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Symptomatic Vertebral Hemangiomas: Treatment by Means of Direct Intralesional Injection of Ethanol[1]

PURPOSE: To describe the technique and results of injecting ethanol directly into symptomatic vertebral hemangiomas.

MATERIALS AND METHODS: Eleven patients with paraplegia ($n = 6$) or radiculopathy ($n = 5$) due to vertebral hemangioma were treated by means of injecting ethanol (5–50 mL) directly into the lesion with computed tomographic (CT) guidance. CT angiograms were essential prior to treatment to identify functional vascular spaces of the hemangioma and direct needle placement.

RESULTS: All hemangiomas were obliterated completely at follow-up angiography and gadolinium-enhanced magnetic resonance imaging. Five of six patients with paraplegia recovered completely: One who was treated recently was walking with assistance. Four of five patients with radiculopathy improved. No immediate complications were associated with ethanol injection. The two patients who received the largest volumes of ethanol, 42 and 50 mL, developed pathologic fractures of the involved vertebrae 4 and 16 weeks after treatment.

CONCLUSION: Direct injection of ethanol into symptomatic vertebral hemangioma is an effective and safe treatment, provided the dose is less than 15 mL.

Hemangiomas of the vertebrae are common lesions seen in 11% of spines at autopsy (1). They are most frequent in the thoracic spine and least frequent in the cervical spine. The majority are asymptomatic and require no treatment. Fewer than 1% of vertebral hemangiomas produce symptoms owing to collapse (pathologic fracture) or cord compression. Cord compression is more likely with lesions that extend into the pedicles and laminae of thoracic vertebrae where the cord occupies most of the volume of the spinal canal.

The current treatment of vertebral hemangiomas is controversial. Transarterial embolization with particulate agents such as polyvinyl alcohol foam (Contour Embolization Particles; Boston Scientific, Natick, Mass) often produces dramatic but usually transient remissions (2–5). Permanent liquid occluding agents such as cyanoacrylate fail to penetrate the vascular spaces of these postcapillary lesions; and because many symptomatic hemangiomas are in the thoracic spine, liquid embolization techniques may be limited by the presence of a radiculomedullary artery arising at the same segmental level. Currently, transarterial embolization usually is performed prior to surgery to decrease intraoperative blood loss but is not performed solely as a therapy. Neurosurgical treatment involves resection of the vertebrae and bracing with bone grafts and metallic prostheses. Even with preoperative embolization, blood loss is often considerable (6–12). Radiation therapy can obliterate hemangiomas (13–15), but the effect is too delayed for patients with cord compression.

Several years ago, we became interested in the treatment of vertebral hemangiomas that cause cord or root compression by means of intralesional injection of ethanol as a sclerosing agent to rapidly obliterate and shrink the malformation, which is a critical requirement in the presence of progressive cord compression (16). The technique was based on the observation that hemangiomas do not penetrate the dura but remain confined by the periosteum of the vertebrae, as evidenced by the typical bilobed posterolateral

intraspinal protrusions tethered centrally by the posterior longitudinal ligament of the vertebral body.

The purpose of this study was to describe the technique and results of injecting ethanol directly into symptomatic vertebral hemangiomas. We treated 11 patients with vertebral hemangiomas that caused cord or root compression with the use of percutaneous intralesional injection of ethanol; the initial two cases have been published (16). We now present our current experience and stress the safety and efficacy of this procedure, provided the volume of ethanol does not exceed 15 mL (17). We also wish to stress the importance of preliminary computed tomographic (CT) angiography to identify the hypervascular components of these often inhomogeneously vascularized lesions.

MATERIALS AND METHODS

Eleven patients (seven women, four men; age range, 29–73 years; mean age, 51 years; mean age of men, 37 years; mean age of women, 58 years) underwent magnetic resonance (MR) imaging (Signa; GE Medical Systems, Milwaukee, Wis) of the involved vertebrae to evaluate the aggressiveness of the hemangioma, as described by Laredo et al (18,19), and to demonstrate encroachment on the spinal canal and intervertebral foramina. T1-weighted spin-echo (400/12-16 [repetition time msec/echo time msec]) images before and after enhancement with gadopentetate dimeglumine (Magnevist; Berlex Laboratories, Wayne, NJ), and gradient-echo images in the sagittal and transverse planes, were the most useful. All patients underwent CT to assess the extent of bone involvement, and the extension of the hemangioma into the pedicles and laminae of the involved vertebrae was noted specifically.

In the last eight patients, we combined selective spinal angiography with CT. The segmental arteries at the level of the hemangioma and one level above and below the lesion were catheterized with a 5-F Mikaelsson catheter (Cook, Bloomington, Ind), and digital subtraction angiography was performed during the manual injection of 8–10 mL of ioxilan (Oxilan 300; Cook) at a rate of 2–3 mL/sec. The feeder arteries were not embolized.

