Review

Indian Journal of Medical Science 2017 April - June;69(2):34-38

Current Strategies in the Management of Symptomatic Vertebral Hemangiomas

Raghavendra S[1], Sandhya K[2]

Abstract

Vertebral Hemangiomas (VH) are common benign vascular tumors, present in as many as 10% to 12% of autopsy specimens(1), the presentation of which varies from asymptomatic lesions often diagnosed incidentally (1,5) to the rare lesions that cause vertebral compression fractures with spinal cord compression. VH typically occurs in the lower thoracic and upper lumbar regions and are more frequent in the female population (7,8). Although malignant degeneration is extremely uncommon, fewer than 5% of them could be labeled "aggressive hemangiomas", presenting with localized spinal pain (60%), fracture (10%), compression of the spinal cord with myelopathy, or radiculopathy (30%). Posterior spinal structures (pedicle, laminae, and spinous processes) involvement is seen in 10-15% of patients, and this is indicative of an atypical, aggressive lesion. Lesions generally become symptomatic when there is neural arch expansion, vertebral body enlargement, or direct compression of the thecal sac or nerve roots (2,3). Cord compression leading to neurological deficits and paraplegia is usually a late event. Management of symptomatic vertebral hemangiomas includes observation, radiotherapy, enbloc excision/spondylectomy, laminectomy with preoperative embolizaton and percutaneous vertebroplasty/Kyphoplasty. We have reviewed the currently available literature regarding the evaluation and management of these lesions with illustrative case examples and propose a treatment approach to be followed.

Keywords: Vertebral Hemangiomas, Vertebroplasty, Surgery, Radiotherapy

Introduction

Vertebral Hemangiomas (VH) are common, vascular benign tumors of the vertebral body, present in as many as 10% to 12% of autopsy specimens [1]. A majority of patients with these lesions are asymptomatic, being detected incidentally during evaluation of other problems. Patients with symptomatic hemangiomas most commonly have pain (60%), neurological compromise (30%), or symptomatic fracture (10%)[2–5] . Although malignant degeneration is extremely uncommon, fewer than 5% of them could be labelled "aggressive hemangiomas", presenting with localized spinal pain, fracture, compression of the spinal cord with myelopathy, or radiculopathy. Posterior element (pedicle, laminae, and spinous processes) involvement is seen in 10-15% of patients, and this is indicative of an atypical, aggressive lesion. Management of symptomatic vertebral hemangiomas includes observation, radiotherapy, enbloc excision/spondylectomy, laminectomy with preoperative embolizaton and percutaneous vertebroplasty/Kyphoplasty. VH typically occurs in the lower thoracic and upper lumbar regions. Backache is the commonest symptom at presentation [4,6] and VH are more frequent in the female population [7,8]. Cord compression leading to neurological deficits and paraplegia is usually a late event. Doppman et al observed paraplegia or paraparesis in six out of 11 cases[9], while Fox et al reported eleven patients with progressive neurological deficits in their series of 59 patients[7]. Lesions generally become symptomatic when there is neural arch expansion, vertebral body enlargement, or direct compression of the thecal sac or nerve roots [2,3].

[1]Department of Orthopaedics, BGS Global Institute of Medical Sciences. No.67, BGS Health and Education City, Uttharahalli road, Kengeri, Bangalore-560060.

[2]Department of Anesthesiology, BMCRI (Super-specialty Hospital), Victoria Hospital campus, Fort, Bangalore- 560002.

Address of Correspondence
Dr.Sandhya K,
Associate Professor, Department of Anesthesia, BMCRI (Super-speciality Hospital), Fort, Bangalore 560002.
Email:sandyaraghu@gmail.com

Case illustration

Figure 1 shows the natural history of aggressive Hemangiomas. This 36 year old female patient presented with intermittent backache in 2008. Radiological evaluation then showed irregular trabecular pattern involving almost the entire L3 vertebral body and the posterior elements. She was advised a biopsy and intervention then which she refused. She again presented in 2015 (Figure 2) with compression fracture of L3 and gradually progressive pain. She eventually underwent posterior pedicle screw fixation from D12 to L5 and decompression of the lesion.

