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Left Parasagittal Solitary Fibrous Tumor Presenting With Lumbar Radiculopathy-Like Symptoms: A Case Report

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Background: Lumbar radiculopathy is typically attributed to degenerative spinal disease, but central lesions can rarely mimic its sensory and motor features. Parasagittal tumors involving the motor and sensory leg regions may present with contralateral weakness, paresthesia, or pain that can be mistaken for peripheral pathology.

Case Report: A 60-year-old man with chronic postoperative lumbar symptoms developed progressive bilateral leg weakness, worsening pain, and new right arm shaking. Lumbar imaging showed no clear correlate, but brain magnetic resonance imaging revealed a large left parasagittal solitary fibrous tumor compressing the leg motor–sensory cortices. Following craniotomy and adjuvant radiation, he experienced marked improvement in strength, sensation, and functional mobility, supporting a cortical source for symptoms previously attributed to radiculopathy.

Conclusions: Persistent radicular-like symptoms unresponsive to spinal intervention should prompt evaluation for central causes, especially when new upper motor neuron signs or multifocal deficits emerge.

Key words: Solitary fibrous tumor; radiculopathy mimic; parasagittal lesion; cortical leg weakness; case report

BACKGROUND

Lumbar radiculopathy is a common clinical presentation often resulting from nerve root compression secondary to degenerative spinal disease. Although lumbar radiculopathy is most commonly caused by peripheral or spinal pathology, central nervous system lesions may rarely produce pseudoradicular or radiculopathy-like motor and sensory deficits (1,2). Lesions involving the parasagittal region of the cerebral cortex, particularly within the precentral and postcentral gyri, can produce contralateral lower extremity weakness, paresthesia, or pain due to involvement of the leg representation area on the medial aspect of the homunculus (3,4).

Solitary fibrous tumors are rare, often highly vascular tumors of mesenchymal origin that account for less than 1% of all intracranial neoplasms (5,6). Parasagittal lesions may compress the supplementary motor area and the leg region of the motor cortex, leading to contralateral motor and sensory deficits (5). While classic symptoms include contralateral leg weakness, sensory loss, or seizures, this case demonstrates an atypical presentation initially indistinguishable from lumbar radiculopathy.

CASE REPORT

The patient was a 60-year-old man with a history of lumbar spinal stenosis, chronic postlaminectomy syndrome, and incomplete paraparesis following L3–L5 laminectomy and fusion performed in 2020. Since that surgery, he had persistent bilateral lower extremity

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spasms, neuropathic pain, and paresthesia that had remained relatively stable with medications including gabapentin, tizanidine, baclofen, and intermittent opioids. He lived independently until late 2023, when progressive low back pain and worsening bilateral leg weakness resulted in his transfer to an inpatient rehabilitation facility. There was no recent trauma, infection, or systemic illness, and his family and genetic history were contributory.

In the month prior to presentation, his longstanding lower-extremity symptoms progressed unexpectedly, accompanied by new right upper extremity weakness and numbness, as well as intermittent episodes of involuntary right upper extremity shaking consistent with focal motor seizures. The patient's "radicular-like" pain was dermatomal, primarily involving the L5 and S1 distributions bilaterally, with burning sensations in the posterior thighs, calves, and soles of the feet. It was not typically shooting or electric in quality but was notably activity-dependent, worsening with prolonged standing or walking and improving when he sat down to rest. Provocative maneuvers such as straight leg raise and facet loading were negative, further suggesting the symptoms might not stem solely from peripheral spinal pathology.

On examination, he had significant bilateral leg weakness (3–4/5), impaired proprioception, diffuse paresthesias, and allodynia out of proportion to peripheral findings. The right upper extremity demonstrated reduced strength as well, along with the intermittent shaking episodes described by the patient. Reflexes were brisk (3+) in the lower extremities compared to baseline, with positive Babinski signs bilaterally and mild spasticity noted in the hip flexors and knee extensors—explicit upper motor neuron (UMN) findings that supported a central lesion rather than pure radiculopathy. His gait was unsafe due to pain and weakness. Sensation was patchy but present throughout.

His lumbar magnetic resonance imaging (MRI) showed only expected postoperative changes without recurrent stenosis or nerve compression. However, brain MRI revealed a large, hemorrhagic, extra-axial mass along the left parasagittal region compressing the primary motor and sensory cortices corresponding to the contralateral leg. The mass arose from the superior left paramedian dura, measured approximately 5.5 × 5 × 4 cm, and was centered in the posterior left frontal region. It was isointense to brain cortex on T1-weighted imaging, heterogeneous and predominantly intermediate signal on T2, with internal restriction of diffusion, uniform avid enhancement, and surrounding vasogenic edema extending to the motor strip. There were internal vascular flow voids and partial drainage to the left internal cerebral vein, along with a 3–4 mm rightward midline shift but no hydrocephalus.

