AJNR

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Radiologic Evaluation of Spinal Cord Fissures

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The clinical course and radiographic studies were reviewed of eight patients with posttraumatic spinal cord fissures (rents in the spinal cord communicating with the subarachnoid space). Five patients had spinal cord fissures associated with symptomatic intramedullary cysts that required shunting to alleviate progressive neurologic deficits and intractable pain. Their metrizamide myelograms showed contrast medium passing immediately from the subarachnoid sac into the spinal cord and their immediate metrizamide CT scans delineated the entire extent of the secondary cord cysts. Intraoperative sonography confirmed the presence of the cord fissures with sizeable cysts, detected adhesions, and guided myelotomies and the subsequent shunting and collapse of the cysts. Since neurologic improvement followed the shunting procedures in all five patients treated for progressive symptoms, it was concluded that early radiologic evaluation of posttraumatic spinal cord fissures with symptomatic cord cysts is crucial in the clinical management of these patients.

Because of the widespread use of the delayed metrizamide CT scan as a diagnostic procedure for identifying contrast collections within the spinal cord, the entity of the symptomatic posttraumatic spinal cord cyst has gained increasing radiologic and clinical attention [1–6]. Further interest in this entity has been stimulated by the recent employment of intraoperative spinal sonography (IOSS) to confirm the presence of these symptom-producing intramedullary cysts and to monitor their decompression during shunt placement [7–9]. The promising clinical results that have been achieved after decompression and shunting of these cysts [5–7, 10–13] have made this increasing recognition of posttraumatic spinal cord cysts very rewarding.

Recently we encountered eight spinal cord injured patients whose spinal cord cysts were caused by the presence of a fissure or rent within the spinal cord communicating with the subarachnoid space. Diagnoses were established by metrizamide myelography and metrizamide CT scanning in all eight patients and also confirmed by IOSS in four and surgical findings in five. Six of these patients had progressive neurologic deficits. The lack of radiologic literature on spinal cord fissures and the clinical improvement we saw in the five patients whose cysts were shunted prompted us to report our experience with spinal cord fissures and secondary cord cysts.

