Original Article

Outcome of dorsolumbar vertebral hemangiomas presenting with neuraxial compression

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Abstract

Background: Hemangiomas are benign vascular tumors associated with proliferation of blood vessels in bone or soft tissue and they are usually incidental findings in vertebrae. When symptomatic, they present with features of radiculopathy, myelopathy, or vertebral fractures. Treatment options are varied, include sole embolization, embolization combined with surgical excision, surgical excision alone, percutaneous ablation, and radiotherapy. We hereby describe a series of seven cases of symptomatic vertebral hemangiomas operated from 2006 to 2009.

Materials and Methods: Their clinical and radiological profile and outcome have been described. All patients were subjected to surgical excision followed by instrumentation. Outcome was assessed at a followup of 2 years following surgery with Frankel grading system.

Results: Seven patients (five females and two males) were included in the study. The mean age was 33.85 years with the mean duration of symptoms of 12 months. All seven cases were symptomatic vertebral hemangiomas with cord compression and underwent surgical excision. Preoperatively, patients with poor Frankel grade such as A and B improved postoperatively to C, D, or E.

Conclusion: Surgical excision of these lesions is difficult due to the tremendous amount of intraoperative bleeding. During surgery, brisk bleeding is usually encountered, but can be brought under control with adequate preoperative preparation and expertize. Preoperative embolization may help to reduce the bleeding, but at times it may be difficult to do if vertebrae are replaced by a solid hard mass. In spite of the risks associated with surgery, it still is the treatment of choice as a single intervention, especially in aggressive vertebral hemangiomas.

Key words: Hemangioma, myelopathy, spinal extradural compressive myelopathy, spinal hemangioma

Introduction

Vertebral hemangiomas are common benign lesions of the spinal column usually asymptomatic and are discovered incidentally or found during evaluation of neck or back pain.[1-3] They comprise 12% of the benign tumors affecting the general population.[4-7] They are congenital vascular malformations and not true neoplasms. They consist of remnants of embryonic capillaries and veins, but unlike congenital vascular malformations, arteriovenous shunting does not occur in these hemangiomas. Rarely they may enlarge and cause pain and neurological deficit because of spinal cord compression, vertebral body or arch expansion, or pathological fracture. Radiology plays a crucial role in the diagnosis and management of symptomatic vertebral hemangiomas. Plain spine films often suggest the diagnosis of vertebral hemangiomas by the presence of coarse vertical striations; however, one-third of the vertebral body must be involved for these findings to be evident. Computed tomography (CT) scan helps in determining the extent of vertebral involvement and magnetic resonance imaging (MRI) provides information regarding the aggressiveness of the disease.

Treatment options for symptomatic vertebral hemangiomas include transarterial embolization, surgical excision, radiotherapy, and percutaneous injection of alcohol or methyl methacrylate. Embolization alone is not quite effective in the treatment of symptomatic hemangiomas causing compression of the neuraxis. Nowadays, preoperative embolization followed by surgical excision is the preferred treatment modality in many centers.

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Access this article online	
Quick Response Code:	Website: www.ijoonline.com
	DOI: 10.4103/0019-5413.101033

However, even with preoperative embolization, blood loss has been considerable. Radiotherapy may be successful in obliterating the hemangiomas, but the effect may be too delayed for patients with cord compression.[8,9] We have analyzed seven cases of vertebral hemangiomas causing significant cord compression and features of myelopathy. All patients underwent corpectomy through a retroperitoneal approach or transthoracic approach followed by stabilization. The problems of differential diagnosis in such cases, their surgery and postoperative management are discussed in this article.

Materials and Methods

We retrospectively collected the data of patients of symptomatic vertebral hemangiomas with neuraxial compression that were managed from 2006 to 2009 and have a followup at our Institute. The demographic, radiological profile and treatment modalities used were analyzed. Patients were evaluated for clinical features such as backache, radiculopathy, myelopathy, sensory deficits, and bowel/ bladder involvement. The patient who opted for other treatment modalities such as embolization or percutaneous ablation were excluded.

A total of seven patients were analyzed preoperatively according to Frankel grading system [Table 1] and compared with the postoperative status. Preoperatively MRI was done in all patients. All seven patients were subjected to surgical excision of the hemangiomas followed by instrumentation.

