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Severe C7-T2 Spinal Cord Compression From a WHO Grade 1 Meningioma With Preserved Neurological Function: A Case Report

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Abstract

Spinal meningiomas are usually slow-growing intradural extramedullary tumors, and most published series link higher spinal canal occupancy to clinical myelopathy. We report the case of a 53-year-old woman with a large left-sided C7-T2 intradural extramedullary WHO grade 1 meningioma who maintained full strength, a stable gait, and normal reflexes despite years of severe cord displacement on imaging. She was first evaluated for chronic neck and upper back spasms and low back pain. Imaging at that visit already showed a left anterolateral intradural extramedullary mass with severe canal stenosis and rightward cord displacement. Because her examination showed no myelopathy, she was managed with surveillance rather than surgery. Over the following years, she developed slowly progressive bilateral paresthesias in the arms and legs, but her strength, gait, and reflexes remained normal, and she never developed classic myelopathic signs. Approximately six years after the initial presentation, she underwent a left C7, T1, and T2 hemilaminotomy and hemifacetectomy with microsurgical gross total resection, guided by intraoperative electromyography, somatosensory evoked potentials, and motor evoked potentials. No neuromonitoring changes occurred during surgery, and pathology confirmed a WHO grade 1 meningioma. On postoperative day 1, her strength remained full, with only mild residual sensory changes in the left fingertips. Severe radiographic cord compression from a slow-growing meningioma can persist for years without producing the motor or gait findings expected at that degree of compression, most likely because gradual growth allows the cord to adapt to progressive displacement. Surgical timing in these patients must take imaging severity and symptom trajectory into account rather than relying on the motor examination alone.

Categories: Neurology, Oncology, Neurosurgery
Keywords: asymptomatic cord compression, cervical cord compression, cervicothoracic spine surgery, intradural extramedullary tumor, spinal canal occupancy, spinal meningioma, who grade 1 meningioma

Introduction

Spinal meningiomas make up a large share of intradural extramedullary tumors and usually grow slowly enough that they are found only after they begin compressing the spinal cord or nerve roots. Larger tumors and higher canal occupancy are generally associated with clinical myelopathy [1,2], and a lesion large enough to severely compress and displace the cord would ordinarily be expected to produce neurological manifestations, particularly at the cervical and cervicothoracic junction [3,4]. Canal occupancy, the proportion of the spinal canal's cross-sectional area filled by a tumor on axial imaging, is one of the measures most often used to estimate this risk.

This patient's course did not follow that pattern. She had a large C7-T2 intradural extramedullary WHO grade 1 meningioma that severely compressed and displaced her spinal cord for years, yet she maintained full strength, a normal gait, and no classic myelopathy until the time of surgery. The gap between her imaging findings and clinical examination is what makes the case worth reporting: it argues against relying on the motor examination alone when deciding whether a slow-growing meningioma requires resection.

Case Presentation

A 53-year-old woman working in public relations, with a history of migraine and gastritis, presented with chronic dorsal neck and upper back muscle spasms and low back pain that had started eight months earlier. Her job involved prolonged daily computer use. At the initial evaluation, she had no upper- or lower-extremity radicular pain, weakness, sensory loss, gait disturbance, long-tract signs, or clinical myelopathy.

MRI obtained at that visit showed a left anterolateral intradural extramedullary mass spanning C7-T2, most consistent with a meningioma. The lesion caused severe spinal canal stenosis with rightward displacement and compression of the cord, although there was no definite signal change within the cord itself. Because her neurological examination was normal, the mass was considered a probable incidental finding and managed with surveillance rather than immediate surgery.

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Over the next several years, she developed slowly progressive paresthesias in both arms and both legs. Her strength remained 5/5 throughout, her gait remained stable, her reflexes remained 2+, and she never developed the long-tract signs or gait changes that would indicate classic cervical or thoracic myelopathy. The clinical course from initial presentation to surgery is summarized in Figure *1*.

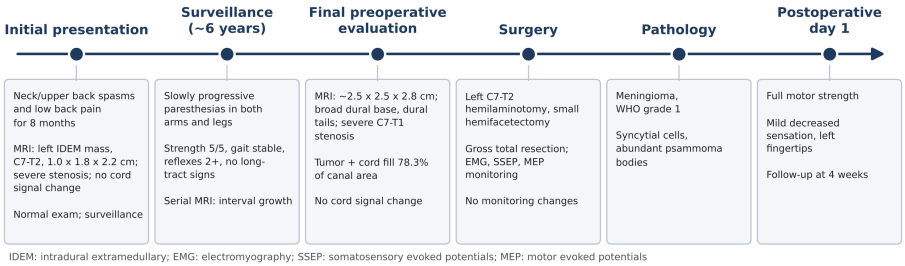


FIGURE 1: Timeline of the clinical course.

