Progressive Posttraumatic Cystic Myelopathy:
Neuroradiologic Evaluation

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The neuroradiologic evaluation and findings in 25 symptomatic patients with surgically proven progressive posttraumatic cystic myelopathy are reviewed. To follow patients with spinal cord injury, neuroradiologic algorithms were developed to confirm and define cystic myelopathy. The algorithm used in the early and mid 1970s relied on the myelographic demonstration of a large cord for suspicion of a cyst. Review of this material found that in progressively symptomatic patients 14 of 25 proven cysts were in large cords. A more recent algorithm used computed tomographic metrizamide myelography. In nine of 11 patients studied in this fashion, the cyst filled with contrast material 2–4 hr after injection, yet it did not communicate with the subarachnoid space at subsequent surgery. The origin of the cyst fluid and mechanism of cyst demonstration with metrizamide may be associated with transneural migration of fluid. This condition must be clinically suspected and radiologically confirmed for surgical treatment (cyst-shunt procedure) if neurologic preservation of function is to be maintained.

Progressive posttraumatic cystic myelopathy is an uncommon, but well documented complication of spinal cord injury. In a 1973 monograph, Barnett and Jousse [1] reviewed their findings in 17 cases and reported an additional 56 cases from the literature, but there has been little reference to this condition in neuroradiologic literature. Barnett and Jousse [1] reported that the posttraumatic cysts in their series did not communicate with subarachnoid space, central canal, or fourth ventricle, and that patients with this disorder may have progressive ascent of their level of loss of residual neurologic function. Recently, Edgar [2] described a shunt procedure for the cyst that was successful in arresting this progression. Technical advances in neuroradiologic evaluation methods, that is, the availability of both water-soluble contrast material for myelography and computed tomography (CT), have changed the workup of clinically suspected patients. Our review was designed to tabulate the neuroradiologic findings in a series of patients with posttraumatic myelopathy from Craig Hospital and Swedish Medical Center in Englewood, Colo. and to record a new unexplained observation: that the posttraumatic cyst fills with contrast material at metrizamide CT.

Materials and Methods

Craig Hospital admits about 350 new patients with spinal cord injury each year and about 500 more for follow-up evaluation. Neuroradiologic studies in 25 patients with surgically proven posttraumatic cystic myelopathy were reviewed. All 25 patients had symptoms and signs of progressive posttraumatic cystic neuropathy that prompted their evaluation and subsequent treatment. All were operated on by one of the authors (R. E. E.) with a technique described elsewhere consisting of a cyst shunting procedure [2]. Neuroradiologic studies reviewed included spine radiography, tomography, myelography with air or positive contrast agents (Pantopaque or metrizamide), CT of the spine with or without metrizamide, diagnostic percutaneous cyst punctures, and cystography with positive contrast injection (Pantopaque). Air myelography by a cervical approach was used in patients in whom a total block

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had been demonstrated with Pantopaque from the lumbar approach. When metrizamide became available, cervical myelography with this agent was used in concentrations of 180–190 mg I/ml using 6–12 ml via the C1–C2 lateral approach with horizontal beam fluoroscopy.

CT was performed with a variety of scanners including the EMI 5005, GE 8800, and Ohio Nuclear Delta CT machines. Most of the CT scans were obtained after cervical myelography with metrizamide. The patient was maintained in the supine 30° head up position, and the scans were most often obtained 3–4 hr later; however, because of transportation or technical CT delays, sometimes the scans were obtained as early as 2 or as late as 5 hr. Slice thickness was 8–13 mm and reconstruction matrix images varied from 256 × 256 to 320 × 320. One patient underwent sequential CT study of a previously demonstrated cyst at 30 min intervals up to 5 hr after installation of the metrizamide via a C1–C2 puncture.

The neuroradiologic findings specifically reviewed were: cord size (defined at myelography); adhesions of the spinal cord; partial or complete block to the flow of contrast material; success of diagnostic cyst puncture and cystography; cyst morphology including size, extent, and location; and the identification of the cyst at CT. The diagnostic sequence of studies and method of diagnosis were reviewed and tabulated and operative findings were also reviewed, specifically for evidence of extension or connection of the cyst to the subarachnoid space.

Results

In 21 of 25 patients, the neuroradiologic workup confirmed the clinical suspicion of progressive posttraumatic cystic myelopathy and shunt surgery was performed. In four patients the neuroradiologic findings were inconclusive or normal, but because of progressive clinical findings and the high suspicion of a progressive posttraumatic cyst, surgery was performed and a cyst was found and shunted.

