

Lower Thoracic Spinal Myeloradiculopathy with Intramedullary Schwannoma in an Elderly Woman: A Rare Case Report

Syawal Muhammad Reyhan *, Ompusunggu Sevline Estethia, Dahlan Rully Hanafi

Department of Neurosurgery, Faculty of Medicine, Padjadjaran University / Dr. Hasan Sadikin General Hospital, Bandung, West Java, Indonesia.

***Corresponding Author:** Muhammad Reyhan Syawal., Department of Neurosurgery, Faculty of Medicine, Padjadjaran University / Dr. Hasan Sadikin General Hospital, Bandung, West Java, Indonesia.

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Abstract

Intramedullary schwannomas are a rare form of spinal tumors. Here, we present a case of a 61-year-old woman who was admitted to our hospital with complaints of weakness in both lower extremities, numbness, urinary and defecation disorders, and radiculating back pain. Physical examination revealed sensory and motoric deficits in the lower extremities, as well as autonomic dysfunctions. Radiological imaging revealed a mass at the lower level of thoracic spinal segment, which was confirmed as a schwannoma through histopathological analysis. The patient underwent laminectomy tumor removal and posterior stabilization. She was also diagnosed with type 2 diabetes mellitus and hypertension. Spinal intramedullary schwannoma is a rare condition that requires a thorough history and physical examination for diagnosis, particularly for patients with neurological impairments. Prompt treatment is essential once the diagnosis is made.

Key words: intramedullary schwannoma ; spinal tumors ; laminectomy ; hyperplastic ; verocay bodies

Introduction

Schwannoma is the most frequent nerve sheath tumor of the spine, and in most circumstances, it tends to be solitary. Referring to some reports, the prevalence of spinal schwannoma is approximately 0.24 cases out of 100.000 individuals; hence, spinal schwannoma is considered to be a very rare medical condition.¹ Sensory nerve roots are the typical sites of schwannoma, which manifests as a globular, well-defined, encapsulated mass that is well-defined and isolated from the other rootlets.²

Complete surgical excision is the gold standard treatment for symptomatic spinal schwannomas since prevents the progression of symptoms, speeds up recovery in most patients, and lowers the likelihood of recurrence. Patients who are poor surgical candidates or those with recurring malignancies may undergo radiotherapy as a last resort.²

Here we report a case of a 61-year-old woman with the chief complaint of progressive neurological loss, including motoric deficits (inability to walk) along with sensory dysfunctions. Autonomic involvements were also complained.

Case Presentation

A 61-year-old woman was referred to our hospital with inability to walk as the chief complaint. This condition was accompanied by numbness,

urinary, and defecation disorders. Based on history taking, these complaints were preceded by radiculating pain at her back in 2017 and in July 2018 she felt weakness on her both lower extremities and the symptoms worsen since then. Prior to admission, she had lumbosacral x-ray on March 17th 2022 and contrast lumbosacral MRI on April 5th 2022 from neurology outpatient clinic at our hospital.

Vital signs showed stage I hypertension, and thoracic examinations were unremarkable. Neurological examinations demonstrated normal awareness (GCS = 15), no nuchal rigidity, and normal pupillary responses.

Neurological examinations revealed impaired sensory functions: hypesthesia at the level of L1 and below. Absence of perianal sensation, voluntary anal contraction, and great toe extension were found during sacral segment evaluations. Motoric functions are described as in Table 1.

The patient also had urinary and fecal incontinence. Laseque sign (-), physiological reflexes are all positive, no clonus nor spasm were found.

However, Babinski sign was positive.

Laboratory tests showed no remarkable results except for high random and fasting blood glucose (366 & 123 mg/dL, respectively).

Elbow flex	5/5	Hip flexion	4/4
Wrist extension	5/5	Knee extension	4/4
Elbow extension	5/5	Ankle dorsiflexion	4/3
Flexion of middle distal phalanx	5/5	Great toe extension	4/3
Finger abduction	5/5	Ankle plantar flexion	4/1

Table 1: Motoric status of the patient

Clinical presentation of the patient showed normal facial feature; second to fourth photos demonstrated motoric function test of the patient; with the last picture showed external condition of patient’s back (Figure 1). On plain chest radiographs, thoracic x – ray showed cardiomegaly. Lumbosacral x – ray revealed normal alignment of the spine segments.

