Case Report of Spinal Cord Astrocytoma Presenting with Extensive Spinal Vertebral Body Destruction

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Key words
- Astrocytoma
- Low-grade glioma

Abbreviations and Acronyms
MRI: Magnetic resonance imaging
SCA: Spinal cord astrocytoma
WHO: World Health Organization

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BACKGROUND: **Spinal cord astrocytomas are rare lesions in adults.**

CASE DESCRIPTION: **In this report, we present the case of a 28-year-old female patient who presented with a 2-year history of back pain and 3-month history of leg weakness. Magnetic resonance imaging of the patient showed an intrinsic expansive spinal cord lesion with extensive vertebral body destruction. Open biopsy from the tumor with limited debulking confirmed World Health Organization grade II astrocytoma. Postoperatively, the patient had mild improvement in neurologic status.**

CONCLUSIONS: **This case represents a rare presentation of a spinal cord astrocytoma with extensive erosion of the vertebral body.**

INTRODUCTION

Vertebral destruction can result from different pathologies. The causes could be infectious, metabolic, or neoplastic conditions.[1] From the neoplastic conditions, 95% of the cases are metastatic tumors.[1] Other than metastasis, differential diagnosis for isolated spine-destroying neoplastic conditions include chordoma, giant-cell tumor, aneurysmatic cyst, osteoid osteoma, osteoblastoma, plasmacytoma, osteosarcoma, Ewing's sarcoma, and neuroblastic tumors.[1] Tumors originating from neural tissue in the spinal canal may rarely cause bony scalloping.[2] There are also few case reports of schwannomas with associated vertebral body destruction.[3,4] Otherwise, it is very rare for tumors originating from the spinal canal neural tissues to cause significant bony changes.

Spinal cord astrocytomas (SCAs) are rare intramedullary lesions.[5] Although in the pediatric population they represent the single most common histologic type of intramedullary lesion, in adults they are less common than ependymomas.[5] Most SCAs are low-grade lesions, but approximately 25% of cases are high-grade, World Health Organization grade (WHO) III and IV.[5] In adults, the lesions rarely present in patients older than the age of 60 years.[6] According to a retrospective review of a 20-year experience with these lesions at Duke University, the mean age at presentation was 22 years with a male preponderance of 70%.[7]

Clinical presentations of astrocytomas include back pain with associated progressive neurologic deterioration. The location of these lesions is nearly evenly divided among the cervical, thoracic, and lumbar spine.[8] The neurologic symptoms are usually asymmetric with a variable degree of motor and sensory symptoms with slow insidious onset, often taking years to diagnose.[8] Radiologic diagnosis is usually made with magnetic resonance imaging (MRI), where a fusiform expansion of the cord extending several body segments often is seen. It is usually isointense to hypointense on T1-weighted sequence and hyperintense on T2 and fluid-attenuated inversion recovery images.[9] Gadolinium enhancement shows heterogeneity with irregular margin. As drop metastasis can occur with this lesion, it is advisable to image the entire neuroaxis when a lesion is found. Plain radiograph is usually normal but may rarely show bony scalloping with thinning out of lamina and medial pedicel erosion.[2]

Treatment of these lesions is controversial. Options of treatment include surgery, chemotherapy, and radiotherapy. If a tissue diagnosis is required to differentiate the lesion from other intramedullary diagnoses such as infection or inflammation, then an open biopsy is recommended. If a distinct tissue plane can be identified at the time of surgery, then it is reasonable to proceed with a more aggressive surgical debulking, but that is rarely the case, as most of these lesions are infiltrative and not easily separated from surrounding neural tissue.[10] For lesions that are grade II or greater, radiation has become the mainstay of treatment with some options for chemotherapy.[11]

In this paper, we present a case of a young adult patient with back pain and progressive weakness of the lower extremities who was found to have an extensive intramedullary spinal cord lesion with associated vertebral body destruction on MRI.