Radiological evaluation

Asymptomatic VHs are rarely detected on plain radiographs (10). Features include regular striations, normal cortices and incomplete involvement of vertebral body (10). In contrast, compressive hemangiomas display irregular trabeculae with lytic zones, poorly defined and expanded cortex and involvement of the entire vertebral body, including the neural arches with soft tissue extension. Based on their study of 57 solitary vertebral hemangiomas, Laredo et al (10) developed the following six criteria to distinguish asymptomatic VH from compressive VH on plain radiographs and Computed Tomograms: involvement of the entire vertebral body; involvement of the neural arch (particularly pedicles); an irregular, honeycomb appearance; expanded and poorly defined cortex; and swelling of the soft tissue.

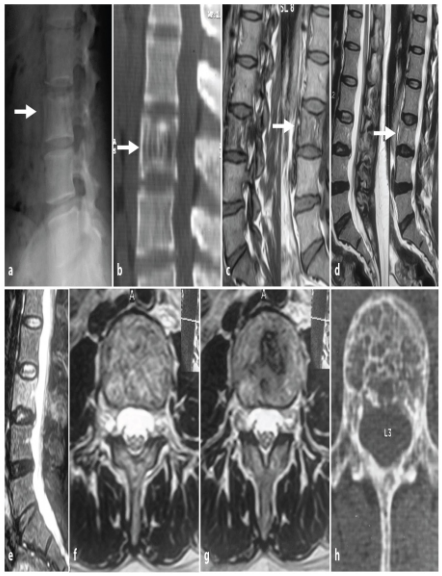

Figure 1: A 36 year old patient with irregular trabecular pattern and poorly defined lytic zone (a, b) involving L3 vertebra. T1, T2 and STIR (c, d, e) MR imaging clearly define the lesion. CT (h) shows involvement of the entire body with irregular honey-comb appearance.

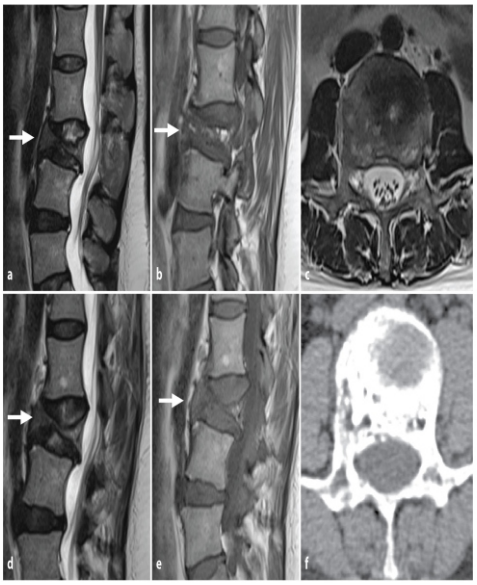

Figure:2 CT and MR Imaging of the same patient as in Figure 1 after seven years shows compression fracture of L3 (arrows), with local kyphosis, but no encroachment of the neural structures (c, f).

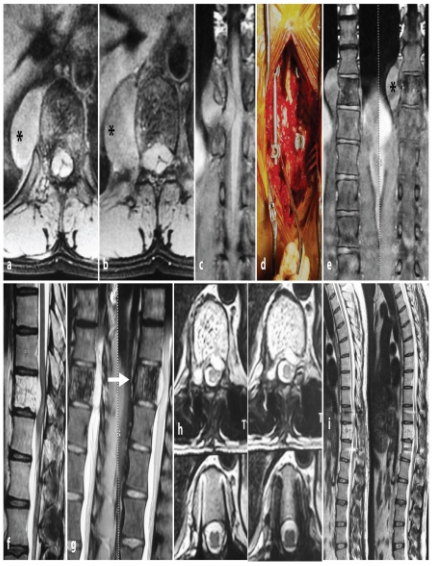

Figure 3: A 33 year old patient shows hemangioma at D11 with soft tissue extension (*), more on the right side. Images of posterior pedicle screw fixation from D10 to L1 and laminectomy of D11 (d, h, i) and decompression of the lesion.

