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MR Appearance of an Ectopic Intraspinal Adrenal Cortical Adenoma

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An ectopic adrenal cortical adenoma presented as an intradural spinal mass in an 8-year-old girl with bilateral leg pain. The patient had no endocrine symptoms of an adrenal adenoma despite abnormally high levels of androstenedione found within the tumor. The purpose of this article is to highlight the diagnostic radiologic aspects of this unusual neoplasm.

The adrenal cortex develops from the coelomic mesoderm in the fourth to sixth week of life as a cluster of cells between the root of the mesentery and the genital ridge [1]. Aberrant adrenal cortical tissue can migrate with gonadal tissue and has been described in a subcapsular location within the testes and ovaries [2]. Adrenal rest tumors have been described in the testes, particularly in patients with endocrine disturbances associated with increased levels of adrenocorticotrophic hormone [3]. Sites of adrenal cortical ectopia also include the mediastinum, retroperitoneal space, pancreas, and kidney [4, 5]. There are two reports of intracranial adrenal glands containing both cortical and medullary tissue [6, 7]. However, ectopic adrenal cortical tissue with or without neoplasia within the spinal canal has not been reported before. Spinal ectopic adrenal tissue has been reported in a patient with spinal dysraphism [8].

Case Report

An 8-year-old girl presented with a 3-week history of bilateral leg pain that radiated down the posterior aspect of her thighs. The pain had increased in severity during the preceding 3 weeks, and at the time of presentation, she was unable to lie down or walk because of the discomfort. She denied weakness, numbness, or tingling. Bowel and bladder function were maintained. Physical examination revealed a well-developed child with rigid paraspinal and lower extremity musculature but without neurologic deficits or evidence of spinal dysraphism. Standard laboratory evaluation showed no significant abnormalities.

Radiologic Evaluation

MR imaging of the lumbar spine was performed with a 1.0-T superconducting magnet (Siemens Magnetom, Iselin, NJ) and a 30-cm surface coil. All images were acquired with a 256×256 matrix. Precontrast T1-weighted sagittal 500/17/2 (TR/TE/excitations), axial 1000/17/1, and T2-weighted sagittal 2500/25,90/1 images were obtained by using standard spin-echo technique. After IV administration of gadopentetate dimeglumine (0.1 mmol/kg), sagittal and coronal T1-weighted (500/17) images were obtained.

All imaging sequences revealed a well-circumscribed, intraspinal mass measuring 1.2 cm anteroposteriorly by 1.3 cm transversely by 2.0 cm vertically, occupying the posterior intradural compartment at the L2 level. On precontrast T1-weighted images, the signal intensity of the mass was slightly less than that of spinal cord soft tissue (Fig. 1A). The signal was inhomogeneous with areas of decreased intensity inferiorly. The mass was moderately hyperintense relative to liver and moderately hypointense relative to subcutaneous fat. T2-weighted images also showed an inhomogeneous signal from the mass with an intensity similar to that of spinal cord soft tissue (Fig. 1B). Nerve roots were stretched ventrally over the mass. After IV contrast administration, T1-weighted images showed marked, slightly heterogeneous enhancement of the mass (Fig. 1C).

Pathologic Findings

The circumscribed, well-encapsulated nature of the mass made complete surgical removal possible. The mass was tethered at both poles by a vascular stalk, and complete removal was accomplished without damage to the cauda equina (Fig. 2A). After pathologic evaluation revealed a steroid-producing tumor of adrenal cortical origin, further blood analysis showed no endocrine abnormalities. Specifically, serum cortisol, androstenedione, DHEAS, estrogen, and pro-

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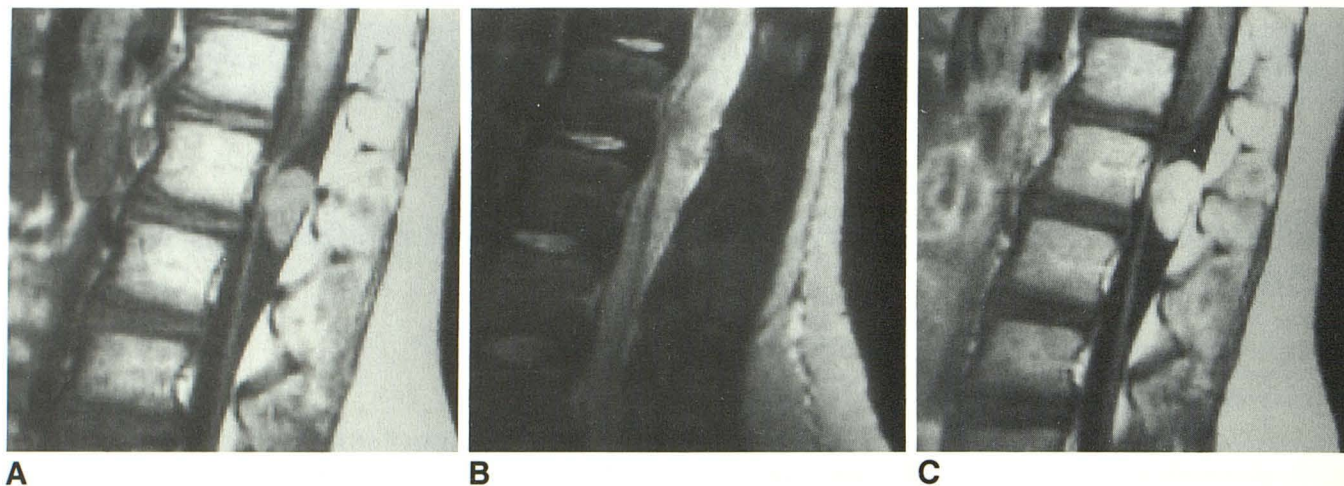


Fig. 1.—A, T1-weighted (500/17) sagittal MR image shows a well-defined intradural mass with an inhomogeneous signal intensity, slightly hypointense to spinal cord soft tissue.

