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Value of Metrizamide CT in the Demonstration of Spinal Arachnoid Cysts

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Spinal arachnoid cyst presents a diagnostic challenge to clinical neurologists as well as radiologists. Although not an infrequent cause of progressive spastic paraparesis in the young and middle-aged, it is often overlooked in the differential diagnosis of progressive myelopathy. This is of concern in that early diagnosis and resection of these benign lesions could prevent progressive and irreversible damage to the spinal cord.

Diagnostic evaluation of spinal arachnoid cysts has evolved with advent of newer technologies. Currently, water-soluble myelography is widely advocated, yielding less morbidity from arachnoidal scarring, which has often been associated with oily contrast media—especially when trapped by or within the cyst [1].

Vonofakos et al. [2], however, argue in favor of oily contrast material, contending that metrizamide myelography could fail to demonstrate an arachnoid cyst owing to the homogeneous dilution of the contrast medium within the cyst and the subarachnoid space. Along with others, they emphasized the efficacy of oily contrast material with use of prone, supine, and erect positions [3, 4].

We report a case in which both Pantopaque and metrizamide myelograms failed to demonstrate a spinal arachnoid cyst that was subsequently delineated by metrizamide computed tomography (CT). Metrizamide CT appears to be the most sensitive method for evaluating the spinal arachnoid cysts.

Case Report

A 54-year-old Hispanic woman was admitted to Beth Israel Medical Center after 8 months of progressive weakness and stiffness of the lower extremities and occasional numbness and tingling of the right leg. For several weeks before admission, the patient had urinary frequency and dribbling incontinence. She denied having back pain, arthralgia, myalgia, or systemic symptomatology.

The patient had normal growth and development until age 8, at which time she had an insidious onset of progressive spastic paraparesis. Dorsal laminectomy performed in Puerto Rico at age 10 (records not available) resulted in gradual functional recovery, and the patient remained in good health until her present illness. The patient denied history of trauma or infection. Family history was negative for neurologic disorders.

General physical examination revealed a mild kyphoscoliosis of the dorsal spine and minimal atrophic changes of the lower extremities. Neurologic examination showed a spastic paraparesis, greater in the left lower extremity than in the right, with bilateral extensor plantar responses and a T6 sensory level to pin and temperature on the right side. Position sense was impaired in the toes, with vibratory loss to the superior iliac crest on the left and to the knees on the right side.

Cerebrospinal fluid (CSF) obtained by lumbar puncture was clear and colorless under initial pressure of 150 mm H_2O with a normal Queckenstedt maneuver. Total protein, 26 mg/dl; glucose, 62/dl; lymphocytes, 2/mm^3; VDRL, nonreactive. All laboratory tests for infectious and collagen disease were negative. Radiographic examination of the spine confirmed a mild kyphoscoliosis of the dorsal spine. Pantopaque thoracic myelography demonstrated findings suggestive of arachnoid adhesions extending from T6 to T8 (figs. 1A and 1B). She was discharged 2 weeks later with a presumptive diagnosis of arachnoiditis of unknown etiology.

The patient's gait disturbance progressively worsened and she was readmitted after 2 weeks. A metrizamide myelogram (figs. 1C and 1D) was obtained, with findings resembling the earlier Pantopaque study. About 2 hr later, metrizamide CT (fig. 2) of the dorsal spine (5 mm thickness at 10 mm intervals) was obtained and demonstrated an enlarged extradural arachnoidal cyst extending from T6 to T8 with compression of the spinal cord. Surgical excision of the arachnoidal cyst was performed. The dura mater was defective posteriorly and the anterior wall of the cyst was adherent to the pia mater with obliteration of the subarachnoid space. Identification of the communication between the arachnoid cyst and the subarachnoid space was impossible. Histologically, there was fibrous thickening of the pia mater and arachnoid membrane with an inflammatory process. Postoperatively, the patient's neurologic condition was stabilized, though spastic paraparesis persisted.