Over approximately six years of surveillance, the mass grew from 1.0 × 1.8 × 2.2 cm to approximately 2.5 × 2.5 × 2.8 cm, while strength, gait, and reflexes remained normal and only slowly progressive bilateral paresthesias developed.

IDEM: intradural extramedullary

Diagnostic imaging

Serial imaging over the surveillance period documented growth of the mass. On initial evaluation, MRI measured the lesion at 1.0 × 1.8 × 2.2 cm. It was a dural-based intradural extramedullary mass at C7-T2 with a dural tail, severe canal stenosis, and rightward cord displacement without cord signal change. By the final preoperative evaluation six years later, the mass measured approximately 2.5 × 2.5 × 2.8 cm, with a broad dural base, prominent dural tails, and severe C7-T1 canal stenosis displacing the cord to the right (Figure *2*). The radiologic impression favored meningioma, although schwannoma or neurofibroma could not be completely excluded. No intramedullary signal abnormality suggestive of compressive myelopathy was identified on any study.



FIGURE 2: Preoperative sagittal MRI showing a dural-based intradural extramedullary mass spanning C7-T2, with a dural tail, severe canal stenosis, and compression of the spinal cord.

On the axial images from the final preoperative study, the outer canal contour and the combined mass-and-cord complex were traced manually using the measurement tool in Sectra UniView (Sectra AB, Linköping, Sweden). At the level of maximal compression, the canal cross-sectional area was 297.64 mm^2, and the combined mass-and-cord complex measured 233.05 mm^2, leaving 64.59 mm^2 of residual canal space and an occupancy of 78.3% (Figure *3*). This ratio describes the proportion of the canal occupied by the tumor and cord together. It is not the tumor-only occupancy metric used by Corell et al. and Yamaguchi et al. [1,2]; therefore, it cannot be compared directly with their published thresholds and is reported here as a descriptive measurement of how little canal reserve remained. A tumor-only occupancy ratio was not calculated, which is a limitation of this report.

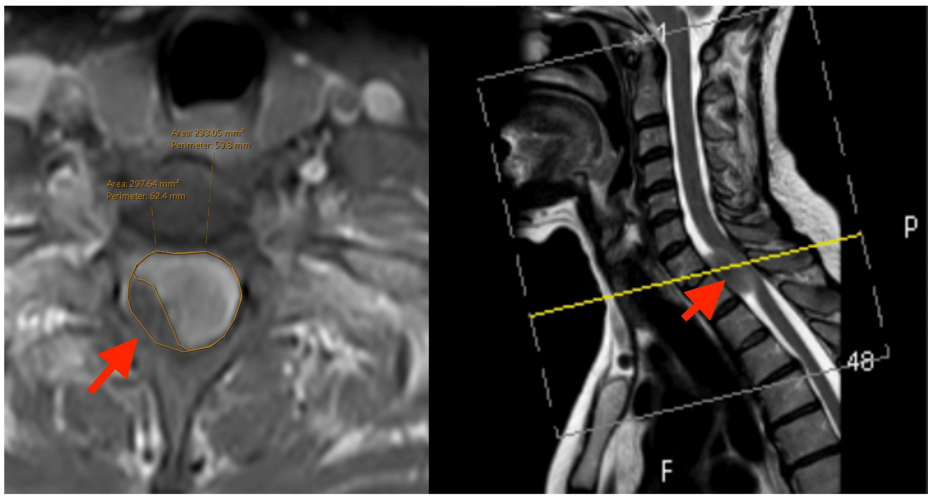


FIGURE 3: Preoperative axial MRI at the level of maximal compression showing manual contours of the outer canal boundary and the combined mass-and-cord complex.

Canal area: 297.64 mm²; mass-and-cord complex: 233.05 mm²; occupancy: 78.3%. The sagittal localizer indicates the axial level.

Surgical intervention

Approximately six years after the initial presentation, the patient, then 53 years old, underwent a left C7, T1, and T2 hemilaminotomy with a small hemifacetectomy and microsurgical gross total resection of the intradural extramedullary tumor. Her head was secured in a Mayfield three-pin holder (Integra LifeSciences, Princeton, New Jersey), and the correct levels were confirmed with fluoroscopy. Intraoperative electromyography, somatosensory evoked potentials, and motor evoked potentials were used throughout the procedure.