B, T2-weighted (2500/90) sagittal MR image shows mass to be inhomogeneous.

C, Postcontrast T1-weighted (500/17) MR image shows marked enhancement of mass, reflecting its high vascularity.

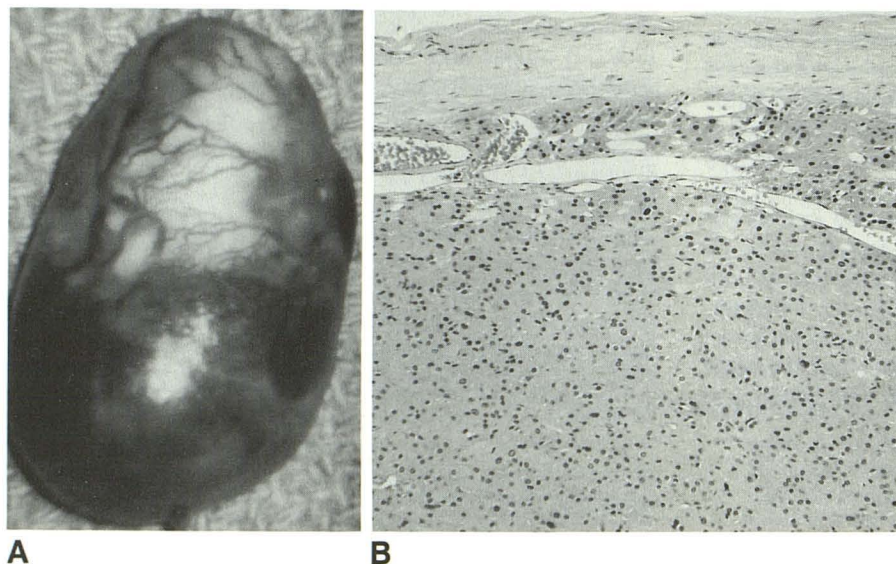


Fig. 2.—A, Gross surgical specimen shows tortuous vessels within capsule. Whitish areas are patchy capsular calcifications.

B, Histologic section shows cells with pale, eosinophilic cytoplasm and numerous dilated, thin-walled blood vessels. Histologic pattern is typical of adenomatous adrenal cortical tissue. (H & E, $\times 200$)

gesterone levels were all within normal limits. Postoperative CT of the abdomen showed normal adrenal glands without evidence of a mass lesion.

The light microscopic appearance was typical of adenomatous adrenal cortical tissue with tumor cells showing pale, eosinophilic cytoplasm and areas of marked vascularity (Fig. 2B). Portions of the tumor showed thin-walled capillaries with sinusoidal dilatation. The mass was surrounded by a fibrous, vascular capsule. Capsular and deep calcifications were identified. Electron microscopy showed features of a steroid-producing tumor.

Radioimmunoassay from frozen portions of the surgical specimen showed the tumor to contain 18 times the normal serum level of androstenedione. Cortisol or aldosterone levels in the tumor were not elevated. The pathologic features were

consistent with a benign tumor. Adrenal medullary tissue was not encountered.

Discussion

MR evaluation of this patient showed a markedly enhancing, slightly inhomogeneous intradural mass with associated stretching of nerve roots. Overall signal characteristics were similar to those of spinal cord soft tissue, and the appearance was most suggestive of a tumor of neural origin such as a neurofibroma, an ependymoma, or a metastasis from an intracranial neoplasm.

The MR appearance correlated well with the gross pathology of the mass, which showed it to be well encapsulated

with areas of marked vascularity and calcification. These characteristics probably accounted for the heterogeneous MR signal. Significant contrast enhancement reflected the vascular nature of the mass. The tumor contained androstenedione at more than 18 times the normal serum level. Despite this, the patient showed no clinical virilization, indicating that the tumor cells were not releasing the steroid into the circulation. Laboratory analysis showed no abnormalities in blood levels of adrenal steroids. Thus, the mass, although hyperfunctional, was nonsecretory.

The appearance of the mass on MR imaging is consistent with the reported characteristics of hyperfunctioning adrenal cortical adenomas involving normally situated adrenal glands. Adrenal adenomas, found incidentally in 10% of the population at autopsy, are generally small with signal intensities equal to or slightly greater than that of liver. Hyperfunctioning adenomas such as this one have been reported to show higher signal intensity than liver does on both T1- and T2-weighted images [9].

This spinal, intradural adrenal cortical adenoma most likely developed from ectopic intraspinal adrenal cortical cell rests. As ectopic cell rests have been described in various areas of the retroperitoneum, some cells may become isolated within the spinal canal during fetal development by traveling with the adventitia of an ingrowing segmental lumbar artery or by following the sheath of an exiting nerve. Ectopia within the spinal canal has not been reported, although intracranial adrenal tissue has been identified on at least two occasions. We think it unlikely that the mass was a metastasis from an adrenal carcinoma in light of its benign appearance histologi-

cally and the normal appearance of the adrenal glands on CT scans. It is further noted that intraspinal metastases of adrenal cortical carcinomas have not been reported.

This case introduces a new consideration in the differential diagnosis of intraspinal, intradural masses. In patients without symptoms of adrenal abnormality, the diagnosis would be difficult to predict, since the MR appearance is not distinctive. However, in the proper clinical setting and with appropriate MR characteristics, the diagnosis might be suggested. Further, in a patient with symptoms of adrenal steroid overproduction but with no demonstrable adrenal lesion, evaluation of the spinal canal may be justified.

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