The natural history and management of symptomatic and asymptomatic vertebral hemangiomas

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Fifty-nine cases of vertebral hemangioma were seen at the Mayo Clinic between 1980 and 1990. Vertebral hemangiomas were discovered incidentally in 35 patients, while pain was the presenting complaint in 13 patients. Five patients presented directly with progressive neurological deficit requiring surgery, and six patients had surgery elsewhere for spinal cord compression and were referred for follow-up evaluation. To better define the natural history of these lesions, a historical review of these patients was conducted; progression of an asymptomatic or painful lesion to neurological symptoms was found in only two cases (mean follow-up period 7.4 years, range 1 to 35 years). New-onset back pain followed by subacute progression (mean time to progression 4.4 months, range 0.25 to 12 months) of a thoracic myelopathy was the most common presentation for patients with neurological deficit. Initially, all 11 patients with spinal cord compression underwent decompressive surgery with full neurological recovery. Recurrent neurological symptoms were observed in three of six patients following subtotal tumor resection and postoperative administration of 1000 cGy or less radiation therapy (mean follow-up period 5.7 years, range 1 to 17 years). No recurrences were noted in four patients who had subtotal excision plus radiotherapy between 2600 and 4500 cGy. One other patient had gross total tumor removal without radiotherapy and has not had a recurrence.

Based on these patients and a review of the literature, the authors 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. Radiation therapy or embolization is an effective therapeutic alternative for patients with severe medically refractory pain. Regular follow-up monitoring for patients with asymptomatic lesions is unnecessary unless pain develops at the appropriate spinal level. It is concluded that management of patients with a progressive neurological deficit should include preoperative angiography and embolization, decompressive surgery with the approach determined by the degree of vertebral involvement and site of spinal cord compression, and postoperative radiation therapy in patients following subtotal tumor removal. Operative management and complications are discussed.

KEY WORDS • vertebral hemangioma • natural history • spine • complication

Vertebral hemangioma is a common and benign lesion of the spinal column which is often discovered incidentally or may be found during evaluation of neck or back pain.2,3,5,4 It has an estimated incidence of 10% to 12% in the population based on large autopsy series2,27,47 and a large review of plain spine films.5 Rarely, vertebral hemangioma may be associated with spinal cord compression requiring surgical intervention.2,3,35,37,39,41 Very little is known about the natural history of these lesions.2,35 One purpose of this study was to analyze retrospectively all patients diagnosed with asymptomatic hemangiomas at our institution over a 10-year period to better define the natural history of these lesions. Our intent was to identify factors that might predict progression of these lesions and to determine follow-up parameters for patients found to have asymptomatic hemangiomas.

Management of hemangiomas associated with pain has generally involved either observation or radiation therapy.3,11,16 Decompressive surgery with or without postoperative irradiation has been the therapy of choice for lesions causing spinal cord compression.1,2,5,20,25,27,40,41,48 In recent years, preoperative embolization has become popular and sometimes curative for lesions causing pain or neurological deficit.21,22,42,45,46 We review our experience with symptomatic hemangiomas and discuss current management principles.

Clinical Material and Methods

Patient Population

All medical and surgical records were reviewed for patients diagnosed as having vertebral hemangioma at our institution during the period 1980 through 1990. All available radiological studies were reviewed. Patients were contacted by telephone to confirm their
TABLE 1
Clinical features of 59 patients with vertebral hemangioma

<table>
<thead>
<tr>
<th>Initial Diagnosis</th>
<th>No. of Cases</th>
<th>Age (yrs) Mean Range</th>
<th>Sex (F:M)</th>
<th>Vertebral Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>asymptomatic lesions</td>
<td>35</td>
<td>9-77 18:17</td>
<td>C-2, T-24, L-10</td>
<td></td>
</tr>
<tr>
<td>lesions associated with pain</td>
<td>13</td>
<td>26-69 9:4</td>
<td>C-1, T-9, L-3</td>
<td></td>
</tr>
<tr>
<td>lesions associated with neurological defect</td>
<td>11</td>
<td>18-64 9:2</td>
<td>C-1, T-10, L-10</td>
<td></td>
</tr>
</tbody>
</table>

medical and surgical histories related to their hemangioma, and to determine any additional information prior to or after their Mayo Clinic visit. In some cases, the hemangioma had been diagnosed and treated elsewhere many years earlier. Specifically, the chronology of pain and neurological symptoms was examined carefully and current symptoms were identified. Embolization, surgery, irradiation, or diagnostic studies performed elsewhere were documented and reviewed.