TABLE 1: **Myelographic Findings with Proven Progressive Posttraumatic Cystic Myelopathy**

Finding	No. Patients (n = 25)
Large cord	14
Normal size cord	11
Partial block to contrast flow	11
Complete block to contrast flow	14
Cord adhesions	25

Plain Films

Spine radiography and tomography did not contribute to the diagnosis of progressive posttraumatic cystic myelopathy, other than by localizing the original injury site.

Myelography

All patients underwent myelography (tables 1 and 2). Seventeen Pantopaque myelograms were obtained via the lumbar route, 11 metrizamide cervical myelograms via the C1–C2 approach, and five air cervical myelograms via the C1–C2 approach (table 2). A partial block was seen in 11 patients and a complete block was noted in 14 (table 1). Adhesions of the spinal cord were evident in all 25 patients

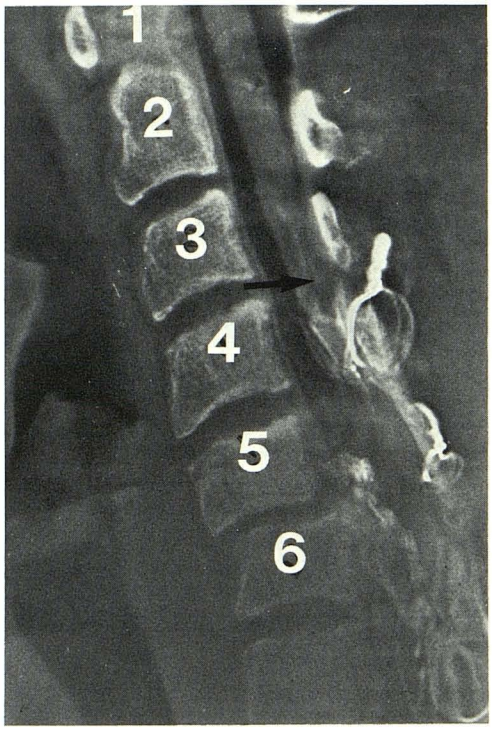


Fig. 1.—Lateral view (xerotomography) air myelogram. Posterior adhesion of cord (*arrow*). Patient had previous laminectomy and posterior wiring for stabilization.

TABLE 2: **Myelographic Findings by Route in 25 Patients with Proven Progressive Posttraumatic Cystic Myelopathy**

Medium, Route	No. Examinations*	Myelography		Cyst Location			Cord Size		Original Injury Level (Block Location)	
		Total Block	Partial Block	Cervical	Cervico thoracic	Thoracic	Normal§	Large	Cervical	Thoracic
Pantopaque, lumbar	17	8	9	12	4	1	7	10	15	2
Air, C1–C2†	5	5	...	5	...	...	4	1	5	...
Metrizamide, C1–C2‡	11	8	3	8	1	2	7	4	9	2

* Five patients had Pantopaque followed by air myelography; three had Pantopaque followed by metrizamide myelography.

† One patient had enlarged cord on air myelography; the cord was of normal size at the site of the block on Pantopaque myelography.

‡ Three patients had Pantopaque and metrizamide myelography. Two had normal cord size on Pantopaque and metrizamide myelography and one had increased cord size with both techniques.

§ Cyst diagnosis was made by cyst puncture, metrizamide cyst filling, or surgery.