Discussion

Spinal arachnoid cysts, although rarely symptomatic, are probably more common than the literature indicates. These cysts are characterized by an insidious, often intermittent

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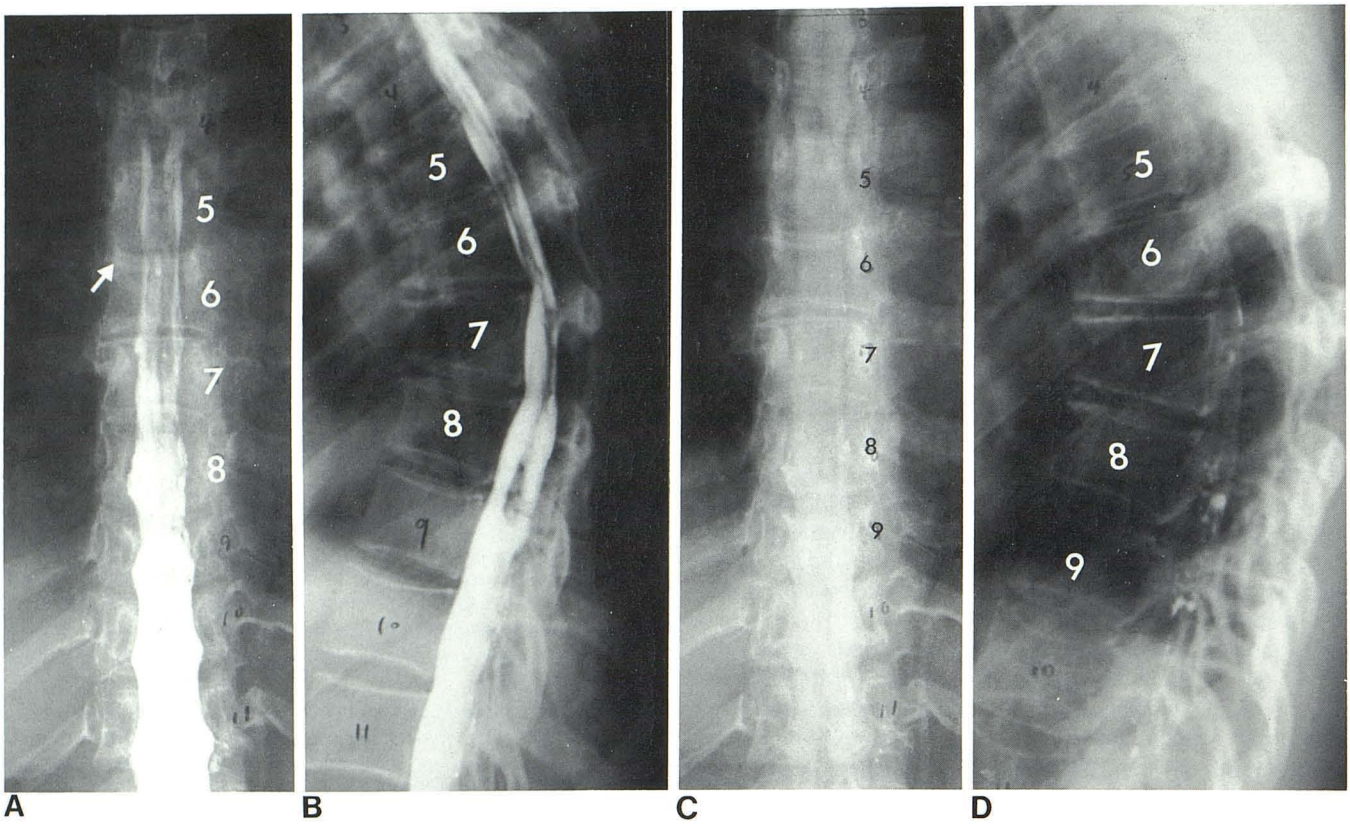

Fig. 1.—Anteroposterior (**A**) and lateral (**B**) Pantopaque thoracic myelograms with patient supine. Pantopaque column is irregular at T6–T8 level, suggestive of arachnoid adhesion. On **B**, it is slightly narrow and distance between contrast column and posterior margin of spinal canal is widened at same level. Note slight thinning of right pedicle (*arrow*) of T6 vertebra and increased interpedicular distance at same level. No Pantopaque-filled arachnoid cyst is evident. Anteroposterior (**C**) and lateral (**D**) metrizamide thoracic myelograms 2 months later show same findings as on the Pantopaque study.

course of widely diverse symptoms and signs of myeloradiculopathy secondary to compression or traction on the spinal cord and nerve roots. It is not unusual for the diagnosis of intraspinal arachnoid cysts to be overlooked while the clinician considers other possibilities [5]. The protean nature and the difficulty of demonstrating these lesions on routine myelography pose a diagnostic problem as reported in our case.