There were 36 female and 23 male patients diagnosed with a vertebral hemangioma at our institution between 1980 and 1990. Table 1 summarizes the clinical features of this group of patients. Six patients had multilevel involvement. Asymptomatic hemangiomas were identified in 35 patients based on radiological studies performed for other purposes. Thirteen patients presented with neck or back pain and were found to have radiological evidence of spinal column hemangioma at a compatible spinal level. Eleven patients presented here or elsewhere with a progressive neurological deficit and were found to have spinal cord compression due to vertebral hemangioma. Five patients underwent surgery here, while six were referred to our institution following decompressive surgery elsewhere for consideration of further therapy.

Diagnostic Evaluation

Plain spine films were obtained in all patients. Coarse vertical vertebral striations or a “honeycomb” appearance were used as criteria for diagnosis of vertebral hemangioma (Fig. 1). If doubt existed, pluridirectional tomography was carried out (Fig. 2). If the diagnosis was still unclear or if neurological symptoms were present, computerized tomography (CT) with or without myelography was performed. Bone scanning and magnetic resonance (MR) imaging were used selectively when indicated to rule out other bone or soft-tissue lesions but were not routinely performed (Fig. 3). Spinal angiography was undertaken in selective cases when the above studies suggested large feeding or draining vessels or when embolization was considered a presurgical adjunct (Fig. 4).
Fig. 4. Retrograde aortic angiography demonstrating a large radicular feeding vessel and a vertebral body hemangioma.

**Therapy**

Patients with asymptomatic lesions were informed of the benign nature of their lesion and no further evaluation was performed. All patients with pain underwent CT scanning to rule out soft-tissue extension and electromyography if pain was radicular in nature. None demonstrated extrasosseous involvement or nerve root irritation, and these patients were followed with annual neurological and radiological examinations here or by their local physician. One patient with severe intractable pain underwent localized spinal irradiation. All patients with spinal cord compression were treated with decompressive surgery while selective patients had preoperative embolization and/or postoperative radiation.

**Results**

**Clinical Follow-Up Findings**

Current contact was available in 30 of the 35 patients with incidental hemangiomas and none had developed symptoms related to their lesion, with an average follow-up period of 6.2 years (range 1 to 35 years). All patients with asymptomatic lesions had plain spine x-ray evidence of intrasosseous vertebral body involvement only, without bone expansion, cortical irregularities, or posterior element involvement. None of the patients had interval changes in the radiological appearance of their hemangiomas.

Follow-up data were available in 10 of the 13 patients initially diagnosed at our institution with a vertebral hemangioma during evaluation for spinal pain. The pattern of pain in all of these patients was generally intermittent, positional, and well localized to the neck or back without radiation. The chronology was that of chronic pain or multiple exacerbations and remissions each year. All 10 patients contacted continued to have complaints of pain. None of these patients had developed a neurological deficit or interval radiological changes, with an average follow-up period of 8.8 years (range 1 to 20 years). Of the three patients who could not be contacted, two had been seen 2 and 4 years after initial diagnosis with no change in their pain or x-ray findings. The last patient was a 33-year-old man who had received 4400 cGy radiotherapy for an L-1 vertebral segment hemangioma with both anterior and posterior element involvement and associated severe intractable low-back pain. He had no back pain or neurological deficit at last contact 5 years postirradiation. All but two patients with painful lesions had isolated vertebral body involvement only: one was the man with the L-1 lesion mentioned above who had total vertebral involvement, and the other patient was a 52-year-old woman with both anterior and posterior element involvement of the C-5 vertebra. She continues to have localized neck pain 9 years after initial diagnosis without x-ray changes.

Current contact was available in 10 of the 11 patients initially found to have a vertebral hemangioma during workup of a progressive spinal neurological deficit. For all patients with neurological symptoms, the mean time interval between onset of symptoms and surgery was 4.4 months (range 0.25 to 12 months). Notably, 10 of these patients had experienced new and localized back pain for weeks to months prior to the development of their thoracic myelopathies. The last patient presented with a cervical radiculopathy related to a C-6 lesion. Table 2 outlines the extent of vertebral involvement and the etiology of spinal cord compression for each of these patients.