Postoperatively, X-ray was done in all cases to assess the adequacy of decompression and instrumentation. All patients were assessed in the wards postoperatively and long term followup was obtained in the outpatient departments or on regular correspondence. Clinical and functional outcomes of these patients were assessed based on a standard format. Followup was done regularly at 3 months, 6 months, 12 months, and then yearly.

Results

Seven patients with five females and two males were included in the study. The mean age was 33.8 years with range from 20 to 40 years. The mean duration of symptoms was 12 months (range of 2–36 months). The clinical signs developed insidiously in two patients and rapidly in five patients (3–5 months). Clinical presentation included low backache in all the patients with associated spastic paraparesis. Grossly the motor power was MRC grade 0 in three patients and grade 3–4/5 in the remaining four patients. Bowel and bladder involvement in the form of urinary retention and constipation was present in three cases. The paralysis was graded by Frankle grade C. All patients were evaluated radiologically with X-rays and MRI. The thoracic vertebrae were involved in five and lumbar vertebrae in two of the patients. Radiology revealed complete destruction of the vertebral body in all seven cases with destruction of posterior elements in one of them. T1-weighted images revealed isointense changes in the destroyed vertebral bodies in three patients and hyperintense changes in four patients. All had T2 hyperintensity changes [Table 2]. All were associated with epidural soft tissue changes with extension and compression onto the thecal sac.

All these cases were treated surgically and we used a retroperitoneal approach in two lumbar lesion and transthoracic approach in five of the cases. Excision of the vertebral bodies was done in the seven cases and all had brisk bleeding during surgery. The blood loss ranged from 1.5 to 3 l. The loss was adequately and immediately replaced with blood and fresh frozen plasma. After excision of the vertebral bodies, the dura was seen thoroughly superiorly, inferiorly, and laterally, indicating adequate decompression. These patients underwent instrumentation in the form of caging and stabilization with screws in the vertebral bodies and rod construct. Postoperatively, the hemoglobin and bleeding parameters were regularly monitored and replaced accordingly. Two patients had abnormal bleeding parameters postoperatively, but were corrected with fresh frozen plasma and vitamin K. At average followup of 24 months, their comparison showed significant improvement in their neurological status [Table 3].

It was found that of the three patients who had a grade A preoperatively, two improved to grade C and one to

Table 1: Frankel grading system

Grade A	Complete neurological injury – no motor or sensory level detected below the level of injury
Grade B	Preserved sensation only – no motor function clinically detected below the level of the injury; sensory function remains below the level of the injury but may include only partial function (sacral sparing qualifies as preserved sensation)
Grade C	Preserved motor non-functional – some motor function observed below the level of the injury, but is of no practical use to the patient
Grade D	Preserved motor function – useful motor function below the level of the injury; patient can move lower limbs and walk with or without aid, but does not have a normal gait or strength in all motor groups
Grade E	Normal motor – no clinically detected abnormality in motor or sensory function with normal sphincter function; abnormal reflexes and subjective sensory abnormalities may be present

Table 2: Clinical profile of patients

Duration of symptoms	Age/sex	Level of compression	Grade of muscle/power	Surgery done	Postoperative status (12 months)
6 mnths	35/m	D2-D3	0/5 both lower limbs. Urinary and bowel incontinence	D2-D3 decompression and transpedicular screw fixation	Sensory improvement upto 40% in both lower limbs with grade 2/5 power in both lower limbs
36 mnths	32/m	L1	bilateral lower limbs- 3/5, 20% sensory loss in right L5 S1	Retroperitoneal approach and L1 corpectomy and caging	Power improved to 4/5 in both lower limbs with improvement in sensory symptoms
3 mnths	40 /f	L1	Both lower limbs – 4/5 Sensory loss of 50% below L1	Retroperitoneal approach and decompression and caging	Power improved to 4/5 in both lower limbs. Patient ambulatory. Sensory improvement present
5 mnths	20/f	D10	0/5 on both lower limbs. Sensory loss of 30% below bowel and bladder involvement present	Right transthoracic decompression and caging	Bladder and Sensory improvement present
4 mnths	35/f	D10	3/5 power in the lower limbs. Sensory deficit of 20 % below L1 dermatome bilaterally	Left posterolateral thoracotomy and D10 corpectomy with caging and instrumentation.	Power improved to 4/5 and patient is ambulatory with support
24 mnths	35/f	D10	0/5 in both lower limbs with bowel and bladder involvement	Left posterolateral thoracotomy and D10 corpectomy with caging and instrumentation.	Bladder, bowel and sensory improvement in postoperative period. Motor power improved to 1/5
6 mnths	40/f	D9	3/5 power in both LLwith sensory loss of 30% below D10 dermatome	Left posterolateral thoracotomy and D9 corpectomy with caging and instrumentation	Power improved to 4/5 and patient ambulatory with support