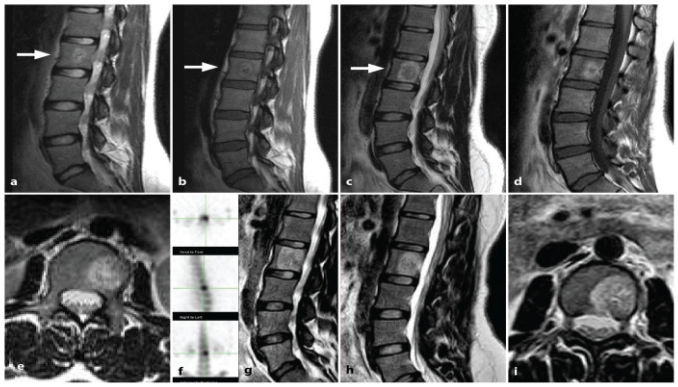

Figure 4: MRI of a 28 year old patient show the progression of the lesion with involvement of left half of L2 vertebra extending up to the base of the pedicle (arrows). Increased uptake on radioisotope scan (f).

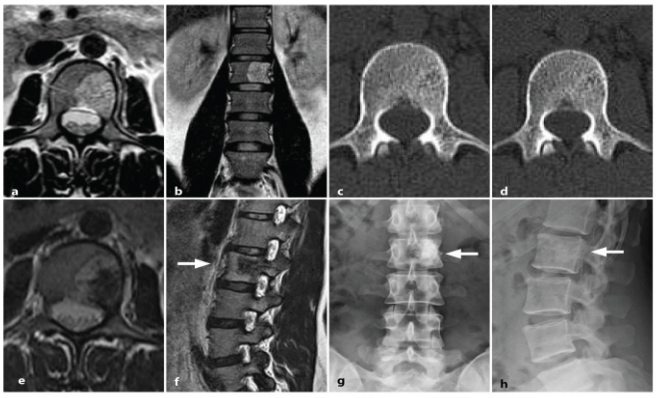

Figure 5: Same patient as in Figure 4 with MR and CT images before Percutaneous Vertebroplasty (a-d). MR images (e, f) and radiographs (g, h) at follow up show no further extension of the lesion with cement in situ (arrows).

Based on this scoring system, a score from 0 to 2 suggests inactive, localized VH, which require routine clinical and radiological follow-up study. On the other hand, a score of 3 or more and the presence of pain in the region of the lesion warrant thorough evaluation including angiogram of the lesion and more aggressive management. Furthermore, increased fat stroma as evidenced by increased signal intensity on T1-weighted MR and mottled, high signal intensity on T2-weighted images is indicative of an increased fatty to vascular tissue ratio, a feature of relatively inactive VH [11,12]. In contrast, low signal intensity on T1-weighted MR images and high signal intensity on T2-weighted MR images indicate relatively aggressive tumors which might be symptomatic and may be complicated later by neurologic compression [11,12]. Selective arteriography of the spinal cord is an obligatory procedure in cases of compressive VH. Laredo et al have described the characteristic arteriographic appearance of these lesions: dilatation of arterioles of the vertebral body, multiple blood pools in the capillary phase, and, finally, intense opacification extending beyond the normal hemi vertebral territory throughout the entire vertebral body [10]. The absence of early venous draining distinguishes compressive VHs from high-flow arteriovenous malformations [10].

TREATMENT

Vhs that are incidentally detected and asymptomatic do not require any treatment. Fox et al reported progression of an asymptomatic lesion to neurological symptoms in only two patients in their series of 59 patients [7] and regular follow-up monitoring for patients with asymptomatic lesions is unnecessary unless pain develops at the appropriate spinal level [7]. They recommend annual neurological and radiological examinations for patients with hemangiomas associated with pain, especially young females with thoracic lesions in whom spinal cord compression is most likely to develop [7].