Materials and Methods

A retrospective review was made of the radiologic and surgical records of eight patients whose spinal cord injuries resulted in a spinal cord fissure with secondary cyst formation. The radiologic studies reviewed included myelograms, immediate (IMCT) and delayed (DMCT) metrizamide CT scans, magnetic resonance (MR) images, and intraoperative and percutaneous spinal sonograms. All myelograms were obtained via lumbar and/or cervical punctures using metrizamide in concentrations of 220–300 mg/ml. The CT scans were obtained on GE 8800 or 9800 CT/T scanners either immediately or as close to 4–5 hr after the standard
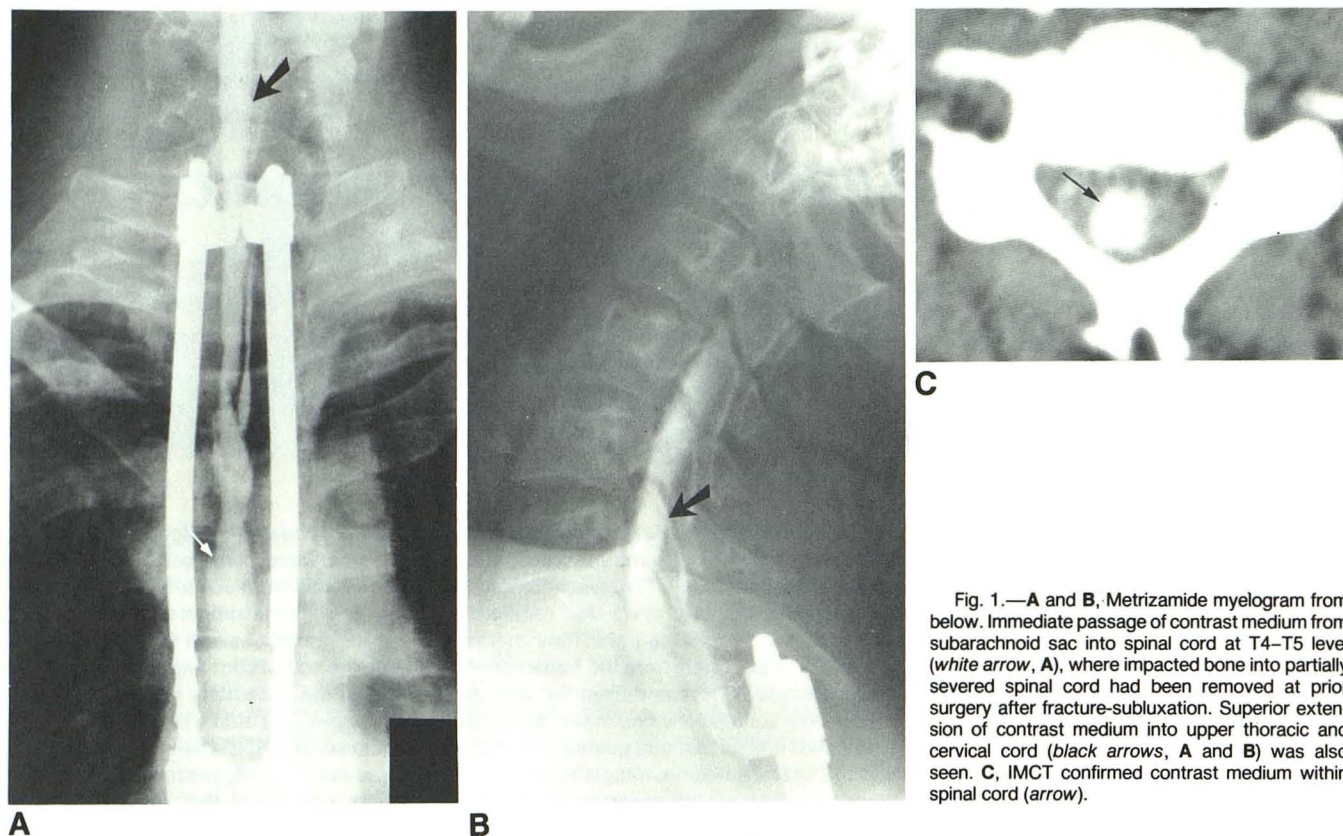
Fig. 1.—**A** and **B**, Metrizamide myelogram from below. Immediate passage of contrast medium from subarachnoid sac into spinal cord at T4–T5 level (*white arrow*, **A**), where impacted bone into partially severed spinal cord had been removed at prior surgery after fracture-subluxation. Superior extension of contrast medium into upper thoracic and cervical cord (*black arrows*, **A** and **B**) was also seen. **C**, IMCT confirmed contrast medium within spinal cord (*arrow*).

metrizamide myelogram as possible. A Siemens Magnetom unit operating at 0.35 T was used with 1-cm-thick sections without a surface coil for examination of the thoracic spinal cord in one patient and with a head coil with 5 mm sections at 2 mm intervals for evaluation of the cervical spinal cord in two patients. The ATL NeuroSector portable real-time unit (Advanced Technology Laboratories, Bellevue, WA) with a 7.5 MHz in-line transducer was used for all IOSS, using the same techniques described in previous reports [9–11]. Percutaneous sonography in one postoperative patient was performed with the same unit and transducer.

Results

Clinical

The eight patients, all men, were 21–62 years old (median age, 24.5 years). Their injuries, cervical in five and thoracic in three, were secondary to motor vehicle accidents in six, a boating accident in one, and unknown in one.