There were only three instances of symptomatic progression of incidental or painful lesions in our series. One 42-year-old woman developed pain 9 years after the incidental discovery of an L-1 lesion. Plain spine films and tomograms showed no change in the intrasosseous body lesion. Two patients developed spinal cord

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**TABLE 2**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Vertebral Involvement</th>
<th>Etiology of Spinal Cord Compression</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>body &amp; both pedicles</td>
<td>ant. soft-tissue extension</td>
</tr>
<tr>
<td>2</td>
<td>entire vertebra</td>
<td>ant. soft-tissue extension</td>
</tr>
<tr>
<td>3</td>
<td>entire vertebra</td>
<td>ant. &amp; post. bone expansion</td>
</tr>
<tr>
<td>4</td>
<td>entire vertebra</td>
<td>ant. &amp; post. bone expansion, ant.</td>
</tr>
<tr>
<td>5</td>
<td>body only</td>
<td>ant. bone expansion</td>
</tr>
<tr>
<td>6</td>
<td>entire vertebra</td>
<td>ant. bone expansion, ant.</td>
</tr>
<tr>
<td>7</td>
<td>posterior ½ of body, all post. elements</td>
<td>ant. &amp; post. bone expansion</td>
</tr>
<tr>
<td>8</td>
<td>entire vertebra</td>
<td>ant. soft-tissue extension</td>
</tr>
<tr>
<td>9</td>
<td>body only</td>
<td>ant. bone expansion</td>
</tr>
<tr>
<td>10</td>
<td>entire vertebra</td>
<td>ant. bone expansion</td>
</tr>
<tr>
<td>11</td>
<td>posterior ½ of body, all post. elements</td>
<td>ant. bone expansion, ant. soft-tissue extension</td>
</tr>
</tbody>
</table>

* Abbreviations: ant. = anterior; post. = posterior.
compression from previously diagnosed vertebral hemangiomas. One of these patients had a known asymptomatic lesion diagnosed 5 years earlier, and the other patient had mid-back pain with an associated hemangioma found 3 years prior to her neurological deficit. The first patient was a 22-year-old woman initially found to have total T-4 vertebral involvement, who later developed soft-tissue extension. The second patient was a 51-year-old woman initially found to have T-5 vertebral body involvement only, followed later by both bone expansion of the vertebral body and posterior soft-tissue extension.

**Surgical Management**

Table 3 summarizes the pathology and management in the 11 surgically treated patients. Two patients (Cases 2 and 8) underwent preoperative embolization. The surgical approach and aggressiveness of bone and soft-tissue resection were based on the extent of vertebral involvement and the site of spinal cord compression by bone expansion and/or soft-tissue extension. Of three patients with isolated vertebral body disease (Cases 5, 7, and 9), one was treated with a vertebral body resection and strut grafting, while the other two received posterior decompression. Eight patients had entire vertebral column involvement with circumferential or anterior compression. Six of these patients underwent decompressive laminectomy with or without soft-tissue excision, while two patients had surgery via bilateral transpedicular approaches resulting in a circumferential decompression of the thecal sac. Three patients underwent posterior fusion following decompression; two received circumferential decompression as noted above, while the third had an extensive decompressive thoracic laminectomy.

Of the 11 surgical patients, total tumor resection was performed in one with no postoperative irradiation while 10 patients had subtotal removal. Of those patients with subtotal excision, five patients had no irradiation, one had 1000 cGy radiotherapy, and four had between 2600 and 4500 cGy radiotherapy. Initially, all 11 patients experienced full neurological recovery following surgery and adjuvant therapy. The mean follow-up period for these patients was 8.7 years (range 1 to 17 years).

Surgical complications were observed in five patients. Profuse intraoperative bleeding occurred in two patients, neither of whom had preoperative embolization (Cases 3 and 6). Postoperative complications included severe arachnoiditis in two patients (Cases 1 and 4), both of whom underwent Pantoque myelography, failed anterior fusion following a one-level vertebral body resection and strut grafting in one patient (Case 5), and a postoperative epidural hemorrhage in one (Case 6).