Radiotherapy

Several reports suggest favorable reports with irradiation for management of medically refractory pain caused by VH [7,13,14]. Similarly, irradiation has been suggested as an initial modality to prevent evolution of neurological symptoms (in patients predisposed to the same-female, thoracic, soft tissue extension etc.)[13]. Radiotherapy could be considered in patients unfit for surgery, especially those with slowly progressing neurological symptoms or signs [15] and as an adjuvant after surgical resection and reconstruction. A dosage of 36-40 Gy with 2 Gy per fraction has been accepted as a standard dose to achieve pain relief [14]. But radiotherapy has a risk of damage to adjacent tissues, radioosteonecrosis and in the cervical spine, there is a risk of inducing sarcomas, of malignant transformation and of damage to the cervical cord and the thyroid [16–18]. Radiological Imaging after radiotherapy is not useful for evaluation of the effects of radiotherapy as the appearance does not change after radiotherapy, even at five years after irradiation [8,13].

Surgery

Surgical decompression with or without post-operative irradiation has to be considered as a treatment of choice in lesions that cause vertebral collapse and spinal cord compression [3,7,19] Profuse intraoperative bleeding is a complication that should be expected and pre-operative embolization of the feeder vessel is indicated to reduce the intraoperative blood loss [2,3]. In a report by Acosta FL, transarterial embolization alone resulted in resolution of back pain [2,3]. Other complications of surgery include epidural hematoma, arahnoiditis, and problems of nonunion in anterior grafting and fusion. Emergency laminectomy after embolization can be performed for progressive neurological compromise [7]. Complete intralesional spondylectomy followed by reconstruction after embolization of aggressive vertebral hemangiomas with circumferential vertebral involvement can be safely accomplished. Such a spondylectomy can also prevent recurrence of hemangiomas.

Case illustration

Figure 3 describes a 33 year old male patient presented with severe backache and gradually progressive neurological weakness in the lower limbs with upper motor neuron signs predominantly on the right side. MR imaging showed a hemangioma at D11 with significant soft tissue extension, more on the right side. He underwent posterior pedicle screw fixation from D10 to L1 and laminectomy of D11 and decompression of the lesion. He was referred for post-operative radiotherapy with subsequent improvement in neurological function.

Intralesional ethanol

Doppman et al reported direct intralesional ethanol under CT guidance and IV sedation to be an effective and safe alternative for rapidly relieving compression and for devascularising the lesions that are causing collapse and compression of the cord and/or root [9]. The technique is based on the observation that hemangiomas do not penetrate the dura but remain confined by the periosteum of the vertebrae. Ethanol causes intralesional thrombosis and destruction of the endothelium followed by shrinkage of the lesion, which thereby decompresses the cord and the nerve root [9]. They recommend forceful injection of ethanol using a volume and rate of injection predicted by the preceding injection of contrast material into the vertebral body. Excessive amount of ethanol may cause aseptic necrosis and pathological fractures and 15ml has been found to be safe with the desired therapeutic effect without pathological fractures [9]. In their opinion, slow injection of ethanol produces thrombosis about the needle tip and can prevent the ethanol from reaching the critical compressive components of the hemangioma.

Percutaneous Vertebroplasty

Vertebroplasty, which was described by Deramond and Galibert for treatment of aggressive hemangiomas, acts by stabilizing pathological bone with an injection of bone cement into the vertebral body [20]. This alleviates severe pain and helps prevent further complications like compression fracture or paraplegia[15]. The exothermic reaction during cement polymerization may destroy adjacent tumor cells [21]. Indications of vertebroplasty now extend to osteolytic vertebral metastasis, osteoporotic vertebral fractures and symptomatic hemangiomas with 90-95% rates of symptom relief [21,22]. It has been proposed even in asymptomatic patients in the presence of radiologically aggressive lesions in order to consolidate the vertebral body to prevent compression or pain due to collapse [18]. Advantages of this procedure include the minimally invasive approach, early relief of symptoms and low rate of complications [22,23]. However, when a patient is paraparetic owing to extension of the hemangioma into the spinal canal, extravasation of methylmethacrylate may fill the intraspinal component too and exacerbate cord compression [9] with irreversible neurological consequences. Such a risk is increased in high-flow angiomatous lesions, and in the presence of intravertebral and paravertebral venous anastomosis (23). Slower introduction of the cement by avoiding pressure peaks might reduce the risk of leakage, probably by obliterating the major connections to the inferior vena cava or azygos system. Percutaneous Kyphoplasty or surgically controlled VP through a 3cm incision and bilateral fenestrations eliminates the risk of complications due to cement extravasation with prompt pain relief and early mobilization in elderly polymorbid patients [24,25].