When the hemangioma involved a lower thoracic or upper lumbar vertebra, the origin of the artery of Adamkiewicz and of any other radiculomedullary feeder artery to the spinal cord was identified. The forceful injection of ethanol into a vertebral hemangioma entails the theoretic risk of retrograde filling of the segmental arteries and potential reflux into a feeder artery to the spinal cord. Vertebral hemangiomas are postcapillary lesions. Although reflux into the feeder arteries is unlikely, we recommend less forceful injection of ethanol when the artery of Adamkiewicz arises at the same level as the hemangioma. We safely and successfully treated two hemangiomas in which a major feeder artery to the anterior spinal artery arose from the same intercostal artery that supplied the vertebral lesion.

After conventional angiography, spinal CT (HiSpeed CT/i; GE Medical Systems) with 3-mm-thick sections through the involved vertebral body, pedicles, and posterior spine was performed during manual injection of 10 mL of ioxilan at 2–3 mL/sec into the right and left segmental arteries at the level of the hemangioma. This required transporting the patient from the angiographic to the CT suite, but it is critical to demonstrate the nonvascularized areas, particularly in patients with large, often centrally fibrotic hemangiomas and those who have undergone radiation therapy. The success of ethanol injection depends on positioning the needle tip in a vascular portion of the hemangioma so that the entire lesion, particularly the component in the posterior elements, can be filled with a single forceful injection.

On the basis of the CT angiogram, the route of the percutaneous needle was planned to ensure that a vascular component of the hemangioma was accessed. For thoracic hemangiomas, we used a transpedicle approach. Lesions in the lumbar vertebrae usually were approached laterally. We did not treat a cervical hemangioma, but Dousset et al (20) described an anterolateral approach for performing cervical vertebroplasty.

The patient was placed prone on the CT table and local anesthesia was administered; a 16-gauge (Geremia; Cook) or 17-gauge (Percucut Bone Biopsy Needle; E-Z-Em, Westbury, NY) bone biopsy needle was introduced into the hemangioma. Hemangiomas are expansile lesions and are often associated with considerable resorption of cortical bone, which facilitates needle penetration. When normal cortex had to be traversed, the core of cortical bone was displaced by using the blunt trocar once the needle tip was properly positioned. This maneuver sometimes required the use of a mallet but was generally successful if the needle was placed in a vascularized portion of the hemangioma. When the needle was correctly placed and the core of cortical bone dislodged, blood flowed back through the needle, often at a rate suggestive of arterial pressure. An aspiration biopsy could be performed at this time to exclude a hypervascular metastasis. However, the history and the typical appearance of hemangioma on conventional radiographs and CT scans always provided diagnostic confidence in our series.

CT of the hemangioma was then performed during the rapid injection of 10 mL of nonionic contrast material through the biopsy needle. We evaluated the filling of the hemangioma with contrast material and determined if any changes in the volume and speed of the ethanol injection were warranted to treat the entire lesion. We extended the CT scanning for several sections above and below the involved vertebra and looked for any evidence of contrast material in the subarachnoid space. Hemangiomas expand slowly and did not, in our experience, breach the periosteum. This dense fascial envelope allowed us to inject ethanol forcefully into portions of the hemangioma that were close to the spinal cord and nerve roots. It was important to verify that the intraspinal and intraforaminal components of the hemangioma were opacified by means of this injection to ensure that the ethanol reached the symptom-producing components of the lesion. When the hemangioma involved the pedicles and laminae, a frequent occurrence in patients with cord compression, we positioned the needle tip close to the junction of the body and pedicle to ensure that ethanol filled the posterior component of the hemangioma.

Opacification of the ethanol was not essential but helpful. We injected 10–12 mL of dehydrated ethanol (Abbott Laboratories, North Chicago, Ill) into 3.75 g of metrizamide (Amipaque; Nycomed Amersham, Princeton, NJ). Complete mixing required agitation for 3–5 minutes. A CT scan obtained 10 minutes after the ethanol injection showed persistent opacification of the hemangioma within which thrombosis had occurred. This provided a map of the extent of ethanol retention within the hemangioma and the obliterative effect that could be expected.

The most critical part of this procedure was to inject the ethanol forcefully by using a volume and rate of injection predicted by the preceding injection of contrast material. A slow injection produces thrombosis about the needle tip and can prevent the ethanol from reaching the critical compressing components

