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Paravertebral neurinoma associated with aggressive intravertebral extension

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Abstract Neurinomas are relatively common benign tumors thought to arise from nerve sheath cells.

Although intraosseous neurinomas may destroy the bone, extraosseous neurinomas with extensive destruction and invasion of bone are considered rare. We present two unusual cases of a benign extraosseous neurinoma that extensively invaded the vertebral body through the nutrient canal.

Keywords Extraosseous neurinoma · Vertebral body · Nutrient canal · Computed tomography · Magnetic resonance imaging

Introduction

Neurinomas are relatively common benign tumors thought to arise from nerve sheath cells. Typically, they are soft tissue masses along sensory nerve roots in the head, neck, mediastinum, and retroperitoneum, or along the cranial nerves [1, 2]. Neurinomas arising in the paravertebral region occasionally appear as a dumbbell-shaped configuration with

enlargement of the intervertebral foramina and posterior scalloping of the vertebral body. Although intraosseous neurinomas have been reported to invade and destroy the vertebral body, resulting in an osteolytic lesion, such a growth pattern is extremely rare in extraosseous neurinomas. We report two cases of extraosseous neurinoma that extensively invaded the vertebral body through the nutrient canal.

Case reports

Case 1

A 9-year-old girl presented with nontender distention of the back which rapidly increased in size. Physical examination revealed a firm, nontender distention to the left of her mid-back. Neurological examination and laboratory data were normal. A chest radiograph showed an



Fig. 1A–E A 9-year-old girl with a benign extrasosseous neurofibroma of the T10 nerve root. **A** Chest radiograph shows a paravertebral mass lesion (*arrow*) that is accompanied by destructive change in the T10 vertebral body and obliteration of its left pedicle. **B** Computed tomography shows that the paravertebral mass extends into the spinal canal in a dumbbell-shaped configuration, causing enlargement of the neural foramina and posterior scalloping of the vertebral body. Extensive destructive change is also identified at the left pedicle. **C** Contrast-enhanced T1-weighted MR image shows a multiloculated paravertebral mass extending into the spinal canal, spinous process (*arrow*), and paravertebral muscles (*arrowhead*). **D** Photomicrograph of the tumor specimen reveals the Antoni type A of growth. (H&E, $\times 100$). **E** Immunohistochemical stains for S-100 protein is positive diffusely

