Lumbar vertebral hemangioma presenting with the acute onset of neurological symptoms

Case report

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Vertebral hemangiomas are common entities that rarely present with neurological deficits. The authors report the unusual case of a large L-3 vertebral hemangioma with epidural extension in a 27-year-old woman who presented with hip flexor and quadriceps weakness, foot drop, and leg pain. The characteristics of the mass on magnetic resonance imaging suggested an aggressive, hypervascular lesion. The patient underwent embolization of the lesion followed by direct intralesional injection of ethanol. Significant resolution of clinical symptoms was observed immediately after the procedure and at her follow-up visits. Follow-up imaging studies obtained 9 months after the procedure also documented a considerable reduction in the size of the hemangioma with minimal loss of vertebral height and a mild kyphosis at the affected level. On repeated imaging studies obtained 21 months postoperatively, the size of the hemangioma and the degree of vertebral body compression were stable. As demonstrated in this case, patients with vertebral hemangiomas can present with acute nerve root compression and signs and symptoms similar to those of disc herniation. Vertebral hemangiomas can be treated effectively with interventional techniques such as embolization and ethanol injection.

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KEY WORDS • embolization • ethanol injection • hemangioma • lumbar spine • radiculopathy

Vertebral hemangiomas are relatively common benign lesions of the spinal column found in an estimated 10 to 12% of the population based on large autopsy series and reviews of plain spine films.10,21 The majority of these tumors are located in the thoracic spine with comparatively fewer lesions in the lumbar and cervical regions; multiple-level involvement occurs in up to 30% of cases.10 While these lesions can arise in patients over a wide age range, vertebral hemangiomas are usually detected in patients in the fourth and fifth decades of life;13 women appear to be more prone to these lesions.10,32

Although vertebral hemangiomas are typically incidental findings, they are symptomatic in 0.9 to 1.2% of cases.13,25,28 Of the symptomatic lesions, fewer than half are associated with neurological deficits, the rest being characterized by pain only.10 Multiple mechanisms can lead to the manifestation of neurological symptoms, including 1) bone expansion caused by the hemangioma; 2) extraosseous extension of the tumor into the epidural space; 3) disturbance of local blood flow; and 4) compression fractures (rarely).10,13,22 Spontaneous malignant transformation of vertebral hemangiomas has not been reported, although there have been rare cases of angiosarcomas arising from hemangiomas following radiation.27

We present the rare case of an aggressive vertebral hemangioma at L-3 acutely presenting with hip flexor and quadriceps weakness, foot drop, and leg pain as a result of the spread of the mass into the epidural space. The hemangioma was successfully treated with embolization followed by an intralesional ethanol injection.

Case Report

History and Examination. This 27-year-old woman with no significant medical or surgical history presented to the emergency department with a 2-week history of lumbar pain and left lower extremity pain that began in the thigh and ascended into her left buttocks and down her left leg. The pain was sharp and limited the patient’s ability to walk

Abbreviations used in this paper: MR = magnetic resonance; VB = vertebral body.
more than a few steps at a time. Neurosurgical evaluation was sought because of the presumed diagnosis of a disc herniation. At admission, the patient described the development of paresthesias isolated to the left foot. She denied all other neurological symptoms, including urinary incontinence or retention, as well as any recent history of trauma.

On neurological examination, the patient demonstrated decreased motor strength in hip flexion (3/5), leg extension (3/5), and dorsiflexion (3/5) on the left. All other muscle groups in her upper and lower extremities were full strength. Dorsiflexion of the left foot elicited pain, but the patient was cooperative with the examination and gave maximal effort on strength testing. Sensation was decreased over the left foot but was otherwise grossly normal. The patient’s left patellar reflex was diminished, and her gait was very unsteady due both to leg pain and weakness. The results of her medical examination were unremarkable.

Hospital Course and Treatment. A T2-weighted lumbar MR imaging study revealed the presence of an enhancing, hyperintense lesion involving the entire L-3 VB and extending into the left pedicle, lamina, and transverse process (Fig. 1A–C). There were also intensely enhancing lobulated structures, hypointense on T1-weighted and hyperintense on T2-weighted imaging, located in the epidural space medial and inferior to the left L-3 pedicle and para-

Fig. 1. Imaging studies obtained at presentation. Sagittal T1-weighted MR images obtained before (A) and after (B) the addition of contrast demonstrating a hypointense, enhancing lesion involving the entire L-3 VB with extension into the epidural space. On axial T2-weighted imaging (C), the lesion is seen to extend into the left pedicle, transverse process, and lamina, as well as the left paravertebral space. The epidural tissue is seen compressing the thecal sac. Sagittal (D), coronal (E), and axial (F) views of the bone architecture on computed tomography imaging show the classic vertical striations due to trabecular thickening attributed to vertebral hemangiomas.
vertebral space. The epidural mass extended into the left L-3 neural foramen and lateral recess, compressing the L-3 and L-4 nerve roots and the thecal sac. Computed tomography imaging demonstrated diffusely thickened, vertically oriented trabeculae in the VB, as well as evidence of bone softening and mild cortical compression (Fig. 1D–F). No changes in VB height or bone alignment were noted. In the aggregate, these imaging findings suggested the diagnosis of an aggressive vertebral hemangioma.