Five patients, all with chronic injury and prior surgery, had fissures associated with cysts that required surgical decompression. The symptoms that warranted shunting consisted of increasing motor and/or sensory deficits in five and intractable pain in four. These symptoms developed within 3–10 months of the spinal cord injury in three patients, within 2 years in one patient, and 20 years after the injury in one patient. The symptoms were noted for 1–2 months before admission in three patients, for 5–8 months before admission in one patient, and for 14 months before admission in another

patient. After shunting, there was improvement in motor and/or sensory function in all five patients and also a decrease or resolution of pain in four. These neurologic improvements persisted during the 8 month follow-up of two patients, the 2 year follow-up of one patient, and the 3 year follow-up of another patient. One patient has had no long-term follow-up, having been recently shunted.

Two patients, both with chronic injury and prior surgery, had stable neurologic deficits. These patients were studied to determine if there were any compressive spinal lesions that, if surgically corrected, would further improve their neurologic status. Their radiographic studies showed fissures with small cord cavitations that required no shunting. Both patients were treated with decompressive surgery for removal of chronically fractured subluxed bony fragments and with anterior cervical fusions. No postoperative neurologic improvement was noted in either patient.

The other patient in the study initially exhibited significant neurologic improvement after an acute spinal cord injury; he was believed at that time to have a fissure with a nonexpansile cyst. Recently this patient has worsened neurologically. The current radiologic and clinical evaluation indicates that the patient now has a cyst that requires shunting.

Radiologic Studies

The metrizamide myelograms in all five patients who were shunted showed the immediate passage of contrast medium
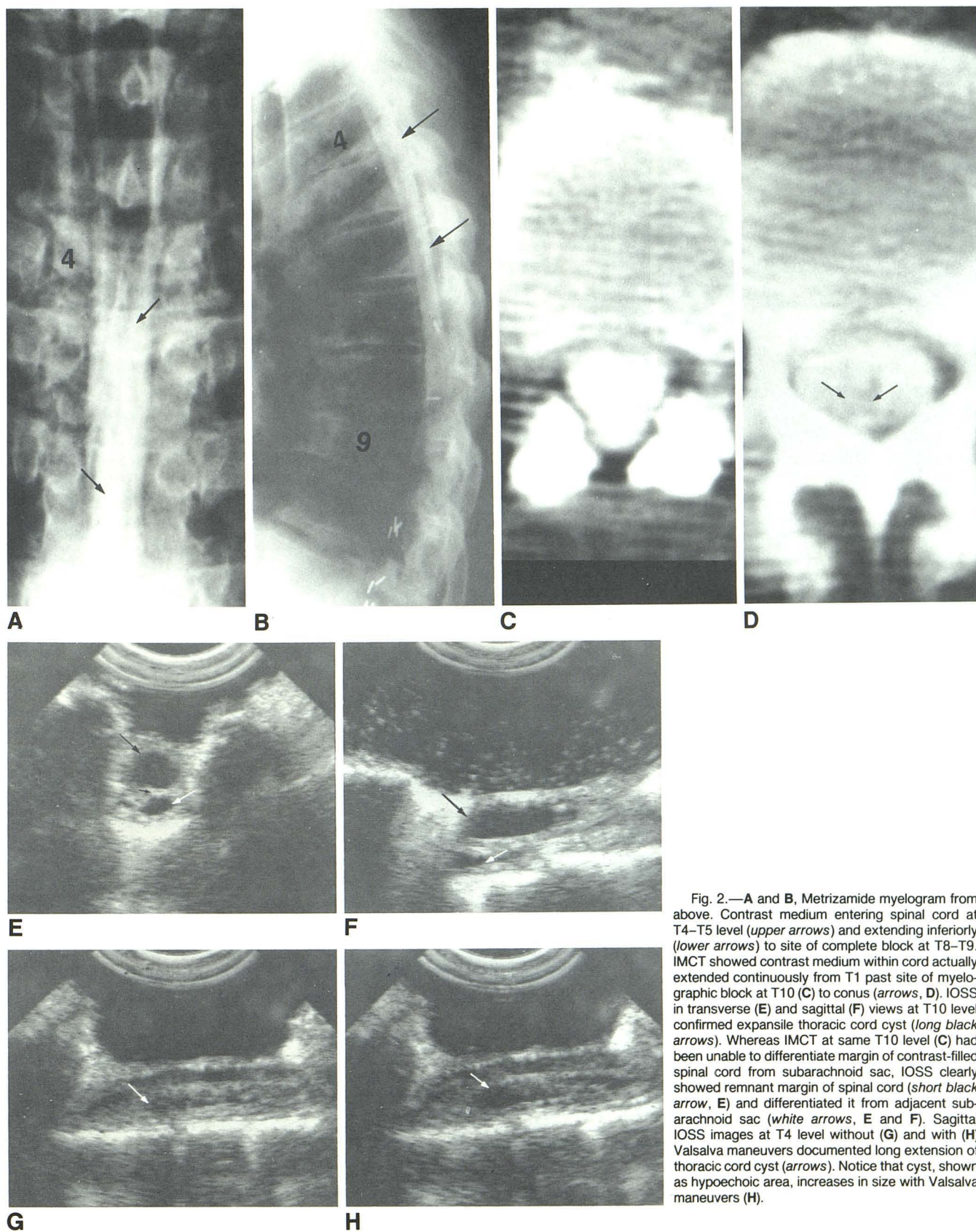
A   B   C   D

