Case Report

Aggressive vertebral hemangioma in early adolescent age group: Atypical presentation with computed tomography and magnetic resonance imaging appearance

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Abstract

Vertebral hemangiomas (VH) are vertebral vascular malformations, which may remain asymptomatic until adulthood. Sometimes, young individuals present with unexplained backache, which poses the diagnostic dilemma. We present a case of 15-year-old female who presented with prolonged unexplained intermittent upper back pain in the dorsal region. She was diagnosed as a case of aggressive VH after ruling out other infective and malignant pathologies and treated with decompressive therapy. The present case highlights the role of imaging studies such as computed tomography and magnetic resonance imaging in such conditions.

Key words: Aggressive, Backache, Computed tomography, Magnetic resonance imaging, Vertebral haemangiomas, Vascular malformations

Hemangiomas (VH) are ertebral generally asymptomatic vascular malformations with a slight predilection for females. Only 3.7% of VH may become symptomatic and show invasion of the spinal canal and/or paravertebral spaces. These are mostly asymptomatic in pediatric age group and become symptomatic in 40-50 years of age. The main presenting feature is a pain due to axial loading either by exercise or housework. Complications are due to encroachment upon the spinal canal and vertebral collapse leading to aggravation of symptoms [1]. Sometimes, back pain is the only presenting feature in adolescents leading to the diagnosis of VH [2]. We present such a case of VH in the adolescent girl who presented with back pain.

CASE REPORT

A 15-year-old adolescent girl of army personnel family presented with dull back pain in the upper back of 2 year's duration. There was a delay in diagnosis and treatment because of the father being away on armed forces duty. There was progressive weakness in both the lower limbs for the last 2 months without any neurological deficit. Plain radiograph of the spine done at other hospital did not reveal any significant abnormality, and she was managed at the pain and physiotherapy clinic on an irregular basis with little temporary relief in pain. Therefore, she was refereed to our hospital for diagnosis and management. On examination, she was conscious, well oriented, and well nourished. Her vitals and general examination were also normal except tenderness along the paravertebral regions over upper dorsal vertebrae (D2-D5). Her neurological examination was within normal limits, and there was no neurological deficit. Rest of the systemic examination was also within normal limits.

Laboratory investigations including complete blood count, renal function test, and liver function test were normal. Patient was subjected to cross sectional imaging of non-contrast computed tomography (NCCT) and magnetic resonance imaging (MRI) of the dorsal spine. NCCT of the dorsal spine in axial (Fig. 1a and b) and sagittal (Fig. 1c) images show expansible lesion of D3 vertebral body involving posterior elements giving characteristic "polka dot" appearance. MR T1W images of the spine showed hyperintensity in body and posterior element of D3 vertebrae which was suppressed on fat sat sequences (Fig. 2a-c). MR T2W images showed characteristic polka dots appearance (Fig. 3a-c). MR TIW contrast enhanced images have shown avid contrast enhancement in the vertebral body and posterior element of D3 vertebra (Fig. 4a-c). Diagnosis of aggressive VH was made on the basis of NCCT and MRI. The case was managed with decompressive laminectomy without pre-surgery embolization. Histopathological examination of the specimen showed displacement of bone by multiple vascular spaces. The

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irradiation of the involved region was advised in follow up for further management. 20 fractions were advised in 4 weeks period in divided doses with a total dose of 40 Gy.

DISCUSSION

VH are called as aggressive hemangiomas because of their extra-osseous extension, bone expansion, disturbance of blood

flow, and occasionally compression fracture of the involved vertebra. These behave like infiltrative mass and can cause mass effect on the spinal cord. Rarely, it can also present with back pain in younger age group. These may be associated with severe cord compression, which gradually leads to neurological deficits. VH presenting with back pain is very rare and Cross et al. reported four cases and one out of which has presented with cord compression [3]. The differential diagnosis of back



Figure 1: Non-contrast dorsal spine computed tomography. Axial (a and b) and sagittal (c) images show expansile lesion of D3 vertebral body and posterior elements (broad arrow in c). Trabecular thickening and intervening decreased density gives the characteristic "polka dot" appearance on axial images (white arrow in a and black arrow in b). Right paravertebral and epidural soft tissue component is seen extending from D2 to D4 vertebrae



Figure 2: Magnetic resonance T1W sagittal (a) and axial (b) images show hyperintensity in body and posterior element of D3 vertebrae (arrow in a and star in b) which is suppressed on fat sat T1W sagittal image (arrow in c)



Figure 3: Magnetic resonance T2W sagittal (a and b) and axial (c) images show hyperintensity in body and posterior element of D3 vertebrae with mild expansion and coarse vertical trabeculae (polka dots). Hyperintense soft tissue component is seen in epidural space and right paravertebral space with thinning and myelomalacic changes in the cord (black arrow in a)



Figure 4: Post-contrast magnetic resonance T1W Sagittal (a and b) and axial (c) images show avid enhancement in vertebral body and posterior element of D3 vertebra with visualization of prominent draining vessels (black arrow in b). Right paravertebral and epidural soft tissue component show similar enhancement. Epidural soft tissue is seen extending from D2 to D4 vertebral level

pain in adolescents includes non-specific pain or muscle strain, herniated disc, spondylolysis, scoliosis and Scheuermann's kyphosis. Less common causes include intramedullary tumors like hemangioblastoma, ependydoma and astrocytoma, infection and sickle cell crisis. Therefore, careful evaluation of these patients is of paramount importance, and VH has to be differentiated from these pathologies [4-7].

Management of these cases also varies depending upon the aggressiveness of the VH. There is an inverse relationship between the amount of intra-osseous fatty stroma and aggressiveness of the lesion [8]. Asymptomatic VH shows high signal intensity on both T1 and T2 sequences that helps to distinguish them from lymphoma and other highly cellular neoplasms as has been seen in our case [9]. Involvement of the posterior elements and the whole vertebral body with less marked pathological compression of the vertebral body points more towards symptomatic VHs when compared to metastatic diseases. Friedman [6] suggested following characteristics to consider aggressive VH: Female sex, mid-thoracic location, marked hyperintensity on T2, intraosseous signal voids and enlarged paraspinal vessel.

Surgical resection using the laminectomy with or without fusion is advocated. In cases with spinal cord compression, a decompressive laminectomy is usually preferred [10].

There is no standard treatment protocol for active or aggressive VH. Initial management includes supportive treatment with analgesics. Radiation therapy or embolization is an effective therapeutic alternative for patients with severe refractory pain with no neurological deficits or with slowly developing neurological symptoms [11]. The region of interest is irradiated with 40 Gy in divided doses over 4 weeks. However, in case of rapidly progressing neurological deficits, surgical decompression with spinal reconstruction is needed [12]. The management protocol of these patients includes preoperative embolization and surgical decompression which is followed by radiation [13]. Annual neurological and radiological examination is recommended for the patients where hemangiomas are associated with pain [2].

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