**Tumor Recurrence**

Three patients suffered recurrent symptoms requiring additional therapy. The average length of time between the first surgery and recurrence was 9.3 years (5, 6, and 17 years). All three patients initially had subtotal tumor removal. Two patients had not received postoperative radiation (Cases 2 and 6) and one patient had been given 1000 cGy (Case 4). Therapy for these patients included further posterior decompressive surgery followed by radiotherapy in Case 6, and a gross total resection in Case 2. The third patient (Case 4) with a recurrent mild thoracic myelopathy was treated with radiation therapy only. The patient with a total resection recovered full neurological function following surgery (Case 2), the patient treated with radiotherapy only was stabilized with a mild thoracic myelopathy (Case 4), and the third had no recovery from her preoperative paraplegia after surgery and irradiation (Case 6).

**Pathological Examination**

Pathological specimens were available in nine of 11 patients. Cavernous hemangioma was found in two cases, capillary hemangioma in one, and mixed in six. Five cases demonstrated both fibrous and fatty stroma.

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**Table 3**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Surgical Procedure</th>
<th>Surgical Resection</th>
<th>Pathology</th>
<th>Adjuvant Therapy</th>
<th>Follow-Up (yrs)</th>
<th>Recurrence Interval (yrs)</th>
<th>Last Neurological Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>laminectomy</td>
<td>subtotal: no STE</td>
<td>no tissue</td>
<td>irradiation (3000 cGy)</td>
<td>8</td>
<td>—</td>
<td>normal</td>
</tr>
<tr>
<td>2</td>
<td>laminectomy</td>
<td>subtotal: no STE</td>
<td>cavernous</td>
<td>embolization</td>
<td>8</td>
<td>6</td>
<td>normal</td>
</tr>
<tr>
<td>3</td>
<td>laminectomy</td>
<td>subtotal: no STE</td>
<td>cavernous</td>
<td>irradiation (2600 cGy)</td>
<td>10</td>
<td>—</td>
<td>normal</td>
</tr>
<tr>
<td>4</td>
<td>laminectomy</td>
<td>subtotal: no STE</td>
<td>mixed</td>
<td>irradiation (1000 cGy)</td>
<td>17</td>
<td>17</td>
<td>mild myelopathy</td>
</tr>
<tr>
<td>5</td>
<td>laminectomy</td>
<td>subtotal: no STE</td>
<td>capillary</td>
<td>none</td>
<td>11</td>
<td>—</td>
<td>normal</td>
</tr>
<tr>
<td>6</td>
<td>laminectomy</td>
<td>subtotal: partial STE</td>
<td>capillary</td>
<td>none</td>
<td>6</td>
<td>5</td>
<td>paraplegia</td>
</tr>
<tr>
<td>7</td>
<td>excision post. elements,</td>
<td>subtotal</td>
<td>capillary</td>
<td>irradiation (4500 cGy)</td>
<td>4</td>
<td>—</td>
<td>normal</td>
</tr>
<tr>
<td>8</td>
<td>laminectomy</td>
<td>subtotal: partial STE</td>
<td>mixed</td>
<td>embolization, irradiation (4000 cGy)</td>
<td>9</td>
<td>—</td>
<td>normal</td>
</tr>
<tr>
<td>9</td>
<td>laminectomy</td>
<td>subtotal: complete STE</td>
<td>no tissue</td>
<td>none</td>
<td>16</td>
<td>—</td>
<td>normal</td>
</tr>
<tr>
<td>10</td>
<td>laminectomy</td>
<td>subtotal: complete STE</td>
<td>mixed</td>
<td>none</td>
<td>1</td>
<td>—</td>
<td>normal</td>
</tr>
<tr>
<td>11</td>
<td>excision post. elements,</td>
<td>total: complete STE</td>
<td>capillary</td>
<td>none</td>
<td>1</td>
<td>—</td>
<td>normal</td>
</tr>
</tbody>
</table>

* Abbreviations: post. = posterior; STE = soft-tissue excision; — = no recurrence.
but all were dominated by numerous cavernous or capillary structures. In the three patients who underwent both studies preoperatively, the CT scans and MR images obtained suggested soft-tissue nonfatty lesions.

