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Why Does C5 Palsy Occur After Prophylactic Bilateral C4-5 Foraminotomy in Open-Door Cervical Laminoplasty? A Risk Factor Analysis

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Abstract

Study Design: Retrospective study.

Objectives: To evaluate the efficacy of bilateral C4-5 foraminotomy in preventing occurrence of postoperative C5 palsy and to identify possible risk factors for its development.

Methods: A total of 70 consecutive patients who underwent open-door laminoplasty with bilateral C4-5 foraminotomy were included. Clinical, radiographic, and operative data was reviewed. Development of postoperative C5 palsy was analyzed.

Results: A total of 54 males and 16 females were reviewed. Mean age was 56 years (range, 30-86 years). The primary pathology was spondylosis in 76% of cases and ossified posterior longitudinal ligament in 21%. Radiographic evidence of C4-5 foraminal stenosis was seen in 81% of the patients. The mean duration of preoperative symptoms was 7 $\pm$ 19 months. Four (5.7%) out of 70 patients developed C5 palsy after open-door laminoplasty with bilateral C4-5 foraminotomy. Multivariate analysis showed that a long duration of preoperative symptoms (>12 months) and the presence of preoperative C4-5 T2-MRI cord signal change were statistically significant risk factors for the development of C5 palsy even after bilateral C4-5 foraminotomy in open-door laminoplasty ($P < .0001$ and $P = .036$, respectively).

Conclusions: Prophylactic bilateral C4-5 foraminotomies do not completely eliminate the occurrence of C5 palsy. Prolonged duration of symptoms and presence of preoperative T2-MRI cord signal change increase the risk for developing postoperative C5 palsy despite foraminotomy.

Keywords

open-door laminoplasty, foraminotomy, cervical complications, C5 palsy, MRI cord signal change, cervical myelopathy

C5 palsy is a well-recognized complication known to occur after cervical laminoplasty.[1-16] It is defined as the new onset of deltoid muscle paralysis after a neuro-decompression procedure.[3,17-20] The incidence of C5 palsy reported after laminoplasty without concomitant nerve root decompression ranges from 5% to 17%[1,2,6,11,21-23] with 92% of the C5 palsy occurring unilaterally.[3] Majority of the motor symptoms occur within the first week after the operation[1,3,8,11,12,24] and are expected to resolve by 12 months.[1,8,11,12,23] Permanent deficit has been reported at 10-year follow-up in up to 30% of C5 palsy in 1 case series.[1] While bilateral foraminotomy performed at the time of laminoplasty may prevent the development of C5 palsy,[3,12,17,20,22,25,26] little has been published on the possible clinical efficacy of bilateral foraminotomy as a prophylactic measure to prevent postlaminoplasty C5 palsy.[27,28] The aim of this study was to examine whether bilateral C4-5 foraminotomy performed at the time of open-door laminoplasty will prevent the development of postoperative C5 palsy, and to identify possible risk factors for its development.

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Materials and Methods

A retrospective review of all patients who had undergone open-door cervical laminoplasty with bilateral C4-5 foraminotomy was carried out. All surgeries were performed by the senior author (K.D.R.) and were reviewed by an independent spine surgeon. Clinical, radiographic, and operative reports were examined. Patient clinical data was recorded with attention to the patients' age and gender, duration of symptoms, neurologic deficits, pathology, and diagnosis. Patient neurological status was described according to Nurick score[29] pre- and postoperatively. Details of the surgery, including number of laminoplasty levels, site of bilateral foraminotomy performed, laminoplasty hinge location, operative time, and blood loss were documented. Radiographs were reviewed for the presence of preoperative C4-5 foraminal stenosis, T2-weighted magnetic resonance imaging (T2-MRI) cord signal change, spinal cord flattening, and pre- and postoperative cervical lordosis. Time to resolution of the C5 palsy symptoms was also recorded. Fischer's exact test and general linear model analyses were used to identify potential risk factors for C5 palsy development after prophylactic bilateral C4-5 foraminotomy in open-door laminoplasty.

Laminoplasty and Foraminotomy Surgical Technique

Details of the senior author's (K.D.R.) surgical techniques have been published[26,30,31] and a brief summary of the critical surgical steps is described below.