CASE REPORT

A 28-year-old woman presented with 2 years history of back pain which has worsened since 8 month prior to her presentation. Over the 3 months before her presentation, she developed weakness of the lower extremities, worst on the left. It had progressed to the point whereby she

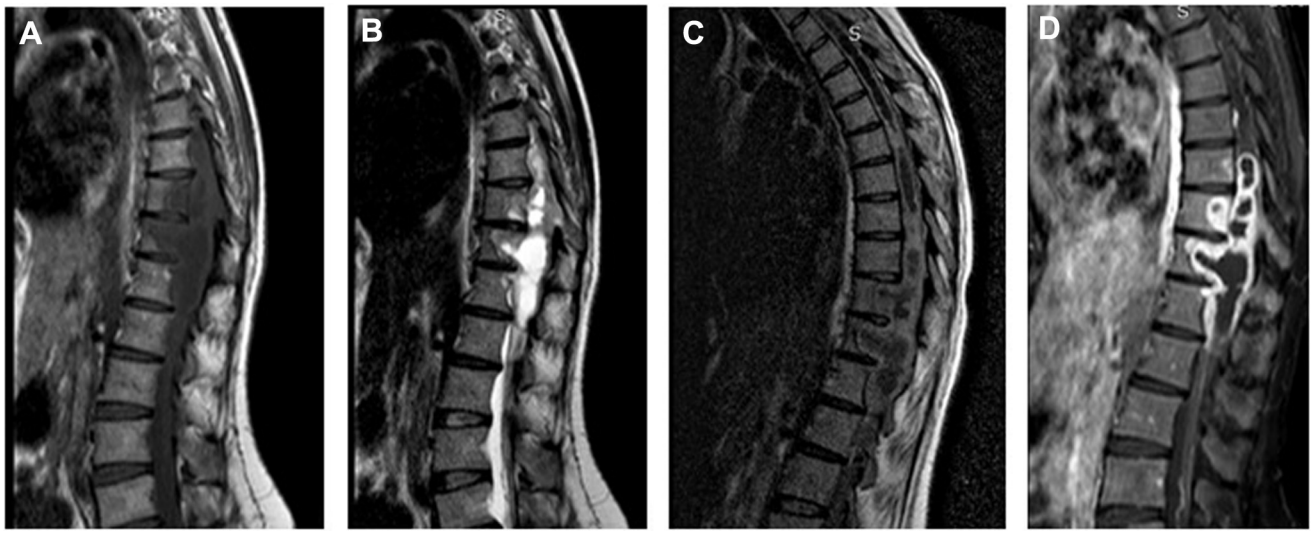

Figure 1. (**A**) T1-weighted sagittal magnetic resonance imaging (MRI) shows a heterogeneous mass with peripherally isointense and central hypointense mass eroding the vertebral body. (**B**) T2-weighed sagittal MRI shows a peripherally isointense and central hyperintense mass eroding the vertebral body. (**C**) Fluid-attenuated inversion recovery shows the intramedullary mass and the cranial segment of the spinal cord with syrinx and cord expansion. (**D**) T1-weighted MRI with contrast shows the peripherally enhancing and central cystic nonenhancing component of the mass.

was unable to ambulate independently in the week before admission. She had no bladder or bowel incontinence. She had no history of trauma, no fever, and no history of known malignancy. At admission to the hospital, she had normal power on the upper extremities but diffuse weakness in the lower extremities: 4+/5 on the right lower limb 4−/5 in the left lower extremity except dorsiflexion of the ankle, which was 0/5 and plantar flexion 3/5. Sensation was intact except for a subjective decrease in sensation on the dorsum of the left foot. The deep tendon reflexes of the knee were +3 bilaterally and ankle reflex was +2 on right and 0 on left. Plantar reflex was equivocal on left and down going on the right.