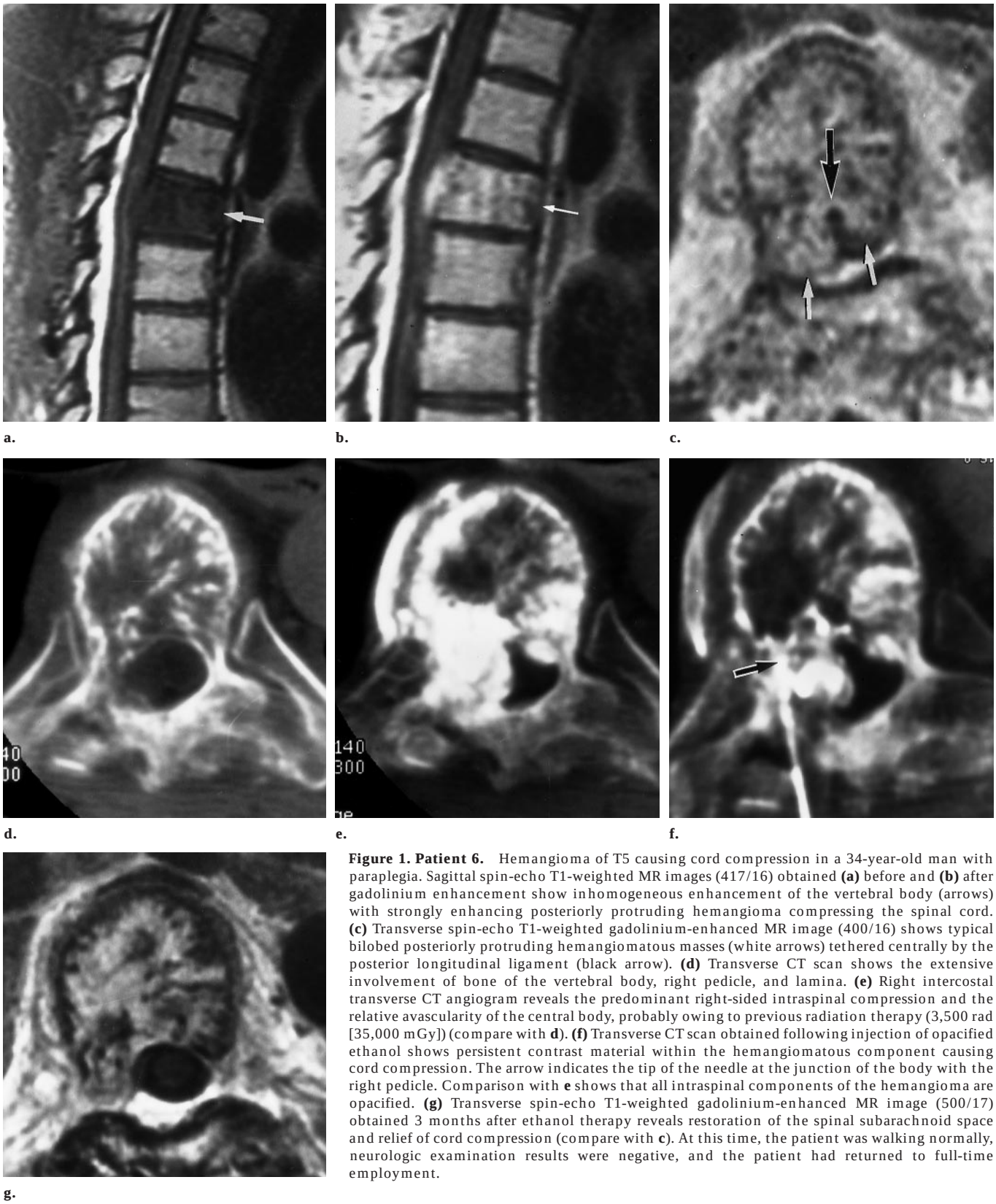

Figure 1. Patient 6. Hemangioma of T5 causing cord compression in a 34-year-old man with paraplegia. Sagittal spin-echo T1-weighted MR images (417/16) obtained **(a)** before and **(b)** after gadolinium enhancement show inhomogeneous enhancement of the vertebral body (arrows) with strongly enhancing posteriorly protruding hemangioma compressing the spinal cord. **(c)** Transverse spin-echo T1-weighted gadolinium-enhanced MR image (400/16) shows typical bilobed posteriorly protruding hemangiomatous masses (white arrows) tethered centrally by the posterior longitudinal ligament (black arrow). **(d)** Transverse CT scan shows the extensive involvement of bone of the vertebral body, right pedicle, and lamina. **(e)** Right intercostal transverse CT angiogram reveals the predominant right-sided intraspinal compression and the relative avascularity of the central body, probably owing to previous radiation therapy (3,500 rad [35,000 mGy]) (compare with **d**). **(f)** Transverse CT scan obtained following injection of opacified ethanol shows persistent contrast material within the hemangiomatous component causing cord compression. The arrow indicates the tip of the needle at the junction of the body with the right pedicle. Comparison with **e** shows that all intraspinal components of the hemangioma are opacified. **(g)** Transverse spin-echo T1-weighted gadolinium-enhanced MR image (500/17) obtained 3 months after ethanol therapy reveals restoration of the spinal subarachnoid space and relief of cord compression (compare with **c**). At this time, the patient was walking normally, neurologic examination results were negative, and the patient had returned to full-time employment.

of the hemangioma. Although patients responded with pain and often a sharp flexion contracture of the spine, we did not encounter neurotoxicity. Previous surgery with opening of the periosteum theoretically can increase the risk of intralesional injection of ethanol; however, patient 6 was treated within 4 weeks after decortication of the posterior elements,

and patient 10 was treated within 12 weeks after a vertebral resection.