The nomenclature used for various types of spinal arachnoid cysts is sometimes confusing [6]. However there is general agreement that the lesions can be simply classified as extradural and intradural cysts based on their relation with the dura mater [6–10]. The perineural cysts that occur along the spinal nerve roots are usually considered a separate entity [8]. There has been extensive discussion about the pathogenesis of the spinal arachnoid cysts, and many theories have been proposed. Intradural cysts are thought to result from uneven distribution of arachnoid trabeculae [11], enlargement of small diverticula in septum posticum [12], and arachnoiditis [9]. In many instances trauma was cited as the predisposing factor in the creation of intradural cysts [7, 11]. Elsberg et al. [13] were the first to report extradural cysts with spinal cord compression and postulated the congenital origin of these cysts. They believed that the extradural cyst originated as a congenital diverticulum of the dura mater or as a herniation of the arachnoid membrane through a defect in the dura mater. Since then observations by others have led to inclusion of laminectomy [14], multiple lumbar puncture, closed injury to the spine [15], and disk surgery [9] as possible etiologic factors. Our case, as confirmed by surgery, was an extradural cyst and its origin may date back to the previous surgery 44 years earlier.

Extradural cysts, predominantly at the thoracic level, are usually large, posterior to the dura mater, and extend the length of several vertebrae. Frequently they produce bony changes as follows: erosion of the pedicles, enlargement of the interpedicular distance, increase in the sagittal diameter of the spinal canal, scalloping of the posterior surface of the vertebral bodies, and thinning of the laminae. However, these findings are not specific and may be seen in other intraspinal mass lesions. In the previous reports, myelography was almost always used before surgery, and there usually existed complete or incomplete block at the cyst level [7]. Most of the case reports lacked precise description of myelographic findings. In their review, Nugent et al. [15] could only find 12 of 45 reported cases to have communication between the cysts and the subarachnoid space. Swanson and Fincher [16] documented an arachnoid cyst developed after laminectomy and found no communication