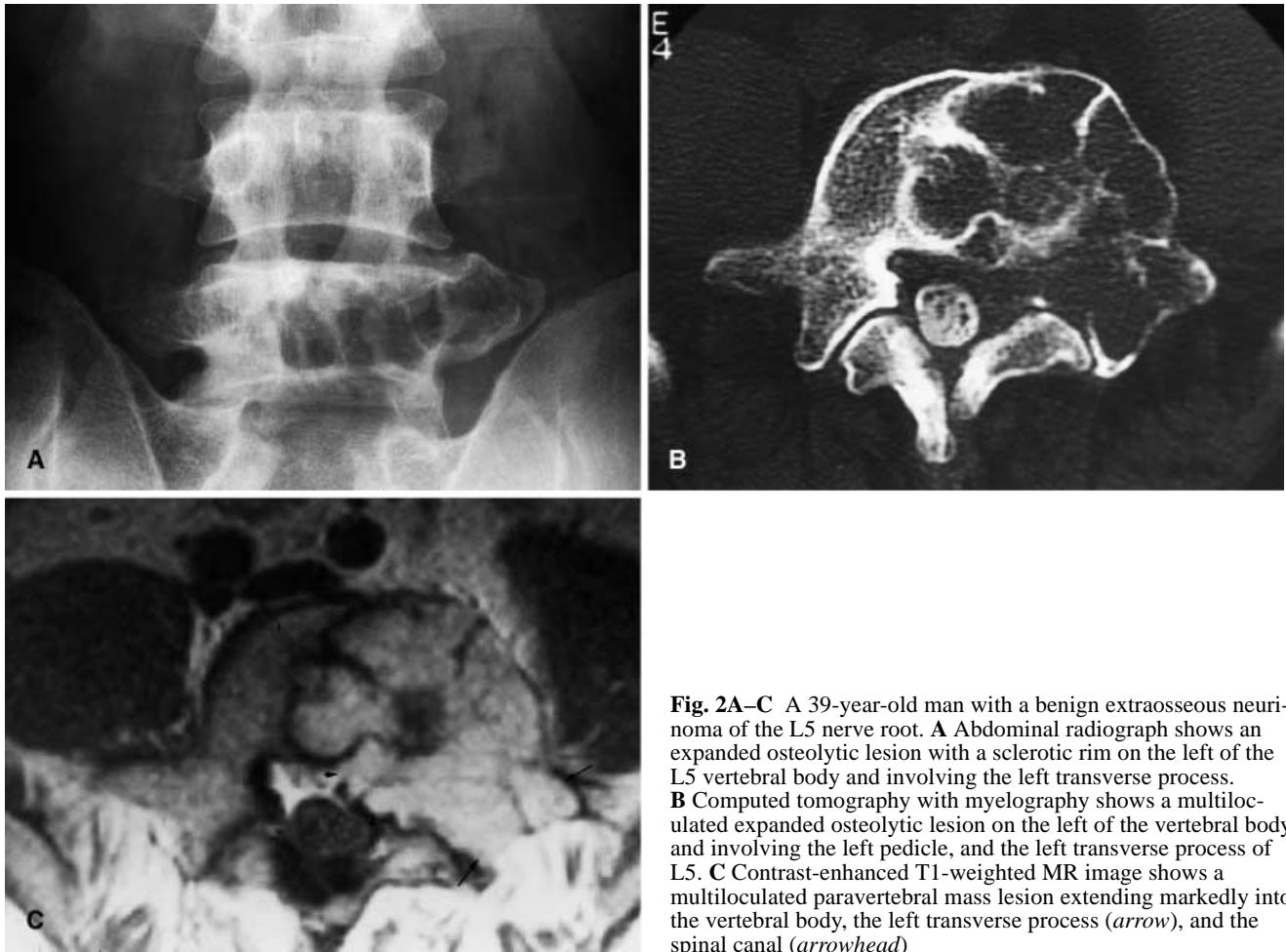


Fig. 2A–C A 39-year-old man with a benign extraosseous neuroinoma of the L5 nerve root. **A** Abdominal radiograph shows an expanded osteolytic lesion with a sclerotic rim on the left of the L5 vertebral body and involving the left transverse process. **B** Computed tomography with myelography shows a multiloculated expanded osteolytic lesion on the left of the vertebral body and involving the left pedicle, and the left transverse process of L5. **C** Contrast-enhanced T1-weighted MR image shows a multiloculated paravertebral mass lesion extending markedly into the vertebral body, the left transverse process (*arrow*), and the spinal canal (*arrowhead*)

expanding osteolytic lesion with a paravertebral mass at the T10 vertebral body and with obliteration of its left pedicle (Fig. 1A). Computed tomography (CT) showed a soft tissue mass arising at the T10/11 intervertebral foramen accompanied by enlargement of the foramen and posterior scalloping of the vertebral body. A portion of this lesion extended into the spinous process and the left transverse process of T10 forming expanding masses with thinning and partial destruction of the cortex. The dural sac was markedly compressed at this level (Fig. 1B). Magnetic resonance (MR) images revealed a multiloculated mass with a dumbbell-shaped configuration that appeared to invade the bone and paravertebral muscles (Fig. 1C).

Although a benign neuroinoma was the most likely diagnosis, we could not exclude a malignant tumor, including a primitive neuroectodermal tumor or malignant nerve sheath tumor, due to the patient's age, its fast growth rate, and associated destructive change in bone. We performed fine-needle aspiration, and pathological examination showed typical findings for neuroinoma. During surgery, these masses were found to arise from the left side T10 nerve root sheath and extend into the vertebral body, spinous process, and left transverse process. These masses were completely removed. Macroscopically, they were well-encapsulated solid tumors with characteristic Antoni A and B type patterns that were consistent with neuroinomas,

and were recognized on microscopic examination (Fig. 1D). The diagnosis of neuroinoma was established by immunohistochemistry, which showed neoplastic cells diffusely positive for S-100 protein (Fig. 1E). Following surgery, the patient had no neurological deficit and no evidence of recurrence was seen during the follow-up period of 1 year.