A review of his recent clinical course clarified this timeline: chronic postoperative leg symptoms that had been stable until November 2023; a month of escalating leg weakness and pain without a clear spinal correlate; the sudden onset of right arm shaking; and, finally in December 2023, identification of the parasagittal mass after brain imaging was obtained.

The patient underwent craniotomy with resection of the mass. Histopathologic examination confirmed a solitary fibrous tumor, CNS WHO grade 1 (7). His immediate postoperative course was notable for early improvement in both right upper extremity and lower extremity strength, strongly suggesting that the cortical lesion had played a major role in his symptoms. He then completed 30 fractions of adjuvant radiation therapy due to the known propensity of solitary fibrous tumors to recur or metastasize. Although he initially exhibited features superficially resembling complex regional pain syndrome type 1 (CRPS-1)—such as temperature asymmetry, allodynia, and severe activity-provoked pain—these symptoms gradually improved with mobilization and therapy and were ultimately felt to reflect postoperative cortical reorganization rather than a primary CRPS-1 process.

Over the following months, he continued to improve with ongoing rehabilitation. By the time of discharge from his acute care and radiation course, he had regained 4–5/5 lower extremity strength bilaterally, his temperature asymmetry had largely resolved, sensation had normalized, and his pain was significantly less severe. He was able to ambulate short distances with assistance, a substantial improvement compared with his preoperative functional status. Follow-up imaging showed satisfactory postoperative changes without early recurrence.

DISCUSSION

This case underscores the importance of maintaining a broad differential when evaluating patients with radicular-type lower extremity pain and weakness, particularly when symptoms persist despite adequate spinal intervention. The patient's presentation initially aligned with lumbar radiculopathy, supported by im-

aging findings of lumbar stenosis and postoperative changes. However, the persistence of symptoms and later onset of right upper extremity weakness prompted reconsideration of a central etiology.

The motor and sensory cortices along the medial aspect of the left hemisphere correspond to the contralateral leg (4). Compression of this region by a parasagittal mass can produce leg-dominant weakness and paresthesia, mimicking a peripheral radiculopathy. Prior studies have documented similar cases of parasagittal meningiomas or gliomas presenting with "pseudoradicular" symptoms (8,9). In this patient, the resolution of leg symptoms following resection strongly supports a cortical origin.

Solitary fibrous tumors are dural-based vascular tumors with a propensity for local recurrence and extracranial metastasis (5,6,10). Their clinical presentation depends on location; parasagittal tumors most often cause contralateral leg weakness, seizures, and sometimes headache due to sagittal sinus involvement (5). In contrast, our patient's predominant initial complaint was lumbar pain with radicular features—an atypical presentation that delayed recognition of the cortical pathology. Brain imaging was delayed because the symptoms appeared as a progression of his known chronic spinal condition, with no immediate red flags like headache. Cognitive bias, particularly anchoring to the established spine pathology, likely played a role, as clinicians focused on reevaluating the lumbar region rather than expanding the workup earlier (11). Pain physicians can avoid similar pitfalls by routinely screening for UMN signs in refractory cases, considering central imaging when symptoms evolve atypically (e.g., multifocal deficits or disproportionate progression), and incorporating multidisciplinary input from neurology early on.

Although a transient diagnosis of CRPS-1 was considered postoperatively because of pain disproportionate to the observed peripheral findings, allodynia, and temperature asymmetry, these features gradually improved with mobilization, rehabilitation, and recovery following tumor resection. The clinical course was considered more consistent with a substantial central neurologic contribution to the patient's symptoms, together with postoperative recovery and deconditioning, rather than definite primary CRPS-1. The precise mechanism underlying the postoperative improvement in pain and sensory symptoms cannot be established from this case alone (12). Nonetheless, the overlap highlights the diagnostic complexity of pain syndromes in patients with both spinal and central pathology.

CONCLUSIONS

This case demonstrates that persistent radiculopathy-like symptoms unresponsive to spinal surgery should prompt evaluation for central lesions, especially when new upper motor neuron signs or multifocal symptoms appear. A multidisciplinary approach integrating neurology, neurosurgery, and rehabilitation medicine is essential for accurate diagnosis and optimized recovery.

Patient Perspective

The patient later shared that he had been convinced "something was wrong" with his spine again, and that he never imagined a brain tumor could explain years of leg pain and the sudden decline that led to his hospitalization. He described noticeable improvement after surgery, stating that he felt like he "finally had an answer that made sense," and he expressed relief that the true cause had been identified.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report and all associated clinical information.

Author Contributions

Thomas Hoang Ngo, DO, contributed to the conception and design of the case report, acquisition and interpretation of clinical information, literature review, and drafting of the manuscript. Andrew Garcia, MD, PharmD, contributed to interpretation of the clinical course, literature review, and critical revision of the manuscript. Peter Minh Huynh, MD, contributed to clinical interpretation, critical revision of the manuscript, and supervision of the work. All authors reviewed and approved the final manuscript and agree to be accountable for all aspects of the work.

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