Eccentric Case of Thoracic Spinal Hemangioma Masquerading as Cauda Equina Syndrome: Clinical Pathophysiological Traits and Management Aspects

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Abstract

We present a case of spinal hemangioma with clinical presentation with lower limb weakness, loss of sensation, back pain, urinary retention and difficulty in bowel movements for 9 months. Patients presenting with lumbar myeloradiculopathy should be carefully evaluated to distinguish it from cauda equina syndrome, spinal dural fistula, spinal cord tumors, and metastasis. Evaluation including MRI brain and CT scan lumbar spine were within normal limits. Further workup including CT scan and MRI thoracic spine revealed T5 vertebral body spinal hemangioma with spina cord compression at T5-T6. The main pathology here is slowly progressive tumor that had power to scrunch the spinal cord and shatter the vertebra, thereby ushering the onset of myeloradiculopathy. Neurosurgery was consulted and they decided to perform a preoperative embolization, and surgical decompression. Recurrence of symptoms despite surgical intervention might necessitate administration of radiation therapy. The neuronal recovery following surgery might be prolonged given the degree of spinal cord injury.

Keywords: *Spinal hemagioma, cauda equina syndrome, spinal cord compression, vertebral fracture, myelopathy, radiculopathy, spinal surgery and radiation.*

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Introduction

Thoracolumbar hemangioma was originally coined by Virchow in 1863 to demonstrate a tumor-like-lesion springing up from the endothelial cell proliferation in the bone marrow of the vertebral body [1]. First and foremost, primordial origin of spinal hemangiomas emanates due to derangement in formation of embryonic blood vessels [2]. The incidence of spinal hemangiomas in antemortem and postmortem studies is approximately 27% and 11% respectively [3, 4]. The estimated prevalence of spinal hemangioma in the general population is around 10%, which is little high from what is broadcasted in the prevailing literature [5]. These tumors tend to be more common in females as compared to males, then again the exact reason for this proclivity is nebulous [4]. It is more age-dependent, with its transpiration dominating in the older age groups; 65 years and higher [5, 6].

Clinical Case

This is a 59-year-old right handed female presented for evaluation of lower extremity weakness. She has past medical history of

HTN, DVT. Past surgical history includes Appendectomy, cesarean section, cholecystectomy. Medications include Apxiban, Levothyroxine, Omeprazole, Oxycodone, and Trazodone. She has hard time defining when the exact symptoms started. She first tells me that her symptoms mildly started as early as May 2024. She can clearly recall that problems worsened on December 25 2024 when she had a bad fall while at work. She had developed lower extremity weakness since that time that required her to use a walker. She developed a right lower DVT in March 2025. During this time, she had developed sensory loss in her legs that seems to have progressively worsened over months. Over the past two days, she developed progressive worsening loss in her lower extremity weakness, she was unable to stand from the toilet. She was not able to pass urine or bowel movement for 4 days. She also associated back pain. Due to this, she went to ER, and was found to have 1L of urine in her bladder and she was unable to pass urine. MRI brain with Gadolinium revealed Mild non-specific white matter disease. CT scan lumbar spine without contrast did not reveal obvious pathology observed in this study. She was diagnosed with paraparesis with myelopathic findings that sounds. On physical examination, she has sensory level in the T-6 which puts her disease process most likely in the T-spine. With that being said, the neurologist decided to move forward with a treatment plan comprising of bed rest, treatment UTI, monitor urinary retention, CT scan without contrast T-spine and MRI T-spine with and without gadolinium. CT scan Thoracic spine revealed T5 vertebral body and right pedicle hemangioma. MRI thoracic spine indicated significant and ultra-severe spinal cord compression at T5 and T6 from extraosseous extension of this hemangioma. There is a T2 signal change within the spinal cord. These findings significant for spinal cord compression centered at T5, leftward deviation of spinal cord and severe thecal sac stenosis. The spinal cord also demonstrates T2 hyperintense hyperintensity from T4-T6 raising concern for myelopathy. The final diagnosis was severely compressive atypical hemangioma with extraosseous extension and significant epidural compression from T4-6, along with a spinal cord injury in the form of T2 intermedullary signal change. Neurosurgery was consulted. The plan was to perform a preoperative embolization followed by surgical resection and stabilization. This would involve transpedicular