Bivector skull traction via Gardner-Wells tong is used to allow easy interchange of intraoperative neck alignment. Neck flexion is achieved through a vector connecting the tong via a rope placed "in-line" along the operating table to a 15-pound single pulley system. This allows "opening up" of the interlaminar space and facet exposure to enable thorough foramen decompression. A change of vector by a separate rope placed over a crossbar on the Jackson table extends the neck. This checks for overcrowding of the laminae and prevents bony blocks from neck extension. The spine is exposed in a standard midline subperiosteal fashion and avoids unnecessary soft tissue violation at either ends of the laminoplasty.

Microscope is used for detailed interlamina-facet complex exposure, foraminotomies and laminoplasty execution. The interlaminar "V" marks the junction where the cranial lamina intersects with the caudal one at the medial facet and the start of the foraminal decompression (Figure 1). A high-speed 3-mm carbide tip cutting burr is used to resect first the overlying inferior facet to uncover the superior facet, which is the cause of the dorsal nerve root impingement. The superior facet is burred out in an "L-shaped" trough with the vertical limb of the 'L" made along the lateral edge of the pedicle and the horizontal limb cranial to the pedicle. Inadequate dorsal nerve root decompression is avoided by careful removal of any residual bony hook at the cranial tip of the superior facet from an incompletely burred out "L-shaped" facet. Care to avoid more than 50% of facet removal is done to minimize postoperative facet instability and copious irrigation is used to avoid thermal trauma to the nerve.

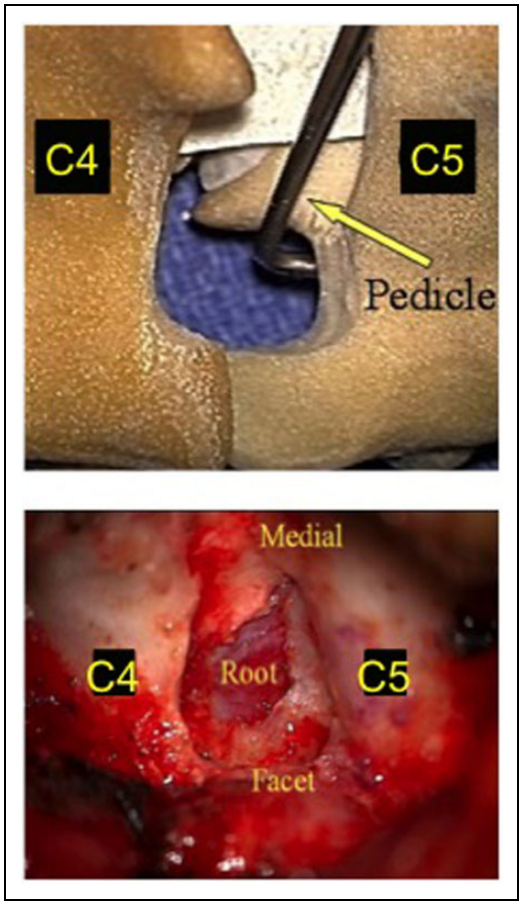

Figure 1. Interlaminar "V" junction where the cranial lamina intersects with the caudal one at the medial facet marks the start of the foraminal decompression.

Open-door laminoplasty is performed after foraminotomies. Bilateral bony gutters are placed near the medial boarder of the pedicle (may be identified through foraminotomies) at the lamina-medial facet junction. The lamina is opened on the clinically more symptomatic side. Elevation of the lamina beyond 60° is avoided to reduce nerve root traction[21,22] and although suture methods have been used in the earlier cases, majority of the laminoplasty is kept open by the use of laminoplasty plates[32,33] without bone graft.

All patients received 8 mg intravenous dexamethasone one hour prior to surgery. No routine steroid was given postoperatively.

Results

A total of 70 patients were retrospectively reviewed. There were 54 males and 16 females with an average age of 56 years (range, 30-86 years). Eighty-seven percent of the patients presented with myelopathic symptoms while 13% had multiple radiculopathy. The mean duration of preoperative symptoms was 7 $\pm$ 19 months. Average preoperative Nurick score was