E   F

G   H

Fig. 2.—**A** and **B**, Metrizamide myelogram from above. Contrast medium entering spinal cord at T4–T5 level (*upper arrows*) and extending inferiorly (*lower arrows*) to site of complete block at T8–T9. IMCT showed contrast medium within cord actually extended continuously from T1 past site of myelographic block at T10 (**C**) to conus (*arrows*, **D**). IOSS in transverse (**E**) and sagittal (**F**) views at T10 level confirmed expansile thoracic cord cyst (*long black arrows*). Whereas IMCT at same T10 level (**C**) had been unable to differentiate margin of contrast-filled spinal cord from subarachnoid sac, IOSS clearly showed remnant margin of spinal cord (*short black arrow*, **E**) and differentiated it from adjacent subarachnoid sac (*white arrows*, **E** and **F**). Sagittal IOSS images at T4 level without (**G**) and with (**H**) Valsalva maneuvers documented long extension of thoracic cord cyst (*arrows*). Notice that cyst, shown as hypoechoic area, increases in size with Valsalva maneuvers (**H**).
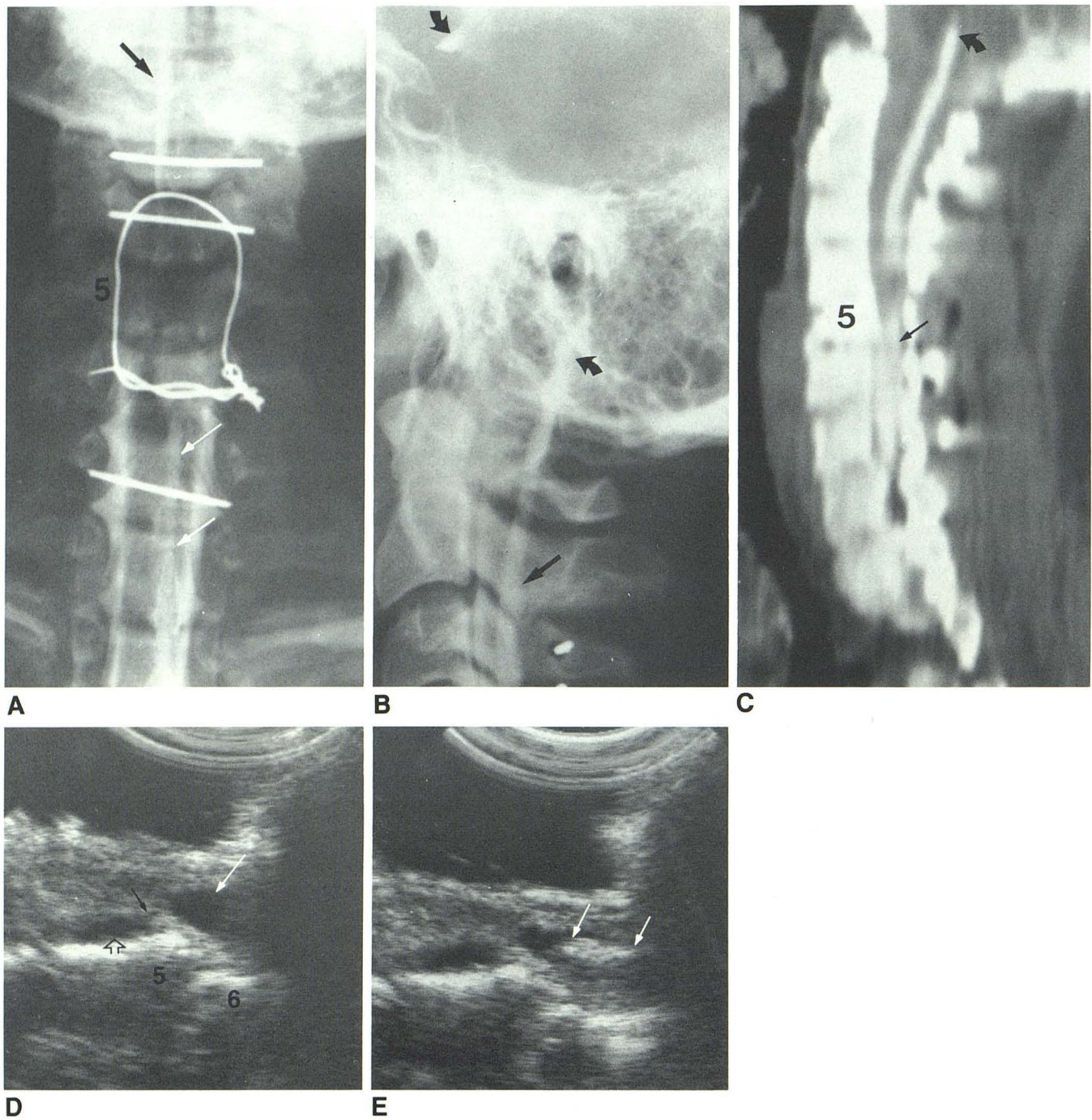
Fig. 3.—Early fluoroscopic spot film (not shown) during metrizamide myelogram from below showed contrast medium entering spinal cord at C5–C6 and extending further craniad. Later overhead AP (**A**) and lateral (**B**) films revealed passage of contrast medium from high cervical cord (*straight black arrows*) into medulla and ventricular system (*curved arrows*). Caudal extension (*white arrows*) into thoracic spinal cord was also seen. IMCT in axial and sagittally-reconstructed (**C**) views documented superior extension of contrast medium within cord from C5–C6 fracture-subluxation site (*straight arrow*) to high cervical cord and medulla (*curved arrow*) and showed its inferior extension to T4. **D**, Initial IOSS in sagittal projection at C5–C6 level showed that contrast medium within cord seen on myelogram and IMCT was secondary to sizeable cyst (*white arrow*) and that cord was tethered at this fracture-subluxation site. Fissure (*solid black arrow*) directly connecting cord cyst at C5–C6 to adjacent dilated subarachnoid sac (*open arrow*) was incompletely imaged on these static images but was well seen on real-time. Smaller superior extension of this cyst at C4 level, separated from larger part of cyst by myelomalacic cord, was shown on other images. **E**, Sagittal sonogram after placement of single shunt tube (*arrows*) confirmed collapse of cyst at C5–C6 level. Intraoperative scanning, however, also showed that this shunt tube could not be easily passed superiorly and that a second smaller shunt tube was needed to decompress cyst superiorly.