A spinal angiogram demonstrated puddling of contrast within the L-3 VB consistent with a hemangioma (Fig. 2). The left L-3 lumbar artery was found to be the primary vascular supply for the lesion and was embolized with polyvinyl alcohol particles. The hemangioma was treated 3 days after the embolization procedure with a direct intraleisional ethanol injection. A bone biopsy needle was inserted percutaneously into the L-3 VB via the left pedicle using intermittent computed tomography guidance. Injection of contrast demonstrated satisfactory opacification of the epidural and foraminal components of the hemangioma and no leakage into the subarachnoid space. A total of 9 ml of absolute ethanol was injected into the VB, the junction of the body and pedicle, and the pedicle itself. The patient tolerated the procedure well and was discharged home the next day. At the time of discharge, her left dorsiflexion strength had improved to 4/5, and her left foot paresthesias had diminished.

Follow-Up Examination. Three weeks after discharge, the pain in the distribution of the L-4 dermatome was significantly improved compared with her pretreatment condition. She continued to report a slight weakness in her left quadriceps muscles. A follow-up MR imaging study obtained at this time documented a significant decrease in the size of the epidural and paravertebral components of the lesion (Fig. 3A and B). The L-3 VB demonstrated decreased enhancement and increased T1- and T2-weighted signal, indicating changes from the ethanol injection. By 9 weeks after her discharge, the patient had completely recovered motor strength in her left leg and required only over-the-counter pain medications to control her intermittent pain.

Nine months after treatment another follow-up MR imaging study was obtained in this patient, which showed continued resolution of the hemangioma. A partial collapse of the L-3 VB had developed in association with mild kyphosis at the L2–3 level (Fig. 3C). Bone edema at the inferior aspect of the L-2 VB caused by abnormal biomechanical forces was also seen. However, the patient’s occasional back and left leg pain had not changed. Because of the risk of a pathological compression fracture at L-3, a second ethanol injection to obliterate the residual hemangioma was not performed. Vertebralplasty with methylmethacrylate was considered at this time but was deferred in favor of observation because of the minor degree of bone changes and the patient’s stable clinical status. Twenty-one months after treatment, the patient remains stable from both a clinical and radiological perspective.

Discussion

Vertebral hemangiomas that cause neurological deficits are rarely located in the lumbar vertebrae. Lesions in this region can cause cauda equina syndrome and compression of the medullary conus. The current case demonstrates the consequences of nerve root compression and establishes that lumbar vertebral hemangiomas can mimic the signs and symptoms of herniated discs. The patient’s hip flexor and quadriceps weakness, along with the diminished patellar reflex, are consistent with the L-3 and L-4 nerve root compression seen on MR imaging. However,
there is no clear radiographic explanation for her foot drop and foot paresthesias, and this finding may have been the result of pain.

The classic radiographic features of vertebral hemangiomas are coarse vertical striations and “honeycombing” due to trabecular thickening. This osseous reinforcement explains why fractures at the site of hemangioma growth are not commonplace. Computed tomography scanning defines bone architecture and is the optimal modality for diagnosing vertebral hemangiomas. Magnetic resonance imaging can be used to evaluate for the presence of spinal cord and nerve root compression as well as tumor aggressiveness. Hemangiomas with a fatty stroma (hyperintense on T1-weighted images) are generally indolent, whereas hypervascular lesions (hypo- or isointense on T1-weighted images, extensive flow void areas) have a higher risk for causing pain or spinal cord compression.

It is not completely clear why some vertebral hemangiomas become symptomatic while most remain asymptomatic. As noted above, hypervascularity correlates with symptomatic hemangiomas, but the biological factors governing increased blood vessel growth in these lesions have not been defined. The authors of one study have suggested that capillary-type hemangiomas are associated with progressive disease to a greater extent than cavernous-type or mixed hemangiomas, but the authors ultimately concluded that the histopathology did not definitively predict whether a hemangioma would be symptomatic. From an epidemiological perspective, patients with symptomatic hemangiomas tend to be younger and female, and their lesions are more likely to be found in the thoracic spine and in the posterior elements. A factor that appears to increase the risk of developing neurological symptoms in previously quiescent hemangiomas is pregnancy. It has been hypothesized that the increase in intraabdominal pressure caused by the growing fetus augments blood flow to the vertebral venous plexus and that increased estrogen levels may enhance endothelial growth in hemangiomas.