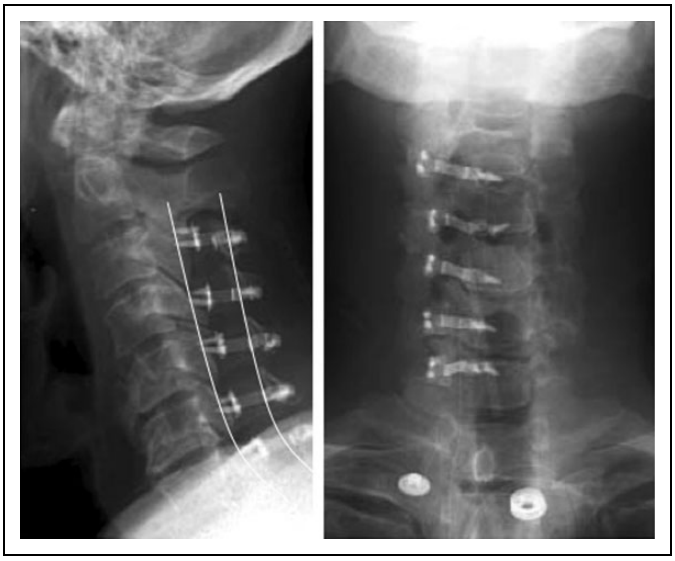

Figure 2. Postoperative cervical radiographs showing bilateral C4-5 foraminotomy.

2.3 (range, 1-5) grades. Spondylotic degeneration was the main pathology in 76% of cases and ossified posterior longitudinal ligament (OPLL) in 21%. Radiographic evidence of C4-5 foraminal stenosis was seen in up to 81% of the patients. Other radiographic findings such as C4-5 flattening of the cord and MRI cord signal change were seen in 30% and 59%, respectively.

Seventy-nine percent of the patients underwent multilevel open-door laminoplasty (5 levels). Twenty-one percent had 4-level laminoplasties done. There was near equal distribution of the side of hinge placement with right-sided hinges done in 46% and left-sided hinges in 54%. All the patients had bilateral C4-5 foraminotomies (Figure 2). Sixty-six percent of the cases had bilateral C4-5 and C5-6 foraminotomies performed. The mean operative time was 168 minutes (range, 90-270 minutes). Mean blood loss was 188 mL (range, 75-900 mL).

Postoperative C5 palsy developed in 4 out of the 70 patients reviewed (5.7%) (Figure 3). All 4 patients were males, nonsmokers and nondiabetics, with a mean age of 56.3 years (range, 52-63 years) at the time of operation. The pathology was spondylotic myelopathy with C4-5 bilateral foraminal stenosis in all four cases. Three out of the 4 patients had MRI cord signal change at C4-5 as well as flattening of the cord. The mean duration of preoperative symptoms was 51 months. However, case 2 had preoperative symptoms for 120 months. If we exclude case 2 and treat it as an outlier, the mean preoperative duration of symptoms is 28 months. The average Nurick grade was 2.5 (range, 1-3) grades. Average follow up was 25.5 months (range, 16-54 months). Three patients with C5 palsy had laminoplasty performed in 4 levels, with 1 case having a 5-level laminoplasty. Cervical lordosis was preserved pre- and postoperatively. Mean operative time was 188 minutes and mean blood loss was 200 mL. Seventy-five percent of postoperative C5 palsy developed in the extremity opposite the side of the laminoplasty hinge. C5 palsy symptoms resolved at 3 months in 2 patients. The remaining 2 patients had resolution of symptoms at 12 and 54 months, respectively. A summary of the characteristics of the 4 patients with C5 palsy is seen in Table 1.

Multivariant logistic regression analysis showed that there was a statistically significant increase in risk for postoperative C5 palsy if the patient had a long duration of preoperative symptoms (>12 months, $P = .0001$). Case 2 had preoperative symptoms for 120 months. In order to determine if this skewed the results, we removed case 2 from the equation and recomputed the data. Statistical significance for preoperative duration was maintained with P value of .003. The presence of T2 MRI cord signal change at C4-5 was another significant risk factor in the development of C5 palsy postforaminotomy with a P value of .036. Other variables such as foraminal stenosis, MRI cord compression, patient's age and gender, diabetes, cigarette smoking, laminoplasty hinge site, preoperative Nurick score, and multilevel foraminotomies did not reach statistical significance. Neither the diagnosis of myelopathy versus radiculopathy nor the pathology of spondylosis versus OPLL have any statistical significance in the development of post foraminotomy C5 palsy (see Table 2).

Discussion

C5 palsy is a known complication of cervical decompression surgery. The overall incidence in literature is between 5% and 17%.[1,2,6,11,21-23] Yifei et al[34] in a 2014 systematic review reported the incidence of C5 palsy after open-door laminoplasty, double-door laminoplasty, and laminectomy as 4.5%, 3.1%, and 11.3%, respectively. Similarly, Kaneyama et al[35] reported a higher incidence of postoperative C5 palsy in the open door laminoplasty group when compared with a double-door laminoplasty (9.6% and 1.4%, respectively).