from the subarachnoid sac through a fissure into the spinal cord and partial filling of a secondary cord cyst (figs. 1–3). The location of the fissure was most easily determined during early fluoroscopy when the cord first began to opacify and before extensive filling of the cord cyst had occurred. The presence of contrast medium within the spinal cord was confirmed in all cases by overhead films in both anteroposterior and lateral projections. The myelograms, although diagnostic of the cord fissures and secondary cysts, failed to reveal the entire extent of cord pathology. In two patients there was a complete block caused by adhesions, and in the other three patients there was incomplete filling of the cord cyst with contrast medium.

Metrizamide CT scanning, however, demonstrated the true extent of cord pathology in all five patients, showing the contrast medium extending within the cord over greater distances than indicated by myelography. In the two patients with complete myelographic blocks metrizamide CT demonstrated the contrast medium extending within the cord beyond the sites of myelographic obstruction (fig. 2). It also showed cord involvement for a greater number of levels above the myelographic block than indicated by myelography. In the three patients without myelographic obstruction, metrizamide CT showed longer segments of cord opacification than seen on myelography.

The contrast medium that was seen within the cord on the metrizamide CT scans extended over 11–13 levels in three patients, over six levels in one patient, and over one level in one patient. This cord pathology was noted at and above the level of injury in all five patients and also below in four.

Although the actual cord fissure was not directly imaged on the metrizamide CT scan, its presence could be inferred in those four patients who were scanned immediately after myelography. The presence of very dense contrast medium within the cord on the IMCT scans of these four patients verified the myelographic impression of a direct communication between the subarachnoid sac and spinal cord. DMCT scans in three of these same four patients showed no significant difference in appearance of the cord pathology. One patient had only DMCT, which documented the presence of a cord cyst.

The appearance of the secondary cyst on the metrizamide scans in all five patients was that of a well defined, very dense collection of contrast medium. In the patient with the longest and largest cyst it was impossible to differentiate the contrast-filled spinal cord from the surrounding subarachnoid sac (fig. 2C). This differentiation was also not possible on the CT scans of the patient who had a complete myelographic block, extensive adhesions with loculation of contrast medium, and a markedly deformed, dilated subarachnoid sac.

MR was performed in two patients preoperatively and in one patient after operative intervention. In one of the preoperative patients MR failed to show well a large cord cyst in the thoracic spine. Resolution was too poor without the use of a surface coil. In two other patients MR provided important diagnostic information. The examinations, performed with a head coil, showed a cervical cord cyst in one patient and in

another documented the continued collapse of a previously shunted cervical cyst.

IOSS was performed in four of the patients whose fissures were associated with symptomatic cysts. In each patient this procedure confirmed the diagnosis of an intramedullary cyst, three of which were expansile and under pressure. The cysts appeared as sharply demarcated hypoechoic lesions, clearly distinguishable from the surrounding echogenic margins of the residual spinal cord parenchyma and adjacent hypoechoic dilated subarachnoid sac (figs. 2E and 2F). The cysts were anchored by adhesions extending from the surrounding cord to the site of prior injury and surgery (fig. 3D). By increasing intradural pressure by the Valsalva maneuvers, these cysts significantly increased in size (fig. 2H). The fissures that directly connected the cysts to the adjacent dilated subarachnoid sac also enlarged during Valsalva maneuvers. Because of their relatively small size compared with the adjacent cord cysts and their expansion during periods of increased intraabdominal pressure, these fissures were best seen during real-time scanning. The continuously changing dynamics of this spinal pathology made it difficult to record the fissures well on the static images (fig. 3D).

In contrast to the fissures, the bony compression caused by the chronic fractures and/or vertebral body subluxations was easily seen on static sonograms (fig. 3D). Also readily apparent was any discontinuity between portions of the cord cysts by myelomalacic segments that appeared hyperechoic and without central echo. This distinction between shuntable cyst and myelomalacic cord had not been made preoperatively on the metrizamide CT scan. Subarachnoid spaces that were loculated and dilated and associated with deformity of the cord were also immediately obvious on IOSS: They appeared as expansile hypoechoic areas adjacent to the spinal cord.