The placement of the needle was performed with conventional sedation: midazolam hydrochloride (Versed; Roche Pharmaceutical, Humaca, PR) and fentanyl citrate (Sublimaze; Elkins-Sinn, Cherry Hill, NJ). The injection of ethanol was always performed with augmented sedation supervised by an anesthesiologist. Deep sedation was achieved with intravenous administration of propofol (Diprivan; Zeneca Pharmaceuticals, Wilmington, Del). Delayed CT scans at 10 and 30 minutes showed persistent opacification within the hemangioma owing to the rapid thrombosis of the hemangiovascular spaces. When blood could no longer be aspirated from the intravertebral needle, the needle was withdrawn. Contrast material–enhanced MR imaging was performed before discharge to look for any persistent enhancement of the hemangioma.

All patients underwent neurologic evaluation, contrast-enhanced MR and CT, and spinal angiography with CT 6 months following therapy. Annually, thereafter, they were examined clinically and with contrast-enhanced MR imaging of the spine.

RESULTS

Figures 1–4 show typical treatment procedures and follow-up studies in four patients, including one patient who developed a pathologic fracture. The Table summarizes the clinical findings and therapeutic results in the 11 patients. The hemangioma involved the entire vertebra (body, pedicles, and laminae) in eight patients and was restricted to the body, a single pedicle, or both in three. Six patients presented with progressive paraparesis. Their lesions were in a thoracic (T4, T5, T9, T12, T12) or upper lumbar (L2) vertebra. Five patients had thoracic (T7, T9) or lumbar (L3, L4, L4) radiculopathy. The two thoracic lesions with radiculopathy involved the posterior portion of the vertebral body and extended into the pedicle, with adjacent extraosseous hemangioma accounting for the intercostal radiculopathy (Fig 4). In all patients with paraparesis, the hemangioma involved the posterior elements and the body of the vertebra.

Patients 3, 5, and 7 had undergone embolization without lasting improvement. Patients 3, 5, and 6 had undergone radiation therapy without improvement. Patients 6 and 10 had undergone surgery without preliminary embolization. In patient 6, laminectomy was abandoned because of profuse bleeding (4 units). He was referred for ethanol therapy 1 month after surgery, as weakness had become severe. Patient 10 had undergone partial resection of the hemangioma with a bone graft and bracing with a blood loss of 16 units. This patient remained paraplegic with intense back pain and was referred for ethanol therapy 3 months following surgery.

All patients underwent intralesional injection of ethanol without immediate complications. Patients 1, 4 ,and 5 underwent two injections because of residual hemangioma demonstrated after the first treatment. In nine patients, the volume of ethanol was 15 mL or less (mean, 10 mL; range, 5–15 mL).

Patients 5 and 7 received 42 and 50 mL, respectively, of ethanol. Patient 5 had a residual hemangioma in spite of embolization and radiation therapy. Injection of ethanol in this patient was interstitial rather than intravascular because of the inability to access a patent vascular channel with the transpedicle needle. Patient 7 (Fig 3) had an extremely vascular lesion that continued to back bleed, which led to increasing ethanol injections up to 50 mL at a single session. Patient 7 had relief of symptoms and resumed normal activity.