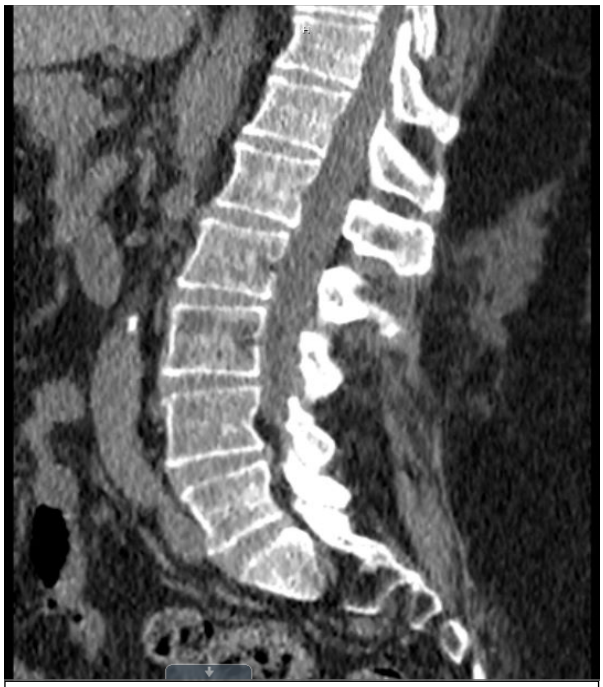

Figure 1: *CT scan Lumbar spine: not revealing obvious pathology observed in this study.*

decompression at T5 & T6, decompression of the neural elements from T4-T6, long segment fusion from T3-T8. Goal of surgery is to halt progression of neurological decline. Recovery of neurological function will take prolonged therapy and rehabilitation given the timeline of symptoms.

Discussion

Most of these spinal hemangiomas are less than 10 mm in diameter [4]. Grossly, 26% of these tumor-like masses are more vulnerable to revamp their size gradually over a period of time, with their growth rate estimated to be < 3mm per 10 years [6]. The proclivity of spinal hemangioma fluctuates in different regions of the spine, with lumbar spine (10%) the highest followed by thoracic (7%) and cervical spine (1%) [6]. On the flip side, some reports pinpoint that thoracic spine is most predominant region for its inception, with T11 & T12 being the most common site [5]. In our patient, the hemangioma has been delineated to T4-T5 level. Routine imaging usually brings to light single spinal hemangioma, although multiple ones are occasionally identified in 33% cases [4, 5, 7]. All the parts of the vertebra including anterior, posterior central and peripheral territories are equally involved with the tumor [5].

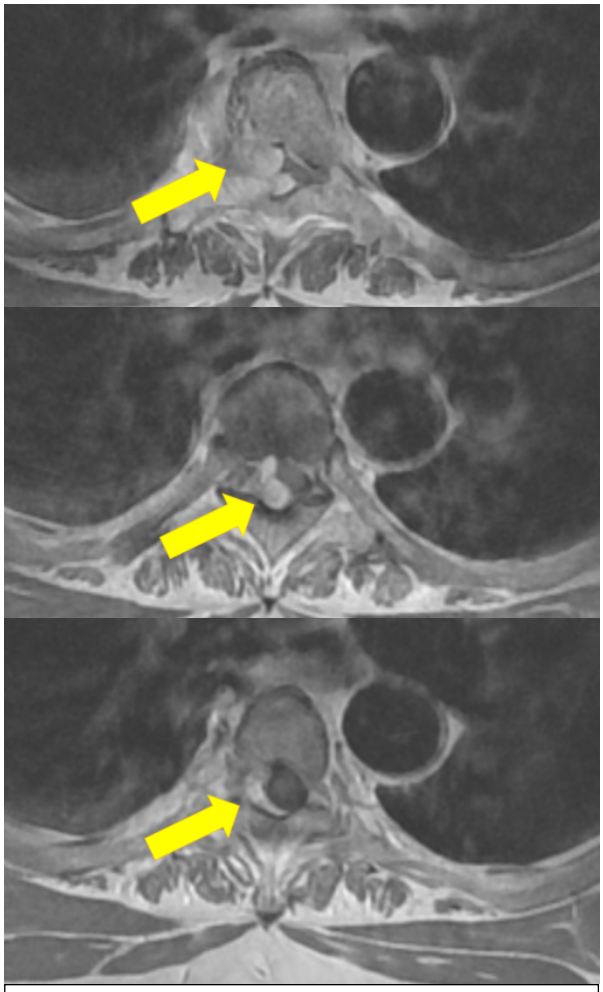

Figure 2B: *MRI Thoracic spine axial view.*

Figure 2 A & B - MRI Thoracic spine:
T2 hyperintense enhancing osseous lesion at T5 vertebral body with involvement of the right pedicle, transverse process and spinous process. In concert with the comparison thoracic spine CT, this reflects a vertebral body hemagioma. Additionally, there is T2 hyperintense enhancing right posterior and right epidural space lesions centered at T5, The posterior soft tissue lesion measures 7.5 mm AP and extends approximately 6.2 cm craniocaudally. The right sided enhancing soft tissue lesion at T5 approximately 1.1 mm x 1.1 x 3.8 cm (APx transverse x craniocaudal). While non-specific the enhancing soft tissue lesion may reflect extraosseous hemangioma, giant cell tumor or soft tissue lesion.