Case illustration

Figure 4 describes a 28 year old lady presented with backache and further evaluation with MRI showed a VH at L2. MRI scans done one year later to document the progression of the lesion show an increase in size involving a significant part of L2 vertebra on the left side extending up to the base of the pedicle, but not invading it. There was no encroachment on the neural tissues. Technetium MDP radioisotope scan showed increased localization of the marker at L2. There was a gradual increase in the pain with tenderness at L2 paraspinal region without any neurological deficits. Radiological workup after two years (Figure 5) showed further increase in the size, which was now involving almost the entire left half of L2, being present along its entire height and also involving the pedicle on the left side. Computed Tomography (CT) scans at the same time showed the typical "polka dot" appearance of the lesion, but there was no cortical erosion. She underwent a percutaneous vertebroplasty under local anesthesia and intravenous sedation. She

had gradual reduction of pain in the first post-operative week and was symptom free at last follow up of 5 years, with a Visual Analogue Score (VAS) score of 0/10. Follow up radiographs and MRI two years postoperatively showed a well localized lesion with cement in situ and no signs of further progression

Conclusion

To summarize, the treatment approach as proposed by Ide et al could be followed For compressive hemangiomas, [15]:
1. Plain films to define and localize the lesion, with MRI to show the degree and extent of cord compression (or myelography if MRI is not available).
2. CT and MRI to establish the precise osseous and extra osseous extension of the lesion, especially in the spinal canal. Spinal cord compromise can be shown by MRI.
3. Angiography (and embolization if necessary).
4. Percutaneous vertebroplasty to consolidate the vertebral body and reduce vascularity prior to surgery.
5. Limited surgery (simple laminectomy and resection of the epidural hemangioma) to reduce residual spinal cord compression.

References

1. Schmonl G, Junghanns H. The human spine in health and disease. New York: Gnune & Stratton; 1971.

2. Acosta FL Jr, Dowd CF, Chin C, Tihan T, Ames CP, Weinstein PR. Current treatment strategies and outcomes in the management of symptomatic vertebral hemangiomas. Neurosurgery. 2006;58(2):287–95.

3. Acosta FL Jr., Sanai N, Chi JH, Dowd CF, Chin C, Tihan T, Chou D, Weinstein PR, Ames CP. Comprehensive Management of Symptomatic and Aggressive Vertebral Hemangiomas. Neurosurg Clin N Am. 2008;19(1):17–29.

4. Pastushyn AI, Slin'ko EI, Mirzoyeva GM. Vertebral hemangiomas: diagnosis, management, natural history and clinicopathological correlates in 86 patients. Surg Neurol. 1998;50(6):535–47.

5. McAllister VL, Kendall BE, Bull JW. Symptomatic vertebral haemangiomas. Brain. 1975;98:71–80.

6. Mohan V, Gupta SK, Tuli SM, Sanyal B. Symptomatic vertebral haemangiomas. Clin Radiol. 1980;31:575–9.

7. Fox MW, Onofrio BM. The natural history and management of symptomatic and asymptomatic vertebral hemangiomas. J Neurosurg. 1993;78(1):36–45.

8. Faria SL, Schlupp WR, Chiminazzo H Jr. Radiotherapy in the treatment of vertebral hemangiomas. Int J Radiat Oncol Bio Phys. 1985;11:387–90.

9. Doppman JL, Oldfield EH, Heiss JD. Symptomatic Vertebral Hemangiomas: Treatment by means of direct intralesional injection of Ethanol. Radiology. 2000;214:341–8.

10. Laredo JD, Reizine D, Bard M, Murland JJ. Vertebral hemangiomas: radiologic evaluation. Radiology. 1986;161(1):183–9.