Spinal MRI was taken a month after Xray showed hypointense spinal cord mass at the level of T11 – T12, with inhomogen strengthening upon contrast administration (T1 weighted). On T2 weighted MRI scan, isointense mass lesion was detected at the spinal level of T11 – T12 (Figure 2).



Figure 1: Physical conditions of the patient.

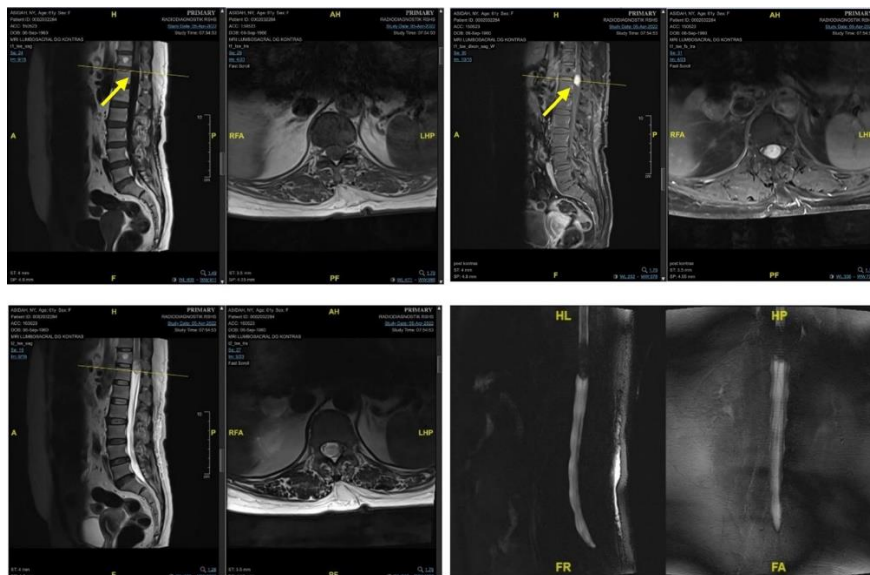


Figure 2: Thoracic segment MRI, showing hypointense spinal cord mass at the level of T11 – T12, with

As the treatment, the patient underwent bilateral laminectomy to the medial facet T11 to T12. Duramater looks white, intact, bulging and no pulsation. We did the durotomy at posterior midline, looks intramedullary tumor mass that filling the spinal canal from posterior to anterior, capsulated, grayish-red and the tumor originating from the nerve radix. The tumor resected 100%, avital rostral and caudal radices are cut. The duramater primary sutured and covered with fibrin glue. We installed 8

pieces (4 levels) pedicle screws at T10 to L1. Planning, preoperative, intraoperative, and postoperative findings are showed as in Figure 7 – 10.

The spinal tumor was successfully resected, with the size of ~ 2 cm (Figure 3), and we referred for histopathological examination to confirm the diagnosis on April 25th 2022, with the tissue size of 2.2 × 1.5 × 1.3cm. Histopathological result was showing hyperplastic, wavy tissue with

spindle – shaped cells, Verocay bodies (+) and arranged in storiform pattern. Lymphocytes and dilated blood vessels were seen. No malignant

cells were detected. The conclusion was schwannoma at vertebrae thoracal 2.