Sonography provided continuous monitoring of the various surgical procedures used in patient treatment. Real-time scanning was used before and after myelotomy, lysis of adhesions, bony removal and shunt placement. Sonography identified the necessity in two of the patients for placement of two shunts to achieve adequate cyst decompression. In one of these patients, buckling of the first catheter caused by a short segment of myelomalacic cord intervening between the cysts was seen with sonography. In the second patient IOSS identified adhesions that were preventing the use of a single catheter to decompress all parts of the cord cyst. After insertion of shunt tube(s) into the cord cyst and adjacent subarachnoid sac sonography documented cyst collapse (fig. 3E). Sonography was also used percutaneously in the followup of one shunted patient. It documented proper shunt position and continued cyst collapse.

In the two patients without progressive neurologic deficits, radiologic studies, although diagnostic, were less strikingly positive than in the patients with fissures associated with symptomatic cysts. In these patients metrizamide myelography did demonstrate the passage of contrast medium from the subarachnoid space into the spinal cord above the site of myelographic block. However, the cord opacification was

seen over only short distances and was subtle in nature. Demonstration of the cord contrast medium was especially difficult on AP views. The subsequent IMCT scans, however, clearly showed contrast medium within the cord. It extended over only one to two levels and was seen at and below the site of myelographic obstruction. The IMCT scans determined cord size, showing cord atrophy in one patient, and also demonstrated cord compression by fractured-subluxed bone. In one patient a DMCT scan was also obtained: Its appearance was similar to that of the IMCT scan except that the cord cyst was less densely opacified. Although these two patients underwent surgery to relieve the bony compression, sonography was not used to evaluate the cord at the site of contrast medium within the cord on the IMCT scans.

In the other patient in the series it was IMCT that enabled the diagnosis of a cord fissure with secondary cyst formation to be established. The metrizamide myelogram did not demonstrate any cord opacification, probably because it was performed with the patient supine and semierect due to the acute and unstable nature of his C1–C2 fracture-subluxation. In this position only the posterior subarachnoid sac filled out well. IMCT, however, revealed a very dense collection of contrast medium within the cord at C1–C2 with superior extension to the medulla and inferior extension to C4. Since the density of the contrast medium in the cord was the same as that in the adjacent subarachnoid space, immediate filling of the cord through an acute rent or fissure in the cord was implied. Positive findings on the follow-up metrizamide myelograms and CT scans over the next 15 months consisted of a very small area of poorly defined contrast medium in the cord at the C2 level on the metrizamide CT scan. This improvement in radiologic findings correlated well with the marked neurologic improvement the patient noted during this same period. A slowly progressive increase in neurologic deficits and the onset of sweating and intense pain later complicated the patient's course. MR 4 years after the original injury confirmed a cyst that accounted for the patient's clinical deterioration.

Discussion

It has been well documented that spinal cord cysts can develop during an interval after a severe spinal cord injury and cause ascending neurologic deficits, intractable pain, and profuse sweating if they progressively expand [1, 3, 5, 6, 10–12]. Many theories have been proposed in the literature to explain their formation [4, 6, 14]. The clinical detection of these primary posttraumatic spinal cord cysts (those that form without an associated fissure) has been aided by the introduction of metrizamide as a water-soluble contrast agent for myelography and by the use of delayed metrizamide high-resolution CT scanning. Radiologic diagnosis has been based on the findings of well defined dense collections of contrast medium within the spinal cord on the CT scan obtained 4 or more hours after myelography [1, 2, 5].