INVESTIGATIONS

Blood workup was within the normal range. Chest radiograph and abdominal ultrasound were normal. MRI of the thoracolumbar spine showed an intramedullary mass that was peripherally T1 isointense and centrally hypointense. On T2, it was a peripherally isointense and centrally hyperintense mass. It extended from the Th8 to Th12 vertebra level and had associated proximal syrinx (Figure 1). MRI also showed extensive vertebral bony destruction from Th9 to Th12. The mass showed intense peripheral enhancement on gadolinium contrast with multiloculated central cystic component (Figures 2 and 3). There was scoliosis of the thoracolumbar vertebrae on the coronal image (Figure 4). On computed tomography scan (Figure 5), it showed multiple vertebral lytic areas with sclerotic borders and decreased vertebral height.

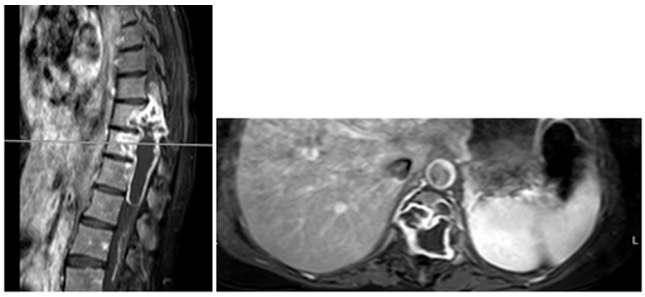

Figure 2. Magnetic resonance imaging with contrast, axial and sagittal images, showing the heterogeneous peripherally enhancing mass with extensive vertebral erosion.

SURGERY

After we evaluated the patient and the imaging, an open biopsy with partial debulking and fenestration of the cystic mass was performed. A midline incision was undertaken over the thoracic spine. At the level of T11−T12, thinned-out laminae were identified and troughs were created on either side across both segments in preparation for a laminoplasty. The laminospinous complex was left intact across the 2 segments and were lifted out in toto. The underlying dura was thinned out. The dura was opened in the midline and an intradural mass was found filling the whole spine canal and with no visible normal-appearing spinal cord. A generous amount for biopsy was taken from the mass; cyst was found, and

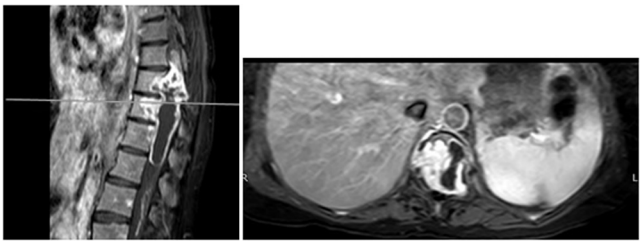

Figure 3. Magnetic resonance imaging with contrast, axial and sagittal images, axial and sagittal images, showing the heterogeneous peripherally enhancing mass with extensive vertebral erosion.

decompressed and had serous fluid content. Because there was an ill-defined boarder between the tumor and the cord, and with lack of intraoperative monitoring at our hospital, no further attempt at more aggressive debulking was undertaken. The dura was closed primarily and the lamina was reattached to the adjacent lamina using silk sutures.

HISTOPATHOLOGY

The biopsy was sent for a pathology examination (Figure 6). Microscopic sections showed hypercellular tumor with hyperchromatic, angulated, elongated nuclei, and fibrillary glial fibrillary acidic protein-immunoreactive processes but no fascicular architecture. No Rosenthal fibers or eosinophilic granular bodies were found. Cellularity was high and with moderate atypia but no necrosis or perivascular pseudorosettes. No ependymal tubules or epithelial membrane antigen, and only nonspecific AE1/AE3 immunostaining was seen. Ki-67 was focally approximately 7%. Less than 1% of the tumor cells showed p53 strong nuclear positivity. IDHI R132 was intermediate.

In addition, calretinin-immunoreactivity and CD34 markers were tested. These should be essentially positive in schwannomas, but they were negative in our case.[12] The pathology conclusion was infiltrative astrocytoma, WHO grade II, not otherwise specified, probable TP53 wild type and ATRX wild type. Isocitrate dehydrogenase I was intermediate.