After dural opening, the tumor was debulked with an ultrasonic surgical aspirator (CUSA, Integra LifeSciences, Princeton, NJ, USA) and dissected free from the surrounding arachnoid, the left-sided dura, and the left C8 and T1 nerve roots. The dura was closed in a watertight fashion with an onlay graft and fibrin sealant. The tumor was solid, grayish, and attached to the left-sided dura. It severely compressed and displaced the cord to the right, consistent with the preoperative imaging findings. Gross total resection was achieved, and no intraoperative neuromonitoring changes occurred at any point during the procedure.

Pathology

The specimen consisted of three irregular fragments of tan-brown, firm tissue measuring 2.0 × 1.2 × 0.2 cm in aggregate. The tumor was composed of syncytial cells with indistinct cell membranes and eosinophilic cytoplasm (Figure *4*). The cells had round, uniform nuclei and a syncytial growth pattern, with eosinophilic cytoplasm. Psammoma bodies were abundant (Figure *5*). Histopathology confirmed a WHO grade 1 meningioma.

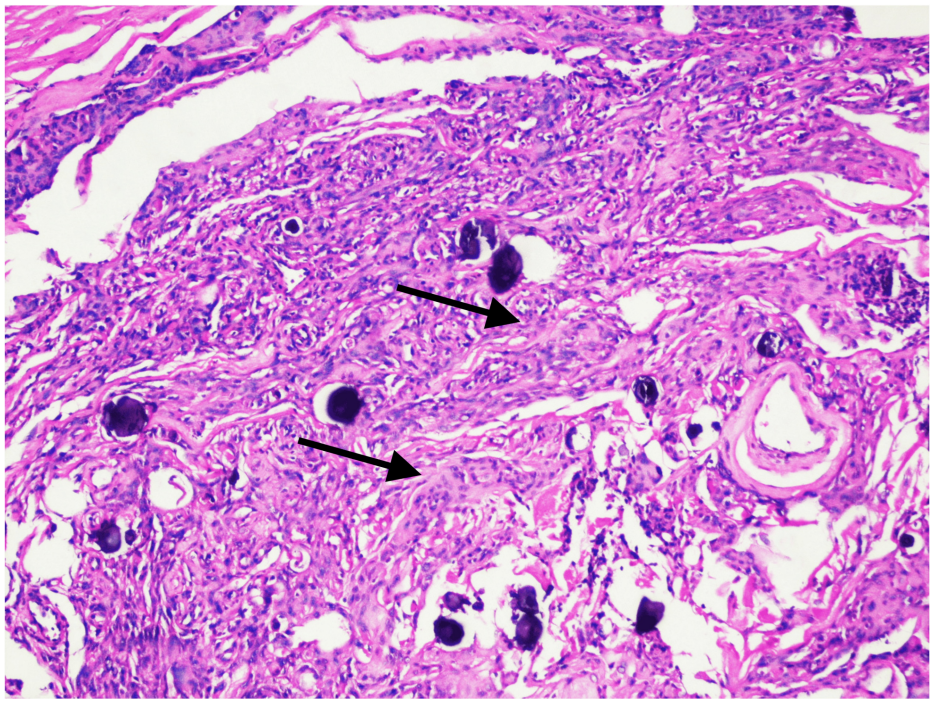


FIGURE 4: Meningioma at 10× magnification showing tumor cells with a syncytial growth pattern, indistinct cell membranes, and eosinophilic cytoplasm (black arrow).