Discussion

Radiological Evaluation

Plain spine films often suggest the diagnosis of vertebral hemangioma by the presence of coarse vertical striations or a "honeycomb" appearance within the vertebral body. One-third of the vertebral body must be involved for the classic findings to be apparent. The disc spaces are generally intact. The presence of pain or neurological symptoms dictates further imaging studies. Vertebral hemangiomas uncommonly result in compression fractures; we observed two compression fractures in our series. Paravertebral soft-tissue involvement can be seen in conjunction with vertebral hemangiomas. These masses may be anterior or posterior to the body aneurysms and myelopathies.

The diagnostic procedure of choice is CT with or without myelography. The characteristic "polka dot" appearance within the vertebral body represents axial cuts through thickened vertebrae. Computerized tomography can determine the extent of vertebral involvement as well as the site of spinal cord compression if present, and the use of intravenous administration of contrast material is valuable to enhance intravertebral, epidural, and paravertebral tumor.

Biopsy guided by CT has been used to differentiate vertebral hemangiomas from metastatic lesions. Bleeding complications have not been reported following biopsy. Recently it was suggested by Laredo, et al., that negative attenuation of a hemangioma indicates a fatty stroma and relative indolence while "soft-tissue" attenuation in noncontrast scans is seen more commonly in symptomatic or compressive lesions. The authors presented biopsy material partially supporting this idea.

Magnetic resonance imaging may also provide additional information regarding the aggressiveness of the hemangioma. Mottled, increased "fat"-signal intensity on both T1- and T2-weighted MR images of intraosseous hemangiomas are characteristic of nonevolutive intraosseous lesions. Conversely, osseous or extraosseous hemangiomas that show an isointense signal on T1-weighted images with increased signal on T2-weighted images are more commonly found with symptomatic lesions. Histological studies have shown that the increased signal on T1- and T2-weighted images of intraosseous hemangioma is due to adipose tissue while extraosseous tissue contains little or any adipose tissue. Flow-void areas in highly vascular extraosseous tumor may also contribute to this appearance. Laredo and coworkers concluded that fat-intensity lesions are generally inactive, while hemangiomas demonstrating soft-tissue intensity on CT and low signal intensity on T1-weighted MR imaging indicates a hypervascular lesion with the potential to compress the spinal cord. Our limited data support this. There are no reports on the use of gadolinium in patients with vertebral hemangiomas. In one of our patients with a symptomatic T3 lesion and an asymptomatic T4 lesion, gadolinium infusion nonspecifically enhanced both lesions as well as the extrasosseous soft-tissue extension (Fig. 3). Spinal angiography may be indicated in the presurgical evaluation of symptomatic lesions. Arteriography is particularly useful in determining the vascularity of the osseous and extrasosseous components of the lesion, to identify feeding and draining vessels, and to identify the blood supply to the spinal cord (Fig. 4); it may aid in diagnosis in some cases. Generally, both osseous and soft-tissue components of the tumor will opacify. Asymptomatic lesions often show a normal or slight increase in vascularity while hemangiomas causing pain or spinal cord compression are associated with moderate to intense hypervascularization. If the feeding vessel does not also supply the anterior spinal artery, it can potentially be embolized as a presurgical adjunct or ligated at the time of surgery, reducing the threat of severe intraoperative hemorrhage. The feeding vessel is usually a branch of a lumbar or intercostal artery that arises proximal to the radicular branches.

Bone scanning may be helpful in differentiating multiple hemangiomas from metastatic spinal column disease; however, neither asymptomatic nor symptomatic hemangiomas generally enhance.

Natural History

Asymptomatic Lesions. As seen in this series and others, patients with incidental lesions may remain asymptomatic for years. There were only two patients in our series whose previously diagnosed incidental hemangiomas later became symptomatic. Both patients developed localized back pain and one progressed over 3 weeks to a thoracic myelopathy (Case 8). Based on these observations, we recommend additional studies or follow-up monitoring for patients with incidental lesions only if pain or a neurological deficit develops in the appropriate spinal segment.