Table 3: Pre and postoperative Frankel grading

Patient	Preoperative grade	Postoperative grade (followup)
1	Grade A	Grade C
2	Grade B	Grade D
3	Grade C	Grade E
4	Grade A	Grade C
5	Grade C	Grade D
6	Grade A	Grade B
7	Grade C	Grade E

grade B postoperatively. One patient who had grade B preoperatively improved to grade D. Three patients who had grade C preoperatively improved to grade E in two patients and grade D in one patient. There was bowel bladder involvement in three patients and all of them improved postoperatively. Four patients had features of intercostal neuralgia, but it gradually resolved over a week following surgery. Postoperative radiology taken on the third and twelfth month after surgery showed the stability of implants and grafts and no spinal deformation was noticed. There was no mortality, and in all cases, the biopsy showed features suggestive of hemangioma.

Discussion

Vertebral (intraosseous) hemangiomas account for 2.3% of spinal tumours and are benign vascular tumors composed of proliferation of blood vessels. They are classified histologically by the predominant type of vascular channel as capillary, cavernous, arteriovenous, or venous type. Middle-aged females are the most commonly affected. The majority of hemangiomas is asymptomatic and is discovered incidentally. They usually have an indolent course, but may cause pathological fractures, hemorrhage, extraosseous extension into the paravertebral space, and rarely, epidural extension. Spinal cord or cauda equina compression is quiet rare. The thoracic region is the most common site for vertebral hemangiomas presenting with features of spinal cord compression.[10-12] Causes of neural compression include: (1) expansion of the involved vertebrae, (2) extension of the tumor into the extradural space, (3) compression fracture, and (4) hemorrhage resulting in extradural hematoma.[13] Of these, the first two are the most common mechanisms. However, in our series, the mechanism was compression fracture of the vertebrae and extension of the tumor into the epidural space leading to spinal cord compression. They had features of myelopathy with bowel and bladder involvement in three cases.

Since all patients presented with features of vertebral collapse, they underwent investigations to rule out metastatic, tubercular, or osteoporotic etiology, but all were inconclusive. Though vertebral hemangiomas have typical X-ray and MRI findings, these were lacking in our cases probably because of the soft tissue component predominating in advanced stages of the disease. It is important to realize that by the time compressive collapse

occurs, the classical radiology of vertebral hemangioma is quiet difficult to be demonstrated. Hence, the differential diagnosis of other epidural compressive lesions such as Pott's spine, metastases, and bony neoplasms must also be borne in mind. They tend to erode the horizontal trabeculae of the vertebral bodies, thus leading to the appearance of accentuated, thickened vertical trabeculae, giving rise to "Corduroy or jailhouse striation" appearance in lateral X-ray or sagittal CT scan.[14] In axial CT scans, they give the appearance of a "Polka-dot" or "spike of bone" pattern because the vertical trabeculae are imaged in cross section.[15] MRI may provide additional information regarding the aggressiveness of the hemangioma. Increased fatty signals with mottled appearance on both T1- and T2-weighted images are suggestive of non-evolving intraosseous lesions [Figure 1a,b]. Conversely, osseous or extraosseous hemangiomas that are isointense on T1-weighted images are commonly found with symptomatic lesions. Laredo concluded that fat-intensity lesions are generally inactive, while hemangiomas demonstrating low or isointense signal on T1-weighted images indicate a hypervascular lesion with the potential to compress the spinal cord.[16] This was true in five of our cases where T1-weighted images were isointense [Figure 2a, b]. Another interesting feature noted was that all patients who had T1 isointense changes had a short duration of symptoms. This indicates that T1 isointensity to a certain extent helps in predicting the aggressive potential of the lesion.