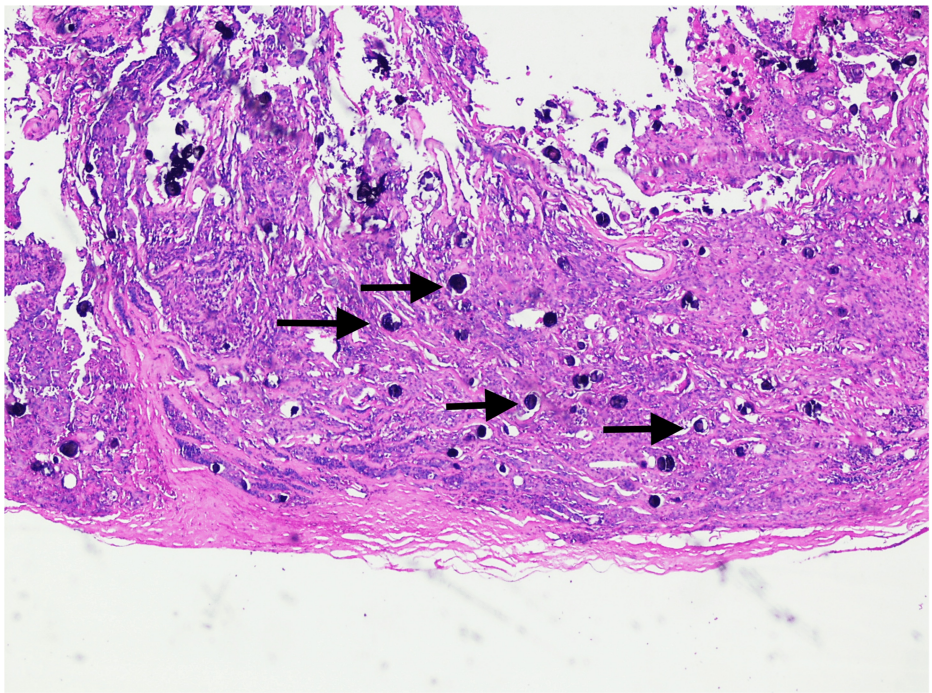


FIGURE 5: Low-power view at 4× magnification showing the lesion adhered to thick fibrous tissue (dura).

Numerous psammoma bodies (black arrow) can be seen.

Postoperative course

On postoperative day 1, the patient was alert and oriented, breathing comfortably, and tolerating oral

liquids, with full motor strength throughout. She had no active radicular pain in the arms and only mildly decreased sensation in the left fingertips. She tolerated sitting upright without a postural headache, and her wound was clean and dry. Follow-up was scheduled for four weeks later.

Discussion

This case shows a mismatch between the severity of cord compression on imaging and the patient's neurological examination. Published surgical series consistently link larger tumors and higher canal occupancy to worse preoperative function. Corell et al., studying 111 patients, found that tumor occupancy above 65% strongly predicted a preoperative motor deficit and argued that even an asymptomatic, fit patient approaching that occupancy should be considered for surgery rather than further observation [1]. Yamaguchi et al. found a similar cutoff of approximately 64% occupancy that independently predicted motor weakness across 53 surgically treated cases [2]. Baro et al., in a multicenter cohort of 90 patients, found that cord and tumor occupancy, cord compression, and tumor volume all correlated with worse preoperative functional status and that T2 cord signal change carried independent prognostic weight [5]. Onishi et al., in a more recent multicenter series, linked preoperative paralysis to sagittal tumor size and sensory disturbance to horizontal occupancy [6]. Tumors at the cervical and cervicothoracic junctions are reported to be almost uniformly symptomatic: Acosta et al. found pain or radiculomyelopathy in all 14 patients with multilevel ventral cervicothoracic intradural extramedullary tumors [3], and case reports of comparably large or giant cervical intradural lesions, such as the giant schwannoma described by Quillo-Olvera et al., generally report progressive weakness rather than preserved strength [4].

The most plausible explanation for why this patient was largely spared is the slow rate of tumor growth. The mass was already causing severe stenosis at presentation and then enlarged from 1.0 × 1.8 × 2.2 cm to approximately 2.5 × 2.5 × 2.8 cm over about six years (Figure *1*). Gradual growth of this kind allows the surrounding neural tissue to adapt through progressive displacement rather than destruction. The cord was pushed and flattened to the right, yet no study ever showed an intramedullary signal change. This is consistent with Baro et al.'s finding that T2 cord signal change tracks functional impairment more closely than occupancy alone [5]: her cord was displaced but showed no imaging evidence of injury. The adaptation was not complete. The slowly spreading paresthesias in all four limbs show that the compression was not clinically silent, and sensory symptoms preceding any motor change are consistent with Onishi et al.'s association between horizontal occupancy and sensory disturbance [6].

Adaptation may not be limited to the cord itself. Kostiuk et al. showed that patients with asymptomatic degenerative cervical cord compression have measurable changes in cerebral functional connectivity, including stronger visual-motor coupling, that appear before any clinical symptoms develop [7]. Their study involved degenerative rather than neoplastic compression, so the mechanism cannot be directly extrapolated, but it suggests that supraspinal reorganization can accompany slow cord compression before symptoms appear. Related work has documented cortical and motor-network reorganization around the time of decompression surgery in patients who were already symptomatic from cervical spondylotic myelopathy [8,9]. Those studies were not designed to investigate why some patients remain asymptomatic, and no functional imaging was performed in this patient; therefore, cortical compensation remains a possible contributor rather than something demonstrated by this case.