Asymptomatic vertebral hemangiomas are generally intraosseous only, without osseous expansion. The thoracic and lumbar spine are the most common locations and a hemangioma is usually localized to the vertebral body without posterior element involvement. Asymptomatic lesions are slightly more common in females and are usually discovered in middle-aged patients. Multilevel involvement has been observed in up to 30% of cases and generally involves two to five noncontiguous levels. Six of the 59 patients in this review had multilevel involvement.

Painful Lesions. Patients with pain as their presenting complaint may have other etiologies for their pain including spondylosis, degenerative joint disease, vertebral disc herniations, or other musculoskeletal disorders. In our series, two of 13 patients with pain had physical signs or x-ray evidence of one of these other causes of pain, including degenerative joint disease in one patient and an L-3 compression fracture in another. Rezine, et al. suggested that, if a painful
vertebral hemangioma is localized to the cervical or lumbar spine but has no posterior element involvement or cortical disruption, then it should be nonolovative. On the other hand, if the painful lesion involves a thoracic vertebra, especially in a young female, and includes posterior element disease, cortical blistering, or soft-tissue extension, then the lesion must be considered evolutive and may have the potential for future spinal cord compression. We had two patients in our series, a 33-year-old man with a lumbar hemangioma and a 52-year-old woman with a cervical lesion, both of which involved both anterior and posterior vertebral elements and caused pain. The patient with the lumbar lesion received 4400 cGy radiotherapy and is currently asymptomatic, while the other patient has been followed closely. Neither patient has developed neurological compromise or radiological progression 5 and 9 years following their initial diagnosis.

**Spinal Cord Compression.** Spinal cord compression may be due to direct neural compression by epidural tumor tissue as an extension from the vertebral body or posterior elements,\(^{5,27}\) by expanded vascular bone,\(^{32}\) by epidural hematoma,\(^{25,27,24}\) by compression fracture of the hemangiomatic vertebra,\(^{2,5,6,10,12}\) or by anomalous vessels draining or feeding the lesion.\(^{7,28}\) A vascular etiology is also possible because osseous tumor compression of the anterior radiculomedullary artery. Diversion of blood flow may also be responsible for neurological symptoms.\(^{27}\)

Patients with neurological symptoms are generally young adults, and the thoracic spine is the site of predilection.\(^{10,20,25,31,41,47}\) Females are more commonly affected than males.\(^{10}\) The lesions are generally localized to one level only. In a large series reported by Nguyen, et al.,\(^{36}\) 54% of patients had total vertebral involvement, 24% had vertebral body involvement alone, and 22% had posterior arch involvement only. Neurological symptoms were due to diffuse narrowing of the spinal canal from soft-tissue extension in one-third of patients and to local bone expansion in two-thirds of patients. Eight of the 11 patients in our series with progressive neurological deficit had anterior and posterior element involvement while three patients had anterior vertebral body involvement only.

The clinical onset of spinal cord compression is often progressive over many months but may be sudden.\(^{9}\) Neck or back pain often precedes the neurological symptoms,\(^{5,10,20,25,26}\) as was observed in 10 of our 11 patients. Thoracic myelopathy is the most common presenting syndrome.\(^{3,35,39}\)

One known risk factor for development of neurological symptoms in quiescent hemangiomas is pregnancy.\(^{31,33,38,53}\) Women usually become symptomatic during the third trimester because of increased intra-abdominal pressure leading to augmented flow within the vertebral venous system or due to the relatively sparse vascular supply of the thoracic spine cord.\(^{21,25}\) Additionally, the endothelial growth-promoting effect of elevated estrogen levels may result in the increased size of a hemangioma during pregnancy.\(^{33,36}\) Fluctuating neurological deficits in women with exacerbations during their menstrual cycle have also been described.\(^{5,13}\)

Two of the 11 patients in our series with spinal cord compression had known vertebral hemangiomas 3 and 5 years prior to the onset of their neurological symptoms. Both were women, aged 51 years (Case 4 with a painful T-5 lesion) and 22 years (Case 8 with an asymptomatic T-4 lesion) and both had lesions involving the entire vertebra. Most progressive lesions have initially demonstrated hemangioma confined to the vertebral body with later x-ray films demonstrating involvement of the posterior elements with associated expansion to bone or soft tissue.\(^{17,42,46}\)