Given this abnormal metamorphosis of endothelial cells during embryonic stages, malformed smaller vasculature including arterioles and capillaries clump together to form a tumor like lesion accompanied by adjoining bone, soft tissue and fibrous tissue [8]. With few exceptions, most of these tumor-like-lesions despite being benign proliferate bit-by-bit to amplify in size [8]. As they attain a palpable and considerable size, they acquire a liability to swap around, erode and subluxate the bony structures as well as compress the spinal cord [8]. These afore

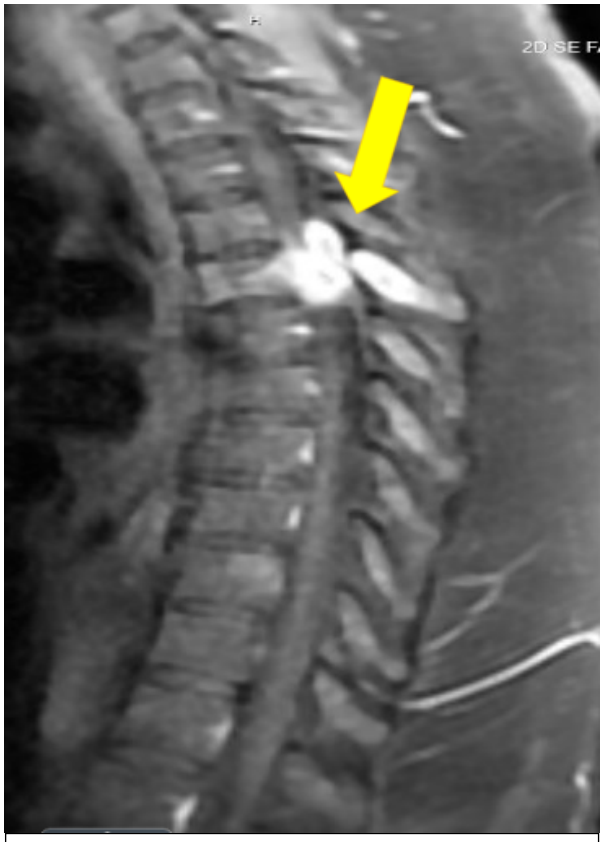

Figure 2A: *MRI Thoracic spine saggital view contrast enhanced.*

mentioned changes will cause a variety of clinical symptoms secondary to spinal cord compression.

According to WHO (World Health organization), the vascular tumors of the bone are classified into benign, intermediary and malignant [9]. Hemangiomas are the textbook example for completely benign tumors due to their inherent slow and progressive growth [9]. Pathologically, spinal hemangiomas are comprised of vascular lined enclosed spaces in close proximity with non-vascular lines canulae encompassed with adipose tissue, smooth muscle, fibrous tissue, bone, hemosiderin and thrombus [10, 11]. Histologically, they are classified into capillary and cavernous hemangiomas based on the presence or absence of adjoining bone [12]. Cavernous hemangiomas are close conglomerations of poorly matured and rudimentary blood vessels without any accompanying bony structures [12]. On the flipside, capillary hemangiomas are agglomerations of primitive capillaries with adjoining bone [12]. Having said that, the growth patterns, invasiveness and clinical presentation of these two types of capillary hemangiomas are still unsettled. Intermediate

variant known as an epitheliod hemangioma was described, which is more prone to be very invasive locally with subsequent lymphoid metastasis [9]. In the last category, angiosarcoma is a typical example of most malignant vascular neoplasm [9]. As per Kato, K. et al, spinal hemangiomas are classified into typical, atypical and aggressive based on their radiological appearance [13]. There are also classified into types I-V based on the presence of bony destruction, soft tissue extension, epidural extension and neurological deficit [14].

Most of the spinal hemangiomas are asymptomatic. However, in few clinical scenarios they tend to grow progressively and encroach the adjoining structures. Having said that, these mushrooming hemangiomas compress and erode flanking vertebral bodies, spinal cord and nerve roots, thus giving rise to myeloradiculopathy. [8, 13, 15]. Our patient presented with classical symptoms of myeloradiculopathy with back pain, limb weakness, urinary retention and difficulty in bowel movements. The mechanism of compressive myeloradiculopathy by spinal hemangiomas can be appreciated by myriad mechanisms including compression vertebral fracture, enlargement of posterior cortex of vertebral cortex, seepage of hemangioma into the epidural space and thereby eliciting the formation of epidural hematoma [16].The spinal hemangiomas with these aggressive traits are more destined to be located in the thoracic spine [17].