Several theories have been proposed to explain the occurrence of postoperative C5 palsy (Figure 4). One of the most widely accepted mechanisms is nerve root traction injury as the spinal cord migrates posteriorly after laminoplasty.[1-3,5,8,12,21,23,25,27,36,37] Unique anatomical features such as an anteriorly located superior facet, a shorter nerve root and a larger distance of posterior shift of the cord at C5 compared to the other spinal levels, further predispose the C5 nerve root to this kind of traction injury.[3,22,23] Preoperative foraminal stenosis has also been proven to be a significant risk factor to developing postoperative C5 palsy.[34] Furthermore, Katsumi et al[28] reported a significant higher incidence of postoperative C5 palsy in patients with C4-5 foraminal stenosis when compared to patients without foraminal stenosis.

Based on the hypothesis of nerve traction and foraminal stenosis as the mechanism of postlaminoplasty C5 motor palsy, the use of prophylactic C4-5 foraminotomy to decompress the C5 nerve root has been proposed as a means of eliminating this complication.[8,12,17,25,27] Baba et al[38] described laminoplasty with foraminotomy for treating cervical myelopathy in the early 1990s. They reported better neurologic improvement of myelopathy and unilateral nerve compression when doing

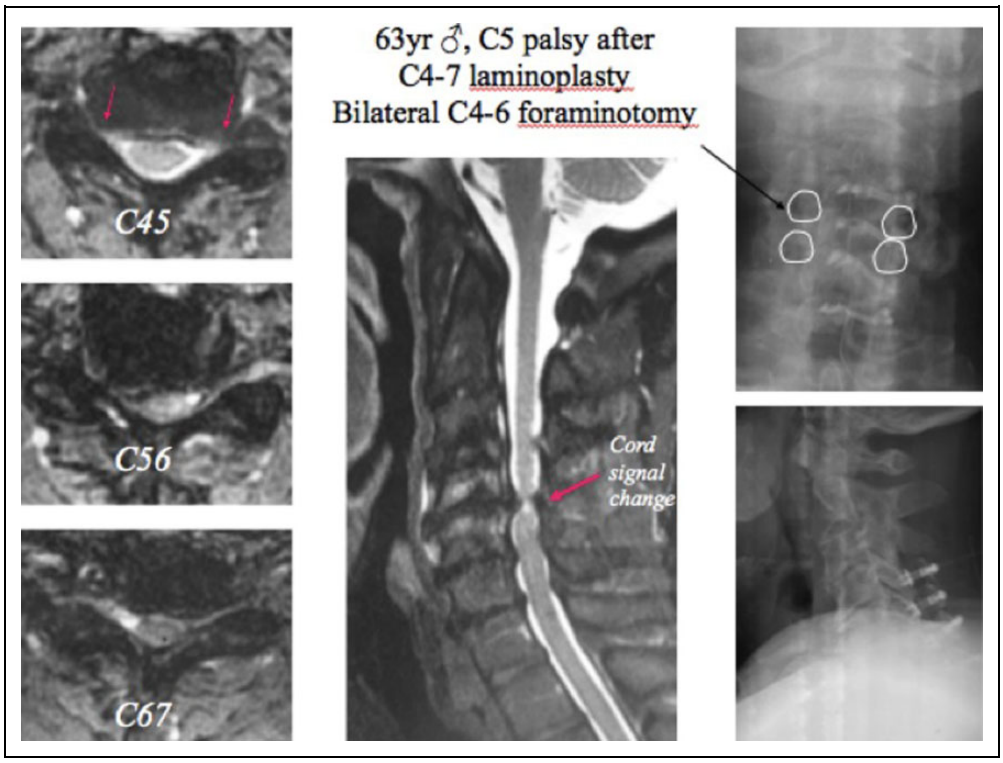

Figure 3. Sample case of a patient who developed C5 palsy after bilateral C4-6 foraminotomy.

Table 1. Characteristics of Patients Who Developed Postoperative C5 Palsy After Open-Door Laminoplasty With Bilateral C4-5 Foraminotomy.