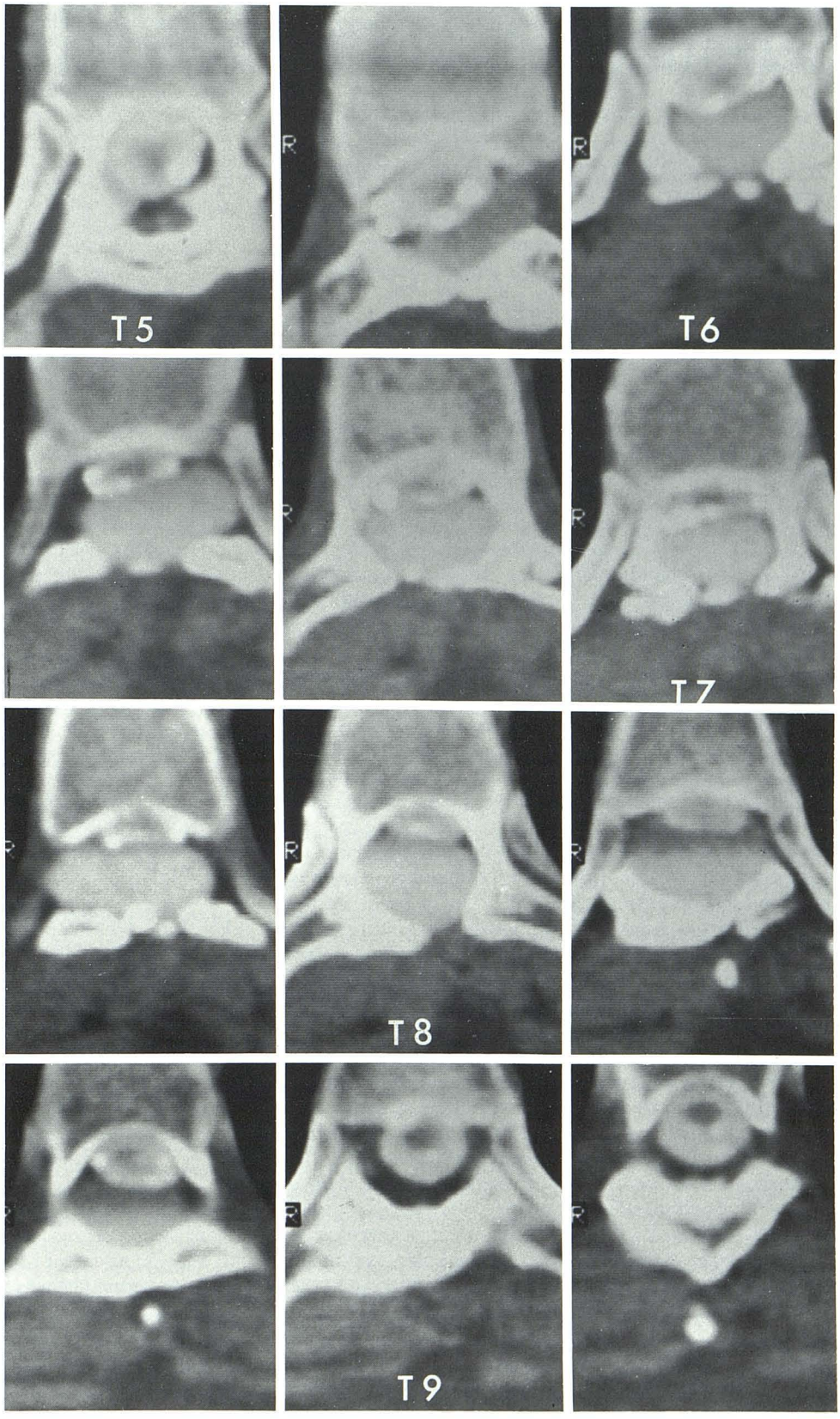

Fig. 2.—Postmyelographic CT scans. Multiple transverse axial images in numeric sequence from T5 to T9. Large arachnoid cyst filled with metrizamide occupies posterior part of spinal canal at T6–T8 level. Spinal cord at same level is compressed, flattened, and atrophic.

with the subarachnoid space when the cyst was inadvertently injected with the contrast medium at myelography. A similar case was described later by Smith and Chavez [17].

Correct diagnosis would be impossible if the cysts are not filled with the contrast medium. The adoption of myelography in prone, supine, and erect positions [3] may help in demonstrating successfully the contrast filling of the cysts as described in later reports [2, 5, 7, 9]. Nevertheless, the extradural cyst reported by Lee and Cancina [4] still failed to show contrast filling in the cyst, and the diagnosis was only made at the time of surgery.

There has been a tendency in the literature [7, 9, 10, 18] to divide the spinal arachnoid cysts into two different categories depending on their relation with the subarachnoid space: (1) communicating cyst or "arachnoid diverticulum" when there is communication between the arachnoid cyst and the subarachnoid space and (2) noncommunicating cyst or "true cyst" when no communication exists. Lombardi and Morello [7] believed that the arachnoid cysts in early stage always communicate with the subarachnoid space but are later secluded from them, accounting for later appearance of symptoms when the cyst enlarged or exerted pressure against the spinal cord. Gortvai [19] attributed valvular mechanism, osmotic attraction of the fluid into the cyst, and secretion of cyst wall for the enlargement of the cyst. Taveras and Wood [20] used the term "occult meningocele" synonymously to indicate a "true cyst" and suggested that when the opening of a meningocele is obstructed the isolated pocket becomes a "true cyst." However, as demonstrated here, with the use of metrizamide CT, the so-called noncommunicating or "true cyst" may have to be redefined.