POSTOPERATIVE COURSE

The patient had an unremarkable postoperative course in hospital. She was given radiotherapy 44 Gy in 22 fractions using cobalt 60 and 2-dimensional planning. At her 3-month follow-up visit, her weakness showed some mild improvement. The power on the left lower extremity became 4/5 but the dorsiflexion power remained 0/5. She was able to walk independently with one cane. Follow-up dynamic radiographic images didn't show mechanical instability. Postradiation MRI, done 4 months after surgery, showed minimal decrease in the tumor size, an otherwise similar appearance.

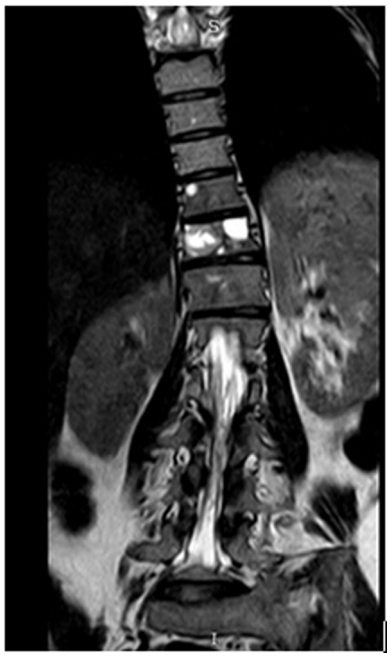

Figure 4. Coronal magnetic resonance scan imaging showing scoliosis.

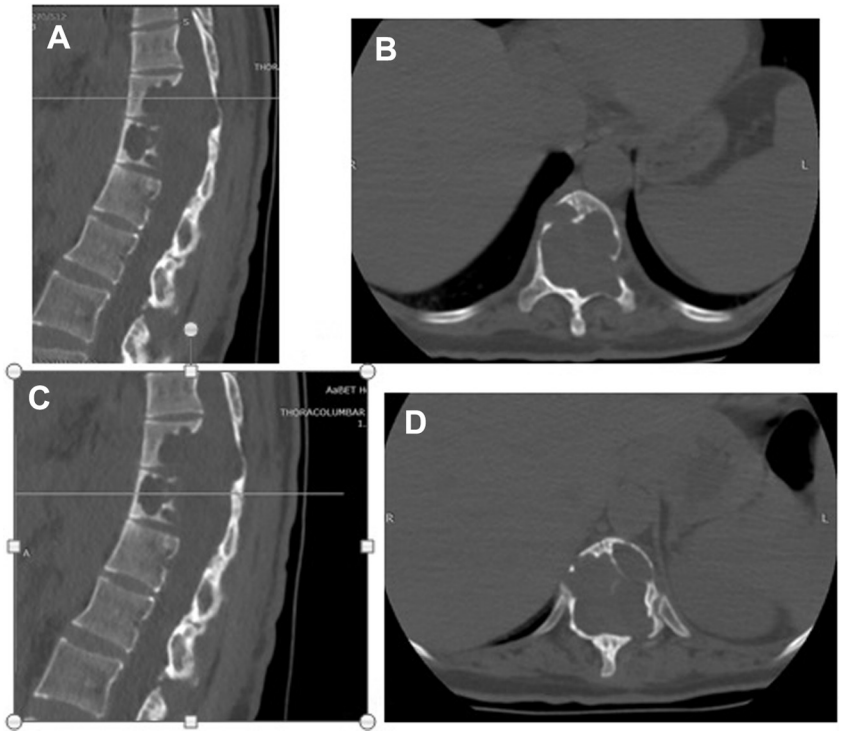

Figure 5. Sagittal and axial computed tomography of the spine at Th11 (**A**–**B**) and Th12 (**C**–**D**) levels shows the vertebral body erosion and thinning out of the lamina; the eroded vertebral body has a sclerotic outline showing the gradual progression of the pathology.