At 16 and 4 weeks after therapy, pa-

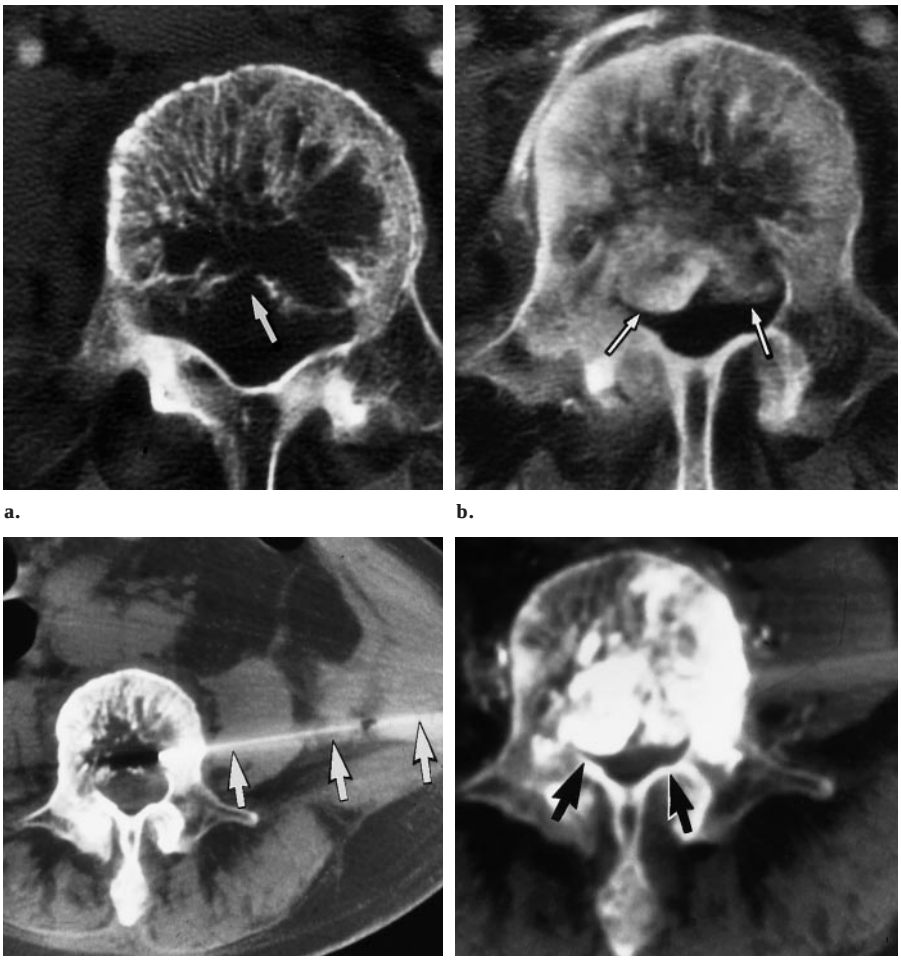

Figure 2. Patient 3. Extensive hemangiomatous lesion of L4 in a 73-year-old woman. Severe back pain and radiculopathy failed to respond to embolization or radiation therapy (3,500 rad [35,000 mGy]). **(a)** Transverse CT scan reveals extensive bone changes with posterior protrusion of cortex into the spinal canal. Note the focal cortical defect (arrow). **(b)** Transverse CT angiogram shows the central body to be avascular, but typical posterior protrusions (arrows) account for the root compression and symptoms. **(c, d)** Transverse CT scans show the needle (arrows in **c**) in the posterior portion of the vertebral body successfully fills both intraspinal components (arrows in **d**) with opacified ethanol. The transpedicle route probably would have failed in treatment of the intraspinal component.