**Management of Patients With Pain**

There are three therapy alternatives for patients with vertebral hemangioma associated with pain only. Patients can be followed with neurological and radiological examinations at regular intervals, or they can be offered either radiation therapy\(^{9,11,16,44,57}\) or embolization therapy\(^{45}\) for medically refractory pain. Various reports have suggested the long-term beneficial effects of either therapy with permanent pain relief in 60% to 100% of patients.\(^{31,44,45,57}\) Radiation therapy has been used successfully for many years while embolization is a relatively new therapeutic procedure for patients with only pain. Twelve of 13 patients in our series displaying pain only, without a soft-tissue compressive component, have been managed successfully with conservative measures and have not demonstrated either radiological or neurological progression. One 33-year-old man with total L-1 vertebral involvement and pain was given irradiation (4400 cGy) with complete resolution of his pain.

**Management of Spinal Cord Compression**

Alternative therapy for patients with vertebral hemangioma and spinal cord compression includes presurgical embolization, surgical bone decompression with total or partial excision of the hemangioma and soft-tissue components, and postoperative radiation therapy in some cases.\(^{3,7,10,46}\) The precise management method is dependent on the spinal level, the location of the lesion within the vertebra, the extent of spinal canal involvement, the neurological condition of the patient, the age of the patient, and the surgical techniques familiar to the surgeon.\(^{2,5,26,27,35,48}\)

**Presurgical Embolization.** Embolization of arterial feeders to vertebral hemangiomas has become popular in recent years due to improved endovascular techniques.\(^{34,46}\) Hekster, et al.\(^{22}\) were the first to report reversal of spinal cord compression following percutaneous embolization of feeding vessels. Others have also reported that presurgical embolization can relieve spinal block, reduce intraoperative hemorrhage, and reverse neurological deficit.\(^{19,35,46,47}\) Embolization has been advocated by some as the sole therapy for spinal cord compression but most surgeons use it as a presurgical adjunct.\(^{7,15,40,42,49,46}\) In the presence of only modest tumor vascularity, or in the absence of discernible feeding vessels, embolization may not be warranted.\(^{7}\) To date,
there are insufficient long-term follow-up data to determine whether embolization alone can cure hemangiomas definitively.\textsuperscript{7,22} Gelfoam particles have been used most frequently and have been shown to temporarily occlude vessels, followed by recanalization in some cases.\textsuperscript{3,4,10,23,34}

Preoperative embolization should be seriously considered for each patient individually based on the extent and location of the lesion, the vascularity as seen by other diagnostic studies, the skill of the angiographers, and the rapidity of the neurological decline. It is especially useful when subtotal or total excision of the vertebral body is contemplated and significantly reduces intraoperative blood loss.\textsuperscript{3,10,24,40}

Surgical Management. Surgical decompression should be undertaken if there is progressive neurological decline.\textsuperscript{1,3,5,40} Rapid neurological decline generally warrants emergency decompression.\textsuperscript{5,26,27,40} When only the posterior elements of the vertebra are involved, radical excision may result in a cure. When only the vertebral body is involved, with or without a soft-tissue component anteriorly, vertebrectomy followed by strut grafting should be performed (Fig. 5).\textsuperscript{13,17,24,40,50,51} If there is vertebral body involvement with little or no bone expansion but a laterally placed soft-tissue component is present, then laminectomy with the removal of the soft-tissue component is performed. Periodic CT is carried out to document whether there is soft-tissue component recurrence or vertebral body expansion. If this occurs, radiotherapy is given. The alternative method is to offer postoperative irradiation after the initial laminectomy and soft-tissue component removal (Fig. 6). Diffuse total vertebral segment involvement will allow only subtotal tumor resection. In our series, laminectomy was the preferred therapy for these cases, with consideration given to postoperative radiation versus careful postoperative follow-up monitoring (Fig. 7).\textsuperscript{26,27,35,40} Circumferential decompression of the spinal canal by a bilateral transpedicular route was also used successfully in two of our patients. A more aggressive approach for predominantly anterior spinal canal compression by soft tissue or bone expansion has been advocated by some (Fig. 8), followed by radiation therapy or observation.\textsuperscript{12,17,30,51} As stated earlier, the surgical approach will depend on several factors, but most importantly on the site of cord compression and the surgical techniques most familiar to the neurosurgeon.