Once the diagnosis of spinal hemangioma is suspected, it is prudent to proceed with further evaluation of the involved spine with CT scan and MRI. In our patient, we detected spinal cord compression at T4-T5 from extraosseous extension of the tumor, thecal sac stenosis and myelopathy. Few tumorous lesions that can impersonate a spinal hemangioma include herniated disks, synovial cysts, granulomatous infections, neurogenic tumors, lymphomas, meningiomas, angiolipomas, pure epidural hematoma, and epidural extramedullary hematopoiesis [18]. On CT scan, the tell-tale signs of spinal hemangiomas can range from neural arch involvement, honey comb pattern, fat accumulation within bony structures and vertebral fractures [19].
There are some specific imaging signs of spinal hemangiomas on MRI that are useful in identifying them. First, there is Polka-Dot sign in which there is appearance of hyperintense vertical trabe-culae sprinkled with hypointense areas caused by fat accumulation [10, 13], Second, there is Coduroy sign formed by hypertrophy of vertical trabeculae intermixed with lesser density of the surrounding bone, thereby providing a Coduroy like cloth appearance [10, 13]. MRI might reveal hyperintense T1 and T2 weighted images due to overaccumulation of fatty tissue and hypervascularity of the lesion [20]. Opposingly, a hypointense T1 weighted image, and hyperintense T2 weighted image might be diagnostic of spinal hemangioma [21]. Lee, J.W. et al had classified spinal hemangiomas based on their MRI features into type A (Cystic lesions with T1 hyperintensity), type B (Cystic lesions with T1 hypointensity), type C (Solid hypervascular mass) and type D (Epidural hematoma) [18]. Fat saturated T1 an T2 weighted lesions might unearth hyperintense and heterogenous lesions [22].
Conversely, aggressive vertebral hemangiomas on MRI may present with features such as vertebral body collapse, underlying osteoporosis, pedicle erosion, irregular honeycomb-like lesions, cortical expansion, associated soft tissue mass, intraosseous fat migration into the neural arch, and focal lytic changes [10, 23-25]. Another feature that helps to differentiate between aggressive and benign hemangiomas is to assess their fat suppression sequences [11]. Few clinical cases might necessitate the usage of angiography in spinal hemangiomas which might reveal capillary and arteriole dilation within the vertebral body and opacification of the vertebral, and blood pooling within capillary vessels [24]. Rarely, advanced imaging studies such as diffusion weighted imaging (DWI) and dynamic contrast enhanced magnetic resonance imaging (DCE MRI) perfusion imaging might necessary to differentiate aggressive hemangiomas from cancer metastasis [26, 27].
Spinal hemangiomas that are asymptomatic can be left alone and monitored through imaging scans at regular intervals. However, the presence of spinal cord compression, and vertebral body erosion is the indication for administration of preoperative embolization, vertobroplasty, laminectomy with or without fusion [8, 13, 16, 28]. Recurrent lesions despite of these aforementioned measures might require reassortment to radiation therapy for pain management and symptom relief [29].

Conclusions

Spinal hemangiomas are vascular tumors that come to existence due to immature growth of capillary blood vessels in the embryonic stage. Despite being benign, they have the potential to enlarge slowly to compress the neighboring spinal cord, nerve roots and vertebral bodies, thereby producing the onset of myeloradiculopathy. The most predisposed site for their origin is thoracic spine, closely followed by lumbar spine and cervical spine. It is important to delineate these tumors from cauda equina syndrome, spinal dural fistula and cancer metastasis. Careful history to ascertain the chronological onset of symptoms is a must. We would like to emphasize the take home message that physical exam findings can frequently can help localize the level of the neurological injury. Our patient had only lumbar spine CTs done, thus delaying the diagnosis. However, a careful examination which revealed a thoracic spinal cord level, persistent lower extremity reflexes, and an upgoing toe, helped localize the lesion to the thoracic spine. This should be supplemented with imaging the entire spine with MRI/CT in the patients with myeloradiculopathy to pin point the location of the tumor. This will help to detect these tumors at earlier stages before they exert their constricting effects on the adjoining spinal cord and vertebra. Although, specific signs such as Polka-Dot sign and Coduroy sign on MRI were indicative, these will not be omnipresent. It is crucial to recognize the aggressive variants by their traits such as honeycombing pattern, vertebral fracture and intraosseous fat accumulation. Spinal surgery to remove them will provide drastic symptomatic relief. Recurrence of symptoms despite these measures compels to seek the opinion of radiation oncology for possible radiation therapy. Despite of all these measures, the degree of neuronal recovery cannot be predicted given the extent of neuronal devastation that is been consolidated. Long and short of it, a high degree of suspicion is imperative for earlier detection of these tumors so that a high priority management can be embarked upon for optimal clinical outcomes in these patients.

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Declarations

Consent for publication: The authors clarify that written informed consent was obtained and the anonymity of the patient was ensured. This study submitted to Swiss J. Rad. Nucl. Med. has been conducted in accordance with the Declaration of Helsinki and according to requirements of all applicable local and international standards.

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- Data Curation: N.A.;
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