation to the extremities or the trunk. Other pathophysiologies of pain include inflammatory factors that generate pain via the nervi nervorum. Other types of pain, such as myalgia and arthralgia, may also exacerbate back pain in GBS.[6]

Limited data exist on the efficacy of caudal block in

Submitted date: 21 March 2022 Revised date: 10 April 2022 Accepted date: 24 April 2022
Correspondence to: Dr. Lin-Fen Hsieh; Department of Physical Medicine and Rehabilitation, Shin Kong Wu Ho-Su Memorial Hospital, B1F., No. 95, Wen Chang Road, Shih Lin District, Taipei City, Taiwan (R.O.C.).
Tel： E-mail：M001026@ms.skh.org.tw; reh6110@yahoo.com.tw. doi: 10.6315/TJPMR.202306_2023(1).0003

treating low back pain associated with GBS. In this study, a case of GBS complicated by quadriparesis and severe low back pain successfully managed using a caudal block was presented.

CASE REPORT

A 65-year-old man was admitted to the emergency department for acute onset of progressive ascending quadriparesis and dyspnea. His neurological manifestations began with numbness in his right toes 1 month before admission to the hospital. Three days after the numbness initiated, the numbness progressed from his right toes to his right thigh, followed by right leg weakness another 2 days later. Left upper and lower limb weakness, mild dyspnea, and dysphagia were noted 12 days after onset.

Brain magnetic resonance imaging (MRI) revealed no acute infarct. The patient developed ascending numbness up to the nipples at 22 days after onset. On that day, a nerve conduction study (NCS) revealed no abnormalities. Because of the worsening dyspnea, quadriparesis, low back pain, and numbness across the trunk and four limbs, he was admitted to the neurology ward for further examination. No recent history of fever, chills, dizziness, vomiting, diarrhea, or symptoms of common cold was noted. The patient denied having slurred speech, diplopia, loss of consciousness, tongue deviation, or facial palsy, and had no urinary or fecal incontinence.

On physical examination, the patient had clear consciousness and was afebrile, with blood pressure of 129/76 mmHg. A muscle strength examination revealed weakness in the four limbs with a Medical Research Council scale score of 2 (right)/4 (left) and 3/4 in the proximal and distal parts of the upper extremities, respectively, and 2/2 and 3/4 in the proximal and distal parts of the lower extremities, respectively. Deep tendon reflexes were absent in the four limbs. A lumbar puncture revealed an elevated total protein level (190 mg/dL). Follow-up NCS at 27 days after onset revealed the absence of bilateral tibial H reflexes and prolonged F-wave latencies of bilateral median nerves, reflecting acute proximal demyelination (Table 1). Motor and somatosensory evoked potentials showed poor waveforms, but the central conduction time was within the normal range. Spine MRI (Figure 1) revealed neither myelitis lesions nor spinal cord compression. Lumbar X-ray showed marginal spurs, reduction of intervertebral space, scoliosis, and osteoporotic change (Figure 2). A second lumbar puncture at 36 days after the onset revealed increased total protein levels (230 mg/dL). Therefore, plasmapheresis was performed for five sessions between 37 and 46 days after disease onset. After treatment, the patient's muscle power improved, and the numbness in his four limbs subsided. A follow-up NCS on day 48 revealed prolonged distal latency and slowed conduction velocity in the nerves in four limbs (Table 2). However, severe low back pain persisted despite taking 75 mg of oral pregabalin three times a day since admission. On day 63, 300 mg of oral oxcarbazepine before sleep was added, and the dose was doubled on day 67 but it had a limited effect on relieving his low back pain. Along with TENS and a hot pack, his low back pain improved, with the Numerical Rating Scale (NRS) score decreasing from 10 to 8. Ultrasound-guided caudal block with 40 mg of methylprednisolone, 2 mL of 1% xylocaine, and 18 mL of normal saline was conducted on day 78 (Figure 3), resulting in considerable relief of low back pain with the NRS score decreasing from 8 to 3. The Oswestry Disability Index dropped from 89.5% to 41.6% after the intervention.

After his discharge on day 80 after symptom onset, follow-ups at every subsequent week via telephone were conducted. One week after the caudal block, his low back pain remained the same. However, on day 12 after the caudal block administration, his pain gradually worsened to an NRS score of 8. On day 22 after the caudal block administration, the pain intensity decreased, with the NRS score lowering to 2, with oral medications unknown. A follow-up NCS study two months after discharge revealed normalized data in the nerves of four limbs (Table 3).