Laminectomy followed by radiotherapy for vertebral hemangioma involving the entire vertebra was reported as yielding a 93% rate of neurological recovery without recurrence in one recent review with a 52-month follow-up period.\textsuperscript{31} Laminectomy without radiotherapy following subtotal resection has resulted in cure rates of 70% to 80%.\textsuperscript{5,26,35,41} Nguyen, et al.,\textsuperscript{41} reported that 45 patients with various combinations of therapy for spinal cord compression had a 75.5% overall favorable clinical course with a mean follow-up period of 52 months; 13% of their patients did not recover significant neurological function following decompressive surgery. They also reported a recurrence rate of 29%, usually within the first 2 years after initial therapy.\textsuperscript{41} This recurrence rate and interval have been reported by others.\textsuperscript{8}

Stabilization of the spinal column is used when the extent of surgical resection may destabilize the spine. It is believed that hemangiomatous bone is stronger than normal bone due to the thickened and sclerotic trabecula, which can be enlarged as much as three times its normal size in reaction to the abnormal blood vessels.\textsuperscript{2,17,18,26,27,35} Patients undergoing vertebrectomy bone resection for total vertebral segment involvement require iliac crest or tibial strut grafts for stabilization. Posterior fusions are not mandatory for these patients, but external immobilization is warranted during strut incorporation. Patients undergoing limited decompressive laminectomies do not need fusion procedures, but patients undergoing aggressive transpedicular approaches (especially bilateral) or extensive laminectomies may require posterior instrumentation for stabilization.

The greatest surgical risk is the potential for intraoperative blood loss and postoperative epidural hematoma.\textsuperscript{5,6,12,17,24,26,27,51} Preoperative embolization and radiotherapy have been used as adjuncts to minimize these complications.\textsuperscript{5,5,5} In our series, two patients suf-
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Fig. 7. Illustration depicting laminectomy alone, the surgical treatment for hemangioma associated with complete vertebral segment involvement without a soft-tissue component. The circumferential bone compression is indicated by arrows.

Fig. 8. Illustration depicting corpectomy with soft-tissue resection and strut grafting, the surgical treatment for hemangioma associated with complete vertebral segment involvement and anteriorly placed soft-tissue component. The circumferential compression is indicated by arrows.

Pathology

Vertebral hemangioma is a lesion of dysembryogenetic origin causing secondary resorption of underlying bone and producing a honeycomb mass within bone.2.18,15,46,55 While most trabeculae are atrophic due to the abnormal blood vessels, some trabeculae become thickened and sclerotic. Microscopically, there are two main types characterized by cavernous or capillary vessels. The cavernous type comprises multiple, large, wall-to-wall, thin-walled vascular spaces lined by flat endothelial cells. The capillary type is composed of numerous capillary channels lined with plump uniform endothelial cells and separated by reactive fibrous tissue. In some cases, adipose tissue may be observed. Malignant degeneration is not known to occur.11 Pathological typing was found to be prognostically significant in our series as our patients with recurrent tumors (Cases 2, 4, and 6) each demonstrated a different microscopic pattern.

Conclusions

Our study confirms that it is rare for incidental lesions or hemangiomas associated with pain alone to progress to spinal cord compression. We observed only two of 59 patients with previously diagnosed asymptomatic or painful lesions who later developed spinal cord compression. Based on these findings, we recommend that patients with painful lesions should be followed with annual examinations. The CT and MR imaging appearance may suggest a potential for progression. Painful hemangiomas may be managed conservatively, with radiotherapy or with embolization. Patients with asymptomatic lesions do not need further evaluation unless pain or a neurological deficit develops at the appropriate spinal level.

Based on our series, we conclude that surgery can be safely performed in patients with vertebral hemangiomas associated with spinal cord compression, and neurological recovery can be excellent. Preoperative embolization should be performed in patients with large feeding vessels to reduce intraoperative blood loss. We
favor a posterior decompressive approach for vertebral hemangioma associated with total vertebral involvement and circumferential spinal cord compression. For patients with either vertebral body involvement only with anterior canal compromise or patients with total vertebral segment involvement with anterior compression, a vertebral body resection and strut grafting should be the therapy of choice. Postoperative irradiation reduces the risk of tumor recurrence in patients following subtotal tumor removal.

References
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