Treatment options for symptomatic vertebral hemangiomas include surgery, transarterial embolization, direct ethanol injection, and vertebroplasty; each with varying degrees of success.[17-19] Surgery is indicated when there is significant or progressive neurological deficit. Surgical options include corpectomy or vertebrectomy if there is significant vertebral body involvement with epidural extension causing spinal cord compression. Patients undergoing corpectomy are subjected to instrumentation to provide spinal stability.[6,7,20] In our series, patients underwent caging and pedicle screw instrumentation in six patients and only pedicle screw fixation in one of them [Figure 3a, b]. Surgery was encountered with significant bleeding ranging from 1.5 to 2.5 l in all these patients. But with timely replacement of the lost blood volume, surgery could be continued and adequate excision achieved. All patients were replaced adequately and timely with fresh frozen plasma, blood, and colloids. Patients were regularly followed up for a period of 2 years with periodic radiological investigations. Postoperative radiology revealed the screws well in place with adequate canal decompression [Figure 4a, b]. All patients had neurological improvement of motor, sensory, or bladder symptoms. This justifies the need for surgery to achieve decompression and stabilization.

Endovascular embolization has also been used by some as a sole therapy for vertebral hemangiomas.[21] Hekser *et al.* were the first to report reversal of spinal cord compression following percutaneous embolization of feeding vessels.[22] However, this may not be helpful in the setting of acute spinal cord compression and neurological deterioration. Embolization followed by surgery is another option that is highly propagated in the present era. However, there are reports where preoperative embolization failed to reduce the intraoperative blood loss. This is due to the fact that though transarterial embolization may occlude the feeding vessels, it may fail to destroy the hemangioma due to an intervening capillary bed that separates the hemangioma from the arterial feeders. Reflux of the embolization material into the lumbar and intercostal arteries may occur and this may lead to spinal cord infarction with severe pain and paresis.[23] In case of

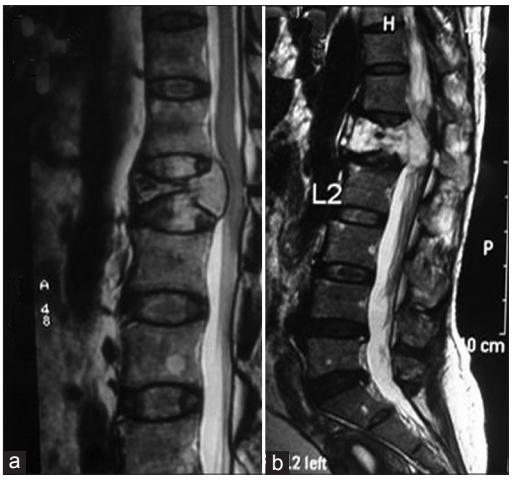

Figure 1: (a) Sagittal T2WI showing L1 collapse with hyperintensity changes within the vertebral body and compression on the thecal sac. (b) Sagittal T2WI showing hyperintensity changes with extradural soft tissue component causing thecal sac compression at L1 level

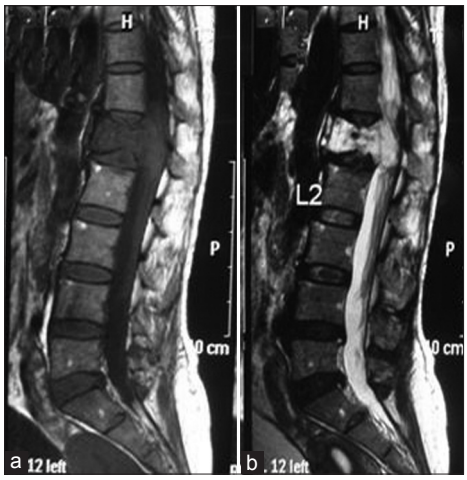

Figure 2: (a and b) Sagittal images with T1 isointensity and T2 hyperintensity changes within the L1 vertebral body with significant extradural soft tissue component. This patient had a short duration of symptoms

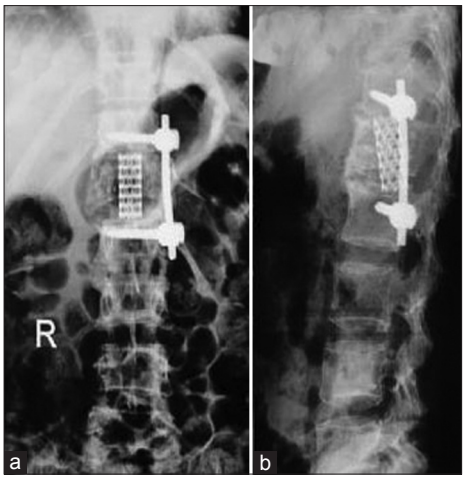

Figure 3: Anteroposterior and lateral postoperative X-rays (a and b) showing adequate excision of the vertebral bodies and instrumentation

recurrent or residual disease, a second attempt is not possible because feeding arteries have already been occluded. At our institute, we attempted preoperative embolization in two patients, but could not succeed because the vertebral bodies were collapsed and converted into a solid mass. Considering the impact of neuraxial compression, we felt it unwise to subject the patient to multiple treatment modalities, and hence direct surgery was adopted in all our patients.