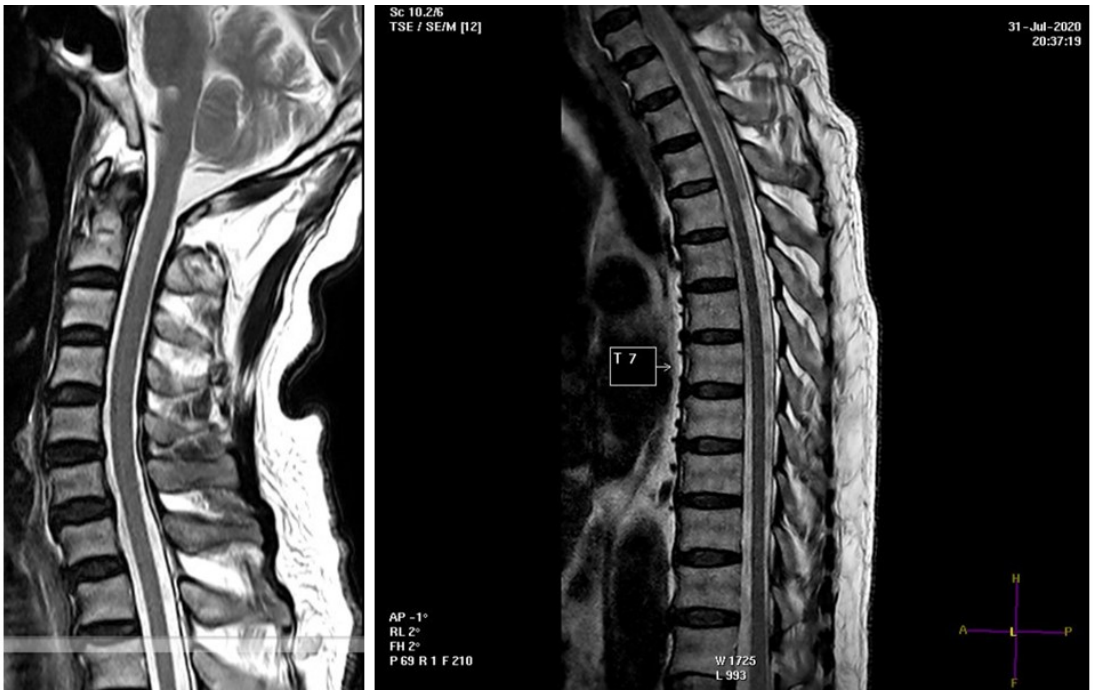

Figure 1. MRI scan without Gadolinium contrast of the cervical and thoracic spine (T2-weighted) showed neither spinal cord enhancement nor spinal cord compression.