11. Laredo JD, Assouline E, Gelbert F, Wybier M, Merland JJ, Tubiana JM. Vertebral hemangiomas: fat content as a sign of aggressiveness. Radiology. 1990;177:467–72.

12. Ross JF, Masaryk TJ, Modic MT, Carter JR, Mapstone T, Dengel FH. Vertebral hemangiomas: MR imaging. Radiology. 1987;165:165–9.

13. Sakata K, Hareyama M, Oouchi A, Sido M, Nagakura H, Tamakawa M, Akiba H, Morita K. Radiotherapy of Vertebral Hemangiomas. ; 1997. 36(7): p. Acta Oncol (Madr). 1997;36(7):719–24.

14. HC Suparna, Vadhiraja BM, Apsani RC, Seetharamaiah T, Fernandes DJ, Rao K, Vidyasagar K. Symptomatic vertebral hemangiomas- results of treatment with radiotherapy. Ind J Radiol Imag. 2006;16(1):37–40.

15. Ide, Gangi A, Rimmelin A, Beaujeux R, Maitrot D, Buchheit E, Sellal F, Dietemann JL. Vertebral haemangiomas with spinal cord compression: the place of preoperative percutaneous vertebroplasty with methyl methacrylate. Neuroradiology. 1996;38:585–9.

16. Donovan DJ, Huynh TV, Purdom EB, Johnson RE, Sneizek JC. Osteoradionecrosis of the cervical spine resulting from radiotherapy for primary head and neck malignancies: operative and nonoperative management. J Neurosurg Spine. 2005;3:159–64.

17. Obana Y, Tanji K, Furuta I, Yamazumi T, Hashimoto S, Kikuchi H, Tanaka S, Ohba Y. A case of malignant transformation in thoracic vertebral hemangioma following repetitive irradiation and extraction. Pathol Int. 1996;46:71–8.

18. V. Dousset, Mousselard H, Monck d'User LD, Bouvet R, Bernard P, Vital JM, Senegas J Caillé JM. Asymptomatic cervical haemangioma treated by percutaneous vertebroplasty. Neuroradiology. 1996;38:392–4.

19. Rich JA, Donohue TC, Mick TJ. Symptomatic Expansile Vertebral Hemangioma Causing Conus Medullaris Compression. J Manipulative Physiol Ther. 2005;28(3):194–8.

20. Galibert P, Deramond H., Rosat P, Le Gards D. Note préliminaire sur le traitement des angiomes vertébraux par vertébroplastie acrylique percutanée. Neurochirurgie. 1987;33:166–8.

21. Chiras J, Barragán-Campos HM, Cormier E, Jean B, Rose M, LeJean L. Vertebroplasty: State of the Art (Article in French). J

Radiol. 2007;88(9):1255–60.

22. Brunot S, Berge J, Barreau X, Ménégon P, Dousset V. Long term clinical follow up of vertebral hemangiomas treated by percutaneous vertebroplasty (Article in French). J Radiol. 2005;86(1):41–7.

23. Guarnieri G, Ambrosanio G, Vassallo P, Pezzullo MG, Galasso R, Lavanga A, Izzo R Muto M. Vertebroplasty as treatment of aggressive and symptomatic vertebral hemangiomas: up to 4 years of follow-up. Neuroradiology. 2009;51:471–6.

24. Wenger M, Markwalder T.-M. Surgically Controlled, Transpedicular Methyl Methacrylate Vertebroplasty with Fluoroscopic Guidance. Acta Neurochir (Wien). 1999;141:625–31.

25. Jones JO, Bruel BM, Vattam SR. Management of Painful Vertebral Hemangiomas with Kyphoplasty: A Report of Two Cases and a Literature Review. Pain Physician. 2009;12:E297–E303.

Conflict of Interest: Nil
Source of Support: None

How to Cite this Article

Raghavendra S, Sandhya K. Current Strategies in the Management of Symptomatic Vertebral Hemangiomas. Indian J Med Sci 2017 April - June;69 (2):34-38.