

SPINAL STENOSIS CAUSED BY EPIDURAL LIPOMATOSIS IN CUSHING'S SYNDROME

STEPHEN J. LIPSON, M.D.,
MOHAMMAD H. NAHEEDY, M.D.,
MICHAEL M. KAPLAN, M.D.,
AND DON C. BIENFANG, M.D.

CENTRIPETAL fat deposition is a well known clinical feature of excessive endogenous or exogenous adrenal glucocorticoids. We describe an unusual case of central spinal stenosis caused by excessive epidural fat in a patient with Cushing's syndrome secondary to prolonged therapy with high doses of glucocorticoids. Only one other case report of this phenomenon could be located in the medical literature.¹

CASE REPORT

A 53-year-old woman with Graves' disease, euthyroid after radioiodine therapy, had infiltrative ophthalmopathy with optic-nerve involvement. When she was treated with prednisone, 150 mg per day, cushingoid features developed rapidly. Two months after the initiation of therapy, with the prednisone tapered to 100 mg per day, the patient reported a low-back ache that radiated to the buttocks and thighs. The pain worsened over months, was exacerbated by walking, and showed a claudication pattern. After six months the prednisone had been tapered to 50 mg per day, and the patient was able to walk only a few steps. She could sit comfortably, could extend her spine to only 20 degrees of flexion with further flexion to 80 degrees, and could tolerate the supine position only very briefly. Straight leg raising reached 90 degrees bilaterally. The lumbar spine was nontender, and the sciatic notches and nerves were tender to palpation. The findings of neurologic examination of the lower extremities were unremarkable except for the absence of deep-tendon reflexes in spite of reinforcement.

Electromyographic findings in the lower extremities included chronic, bilateral denervation in the L4 and L5 distribution. Radiographs of the lumbosacral spine revealed diffuse osteopenia without evidence of collapse. A lumbar metrizamide myelogram revealed a complete extradural block at L4-5 with ventral indentation at the disk space and a prominent epidural space posterior to the body of L4 (Fig. 1). Computed tomography of the lumbar spine failed to reveal metrizamide below L4-5. The bony canal and foramina were of normal dimensions and the intracanal soft-tissue density measured -80 to 0 Hounsfield units, indicative of fat density.

A lumbar bilateral laminectomy was performed at the L4 and L5 levels. Copious epidural fat herniated through the incision in the ligamentum flavum. Abundant epidural fat was found throughout the area exposed. The L4-5 disk appeared normal and the L5-S1 disk had a diffuse degenerative bulge. Histologic examination of the fat revealed normal adult adipose tissue. After operation, the patient had immediate relief of buttock and thigh pain and was able to walk, to extend her spine fully, and to lie supine.

DISCUSSION

Centripetal fat deposition in Cushing's syndrome most commonly occurs in the subcutaneous areas of the face, neck, and trunk.² Mediastinal fat deposition in Cushing's syndrome is also well known³ and can be easily demonstrated by means of computed tomogra-



Figure 1. Metrizamide Lumbar Myelogram Demonstrating the Complete Extradural Block at L4-5 (Arrow).

phy.⁴ In this case, computed tomography revealed the epidural fat in the lumbar canal and served as a valuable adjunct to myelography.

Lumbar central spinal stenosis, presenting with symptoms of spinal claudication, is caused by lumbar-root entrapment dictated by the anatomy of the bony spinal canal, neural foramina, and intervertebral disks.⁵ The epidural fat in stenotic segments is usually diminished or absent, and excessive epidural fat as the cause of stenosis is most unusual. In these patients, epidural lipomatosis almost certainly is a manifestation, although rare, of glucocorticoid excess and should be considered in the differential diagnosis of spinal stenosis.

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From the departments of Orthopedic Surgery, Radiology, Medicine, and Ophthalmology, Harvard Medical School, and Peter Bent Brigham Hospital, Boston, MA (address reprint requests to Dr. Lipson at 721 Huntington Ave., Boston, MA 02115).