

Compression Fracture of the Hemangiomatous Vertebra Leads to Clinical Lumbar Spinal Stenosis: Case Report

Hemanjiyomatöz Vertebranın Kompresyon Fraktürüne Bağlı Lomber Spinal Stenoz Olgusu

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Geliş Tarihi/Received: 05.09.2006
Kabul Tarihi/Accepted: 23.01.2007

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ABSTRACT Vertebral hemangiomas are not infrequent on radiological evaluation of the spine. Symptomatic vertebral hemangiomas can mimic various neurological conditions with motor and sensory deficits. Here we report a male patient with lumbar spinal stenosis symptoms due to compression fracture of the hemangiomatous L4 vertebra. A possible spinal cord injury was prevented by early diagnosis and appropriate treatment of the vertebral hemangioma.

Key Words: Spine; hemangioma; fractures, compression; spinal stenosis

ÖZET Vertebral hemanjiomalara omurganın radyolojik incelemesi sırasında rastlanabilir. Semptomatik vertebral hemanjiomalar duyuşsal ve motor kayıp ile sonuçlanabilecek nörolojik problemlere neden olabilirler. Bu yazıda kompresyon fraktürüne bağlı olarak spinal stenoz bulgularıyla ortaya çıkan L4 hemanjiyomatöz vertebra olan bir erkek hasta bildirilmiştir. Hastada erken tanı ve uygun tedavi sayesinde vertebral hemanjiyomanın yol açabileceği spinal kord yaralanması önlenmiştir.

Anahtar Kelimeler: Vertebral hemanjiyoma, kompresyon fraktürü, lomber spinal stenoz

Türkiye Klinikleri J Med Sci 2008, 28:408-410

Vertebral hemangioma is a benign tumor originating from vascular tissue. It generally does not produce symptoms. Asymptomatic vertebral hemangiomas are incidental findings in radiographic studies with a prevalence of 10-12%.¹ Symptomatic vertebral hemangiomas frequently present with back pain.² Rarely, vertebral compression fracture with neurological deficit develops.³⁻⁸ In this article, we present a case of vertebral hemangioma with compression fracture.

CASE REPORT

A 64-year old male Caucasian internal architect presented with low back pain. He has had pain for approximately one year. The pain intensity increased after walking a distance of 300 meters. He mentioned that his pain was relieved when he stopped and flexed his trunk. He experienced the first episode of pain while he was trying to lift furniture during home decoration. On physical examination, there was loss of sensation in the L2, L3, L4 dermatome areas. Deep tendon reflexes and muscle tonus were normal and

muscle strength was 4/5 at the iliopsoas and the quadriceps on the muscle test. Non-steroidal anti-inflammatory drugs (NSAIDs) were administered with the diagnosis of lumbar spinal stenosis. The patient did not respond to medical treatment and thus, further radiological studies were undertaken. Magnetic resonance imaging (MRI) study of the lumbar region revealed a compression fracture at the L4 vertebra. In the sagittal section, vertebral height had decreased by 50%. On the T2 weighted images, hyperintensity was observed at the L4 vertebral corpus and spinous process (Figure 1). The axial section demonstrated that the fractured vertebra had protruded into the spinal canal, compressing the dural sac (Figure 2). Vertebral angiography was planned with the suspicion of hemangioma.



FIGURE 1: T2 weighted sagittal image reveals hyperintensity of the hemangioma and expansion of the L4 vertebra toward the spinal canal due to compression fracture.

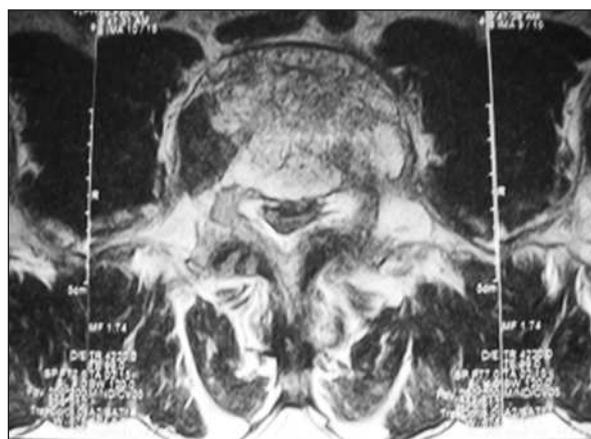


FIGURE 2: T2 weighted axial image shows hyperintensity of the hemangioma and canal stenosis due to the collapse of the vertebra.