More recently, operative findings and the use of IOSS and MR have further refined the criteria used in the diagnosis of primary cysts [5, 7, 9, 15]. A spectrum of cord pathology has

been noted after spinal cord injury ranging from cord cavitations that are small, nonconfluent, nonexpansile, and not associated with progressive neurologic symptoms to that of cord cavitation that is of a sizeable or expansile nature and is associated with worsening of neurologic status. The use of IOSS and MR has made it evident that not every collection of contrast medium within the cord seen on the delayed metrizamide CT scan represents a shuntable cyst [7, 9, 15, 16]. These procedures have allowed for the differentiation of myelomalacia and cystic degeneration of the cord from shuntable cord cysts [7, 9, 15, 16].

In contradistinction to those patients whose posttraumatic cysts have formed without fissures, we have noted a group of patients whose cysts have developed because of fissures in the spinal cord. Our patients differ from those with primary cysts, then, mainly in their mode of development rather than in their clinical presentation, spectrum of pathology, or major radiologic findings. In our patients the original severe injury, usually associated with a marked fracture-subluxation, created a large rent or fissure in the spinal cord that allowed for the direct communication of the spinal cord with the adjacent subarachnoid space. With time, adhesions formed that tethered the spinal cord, allowing for ingress of cerebrospinal fluid (CSF) into the cord during Valsalva maneuvers without equal egress of fluid. Progressive enlargement of a cord cyst, usually with extension over 6–12 levels, then ensued in most of our patients and resulted in increasing neurologic deficits and pain. Smaller rents in the spinal cord, perhaps related to less severe cord injury and less marked tethering by cord adhesions, might explain the lack of progressive symptoms seen in two of our patients and their less striking radiologic findings, with only one to two levels of cord involvement.

Evidence supporting the presence of these large rents or lacerations of the spinal cord came directly from surgery in two of our patients. In one patient a thoracic laminectomy, performed within 3 weeks of a T4–T5 fracture-subluxation with acute cord injury, revealed a fragment of bone actually impacted into and partially severing the spinal cord. Five months later this patient had symptoms of an expansile spinal cord cyst. Metrizamide myelography demonstrated contrast medium passing immediately from the subarachnoid sac into the spinal cord at the same site at which a rent in the spinal cord had been identified at surgery. In another patient a markedly disrupted, torn spinal cord was also seen at surgery at the level of original injury, which allowed for the direct ingress of CSF into the spinal cord and for secondary cord cyst formation.

Additional evidence supporting the presence of spinal cord fissures in our patients was found during radiologic workup. The immediate passage of contrast medium during metrizamide myelography from the subarachnoid sac into the spinal cord at the level of fracture-subluxation and cord tethering, a finding not seen in those with posttraumatic spinal cord cysts, indicated the presence of a significant rent in the spinal cord. The IMCT scans confirmed the presence of contrast medium within the cord and showed it to be of a density similar to that of the metrizamide in the adjacent subarachnoid space. In contrast to those with primary cysts, no scanning delay

was needed to demonstrate the metrizamide in the cord. The fissure itself was actually shown during real-time IOSS and was seen to enlarge along with the adjacent cord cyst during repeated Valsalva maneuvers. IOSS also confirmed the direct communication of the fissure with the dilated subarachnoid sac and demonstrated its occurrence at the site of cord tethering and fracture subluxation.

As a result of our study we offer six conclusions:

1. Posttraumatic spinal cord fissures are a potential source of increasing morbidity in victims of spinal cord injury.

2. When associated with expansile intramedullary cysts, they may cause increasing or new neurologic deficits, sweating, and intractable pain from several months to many years after the original injury.

3. Posttraumatic and/or postsurgical adhesions may be important in their development (i.e., through a ball-valve mechanism).

4. Diagnosis is established preoperatively by metrizamide myelography and IMCT. In the future, MR will play an increasingly important diagnostic role.

5. IOSS plays a crucial role in the surgical management of these lesions.

6. Cyst shunting and lysis of adhesions improves neurologic status and causes resolution of or a significant decrease in pain.

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