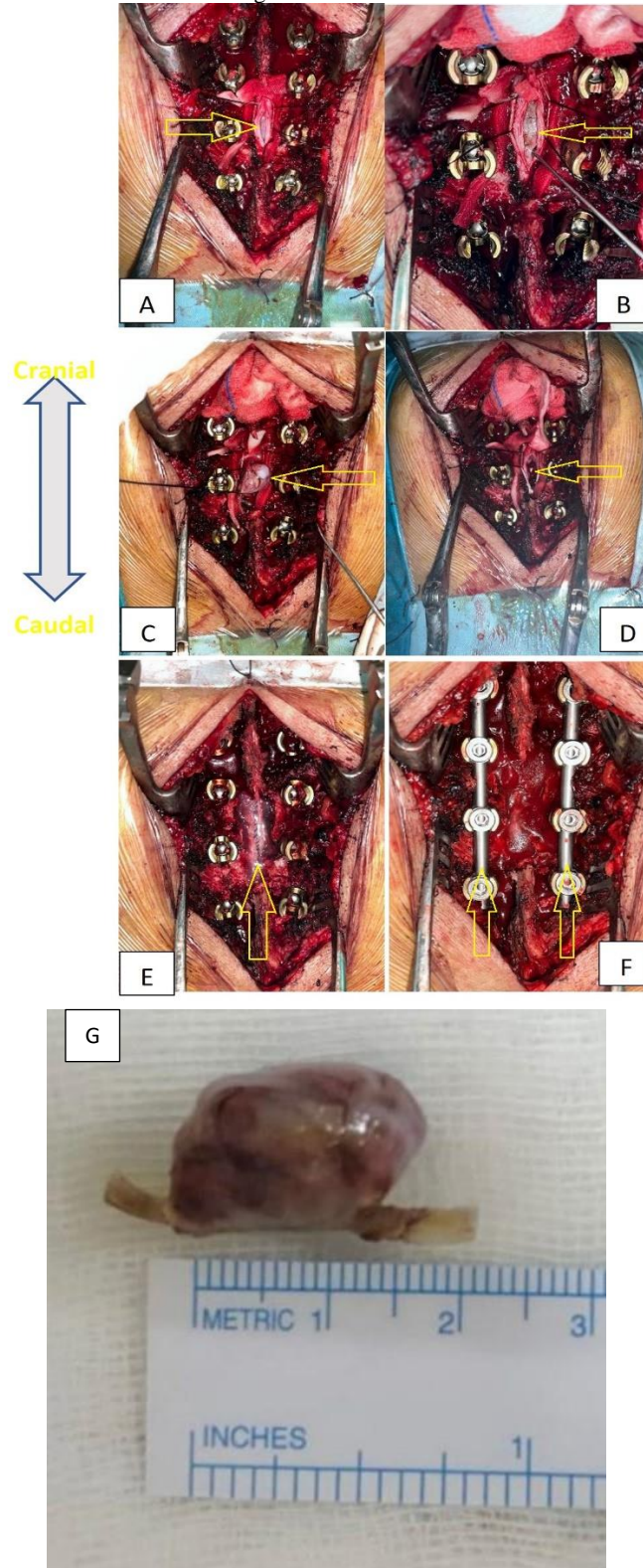


Figure 3. Intraoperative findings (tumor resection process). A-B = dural incision ; C= tumor resection ; D-E = dural closure, F = spinal instrumentation. G. Resected tumor.

Based on history, physical, and the results of supporting examinations, we diagnosed the patient for having myeloradiculopathy due to schwannoma

at the level of T11 – T12, accompanied by stage I hypertension and diabetes mellitus.

Discussion

According to their anatomic locations, spinal tumors can be classified as intramedullary, intradural extramedullary, or extradural; that include meningiomas, neurofibromas, and schwannomas.^{3,4}

Schwannoma is a benign tumor that grows slowly (as the other benign masses) and is encapsulated. The mass is made entirely of schwann cells that are derived from the neural crest (also known as neurinomas of “verocay”, and neurilemmomas), due to the histopathological findings. Any myelinated central or peripheral nerve with schwann cells can give rise to the tumor. Schwannoma is categorized by the World Health Organization as a grade I benign tumor. 90% of the time, schwannomas are isolated. Attention should be paid to syndromic relationships when there are many tumors in the same patient (neurofibromatosis type 2, schwannomatosis, and carney complex).^{5,6,7} In our patient, the histopathological finding showed the appearance of verocay bodies; in accordance with the conventional slogan ‘verocay bodies’ as stated by the most literatures.