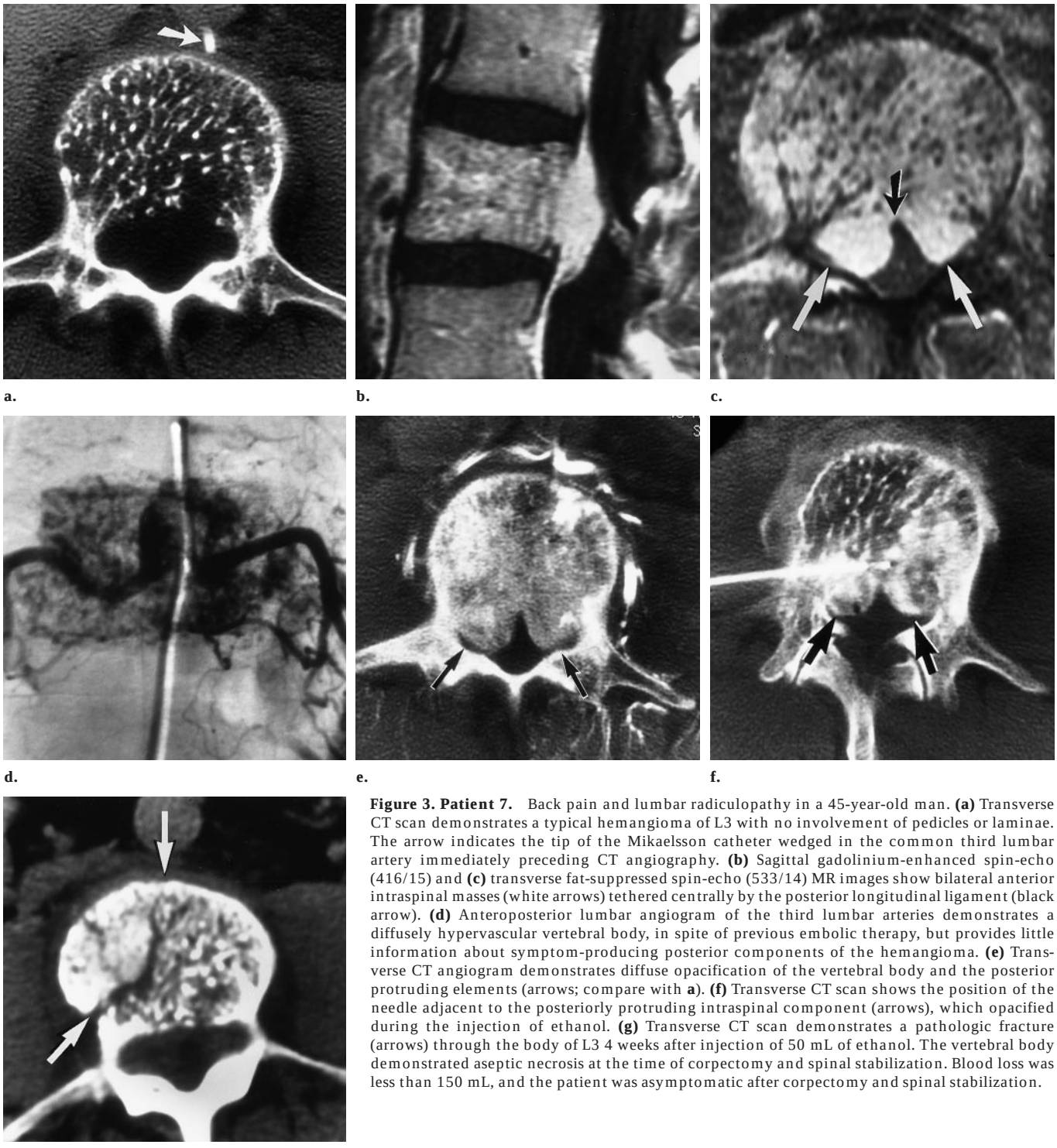

Figure 3. Patient 7. Back pain and lumbar radiculopathy in a 45-year-old man. **(a)** Transverse CT scan demonstrates a typical hemangioma of L3 with no involvement of pedicles or laminae. The arrow indicates the tip of the Mikaelsson catheter wedged in the common third lumbar artery immediately preceding CT angiography. **(b)** Sagittal gadolinium-enhanced spin-echo (416/15) and **(c)** transverse fat-suppressed spin-echo (533/14) MR images show bilateral anterior intraspinal masses (white arrows) tethered centrally by the posterior longitudinal ligament (black arrow). **(d)** Anteroposterior lumbar angiogram of the third lumbar arteries demonstrates a diffusely hypervascular vertebral body, in spite of previous embolic therapy, but provides little information about symptom-producing posterior components of the hemangioma. **(e)** Transverse CT angiogram demonstrates diffuse opacification of the vertebral body and the posterior protruding elements (arrows; compare with **a**). **(f)** Transverse CT scan shows the position of the needle adjacent to the posteriorly protruding intraspinal component (arrows), which opacified during the injection of ethanol. **(g)** Transverse CT scan demonstrates a pathologic fracture (arrows) through the body of L3 4 weeks after injection of 50 mL of ethanol. The vertebral body demonstrated aseptic necrosis at the time of corpectomy and spinal stabilization. Blood loss was less than 150 mL, and the patient was asymptomatic after corpectomy and spinal stabilization.

tients 5 and 7, respectively, developed compression fracture of the treated vertebrae; patient 5 was working in her garden and patient 7 had returned to weight lifting. The patients underwent vertebral resection with bone grafting and insertion of a Harms Cage (DePug Acromed, Cleveland, Ohio) and a Z-Plate (Sofamor Danek, Memphis, Tenn). Blood loss at each procedure was less than 200 mL. Histologic examination revealed aseptic necrosis of bone without residual hemangioma.

In all patients, spinal MR imaging before discharge showed nonenhancement and shrinkage of the hemangioma and restitution of the cerebrospinal fluid space surrounding the cord. Improvement of symptoms often began within 1–2 days after treatment. Five patients with paraparesis were able to walk without support. Patient 11 had been followed up for only 4 months at the time of this writing, but the hemangioma had shrunk and she was walking with a walker.

The five patients with radiculopathy had partial (n = 3) or complete (n = 2)

relief of pain. In three instances, the expanding hemangioma displaced cortex into the vertebral foramina of the involved nerve root. Although the hemangioma was devascularized, treatment of the residual radiculopathy may require foraminotomy; residual radiculopathy may be due to osseous encroachment, which would not be expected to respond to ethanol ablation. Patient 8 (Fig 4) was involved in workers' compensation, and although the hemangioma was completely obliterated, he continued to complain of local back pain.

Follow-up was 15–76 months (mean, 40.6 months), excluding patient 11. All hemangiomas remained obliterated, and all patients maintained their improved status. Pathologic fractures requiring surgical bracing occurred in two patients within 16 weeks after therapy; none of the remaining patients had evidence of pathologic fractures. The other patients maintained the normal height of their vertebral bodies with no change in the pattern of ossification on CT scans.