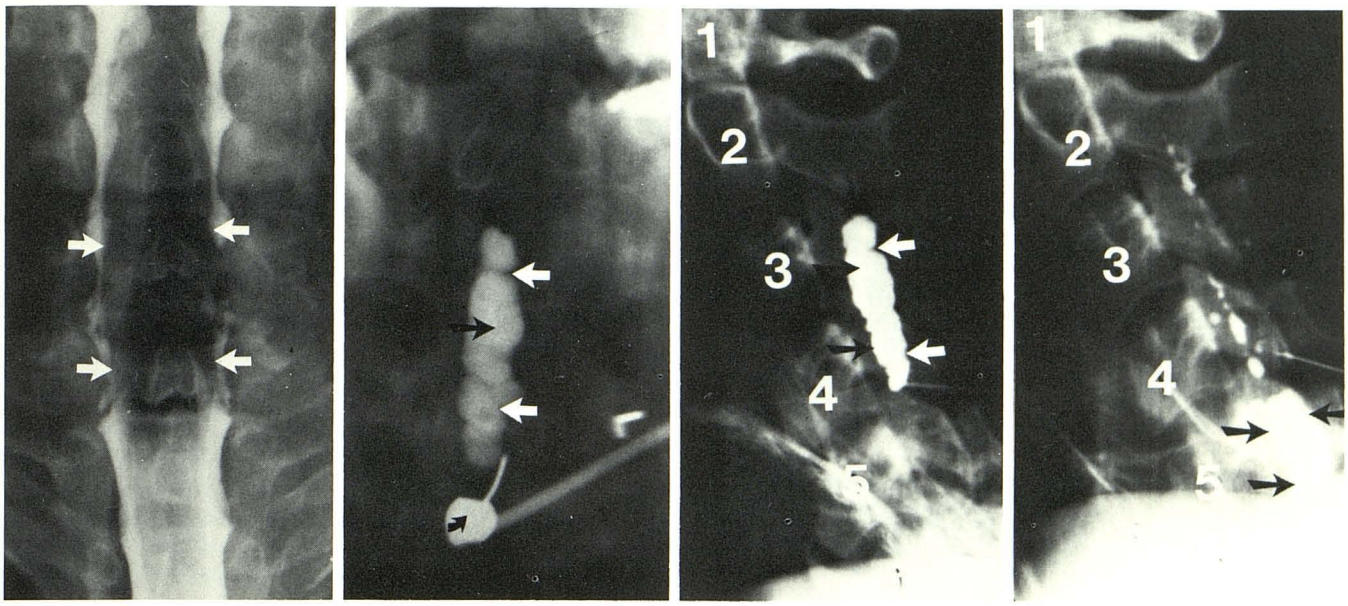


Fig. 2.—**A,** Pantopaque myelogram. Fusiform enlargement of cord (*arrows*). **B–D,** Percutaneous cyst puncture and Pantopaque cystogram in same patient. **B,** Frontal view. Pantopaque in central cyst (*straight black arrow*); glial marginal septations (*white arrows*); percutaneous needle (*curved arrow*). **C** and **D,** Lateral views, percutaneous cystogram; central cyst (*black arrows*); glial septations (*white arrows*). Contrast material in cyst changed in position with table tilt between **C** and **D.**

TABLE 3: **CT Findings**

CT Study	No. Examinations	Cord Size at Myelography		Cyst Filling with Contrast	CT, Technical Artifact	Cyst Location	
		Normal	Large			Cervical	Thoracic
Without metrizamide	3	1	2	...	...	3	0
With metrizamide, cyst filling	9	6	3	9	...	8	1
With metrizamide, no cyst filling	2	1	1	...	1	1	1

(fig. 1). The cord size was interpreted as large in 14 (56%) patients and normal or borderline in 11 (44%) (fig. 2). There were no serious complications in the metrizamide myelographic studies.

Percutaneous Cyst Puncture

Eight attempts at diagnostic percutaneous cyst puncture were made and six were successful in filling the cyst with contrast material. One study was initially interpreted as successful for cyst filling but on a later review was interpreted as an extradural injection. One attempt was unsuccessful. The two unsuccessful examinations were in myelographically normal-sized spinal cords; all six successful punctures were in patients with myelographically large spinal cords (fig. 2).

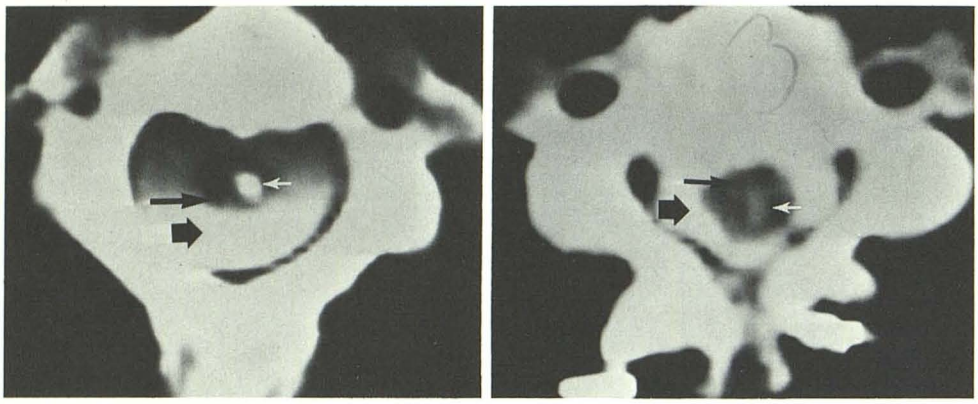
CT

CT scans were obtained in 14 patients, 11 after metrizamide myelography (table 3). In nine patients the cyst filled with contrast material (fig. 3). In three of these nine patients the cords were large, and in six patients the cords were of normal size. In one of the 11 metrizamide studies the cord was not visualized in the area of interest but the patient had a large cord seen on myelographic films preceding CT. In another patient, Pantopaque interfered with the CT study, although the cord had been seen to be normal on the previous Pantopaque myelogram.