Several features help to explain the large size of our patient’s hemangioma, its aggressive growth, and the acute
onset of neurological deficits. The MR imaging characteristics of the mass were consistent with a hypervascular lesion, and the patient’s demographic profile, a young woman with a lesion involving the posterior elements, fit the expected picture of a symptomatic case. Moreover, although the patient was not pregnant at the time of her presentation, she had recently given birth.

The management of vertebral hemangiomas depends on the clinical presentation of each case. Asymptomatic lesions progress infrequently, and patients with vertebral hemangiomas can be observed with no further workup required unless pain or neurological deficits arise. Multiple approaches are available for treating symptomatic hemangiomas when conservative therapy is no longer a viable option. Although radiation therapy is primarily used to relieve hemangioma-associated pain, the authors of some studies have suggested that it can also improve neurological symptoms in up to 72% of patients. However, radiation necrosis of the spinal cord is a significant drawback to the use of this modality, and patients with acute spinal cord compression may not derive any benefit because of this delayed effect. Therefore, vertebroplasty with methylmethacrylate effectively treats hemangiomas that cause pain. Although it affords the benefits of vertebral column stabilization, this technique can exacerbate spinal cord compression, especially in cases of extensive hemangioma growth into the spinal canal. Leakage of cement material into the spinal canal can also cause direct damage to neural tissue. Some authors recommend that vertebroplasty should not be used in patients who present with neurological deficits, but others contend that the combination of vertebroplasty and decompressive surgery can successfully deal with these cases. Surgical treatment is reserved for cases involving spinal cord compression with progressive neurological deficits. Interventions are chosen based on the size and location of the mass and may range from decompressive laminectomy to vertebral corpectomy with subsequent reconstruction. Presurgical embolization of the vessels feeding the hemangioma is a useful adjunct to reduce intraoperative blood loss. Embolization does not obliterate the hemangioma and thus leads to only temporary symptom relief. This technique carries the risk of spinal cord infarction from the emboclusion of vessels that feed both the hemangioma and the cord.

Intralesional injection of ethanol under radiographic guidance has been advocated as a therapeutic alternative. First described by Heiss et al., this procedure causes intravascular thrombosis and endothelial destruction within the hemangioma, leading to devascularization and subsequent shrinkage of the lesion. Potential benefits of ethanol injection include the minimal technology required to perform the procedure, obliteration of the lesion without significant blood loss, and rapid and sustained clinical improvement in most patients. The few studies of intralesional ethanol injection in the literature have described encouraging results. Resolution of symptoms can be seen in 64 to 93% of patients, and radiographic obliteration or reduction in lesion size is seen in 82 to 86% of patients.

Complications related to intralesional ethanol injections are relatively uncommon but can have significant consequences. To minimize these risks, Heiss et al. noted that it is imperative to use test injections of a contrast agent in order not only to delineate the hemangioma morphology but also to detect any leakage of injected material into the subarachnoid space. Other authors have suggested that ethanol opacification with metrizamide powder can further reduce the risk of inadvertent penetration of ethanol into the subarachnoid space or epidural venous plexus.

There have been three cases of VB collapse, thought to be secondary to ethanol-induced osteonecrosis, requiring surgical intervention occurring anywhere from 4 weeks to 5 months after treatment; all instances of pathological fractures were associated with the injection of large quantities of ethanol (30–50 ml). Doppman and colleagues suggested that less than 15 ml is a safe volume for injection, but two cases of asymptomatic loss of vertebral height have been described using this guideline. In the acute setting, Niemeyer et al. have reported the development of Brown-Séquard Syndrome 1 hour after intralesional ethanol injection. The authors attributed this condition to thrombosis of the epidural veins that resulted in spinal cord ischemia, although subarachnoid spread of ethanol could not be ruled out entirely. Other complications related to this procedure include the development of a paravertebral abscess after extravasation of contrast into the pleural space and paravertebral tissue and hemangioma recurrence within a month.

The results in our patient demonstrate the usefulness of ethanol injection in treating large aggressive vertebral hemangiomas and provide further evidence that the injection of a moderate amount of ethanol can be associated with changes in vertebral architecture. Larger studies with longer follow-up will be required to fully appreciate the range of early and late complications associated with intralesional ethanol injection, as well as its long-term efficacy.

References

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