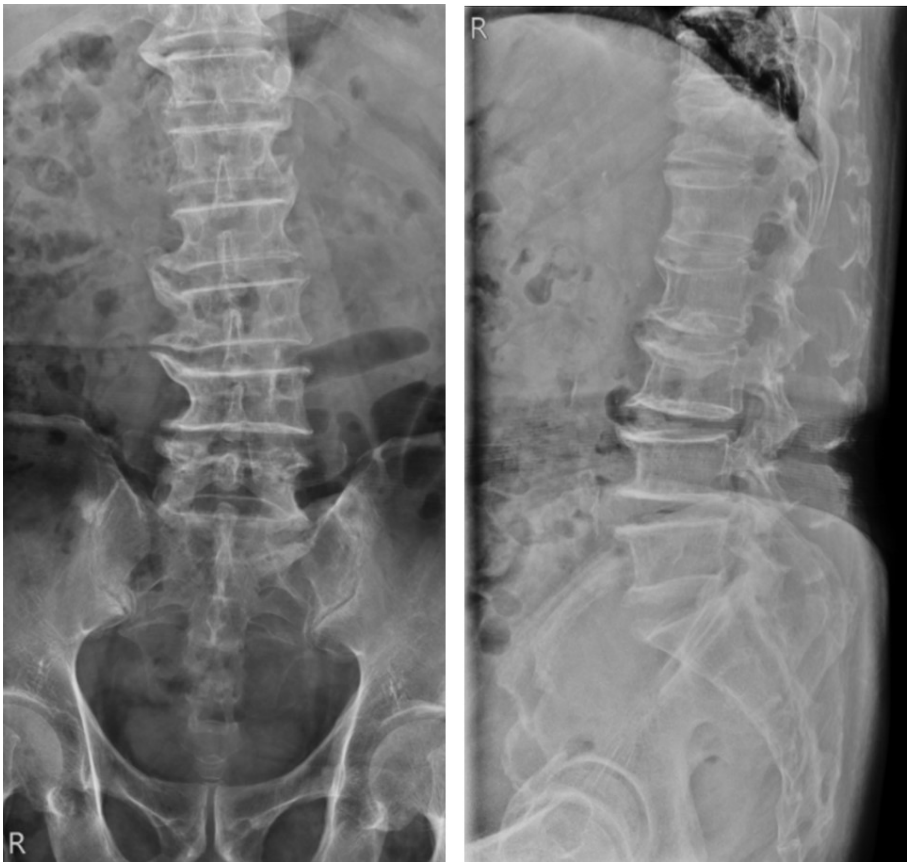

Figure 2. Lumbar X-ray showed marginal spurs, reduction of intervertebral spaces, scoliosis, and osteoporotic change.

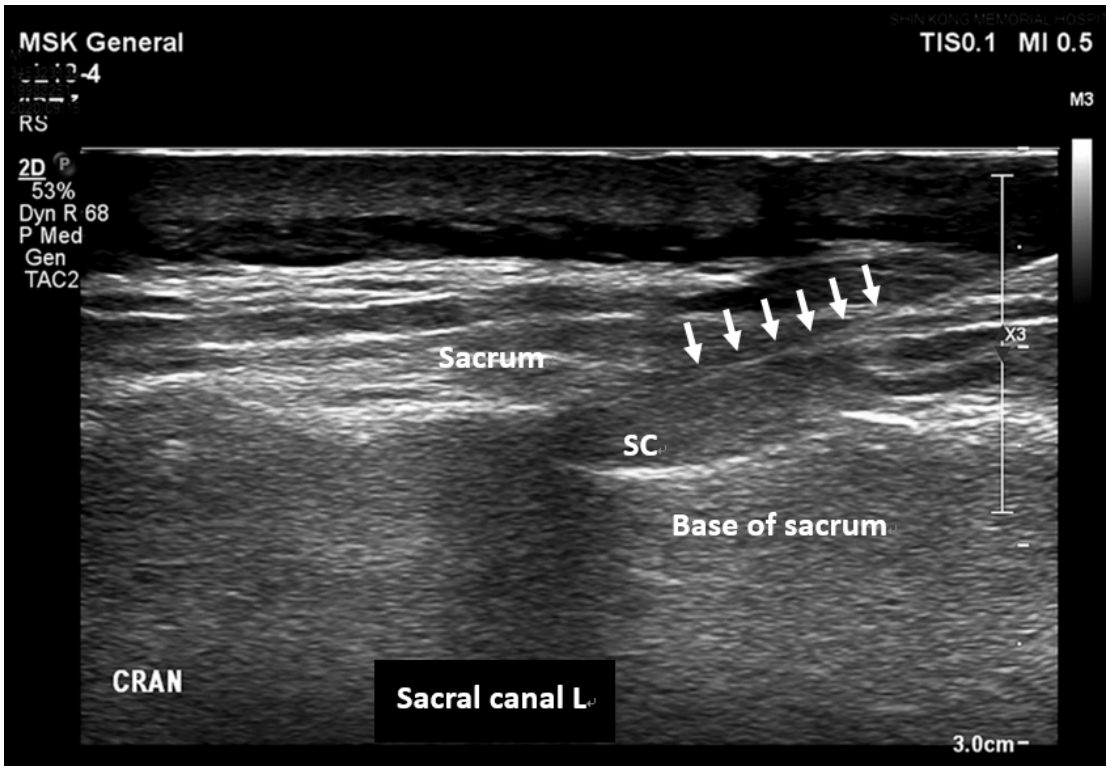

Figure 3. Longitudinal view of the sacral canal during ultrasound-guided caudal epidural block. (White arrow: the needle; SC: sacral canal; CRAN: the cranial side; L: longitudinal view)

Table 1. (2020.7.30, 27 days after symptom onset)