In the three patients who had CT scans without metrizamide myelography, the lesions were in the cervical region and neither the spinal cords nor the cysts were identified (one patient was studied on a GE 8800 scanner and two on Ohio Nuclear scanners). Two of these three patients had large cords seen on previous Pantopaque myelography.

Diagnosis to Procedure Sequence in Abnormal Neuroradiologic Workup Group

In five patients a large cord was demonstrated on contrast myelography. Subsequent diagnostic percutaneous cyst puncture was followed by surgery. In seven patients a large cord demonstrated on contrast myelography and was subsequently confirmed by surgery. In nine patients the cyst



A B

Fig. 3.—Two different patients with progressive posttraumatic cystic myelopathy cyst demonstrated at CT Metrizamide study. Metrizamide in subarachnoid space (*short black arrow*) and cyst (*white arrow*). Spinal cord (*long black arrow*).

filled after metrizamide CT. Subsequent surgery confirmed the cysts. One of these patients also had a successful cyst puncture.

Cyst Morphology

All of the posttraumatic cysts in this series originated at or near the site of cord trauma, and 22 of 25 extended cranially. One of the 25 cysts extended caudally and two more extended both cranially and caudally. Circumferential glial sepatations were commonly seen at contrast cystography in the six successful studies (fig. 2). There were 18 cervical cord cysts, five cervicothoracic cysts, and two thoracic cysts.

Discussion

Barnett and Jousse [1] estimated that progressive posttraumatic cystic myelopathy might be expected in about 2% of patients with traumatic spinal cord injury and concluded that the ascending myelopathy was due to progressive cavitation within the central spinal cord. Frequent clinical symptoms and findings in these patients included the development of new pain, ascending neurologic deficits, asymmetric amyotrophy, increasing spasticity, hyperhydrosis and alternating Horners syndrome, severe autonomic dysreflexia, and disassociated sensory loss.

The cavitation or cyst in the cord frequently caused spinal cord enlargement [1], and before CT and metrizamide became available the aim of the neuroradiologic evaluation was to identify this enlarged cord using either Pantopaque myelography from the lumbar route or, if a complete block was found, by using air myelography from the cervical approach. At our institution in the early 1970s, the confirmation of a posttraumatic cyst in a large cord identified at myelography was by a diagnostic percutaneous cyst puncture and cystography with Pantopaque (fig. 2). This technique, although successful in all six patients with large cords, was discontinued because of the potential risk of the procedure and because of its failure in two cases in which the cord was of normal size, yet a cyst was found at subsequent surgery, albeit eccentric in location.

This neuroradiologic approach (fig. 4) was used until CT and metrizamide became available. The major limitation was that it only identified large cords as abnormal. Our review has since established that with this approach a high false-negative rate is possible *since cyst-containing cords are often normal in size (11 of 25 in this series).* In addition, posttraumatic and postsurgical adhesions often make identification of cord size difficult and inaccurate at conventional myelography.

In 1975, after CT identification of spinal cord syrinx by DiChiro et al. [3], CT was carried out on our patients. However, the cysts were not identified, and in three patients CT without metrizamide did not identify the spinal cord or the cervical cysts that were subsequently proven at surgery. Two of these patients had large cords previously demonstrated at cervical myelography. When metrizamide became available, C1–C2 cervical myelography was carried out in an attempt to identify large cords harboring cysts. Delayed CT was also carried out on these patients at 2–5 hr post-myelography on the premise that the metrizamide would absorb into the spinal cord [4] and enhance visualization of a cyst as a low attenuation defect in the "enhanced" cord. Unexpectedly, a cystic structure was opacified with contrast material in nine of 11 subsequently surgically proven cysts (fig. 3). Although CT is very informative, myelograms are still obtained because a large cord may still be demonstrated and because CT may be nondiagnostic because of technical factors (motion, machine failure, etc.). The current algorithm is diagrammed in figure 5.