	Sex	Follow-up (mo)	Age at Operation (y)	Operation Level	Hinge Side	Side of C5 Palsy	Duration of Symptoms (mo)	Preoperative Nurick Grade	C4-5 Cord Compression	T2-MRI Cord Signal Change at C4-5	C5 Palsy Resolved (mo)
Case 1	M	24	53	C3-7	L	R	36	1	N	Y	3
Case 2	M	54	57	C3-7	R	L	120	1	Y	Y	54
Case 3	M	16	52	C3-7	R	R	12	2	Y	N	3
Case 4	M	16	63	C4-7	L	R	36	3	Y	Y	12

Abbreviations: M, male; F, female; N, no; Y, yes; L, left; R, right; MRI, magnetic resonance imaging.

laminoplasty with concomitant foraminotomy. Their study, however, was limited to unilateral foraminotomy and was thus less effective for bilateral radiculopathic symptoms. Ikata et al[39] advocated a sagittal splitting method of laminoplasty with bilateral foraminotomy as a solution to this problem. Sakaura et al[3] reported a 0% and 1.1% incidence of C5 palsy after open-door laminoplasty with foraminotomy in a series of 76 and 281 patients, respectively.

Komagata et al,[27] in a retrospective study, reported the incidence of C5 palsy in 230 patients with or without foraminotomies after laminoplasty. Foraminotomy was performed in 162 of 460 laminoplasty gutters. C5 palsy was observed in 1 (0.6%) gutter with foraminotomy and in 12 (4.0%) without foraminotomy. Only 54 patients in the series had bilateral foraminotomies and others had unilateral foraminotomy. There was no mention of whether the reported C5 palsy was from patients with bilateral or unilateral foraminotomy and what their surgical indications for unilateral foraminotomy were.

Katsumi et al[28] prospectively compared 141 cases with cervical myelopathy who underwent open-door laminoplasty and prophylactic bilateral C4-5 foraminotomy with another 141 patients who did not have foraminotomy done at the time of laminoplasty. They reported that there was a significant decrease in incidence of postoperative C5 palsy in the foraminotomy group vs no foraminotomy group (1.4% vs 6.4%).[28] However, the use of bilateral foraminotomy still did not totally eliminate the complication as there were 2 patients who developed C5 palsy even after bilateral prophylactic C4-5 foraminotomy. The authors suggested that there may be other spinal cord factors that can contribute to the occurrence of C5 palsy.

Table 2. Multivariate Analysis of Risk Factors for Development of Postoperative C5 Palsy.

Risk Factors	*P*
Foraminal stenosis	1.000
Overall MRI cord signal change	.6369
MRI cord compression	.6086
Patient age	.9260
Gender	.5672
Diabetes	1.0000
Cigarette smoker	.3008
Diagnosis (myelopathy vs radiculopathy)	.2529
Pathology (spondylosis vs OPLL)	.3295
Preoperative Nurick score	.4796
Laminoplasty hinge site	1.0000
Multilevel foraminotomies	.8630
Preoperative symptoms duration	<.0001[a]
Preoperative symptoms duration (excluding case 2)	.0027[a]
T2 MRI cord signal change at C4-5	.0355[a]

Abbreviations: MRI, magnetic resonance imaging; OPLL, ossification of posterior longitudinal ligament.
[a]Statistically significant ($P < .05$).

In our study, the incidence of postoperative C5 palsy after open-door laminoplasty with bilateral C4-5 foraminotomy is 5.7%. Spinal cord risk factor analysis including overall MRI cord signal change, MRI cord compression, diabetes, cigarette smoking, pathology (spondylosis vs OPLL), diagnosis (myelopathy vs radiculopathy), and preoperative Nurick score did not show any significant relationship in the occurrence of postoperative C5 palsy. Only 2 factors were found to be of statistical significance in the development of postforaminotomy C5 palsy. These were prolonged duration of preoperative symptoms and increase in T2-MRI cord signal change at C4-5 spinal level.

Various inherent spinal cord pathologies as a cause of C5 palsy has been purposed in literature. Mechanisms include cord ischaemia,[3,21] reperfusion injury,[24,37] and impairment of spinal cord gray matter.[2,11,37] Chiba et al[24] reported worsening of postoperative MRI spinal cord changes in patients who develop C5 palsy and suggested cord ischemia and reperfusion injury as an etiology of C5 palsy development. Sasai et al[17] reviewed 111 patients who underwent laminoplasty and found that patients who had preoperative electromyographic changes were more likely to develop postoperative C5 palsy. This suggests that preexisting subclinical cord pathology is a risk factor for C5 palsy development. Increased cord signal intensity in MRI has been reported to suggest irreversible changes of the spinal cord.[40] Takashima et al[40] considered it to be reflective of edema, myelomalacia, or cord gliosis secondary to a long-standing compressive effect on the spinal cord. Ikegama et al[41] reported that patients with preoperative MRI spinal cord signal change who had postoperative segmental motor paralysis had wider paralyzed muscle domains, weaker muscle strength, and longer recovery periods. These findings substantiate our study results, which showed that prolonged duration of symptoms and presence of T2-MRI cord signal change to be significant risk factors in postoperative C5 palsy development. The presence of preoperative MRI cord signal changes represents preoperative subclinical neural tissue damage thus making individuals prone to postoperative C5 radiculopathy from