Nerve stimulated	Stimulation site	Recording site	Amplitude (Motor, mV; Sensory, μ V)		Latency(ms)		CV (m/s)		F-wave latency (ms)	
			Rt	Lt	Rt	Lt	Rt	Lt	Rt	Lt
Median (m)	Wrist	APB	8.8	7.7	**4.39**	3.79			**26.1***	**26.8**
	Antecubital fossa	APB	8.6	7.6	8.33	7.58	53.3	55.4		
Ulnar (m)	Wrist	ADM	12.6	10.0	3.29	3.15			24.7	23.1
	Below elbow	ADM	12.3	9.5	7.29	6.34	51.3	61.1		
	Above elbow	ADM	12.3	9.2	9.33	7.96	51.5	61.7		
Median (s)	Wrist	Middle finger	28.2	16.0	3.32	2.76	42.2	50.7		
Ulnar (s)	Wrist	Little finger	31.4	20.0	3.41	2.83	41.1	49.5		
Tibial (m)	Ankle	AHB	16.7	12.6	3.41	3.74			42.1	42.1
	Popliteal fossa	AHB	11.6	8.8	12.1	11.8	47.2	49.0		
Peroneal (m)	Ankle	EDB	5.2	5.2	3.88	3.75			41.1	41.0
	Below fibula	EDB	4.9	4.8	10.3	9.71	49.8	52.0		
	Lateral popliteal fossa	EDB	4.7	4.4	12.3	12.4	50.0	44.6		
Sural (s)	Calf	Posterior ankle	12.0	15.6	2.79	2.68	46.6	48.5		

Follow-up NCS at 27 days after onset revealed the absence of bilateral tibial H reflexes and prolonged F-wave latencies of bilateral median nerves, reflecting acute proximal demyelination. (Abbreviations: m, motor study; s, sensory study; Rt, right; Lt, left; CV, conduction velocity; APB, abductor pollicis brevis; ADM, abductor digiti minimi; AHB, abductor hallucis brevis; EDB, extensor digitorum brevis)

* The boldfaced words represent the abnormal data.

Table 2. (2020.8.20, 48 days after symptom onset)

Nerve stimulated	Stimulation site	Recording site	Amplitude (Moto, mV; Sensory, μ V)		Latency(ms)		CV (m/s)		F-wave latency (ms)	
			Rt	Lt	Rt	Lt	Rt	Lt	Rt	Lt
Median (m)	Wrist	APB	10.5	9.4	**4.63***	**4.33**			**28.2**	**28.3**
	Antecubital fossa	APB	10.4	9.3	8.71	8.21	52.7	54.1		
Ulnar (m)	Wrist	ADM	10.6	8.9	**3.46**	**3.42**			**29.0**	**32.0**
	Below elbow	ADM	10.4	8.9	6.96	7.13	55.7	51.2		
	Above elbow	ADM	10.4	8.7	8.89	9.06	51.8	51.8		
Median (s)	Wrist	Middle finger	26.0	19.2	3.28	3.18	42.7	44.0		
Ulnar (s)	Wrist	Little finger	29.2	26.3	3.24	2.99	43.2	46.8		
Tibial (m)	Ankle	AHB	6.0	8.7	4.63	4.24			**50.6**	**51.0**
	Popliteal fossa	AHB	4.8	5.4	14.1	12.8	**41.2**	46.7		
Peroneal (m)	Ankle	EDB	5.8	4.1	4.97	4.5			45.8	45.7
	Below fibula	EDB	5.4	4.0	12.0	11.0	45.5	45.4		
	Lateral popliteal fossa	EDB	5.4	3.9	14.2	13.5	45.5	44.0		
Sural (s)	Calf	Posterior ankle	11.6	14.8	3.22	3.22	40.4	40.4		

A follow-up NCS on day 48 revealed prolonged distal latency, slowed conduction velocity, and prolonged F-wave latencies in bilateral nerves in four limbs (Abbreviations: m, motor study; s, sensory study; Rt, right; Lt, left; CV, conduction velocity; APB, abductor pollicis brevis; ADM, abductor digiti minimi; AHB, abductor hallucis brevis; EDB, extensor digitorum brevis)

*The boldfaced words represent the abnormal data.