Following angiography, arterial embolization was performed on the feeding vessels of the hemangioma. On the following day, corpectomy and bone graft interposition with posterior fusion at L4 was performed. Back pain relieved in the postoperative period; however, muscle strength was 2/5 and 3/5 at the left hip flexors and knee extensors, respectively. There was also loss of sensation in the L2-3-4 dermatomal areas. The patient complained of left leg pain and participated in a physical therapy and rehabilitation program consisting of electrical stimulation to the quadriceps muscle and strengthening exercises. Fusion of the bone tissue was observed on the control radiographic evaluation. Amytriptyline 10 mg/day orally was administered for the neuropathic pain at his left leg. His medication was changed to oral gabapentin 300 mg/day, due to inadequate response to the former regimen. Later, gabapentin dose was increased to 3 x 300 mg/day. His pain did not bother him anymore. Neurophysiologic study performed at postoperative 9 months revealed partial axon loss in the upper section of the lumbar plexus. At postoperative 12 months, muscle strength was 4/5 at the iliopsoas and 5/5 at the quadriceps. There was loss of sensation in the L4 dermatome area. Deep tendon reflexes and muscle tonus were normal. The patient is currently on oral gabapentin 900 mg/day. Now he is doing well and is able to work in his business.

DISCUSSION

Neurological symptoms tend to develop later than back pain in symptomatic vertebral hemangiomas. The neurological deficit may either be due to compression of the expanding tumor tissue or to vertebral compression fracture.⁵ The patient we report complained of back pain that had started a year ago and later he developed sensory loss in the L2,3,4 dermatome area. Vertebral fracture probably had occurred during minor spinal trauma while he was working. The fact that the hemangioma had invaded the vertebrae totally, including the corpus and the posterior elements has facilitated pathologic fracture. Spinal cord lesions resulting from vertebral compression fractures were reported in

previous studies.⁴⁻⁶ The outstanding feature of the case we present was the fact that vertebral collapse had resulted in spinal stenosis. Ahn et al reported a case with lumbar hemangioma causing neurogenic claudication and cauda equina syndrome.⁸

Symptomatic vertebral hemangiomas are most frequently reported at the lower thoracic region and secondly the lumbar region.⁹ They frequently involve a single vertebra. Multiple involvement is rare.¹⁰ Hemangiomas may be located at any region of the vertebra or may involve all three columns.

Vertebral hemangiomas are diagnosed with radiological studies. On plain x-ray, thickening at the vertebral trabeculae is observed. This is termed "jailhouse striations". Axial computerized tomography (CT) reveals cystic vascular structures as "polka dots". CT is a useful technique for showing bone lesions. MRI helps to determine the level of the lesion and identification of the neurological lesion. Hyperintensity on T1 and T2 weighted images is due to vascular structures, adiposity and interstitial edema.⁵ Reports indicate that 50% of hemangiomas demonstrate hyperintensity at T1 weighted images.¹¹

Surgical decompression, vertebroplasty, radiotherapy, intralesional ethanol or methyl methacrylate injection may be used for the treatment of symptomatic vertebral hemangiomas.^{7,8,10,12} Total dose for radiation therapy should be 40 Gy.¹² Em-

bolization may be used concurrently with surgical intervention or alone. Despite various risks, embolization may decrease vascularity and the size of the tumor and may relieve neurological symptoms without other interventions.⁴ This was the case in our patient, where after embolization back pain relieved considerably. Depending on the extent of the lesion, the surgical procedure may include decompressive laminectomy or corpectomy.⁴ Radiotherapy is not required after total resection of the hemangioma. Radiation therapy may be considered following incomplete resection, in order to prevent recurrences.³ In our case, complete resection was achieved with success. Ethanol injection into the lesion is another approach in symptomatic vertebral hemangiomas. The volume of alcohol should be limited to 15 mL, since higher doses carry the risk of developing aseptic necrosis and pathologic fracture. Ethanol injection was reported to be a safe and effective method of treatment.¹⁰

In conclusion, vertebral hemangiomas should be considered in the differential diagnosis of back pain. Hemangioma related thickening of the vertebral trabeculae may increase the durability of the vertebra to compressive stress. However, it must be kept in mind that extensive vertebral involvement by the hemangioma may lead to pathologic fractures. We therefore, recommend consideration of therapeutic options before neurological symptoms develop.

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