The exact mechanism of posttraumatic cyst formation and extension, as well as the fluid source, is unknown. The review of Barnett and Jousse [1] emphasized that a cyst connection to the central canal or subarachnoid space is absent [1]. McLean et al. [5] suggest that at the time of trauma, contusion, myelomalacia, and/or hemorrhage occurs in the spinal cord and in the course of healing a glial-lined cavity in the traumatized zone is produced. Adhesion of the cord by scars secondary to trauma was found in all of our proven cases and such adhesions or tethering may subject the cord to unusual stress by the common pressure changes associated with the Valsalva maneuver, coughing, or sneezing. duBoulay et al. [6] observed that extension of a cyst cavity in the spinal cord can probably be explained by changes in spinal venous pressure, squeezing, and elon-

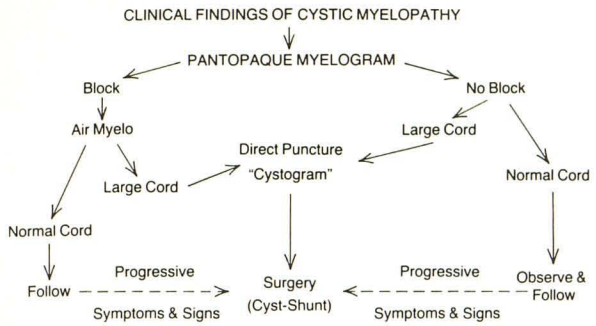


Fig. 4.—Historical algorithm: clinical findings of cystic myelopathy.

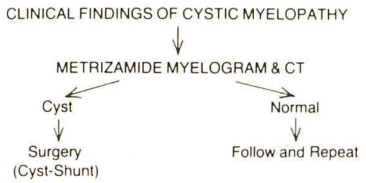


Fig. 5.—Current algorithm: clinical findings of cystic myelopathy.

gating a cavity with the increase in spinal extramedullary pressure that occurs with straining or coughing. Presumably, these extensions cause pressure on adjacent neural tissue and progressive symptoms and signs ensue. Clinical observations support this explanation showing that patients with this disorder often report a sudden development of new symptoms after a violent sneeze [1].

The Gardner and McMurry [7] theory for production of progressive posttraumatic cystic myelopathy suggests that the noncommunicating cyst of Barnett is a nonentity and that the 17 cases observed by Barnett and Jousse were actually occult communicating hydromyelia that occurred in a susceptible substrate population possessing congenital rhombencephalon hernias with subsequent traumatic subarachnoid block producing a hydromyelia. The Gardner and McMurry theory is not readily accepted since a review of autopsy material in which a connection to the central canal was sought was not found in five proven of progressive posttraumatic cystic myelopathy. No rhombencephalon abnormality was seen in our five patients who had cervical air myelography.

The fluid in the cyst resembles cerebrospinal fluid [1]. It is possible that the fluid may enter the cyst from the Virchow-Robin spaces from the subarachnoid space as suggested by Ball and Dayan [9] and Edgar [2], or it may be formed and resorbed by glial lining cells [1]. The demonstration of the contrast material in posttraumatic cystic myelopathy cysts 2–5 hr after intrathecal placement of metrizamide in nine patients in our series may support the theory for transneural migration of fluid to the cyst, as suggested by Edgar [2]. Winkler and Sackett [10] suggested that cerebrospinal fluid spaces and extracellular spaces are one fluid compartment with gaps in membranes allowing water-soluble molecules to pass. No posttraumatic communication to either the fourth ventricle, the subarachnoid space, or the central canal was found in any of the 25 operated cases in this series. The exact mechanism of contrast cyst filling and the time relations are unknown and need further study and evaluation. In one patient with a cyst studied with metrizamide and CT, the cyst filled maximally at 3 hr and was not well identified on earlier or later studies in the time sequence. Factors influencing cyst filling probably include: concentration of Metrizamide, position of the patient during the interval time prior to CT, presence or absence of subarachnoid block, and presence or absence of arachnoid adhesions and arachnoiditis. As improved resolution CT becomes more universally available, nonmetrizamide CT may be able to identify progressive posttraumatic cystic myelopathy as it successfully identified syringomyelia cavities [11, 12].

Progressive posttraumatic cystic myelopathy is an uncommon but potentially devastating complication of spinal cord injury. It must be "clinically recognized" and radiographically confirmed for surgical treatment. The current algorithm of neuroradiologic study of patients with suspected progressive posttraumatic cystic myelopathy emphasizes the use of CT with metrizamide to identify the cyst. With improved CT resolution, it may be possible to identify these posttraumatic cysts without metrizamide.

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