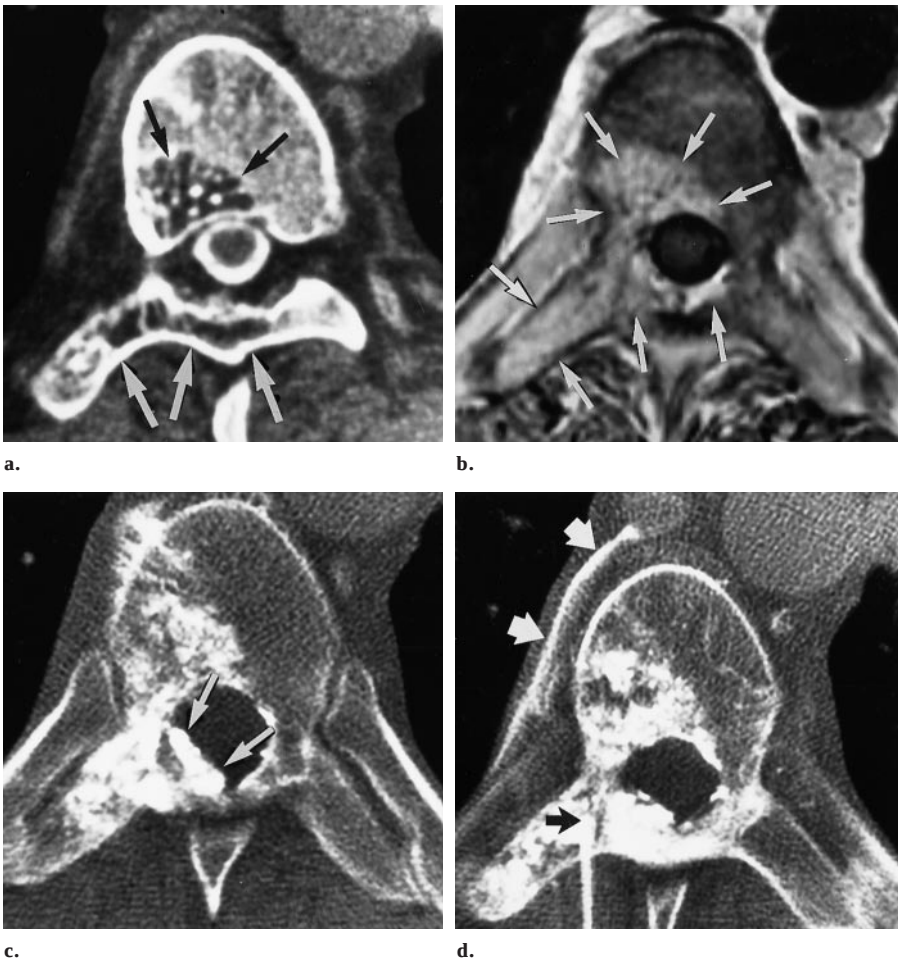

Figure 4. Patient 8. Right T7 radiculopathy and hemangioma involving the right body, pedicle, and lamina of T7 in a 41-year-old man. Note the absence of invasion of the spinal canal by the hemangioma (arrows) on both the **(a)** transverse CT myelogram and **(b)** transverse gadolinium-enhanced spin-echo intermediate-weighted MR image (1,000/15). **(c)** Transverse CT angiogram shows hemangiomatous involvement of the right pedicle and lamina with a posterolateral intracanalicular component (arrows). **(d)** Transverse CT scan of the hemangioma shows placement of the needle (black arrow) at the junction of the pedicle and lamina with filling of all components of the hemangioma. Note filling of a right intercostal vein (white arrows), which indicates that less than 10 mL of ethanol will be adequate. Subsequent injection of 3 mL of ethanol obliterated the malformation.

DISCUSSION

Symptomatic vertebral hemangiomas are uncommon lesions. Radiographs may fail to show the typical striated bony appearance, particularly in the thoracic spine. Patient 1 was referred with a diagnosis of spinal multiple sclerosis, and the hemangioma was revealed only at the time of CT myelography, which demonstrated a total subarachnoid block and the typical appearance of hemangioma. Cord compression is a neurosurgical emergency; embolization followed by resection has been the treatment of choice, but even following embolization, surgery is often associated with profuse hemorrhage. In our experience, injection of such lesions with ethanol can be performed without morbidity and has led to improvement of symptoms within days of the procedure.

Critical to the effectiveness of percutaneous ethanol injection is the requirement that the ethanol be injected into the vascular spaces of the hemangioma. In older patients, hemangiomas tend to become centrally fibrotic, and although the bone changes may be extensive, the hypervascular component responsible for cord or root compression may be very small. It is, therefore, important to determine before therapy the extent of the hemangioma, which does not always correspond to the bone changes, especially in patients who have undergone radiation therapy.

The normal vasculature of the vertebral body permits aspiration of blood. In the past, intramedullary injection of contrast material has been used to opacify veins of the lower extremities and pelvis. To locate hemangiomatous vessels, CT angiography should be performed before the ethanol injection to ensure that the needle tip is placed in the functional vascular spaces of the lesion. We generally position the needle tip in the posterior half of the vertebral body, close to the junction of body and pedicle, because filling the hemangiomatous components responsible for cord compression is critical to the relief of neurologic signs and symptoms. Injection of nonionic contrast material prior to injection of ethanol will demonstrate the correct position of the needle tip and the completeness of filling of the hemangioma.

Injection of contrast material during the preliminary CT scan of the hemangioma and the subsequent injection of ethanol should be forceful enough to opacify the entire network of abnormal vessels. The forceful injection of ethanol is painful, and deep narcosis with the supervision of an anesthesiologist is strongly recommended. Proximity of the spinal cord and nerve roots to the needle tip may be of concern, but the periosteal barrier protects them from neurotoxicity, even following forceful ethanol injections. Systemic effects of the ethanol have not been observed.