Table 3. (2020.11.25, 4 months after symptom onset; 2 months after discharge)

Nerve stimulated	Stimulation site	Recording site	Amplitude (Motor, mV; Sensory, μ V)		Latency(ms)		CV (m/s)		F-wave latency (ms)	
			Rt	Lt	Rt	Lt	Rt	Lt	Rt	Lt
Median (m)	Wrist	APB	9.2	10.1	**4.42***	4.00			23.7	24.4
	Antecubital fossa	APB	8.9	9.5	8.08	8.13	56.0	50.8		
Ulnar (m)	Wrist	ADM	7.8	8.5	2.88	3.08			23.2	24.9
	Below elbow	ADM	7.3	8.4	6.17	6.69	51.3	51.2		
	Above elbow	ADM	7.3	8.3	7.71	8.17	68.2	67.6		
Median (s)	Wrist	Middle finger	25.5	16.6	2.83	2.96	49.5	47.3		
Ulnar (s)	Wrist	Little finger	17.9	9.9	2.76	2.47	50.7	56.7		
Tibial (m)	Ankle	AHB	**4.0**	6.1	3.92	4.32			44.6	44.6
	Popliteal fossa	AHB	2.5	5.0	12.0	12.0	44.6	47.5		
Peroneal (m)	Ankle	EDB	4.7	3.8	4.46	3.46			43.9	43.4
	Below fibula	EDB	4.2	3.6	10.6	9.58	45.6	44.9		
	Lateral popliteal fossa	EDB	3.4	3.5	12.8	11.6	40.9	54.5		
Sural (s)	Calf	Posterior ankle	8.4	7.9	2.73	2.60	47.6	50.0		

A follow-up NCS study two months after discharge revealed normalized data in the four limbs. (Abbreviations: m, motor study; s, sensory study; Rt, right; Lt, left; CV, conduction velocity; APB, abductor pollicis brevis; ADM, abductor digiti minimi; AHB, abductor hallucis brevis; EDB, extensor digitorum brevis)

*The boldfaced words represent the abnormal data.

DISCUSSION

A caudal epidural block (or caudal block) is a technique used for the management of acute or chronic low back pain unresponsive to conservative medical treatment.[7] Caudal block is performed by placing a needle through the sacral hiatus to deliver medication to the epidural space, providing sensory blockade of the lumbosacral roots. The sacral hiatus is located at the caudal part of the sacrum and is covered only by skin, a subcutaneous fatty layer, and the sacrococcygeal ligament. The dural sac typically terminates between the S1 and S2 vertebrae.[8] The apex of the sacral hiatus is most commonly located at the S4 level. The distance between the dural sac and the apex of the sacral hiatus (average 32 mm, ranging from 5.8 to 60.0 mm)[9] provides a safety zone for needle placement without injuring the dural sac. The mean anterior-posterior diameter of the sacral hiatus at its apex ranges from 4.6 to 6.1 mm. The injection is difficult to perform if the diameter is <1.6 mm.[10] Epidural transfer of the local anesthetic solution is affected by different concentrations and volumes, which determine the onset of the action, the level of the analgesia, and the duration of the sensory blockade. Complications associated with caudal blocks include infection, hypotension, injury to nerve roots, perforation of the rectum, and subdural or intravascular injection. Ultrasound guidance can ensure the correct placement of the needle into the sacral canal, which can improve the clinical outcomes to between 96.9% and 100%.[8] In addition, evaluation of the sacral canal's anterior-posterior diameter before injection can aid in predicting the effects of the treatment. In this case report, an ultrasound-guided caudal epidural block targeted to the hypoechoic structure between the sacrococcygeal ligament and the base of the sacral bone that effectively relieved low back pain in GBS without causing complications was presented (Figure 3). The color Doppler detected unidirectional flow at the lumbar spine, which helps to determine the success of a caudal block.

Regarding the treatment of pain in GBS, a randomized controlled trial in 2002 revealed significantly lower mean pain scores on day 7 in the gabapentin phase compared with the endpoint of the placebo phase.[11] Another study in 2005 showed that participants in the gabapentin group had significantly lower median pain scores on all treatment days compared with those in the placebo and carbamazepine groups.[12] In addition to studies involving oral medication, a case report in 2020 documented a case in which a 22-year-old man's acute neuropathic pain in GBS was successfully relieved via peripheral nerve stimulation by using implanted electrodes.[3] Furthermore, a case report presented successful control of radicular pain with oral prednisolone therapy (0.7 mg/kg/day) in a pediatric patient with GBS.[13] Moreover, two case reports in 2018 described the low back pain considerably alleviated after a standard regimen of intravenous immunoglobulin.[5] Rehabilitation programs, such as TENS and desensitization therapy, were also recommended.[4] This is the first case report to document caudal epidural block relieving pain related to GBS. In this case report, the procedure was performed with ultrasound guidance, ensuring correct placement of the needle at the target place, and it might have contributed to the treatment's effectiveness and safety. Given that nerve roots damaged by the aberrant immune response to infection may explain the presence of radicular nociceptive nerve pain in the low back, the mechanism allowing caudal epidural block to relieve pain in GBS may be that the local anesthetic solution blocks the nerve roots after entering the epidural space.