The practical point is that her motor examination, on its own, would have argued for continued observation right up until surgery. What argued against observation was the imaging: years of severe, slowly worsening cord displacement, with the tumor and cord together occupying 78.3% of the canal at the level of maximal compression. Because that ratio is not the tumor-only measure used in the occupancy series, this case cannot be assessed against the 64-65% thresholds proposed by those authors, and we make no claim that it crossed them. What the measurement does establish is how little reserve remained around a cord that was still functioning normally. Waiting for a motor deficit to appear before operating on a lesion like this risks performing surgery only after the cord has already been injured rather than before.

Conclusions

Severe spinal cord compression from a slow-growing intradural extramedullary meningioma can persist for years without producing the myelopathy that the degree of compression would predict, most likely because slow growth gives the cord time to adapt to progressive displacement. A normal motor examination in this setting does not mean the cord is safe, and it should not be the main reason to continue surveillance. When imaging shows severe or progressive cord displacement with little residual canal space and sensory symptoms are slowly spreading, those findings should weigh more heavily in surgical timing than preserved strength. Although a single case cannot define optimal surgical timing, and the absence of cord signal change on MRI does not exclude subclinical cord injury, this case supports considering resection before a motor deficit develops because a deficit that appears before surgery may not fully recover.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the

work.

Concept and design: Charlie Vidal, Jorge Lastra Power, Pamela Vazquez Negron, Carlos Weber Seda

Acquisition, analysis, or interpretation of data: Charlie Vidal, Jorge Lastra Power, Pamela Vazquez Negron, Carlos Weber Seda

Drafting of the manuscript: Charlie Vidal, Jorge Lastra Power, Pamela Vazquez Negron

Critical review of the manuscript for important intellectual content: Charlie Vidal, Jorge Lastra Power, Pamela Vazquez Negron, Carlos Weber Seda

Supervision: Jorge Lastra Power

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Corell A, Cerbach C, Hoefling N, Björkman-Burtscher IM, Jakola AS: Spinal cord compression in relation to clinical symptoms in patients with spinal meningiomas. Clin Neurol Neurosurg. 2021, 211:107018. 10.1016/j.clineuro.2021.107018
2. Yamaguchi S, Menezes AH, Shimizu K, Woodroffe RW, Helland LC, Hitchon PW, Howard MA: Differences and characteristics of symptoms by tumor location, size, and degree of spinal cord compression: a retrospective study on 53 surgically treated, symptomatic spinal meningiomas. J Neurosurg Spine. 2020, 32:931-40. 10.3171/2019.12.SPINE191237
3. Acosta FL Jr, Aryan HE, Chi J, Parsa AT, Ames CP: Modified paramedian transpedicular approach and spinal reconstruction for intradural tumors of the cervical and cervicothoracic spine: clinical experience. Spine (Phila Pa 1976). 2007, 32:E203-10. 10.1097/01.brs.0000257567.91176.76
4. Quillo-Olvera J, Lin GX, Kim JS: Severe spinal cord compression by pure giant intradural schwannoma of cervical spine. World Neurosurg. 2018, 110:17-9. 10.1016/j.wneu.2017.10.106
5. Baro V, Moiraghi A, Carlucci V, et al.: Spinal meningiomas: influence of cord compression and radiological features on preoperative functional status and outcome. Cancers (Basel). 2021, 13:4183. 10.3390/cancers13164183
6. Onishi E, Fujibayashi S, Otsuki B, et al.: Risk factors for preoperative neurological impairment in patients with spinal meningioma: a retrospective multicenter study. J Clin Neurosci. 2024, 126:187-93. 10.1016/j.jocn.2024.06.021
7. Kostiuk A, Jafari M, Wang C, Mayer E, Salamon N, Holly LT, Ellingson BM: Functional alterations across motor, visual, and attention cerebellar and cortical networks in patients with asymptomatic spinal cord compression. J Neurosurg Spine. 2025, 44:45-54. 10.3171/2025.5.SPINE2544
8. Ryan K, Goncalves S, Bartha R, Duggal N: Motor network recovery in patients with chronic spinal cord compression: a longitudinal study following decompression surgery. J Neurosurg Spine. 2018, 28:379-88. 10.3171/2017.7.SPINE1768
9. Green A, Cheong PW, Fook-Chong S, et al.: Cortical reorganization is associated with surgical decompression of cervical spondylotic myelopathy. Neural Plast. 2015, 2015:389531. 10.1155/2015/389531