Vertebrectomy for treatment of vertebral hemangioma without preoperative embolization

Case report

TONY FEUERMAN, M.D., PAUL S. DWAN, M.D., AND RONALD F. YOUNG, M.D.

Division of Neurosurgery, University of California at Los Angeles (UCLA), Los Angeles, and Harbor/UCLA Medical Center, Torrance, California

Vertebral hemangiomas have usually been treated by resection following preoperative arterial embolization. A case is presented in which no feeding tumor vessels were demonstrable angiographically. The tumor was resected by an arterolateral transthoracic approach without preoperative embolization. There was progressive postoperative improvement of the myelopathy.

Key Words • hemangioma • vertebral tumor • myelopathy • vertebrectomy

Vertebral hemangioma is a common tumor of the spine that is rarely symptomatic. When symptoms of myelopathy and/or radiculopathy do occur, they have been successfully treated with radiation therapy, decompressive laminectomy, embolization of feeding vessels, and embolization followed by vertebral body resection.11 We describe a case in which a vertebral hemangioma was resected without preoperative treatment with either embolization or radiation therapy.

Case Report

This 62-year-old right-handed woman suffered the onset of lower-back pain radiating into both thighs 7 months prior to admission. During the next 2 months, she noted pain and numbness of both calves. The pain was relieved by recumbency and was not worsened by the Valsalva maneuver. Over the next 2 months, the patient described clumsiness and weakness of her lower extremities. She had no symptoms of sphincter dysfunction. Eventually, she was able to walk only one block without resting. Her primary complaint at the time of presentation was inability to perform her work as a housekeeper because of lower-extremity weakness.

Examination. The patient was obese with normal vital signs. There was tenderness to palpation over the T9–12 spinous processes. The range of motion of the spine was normal, and there were no visible spinal deformities. Mental status and cranial nerve examinations were normal. Motor examination revealed mild weakness in the proximal muscle groups in the left lower extremity. Muscle tone and bulk were normal. Gait was broad-based and ataxic, with a tendency for the patient to fall to either side with tandem gait. Pain sensation was decreased bilaterally below the T-11 level. Position and vibratory sensation were decreased in both feet. Tendon reflexes were normal except for the right ankle reflex, which was increased slightly. Plantar reflexes were flexor bilaterally.

Plain x-ray films of the spine demonstrated vertical striations and sclerotic trabeculation of the T-10 vertebral body (Fig. 1 left). Computerized tomography showed a high-density stippled pattern within the body

![Fig. 1. Left: Plain x-ray film of the T9–11 vertebral region, anteroposterior view, showing trabeculations within the body of T-10. Right: Computerized tomography scan of T-10 with intravenous contrast material demonstrating a stippled pattern indicating the tumor.](image-url)
Treatment of vertebral hemangioma

and pedicles of the T-10 vertebra with narrowing of the spinal canal by the thickened pedicles. Anterolateral extension of the tumor into the soft tissue was seen (Fig. 1 right). Metrizamide myelography, performed via lumbar puncture, revealed a complete extradural block at the inferior border of T-10. To define the superior aspect of the tumor, metrizamide was introduced through a lateral C1–2 puncture. An extradural block was also noted superior to T-10. Spinal angiography showed that the tumor was supplied bilaterally by the T-10 intercostal arteries with only a slight tumor blush (Fig. 2 left). The artery of Adamkiewicz was identified on injection of contrast material into the left T-9 intercostal artery but did not supply the tumor (Fig. 2 right).

Operation. The thoracic cavity was entered anterolaterally through the T9–10 interspace, and the left lung was deflated with the aid of a double-lumen endotracheal tube. Following reflection of the pleura, an x-ray film was obtained to confirm the location of the tumor. On palpation, the body of T-10 was noted to be hyperostotic anteriorly and laterally with an irregularly raised cortical surface. The vertebrectomy was started by removing the entire left lateral aspect of the corpus with a high-speed drill and a 7-mm gouge. Immediately after the cortical bone was removed, moderate bleeding occurred which was easily controlled with impaction of bone wax. A high-speed drill was then employed to completely remove the vascular tumor and the surrounding cancellous bone. The T9–10 and T10–11 intervertebral discs were removed, as were the inferior endplate of T-9 and the superior endplate of T-11. The remaining fragments of cortical bone were elevated from the thickened posterior longitudinal ligament. A bone graft was harvested from the left iliac crest and impacted between the T-9 and T-11 vertebral bodies. Total blood loss was approximately 1000 ml. Pathology was consistent with vertebral hemangioma.

