Symptomatic Deposition of Epidural Fat in a Morbidly Obese Woman

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This case is reported as a medical rarity. We describe a morbidly obese female (without Cushing syndrome) in whom the excessive deposition of normal epidural fat in the spinal canal caused spinal cord compression.

Case Report

A 33-year-old hypertensive, diabetic woman had a 6 year history of progressive lower extremity weakness. More recently she had developed dysesthetic pains in both lower extremities associated with urinary and fecal incontinence.

Physical examination revealed a grossly obese female weighing 174 kg. Normal motor and sensory function was noted in the upper extremities, but flaccid paralysis and areflexia were evident in both lower extremities. There was also fasciculation of the quadriceps femoris muscle bodies in response to direct percussion. Proprioception and vibratory sensation were absent below the anterior superior iliac spines, and there was pinprick sensory demarcation at the T6 dermatome. Minimal anal sphincter tone persisted.

A Pantopaque myelogram revealed complete block at T9 associated with dorsal extradural impression. Air (5 cm³) was introduced into the lumbar subarachnoid space to force some of the contrast material cephalad beyond the obstruction [1], after which the cephalic end of the extradural lesion was localized at T3 (fig. 1). It was apparent that there was a dorsally situated extradural mass but its exact nature was indeterminate, the thought being that it might represent an epidural lymphomatous infiltrate.

Thoracic laminectomy extending from T1 to T10 exposed a thick layer of normal-appearing nonencapsulated epidural fat measuring 5 mm at its greatest depth. This layer thinned considerably at the upper and lower extremes of the exposed epidural space, but where the fat was thickest it appeared to significantly compress the dura

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Fig. 1.—A, Supine thoracic myelogram, frontal view. Compression of Pantopaque column from T3 to T9. B, Myelogram, lateral view, patient prone, head elevated. Obstruction does not present as abrupt cutoff because (as in A) line of obstruction is oblique rather than transverse, mass extending further cephalad on patient's right side. C, Myelogram, lateral view, patient prone, head down. Ventral displacement of spinal cord.
and its contents. Dural incision exposed arachnoidal thickening and the underlying spinal cord appeared atrophic.

Postoperatively the patient was unable to maintain adequate ventilation without mechanical assistance. After several cardiopulmonary arrests and having in the interval displayed no neurologic changes, she died 75 days after operation. Permission for autopsy was not granted.

Discussion

Neurologic deficit secondary to fat deposition in the epidural space is a rare complication of corticosteroid therapy [2–6]. Epidural lipomas have been described by various authors as well demarcated, usually highly vascular neoplasms which do not resemble normal fat [7–10]. The possibility that these neoplasms might be related to obesity has been suggested, but correlation has been vague at best. In our case, for which there is no precedent in the literature, dural compression was apparently secondary to gradual overgrowth of epidural fat as a by-product of obesity per se.

REFERENCES