Clinical Findings in 11 Patients with Symptomatic Vertebral Hemangiomas Treated by Means of Intralesional Injection of Ethanol

Patient No./Sex/ Age (y)	Level	Extent	Symptom	Duration of Symptom (mo)	Prior Therapy	Total Amount of Ethanol Injected (mL)	No. of Procedures	Obliteration of Hemangioma*	Relief of Symptoms	Complication	Follow-up (mo)
1†/F/54	T4	Holovertebral	Paraparesis, walked with walker	12	None	15	2	Complete	Complete	None	76
2†/F/64	T12	Holovertebral	Paraparesis, neurogenic claudication, walked with cane	3	None	12	1	Complete	Complete	None	68
3/F/73	L4	Holovertebral	Lumbar radiculopathy, treated with morphine pump	18	3,000 rad (30,000 mGy), embolization	10	1	Complete	Partial, occasional use of nonnarcotic analgesics	None	47
4/F/42	L4	Holovertebral	Lumbar radiculopathy	12	None	9	2	Complete	Complete	None	46
5/F/64	T12	Holovertebral	Paraparesis, used wheelchair	66	3,000 rad (30,000 mGy), embolization	42	2‡	Partial	Improved, no pain, walks with walker	Compression fracture of T12 16 weeks after therapy; corpectomy and fusion	39
6/M/34	T5	Holovertebral	Paraparesis, walked with walker	7	960 rad, (9,600 mGy), laminectomy abandoned because of profuse bleeding	9	2	Complete	Complete	None	37
7/M/45	L3	Body	Lumbar radiculopathy, back pain	12	Embolization	50	1	Complete	Partial	Compression fracture of L3 4 weeks after therapy; corpectomy and fusion	36
8/M/41	T7	Hemivertebra and pedicle	Intercostal radiculopathy	3	None	5	1	Complete	None§	None	24
9/F/45	T9	Body and pedicle	Intercostal radiculopathy	1	None	5	1	Complete	Complete	None	18
10/M/29	L2	Holovertebral	Paraplegia, intense back pain, used wheelchair	6	Resection with bracing, profuse hemorrhage	10	1	Complete	Complete	None	15
11/F/65	T9	Holovertebral	Paraparesis, walked with walker	8	None	13	1	Complete	Partial, walks with walker	None	4

* Obliteration of hemangioma proved on negative intercostal-lumbar angiogram and negative contrast-enhanced CT and MR images 3 months after therapy.
† Previously reported in reference 16.
‡ The injection of ethanol was interstitial because vascular space could not be accessed.
§ This was a workers' compensation case, and symptoms did not improve in spite of obliteration of the hemangioma.

Injection of ethanol causes intralesional thrombosis and destruction of the endothelium that composes the hemangioma. Devascularization is followed by shrinkage of the lesion, which thereby decompresses the cord and nerve root. However, an excessive amount of ethanol may cause aseptic necrosis; the patients who received volumes of 42 and 50 mL developed pathologic fractures that required surgical bracing. We now limit the volume of ethanol to 15 mL and have achieved the desired therapeutic effect without encountering further pathologic fractures.

There has been considerable interest recently in the treatment of vertebral hemangiomas by means of the percutaneous injection of methyl methacrylate (20–26). The needle placement and injection of methyl methacrylate generally are performed with fluoroscopic guidance to monitor for leakage, either into draining veins or posteriorly into the spinal canal. This technique is ideal for stabilizing vertebral bodies at risk of collapse or for alleviating severe local pain. However, when a patient is paraparetic owing to extension of the hemangioma into the spinal canal, methacrylate may fill the intraspinal component and exacerbate cord compression. Laminectomy may be required immediately after methacrylate casting to decompress the spinal canal, but it does not remove the new solid component responsible for compression. In addition, when the pedicles and laminae of the vertebrae are involved by the hemangiomatous process, which is the situation most frequently associated with cord compression, the surgeon may still encounter considerable intraoperative bleeding from the posterior elements.

For vertebral hemangiomas, Cotton et al (26) recommended a combination of methacrylate injection into the vertebral body and cyanoacrylate injection into the posterior elements to facilitate subsequent laminectomy. The use of methacrylate requires the placement of large (10–12 gauge) needles and considerable experience on the part of the physician to fill the vertebral body before the material polymerizes. In addition, the methacrylate must be opacified with barium, tantalum, or tungsten to enable online monitoring of its distribution. Methacrylate is an ideal agent for a patient with impending collapse due to hemangioma of the vertebral body without cord or root compression or involvement of the posterior vertebral elements.

Methacrylate restores vertebral strength and prevents pathologic fractures, a com-

mon complication of extensive hemangiomas of the vertebral body. When cord or nerve root compression is present, however, one seeks to shrink hemangiomatous elements away from these critical structures rather than produce a cast that may require subsequent decompression surgery and resection of intraspinal or intraforaminal methacrylate. Only a larger series will demonstrate whether intralesional injection of ethanol in moderate quantities can resolve hemangioma without risk of pathologic fracture.

Direct injection of ethanol into vertebral hemangiomas that cause cord or radicular compression is a simple, safe, and effective technique for rapidly relieving compression and devascularizing the hemangioma. A moderate dose of ethanol (<15 mL) obliterates the hemangioma without affecting stability. Early diagnosis and treatment of the intraspinal extension of the hemangioma is critical to prevent permanent and irreversible neurologic deficit.

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