Postoperative Course. The patient was immobilized in a plastic body brace. On the 3rd postoperative day, she began to walk with assistance. By the time of discharge on the 10th postoperative day, she was walking well without assistance. At her 2-month follow-up examination, she reported resolution of her pain and improved ability to walk. The sensory deficit had also partially resolved. Postoperative x-ray films showed early evidence of fusion (Fig. 3).

Discussion

Vertebral hemangiomas are a common incidental finding at autopsy. Their incidence has been estimated at 11%, but they are rarely symptomatic. A review of the literature reveals less than 200 reported cases. When symptoms do occur, they are most often due to compression of the thoracic spinal cord, presumably because the spinal cord width:spinal canal ratio is greater in the thoracic region than in the cervical region. However, several symptomatic cervical hemangiomas have been reported. Occurrence in children is rare.

Vertebral hemangioma is one of the causes of epidural spinal cord compression, and multiple hemangiomas may mimic metastatic disease. Vertebral hemangiomas have been reported in the multisystem Klippel-Trenaunay-Weber and Kasabach-Merritt syndromes.

The diagnosis of vertebral hemangioma can often be made by the characteristic appearance on x-ray films. The vertebra carries vertical striations and trabeculations; these are most commonly seen in the vertebral body but may be present throughout all elements. Vertebral collapse and erosion of the pedicles are not uncommon. Computerized tomography scanning demonstrates a stippled pattern in the affected portion

![Fig. 2. Left: Angiogram of the left T-10 spinal artery showing the blood supply to the tumor. Only a slight tumor blush is present. Right: Angiogram of the left T-9 spinal artery identifying the artery of Adamkiewicz (arrow). This vessel does not supply the tumor.](image1)

![Fig. 3. Postoperative x-ray film of the operative site, anteroposterior view, showing early evidence of fusion.](image2)
of the vertebra. Myelography is helpful in delineating the extent and mechanism of compression of the dural sac.

Myelopathy may be caused by four basic mechanisms: 1) an epidural soft-tissue mass compressing the spinal cord, 2) expansion of the involved vertebra to compress the spinal cord, 3) a compression fracture, or 4) epidural hemorrhage. Surgical therapy is directed toward relieving the anatomical cause of myelopathy. Epidural hemorrhage presents as an acute spinal cord compression and has been successfully treated by immediate laminectomy and drainage of the epidural blood. The other three mechanisms of myelopathy present chronically with bone or tumor compressing the dural sac. Two different operative approaches have been used. Laminectomy allows indirect decompression of the spinal canal, although it does not directly relieve the cause of compression. Posterior spinal fusion has been used with laminectomy in cases where laminectomy alone would cause instability. Alternatively, the tumor and/or bone fragments can be approached directly by a vertebral body resection by either an anterolateral thoracotomy or a lateral costotransversectomy.

Spinal angiography is important in preoperative planning for resection of a vertebral hemangioma. It is necessary to determine the relationship between blood supply to the tumor and the spinal cord so that the arterial supply of the spinal cord can be spared while tumor vessels are sacrificed. The artery of Adamkiewicz should be identified in cases of vertebral hemangioma in the lower thoracic area as it is the major artery to the spinal cord in that region. There are usually well-defined tumor feeding vessels which may be embolized. Embolization alone has been reported on occasion to relieve symptoms of myelopathy. More importantly, it reduces the blood supply to these usually vascular tumors so as to allow resection. As with other vascular tumors and malformations, our case demonstrates that major feeding vessels may not be delineated on angiography, making preoperative embolization impossible. Vertebral body resection has previously been considered impractical without preoperative embolization because of the extreme and rapid bleeding that would be encountered. Decompressive laminectomy and/or radiation therapy has been used in such cases, with vertebral body resection reserved for patients in whom embolization of feeding vessels could be performed preoperatively. The angiographically demonstrated decreased vascularity of the tumor in our case allowed vertebral body resection without excessive hemorrhage. The possibility and method of tumor resection should be judged not only by whether or not the feeding vessels can be embolized but also by the vascularity of the tumor as demonstrated on angiography. If the vascularity can be reduced by embolization or if the usual angiographic vascular stain is not excessive, vertebral hemangiomas may be successfully resected, as our case demonstrates.

References


Manuscript received August 21, 1985. Accepted in final form March 4, 1986. Address reprint requests to: Tony Feuerman, M.D., Division of Neurosurgery, UCLA School of Medicine, 10833 LeConte Avenue, Los Angeles, California 90049.