The possible reason for the recurrence of pain after the initial caudal block in our patient may be the short duration of corticosteroid effect. Although steroids exhibit analgesic effect through blocking the afferent C fiber nociception and reducing the nerve root inflammation, a study revealed that the epidural steroid injection is more effective for relieving lumbosacral radicular pain than the conservative treatments in short-term (≤1 month) and intermediate-term (1 to 3 months), but the effect was not maintained at long-term (6 months to ≤1 year) follow-up.[14] In addition, arthralgia or muscle pain due to immobilization may be a cause of the patient's recurrent low back pain.[5]

Additional studies may compare the safety and efficacy between ultrasound-guided and fluoroscopy-guided caudal epidural blocks. In addition, more experience in the repetition of caudal epidural blocks in cases when pain recurs after the injection should be documented.

CONCLUSION

Caudal epidural block is a suitable choice for the treatment of low back pain in GBS if oral medication fails to relieve the symptoms.

ACKNOWLEDGMENT

Consent was obtained from the patient for this report. This manuscript was edited by Wallace Academic Editing.

REFERENCES

1. Leonhard SE, Mandarakas MR, GondimFAA, et al. Diagnosis and management of Guillain–Barré syndrome in ten steps. Nature reviews Neurology 2019;15:671-83.
2. Liu J, Wang LN, McNicol ED, et al. Pharmacological treatment for pain in Guillain-Barré syndrome. Cochrane Database Syst Rev 2015;4:CD009950.
3. Albright-Trainer B, Provencio JJ, O'Connor CM, et al. A Case Report of Peripheral Nerve Stimulation for Acute Neuropathic Pain in Guillain-Barre Syndrome. A&A Practice 2020;14:e01315.
4. Khan F. Rehabilitation in Guillian Barre syndrome. Aust Fam Physician 2004;33: 1013-7.
5. Ding XJ, Jiang H, Hu XY, et al. Guillain-Barré syndrome and low back pain: two cases and literature review.Open Med (Wars) 2018;13:503-8.
6. L Ruts, J Drenthen, J L M Jongen, et al. Pain in Guillain-Barre´ syndrome. A long-term follow-up study. Neurology 2010, 75:1439-47.
7. Sanghvi C, Dua A. Caudal Anesthesia. StatPearls Publishing 2022. Available from: https://www.ncbi.nlm.nih.gov/books/NBK551693/
8. Kao SC, Lin CS. Caudal Epidural Block: An Updated Review of Anatomy and Techniques. Biomed Res Int 2017;2017:9217145.
9. Aggarwal A. Kaur H, Batra YK, et al. Anatomic consideration of caudal epidural space: A cadaver study. Clin Anat 2009;22:730-7.
10. Chen CP, Wong AM, Hsu CC, et al. Ultrasound as a screening tool for proceeding with caudal epidural injections. Arch Phys Med Rehabil 2010;91:358-63.
11. Pandey CK, BoseN, Garg G, et al. Gabapentin for the treatment of pain in Guillain Barré syndrome: a double-blinded, placebo-controlled, crossover study. AnesthAnalg 2002;95:1719-23.
12. Pandey CK, Raza M, Tripathi M, et al. The comparative evaluation of gabapentin and carbamazepine for pain management in Guillain-Barré syndrome patients in the intensive care unit. AnesthAnalg 2005;101: 220-5.
13. Kajimoto M, Koga M, Narumi H, et al. Successful control of radicular pain in a pediatric patient with Guillain-Barre´ syndrome. Brain Dev 2015;37: 897-900.
14. Yang S, W Kim, HH Kong, et al. Epidural steroid injection versus conservative treatment for patients with lumbosacral radicular pain: A meta-analysis of randomized controlled trials. Medicine 2020;99: e21283.