Case 2

A 39-year-old man presented with a history of moderate lumbar pain for 1 month. General physical examination, neurological examination, and laboratory data were insignificant. An abdominal radiograph showed an expanding osteolytic lesion with a

sclerotic rim at the left side of the L5 vertebral body and the left transverse process and obliteration of its left pedicle (Fig. 2A). CT with myelography showed remarkable enlargement of the L5/S1 intervertebral foramen and loculated posterior scalloping of the L5 vertebral body. The dural sac was compressed and displaced posterolaterally at the L5 level (Fig. 2B). MR images demonstrated a dumbbell-shaped mass at the left L5/S1 intervertebral foramen and loculated masses invading the L5 vertebral body, the left transverse process, and the paravertebral muscles (Fig. 2C). Malignant nerve sheath tumor was suspected due to the presence of invasion into the vertebral body and the adjacent soft tissues.

Fine-needle aspiration was performed and the diagnosis of benign neurinoma was pathologically established. The patient's investigation was negative for neurofibromatosis. There has been no evidence of recurrence during a follow-up period of 3 years.

Discussion

Neurinomas are most common in young and middle-aged adults, although they can occur at any age. Neurinomas can arise sporadically in patients who have no evidence of a genetic predisposition; however, morphologically identical tumors occur in patients with neurofibromatosis [1, 2]. Their clinical presentation is variable. Small neurinomas are usually asymptomatic, and clinical symptoms include localized pain or tenderness, localized swelling, or even a pathological fracture, depending on the size and site of the tumor [1].

Neurinomas, which gradually increase in size, are occasionally accompanied by pressure erosion of the adjacent bone, resulting in concave deformity of the bony surface or enlargement of the canal. Extensive destruction or erosion of the

bone is usually considered uncommon for neurinomas. Neurinomas can involve bone by three possible mechanisms: (1) an extraosseous tumor causing secondary erosion of the bone, (2) a tumor arising centrally within the bone (intraosseous neurinoma), or (3) a tumor arising within the nutrient canal and growing in a dumbbell-shaped configuration, resulting in enlargement of the canal [3]. As previously mentioned, the extensive destruction and erosion into the vertebral body in our two cases are different from typical secondary changes of neurinomas against the adjacent bone.

Recently several cases of intraosseous neurinomas have been reported [4, 5]. Intraosseous neurinomas account for less than 0.2% of primary bone tumors, which are most commonly seen in the mandible, possibly because of the long course of the sensory nerves within this bone. Other reported sites include vertebral bodies, ulnar, humerus, femur, and sacrum [3, 4, 5]. Neurinomas are essentially lytic lesions with sclerotic margins or cortical erosion without periosteal new bone formation. In intraosseous neurinomas, the histological features, although similar to those of other neurinomas, may be obscured when the lesions are highly cellular, and the Antoni type A and B patterns are subtle [3]. Generally, intraosseous neurinomas are referred to as neurinomas that primarily occupy the intraosseous region. In our two cases, a diagnosis of intraosseous neurinoma was inappropriate because the main part of the lesion was in the extraosseous paravertebral region.

We speculate that the third mechanism (a tumor extending into the nutrient canal) contributed to extensive expansion into the vertebral body in our cases. Sherman [6] detected nerves within the bone marrow of the vertebral bodies in two adult women. Chang et al. [7] described both myelinated and nonmyelinated fibers, usually associated with vessels, in bone marrow from a

human fetus. Pedersen et al. [8] also found nerve filaments arising from the recurrent branch of each spinal nerve root with their accompanying blood vessels entering the vertebral body. In our two cases, the surgical findings indicated that the main parts of the lesions were extraosseous and were considered to originate in a spinal nerve root. Expansion into the vertebral body may represent growth of a neurinoma along the branch of the spinal nerve root entering the vertebral body through the nutrient canal. We found only one case report in the English literature in which an extraosseous neurinoma destroyed and expanded the vertebral body [9]. Although it is an extremely rare manifestation, there should be awareness that an extraosseous spinal nerve root neurinoma may expand into the vertebral body through the nutrient canal.

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