The majority of schwannomas, including those that occur within the spinal cord are sporadic. Neurofibromatosis type 2, schwannomatosis, and the carney complex are three diseases where schwannomas can arise, and both sporadic and syndromic schwannomas may have a genetic cause. 3 percent of syndromic schwannomas are caused by neurofibromatosis type 2 (NF2), 2% by schwannomatosis and 5% by meningiomatosis with or without neurofibromatosis type 2.^{8,9}

Referring to genetic studies, the NF2 gene on chromosome 22 is crucial for the growth of both sporadic and syndromic schwannomas. The merlin protein is produced by the NF2 gene (schwannomin). The NF2 gene is inactivated as a result of certain gene mutations, which prevents the merlin protein from being produced. The majority of schwannomas show both NF2 alleles to be inactive. In individuals with Carney complex, there may be a decrease in PRKAR1A expression, while mutations and inactivation of SMARCB1 can occur in spinal schwannomas.^{8,9} We didn’t conduct chromosomal nor genetic studies on our patient.

Clinical manifestations, such as radiculopathy and neurogenic claudication, as well as progressive sensory loss and back pain radiating from the tumor level, are the two primary symptoms of schwannomas.^{3,4} Rodgers et al., and Mohme et al., in their observational and report studies stated that localized back pain is more common in patients with unilateral localized spinal tumors than with concentric growths, which are linked to distributed pain and motor impairments in thoracic schwannoma; these findings are most prevalent in the fourth and fifth decades of life, with a reported male-to-female ratio of 1:1, despite of a recent observation of a modest male preponderance in one of the largest datasets.^{4,8} Our patient was having radiculating back pain in 2017 (four years apart prior to admission), by which her symptoms worsen since then. She also had motor impairment that was proven by the inability to walk and move her both lower extremities independently.

Besides motor and sensory impairment, autonomic dysfunction also must be taken into account among patients that having spinal mass due to its compressive and debilitating effects toward micturition and defecation physiology; in our patient, we found she had urinary and fecal incontinence. Yang et al.¹⁰ and Mattar et al.¹¹ also reported the same findings as our patient had.

There are several available treatment choices available. As for small sized tumor, it is preferable to keep these benign lesions under close observation because resection in a challenging situation could cause a major physiological disadvantage. Radiological modalities that moreover, especially in patients with comorbidities, such as hypertension and longstanding T2DM, surgical interventions might resulted in profound tissue injury.⁸

However, despite of its invasiveness, several clinical trials and systematic reviews stated the surgical intervention is the gold standard for spinal schwannomas.^{2,12,13} Surgical resection, as conducted in our study, can stop tumor progression and hence preventing further neurological deficits.

It also improve patient’s quality of life and to reduced neurological – related symptoms.^{12,13}

In the majority of individuals, surgical removal utilizing a typical midline posterior route is possible. By separating the tumor from the nearby neural structures (spinal cord and/or nerve roots) and then gently dissecting it from an afferent nerve root, the mass is completely removed. Cutting the nerve root during schwannomas surgery is a generally popular option, despite controversy. The majority of publications claim that cutting a nerve root does not significantly raise the likelihood of postoperative neurological impairments. Keep in mind that sensory root origin is significantly more prevalent and that functional compensation by neighboring spinal roots has been proven.^{2,14,15}

Several surgical techniques are also available, one of them is laminectomy to freely resect the underlying tumor. A laminectomy was carried out to levels above and below the tumor using a midline incision and dissection of the bilateral subperiosteal muscles. Sometimes an extraforaminal tumor could not be removed through the laminectomy site, so a radical facetectomy was done. The paraspinal muscle and skin were stitched together after the tumor was removed using microsurgical methods.^{14,15}

In cases of radical facetectomy or spinal instability, we performed posterior rigid stabilization usin posterior instrumentation, and a laminoplasty was carried out utilizing a plate or laminar screw.^{14,15}

Conclusion

Spinal intramedullary schwannoma is a rare medical condition. A thorough history taking, physical and supporting examinations are needed to diagnose its existence. Once the diagnosis is made, treatment must be initiated, particularly for those who have neurological impairments.

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