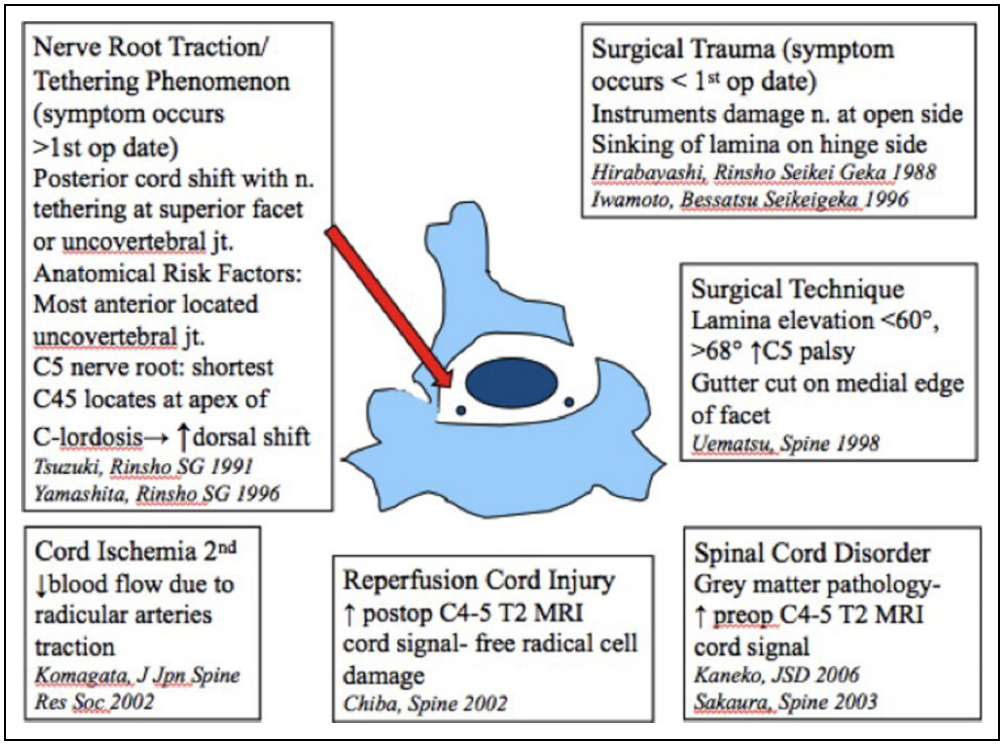

Figure 4. Theories for the development of postoperative C5 palsy.

minimal traction on the nerve root during dorsal cord migration after laminoplasty.

A limitation of the current study is its retrospective nature and the lack of a matched control cohort of laminoplasty patients without foraminotomy. It would also have been valuable to assess the amount of posterior cord drift post foraminotomy and its relationship with the development of C5 palsy as this is one of the most commonly accepted theories of this complication. However, this was not possible in this study since routine postoperative MRI was not done for all patients.

Our study raises awareness that routine bilateral C4-5 foraminotomy in open-door laminoplasty does not prevent the development of postoperative C5 palsy. For patients with foraminal stenosis we would still do prophylactic foraminotomy to try to decrease this incidence. However, C5 palsy may still occur in patients with prolonged duration of symptoms and presence of high intensity cord signal changes at C4-5. In our series, the reason behind this complication may not be related to nerve root traction or the tether theory but due to intrinsic spinal cord pathology. No further surgical management is recommended for this subset of patients who develop C5 palsy. Medical management to reduce inflammation, for example, Riluzole, may perhaps be beneficial. Overall, patients should be counseled accordingly about the risk of developing C5 palsy despite existing measures to prevent it.

Conclusion

The etiology of postoperative C5 palsy remains multifactorial. Performing prophylactic bilateral C4-5 foraminotomy does not eliminate the development of postoperative C5 palsy. Inherent sublinical cord pathology contributes to the development of this complication. Prolonged duration of preoperative symptoms and C4-5 T2-MRI cord signal change appear to be statistically significant risk factors for C5 palsy development even after bilateral C4-5 foraminotomy.

Declaration of Conflicting Interests

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