The diagnosis in our case was made preoperatively by metrizamide CT. We postulate that Pantopaque myelography failed to demonstrate the cystic lesion owing to the extremely narrow communication between the cyst and the subarachnoid space that prevented the viscous contrast medium from passing through. Failure of metrizamide myelography was presumably due to the fact that after mixing with the CSF, metrizamide was of suboptimal density in the arachnoid cyst. This explanation can also be applied in the case reported by Vonofakos et al. [2] in which metrizamide myelography failed to show the arachnoid cyst and no CT study was pursued. In our case, although the CT study was carried out about 2 hr later, it would still be doubtful that radiography of the thoracic spine at that time would have demonstrated contrast filling in the cyst. As has been known, CT is very sensitive for soft-tissue densities and therefore is more efficacious in demonstrating an arachnoid cyst, even when it contains diluted contrast medium. Furthermore, the anatomic relation of the arachnoid cyst to the meninges and the vertebrae can be distinctly outlined. Thus, it seems appropriate to stress the importance of metrizamide CT in the assessment of spinal arachnoid cysts as it is the definitive study in providing an accurate diagnosis.

REFERENCES

1. Baker A, Strand R. Congenital spinal disorders. In: Wolpert SM, ed. *Central nervous system approaches to radiologic diagnosis.* New York: Grune & Stratton, **1979**:163–189
2. Vonofakos D, Grau H, Steudel W. Multiple arachnoid cysts: the role of oily contrast medium. *Surg Neurol* **1981**;15:125–127
3. Teng P, Rudner N. Multiple arachnoid diverticula. *Arch Neurol* **1960**;2:126–134
4. Lee MG, Cancina JE. Extradural spinal arachnoid cysts. *West Indian Med J* **1980**;29:67–72
5. Raja IA, Hankinson J. Congenital spinal arachnoid cysts. *J Neurol Neurosurg Psychiatry* **1970**;33:105–110
6. Beehar M, Beks JWF, Penning L. Intradural arachnoid cysts with scalloping of vertebrae in the lumbosacral region. *Act Neurochir (Wien)* **1972**;26:275–283
7. Lombardi G, Morello G. Congenital cysts of the spinal membranes and roots. *Br J Radiol* **1963**;36:197–205
8. Tarlov IM. Spinal perineural and meningeal cysts. *J Neurol Neurosurg Psychiatry* **1970**;33:833–843
9. Hoy RJ, Faulder KC. Spinal arachnoid cysts. *Australas Radiol* **1968**;12:344–354
10. Fortuna A, LaTorre E, Ciappetta P. Arachnoid diverticula: a unitary approach to spinal cysts communicating with the subarachnoid space. *Acta Neurochir* (Wien) **1977**;39:259–268
11. Teng P, Papatheadorou C. Spinal arachnoid diverticula. *Br J Radiol* **1966**;39:249–254
12. Perret G, Green D, Keller J. Diagnosis and treatment of intradural arachnoid cysts of the thoracic spine. *Radiology* **1962**;79:425–429
13. Elsberg CA, Dyke CG, Brewer ED. The symptoms and diagnosis of extradural cysts. *Bull Neurol Inst NY* **1934**:3:395–417
14. Hyndman OR, Gerber WF. Spinal extradural cysts, congenital and acquired. Report of cases. *J Neurosurg* **1946**;3:474–486
15. Nugent GR, Odom GL, Woodhall B. Spinal extradural cysts. *Neurology* (NY) **1959**;9:397–406
16. Swanson HS, Fincher EF. Extradural arachnoid cysts of traumatic origin. *J Neurosurg* **1947**;4:530–538
17. Smith GW, Chavez M. Lumbar extradural cysts—congenital. *Arch Neurol Psychiatry* **1958**;80:436–440
18. Ü KS, Baloh RW, Weingarten S. Intradural arachnoid cyst presenting with cord compression. *Bull Los Angeles Neurol Soc* **1972**;37:178–183
19. Gortvai P. Extradural Cysts of the Spinal Canal. *J Neurol Neurosurg Psychiatry* **1963**;26:223–230
20. Taveras JM, Wood EH. *Diagnostic neuroradiology*, vol 2, 2d ed. Baltimore: Williams & Wilkins, **1976**:1144–1150