Radiation therapy is also an effective mode of treatment, especially in lesions associated with pain. Vertebral hemangiomas are radiosensitive tumors and reports have shown symptomatic improvement following radiotherapy.[24] However, the effects of radiotherapy are delayed and radionecrosis of the spinal cord and vertebral bodies still remains a risk.

Percutaneous ablation with ethanol or methyl methacrylate constitutes some of the newer options in the treatment of vertebral hemangiomas. Injection of ethanol causes intralesional thrombosis and destruction of the endothelium of the hemangioma. This leads to devascularization of the hemangioma, and thereby reduces its size alleviating the cord compression. However, for its success, it is necessary that the ethanol is injected into the vascular spaces of the hemangioma. This is critical in elderly patients as bone is quiet fibrotic and the vascular component of the hemangioma may be small. Moreover, the technique seems to be cumbersome as the needle has to be localized with the help of prior and intraoperative angiography. Goyal *et al.* in their study of 14 patients suffering from symptomatic vertebral hemangiomas reported neurological deterioration in three patients.[25] One developed infection due to extravasation of contrast material into paraspinal tissues, another a recurrence at 1 month followup, and the third patient developed vertebral fracture 5 months following the

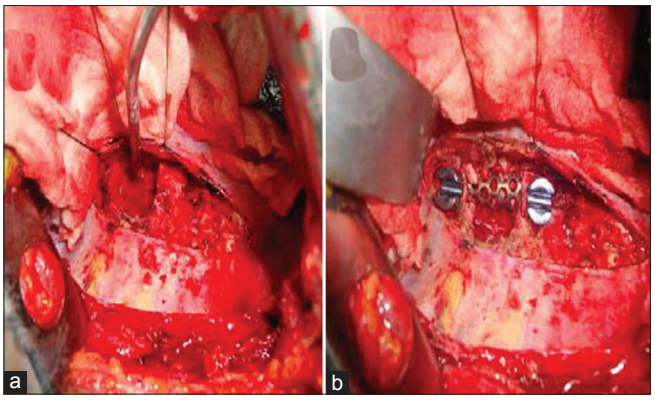

Figure 4: Intraoperative images showing (a) the gutter created after adequate excision of the vertebral bodies. (b) Cage filled with autograft placed in the gutter created after excision of the vertebral hemangioma

procedure. Niemeyer *et al.* have reported a case of Brown–Sequard syndrome following alcohol ablation.[26] Alcohol ablation deals with the vertebral hemangioma per se, but does not restore the strength of the vertebral body that has been fractured. So, this has to be supplemented with methyl methacrylate injection or instrumentation.

Injection of methyl methacrylate is usually performed under fluoroscopic guidance and is indicated for stabilizing vertebral bodies at risk for collapse or to reduce pain. However, when a patient is paraparetic, methyl methacrylate may extend into the spinal canal and exacerbate spinal cord compression that is already present. In such cases, it is usually combined with laminectomy.[27]

Conclusion

Vertebral hemangioma are generally asymptomatic. They may present with thecal and neuraxial compression if aggressive. The presence of T1 isointensity in these may be suggestive of their invasive nature. These lesions if excised and stabilized with adequate instrumentation may show good neural recovery. Severe blood loss is a major intraoperative problem in these surgeries. However, surgery with adequate blood replacement can be done in some cases. In others, preoperative embolization followed by definitive surgery is an option. Though the treatment modalities are many and have their own pros and cons, to use them singly or in combination is a difficult decision to be taken by the treating surgeons.

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How to cite this article: Nair AP, Kumar R, Srivastav AK, Sahu RN, Kumar B. Outcome of dorsolumbar vertebral hemangiomas presenting with neuraxial compression. Indian J Orthop 2012;46:536-41.

Source of Support: Nil, **Conflict of Interest:** None.