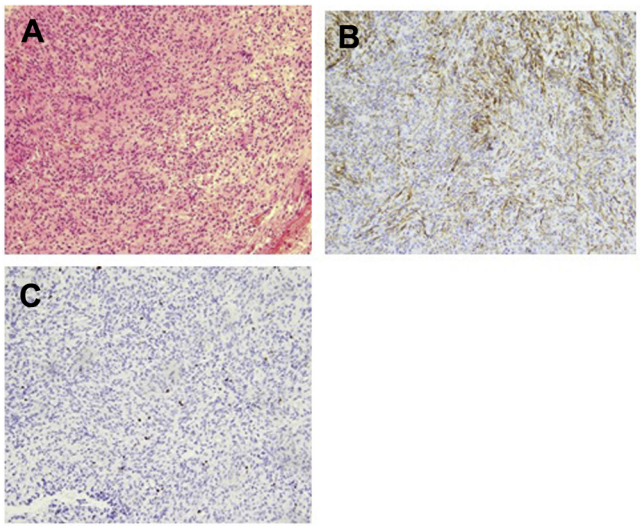

Figure 6. (**A**) Hematoxylin and eosin staining shows a hypercellular tumor with hyperchromatic, angulated, elongated nuclei. (**B**) Glial fibrillary acidic protein (GFAP) showing fibrillary GFAP-immunoreactive processes. (**C**) Ki-67 shows a low mitotic figure.

DISCUSSION

Tumors originating from neural tissue in the spinal canal may rarely cause bony scalloping, but it is very rare to cause vertebral body destruction.[2] There are few case reports of schwannomas causing extensive vertebral body erosion.[3,4] We report a case of spinal cord astrocytoma with extensive erosion of the vertebral body.

SCAs have an infiltrative nature with poor demarcation between tumor and cord. It is usually difficult to attain gross total tumor extirpation without significant neurologic complications. Gross total resection is only accomplished in 12% of WHO grade II and 0% of grade III and IV.[8] In this case, more aggressive debulking was avoided for fear of postoperative neurologic deterioration because of the extent of the tumor and the difficulty differentiating it from normal tissue. We undertook a laminoplasty to minimize the risk of postoperative vertebral instability. Fortunately, the patient had some mild postoperative neurologic improvement, probably from the decompression as a result of the laminotomy and cyst fenestration. From a functional standpoint, this improvement did allow her to regain her ability to ambulate independently.

Histopathologic findings in our patient confirmed that it represented a grade II astrocytoma, which is consistent with what would be the most common grade of lesion in a patient in this age range.[5] Considering the rare presentation, immunohistochemistry confirmation is mandatory. We performed glial fibrillary acidic protein testing, which showed immunoreactive processes. To rule out other pathologies such as schwannoma, calretinin and CD34 marker tests were done. These tests usually are expected to be positive in schwannoma and neurofibroma, respectively, but they were negative in our case.[12]

SCAs are intramedullary tumors with usual eccentric locations. They often are found to extend to multiple levels within the spinal cord. As the majority of these lesions are low grade, they are typically slow growing. One can imagine that as with any slow-growing lesion and increasing localized pressure, some bone remodeling could occur as part of this growth. This is commonly seen in other benign intradural spinal pathologies such as arachnoid cysts and schwannomas, where widening of the spinal canal with scalloping of the vertebral lamina occurs. To our knowledge, however, this is the first report of an intramedullary lesion causing extensive erosive changes in the spine.

Indeed, there are a couple of cases reported in the cranial literature of astrocytomas causing bone remodeling. In 2007, Khan and Hashmi[13] reported on a case of an adolescent with a low-grade astrocytoma causing calvarial erosion. More recently Handzhiev et al.[14] reported on an intracranial case of a low-grade intracranial astrocytoma causing localized dural and bone destruction. These authors postulated that with loss of cerebrospinal fluid in the area, chronic pulsations of the brain on the adjacent calvarium led to gradual erosion of the inner table. A similar phenomenon could certainly be underway in the spinal case reported here.

CONCLUSIONS

SCAs are rare intramedullary lesions in adults. Although other benign spinal lesions have been described causing erosive lesions of the spine, this is, to the best of our knowledge, the first case report of intramedullary astrocytoma resulting in such dramatic changes to the surrounding bones of the spine.

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