Intradural arachnoid cysts of the spinal canal associated with intramedullary cysts

Brian T. Andrews, M.D., Philip R. Weinstein, M.D., Mark L. Rosenblum, M.D., and Nicholas M. Barbaro, M.D.

Department of Neurological Surgery, School of Medicine, University of California, San Francisco, California

Five patients had intradural arachnoid cysts of the thoracic spinal canal associated with syringomyelia or posttraumatic intramedullary spinal cord cysts. Three cases were diagnosed 6 to 18 years after spinal surgery and two 14 to 17 years after spinal cord trauma. In each case, delayed progression of symptoms led to the identification of the lesions. The diagnosis was assisted by the use of myelography and delayed computerized tomography scanning in two cases and by magnetic resonance imaging in all five. In each case, the arachnoid cyst appeared to compress the spinal cord or nerve roots; in three cases, the syrinx cavities appeared to exert a significant mass effect. In the two trauma-related cases, the intramedullary cysts were small and may have represented areas of cystic myelomalacia. In four cases, intraoperative real-time ultrasonography helped to localize the arachnoid and intramedullary cavities. All five patients were treated by fenestration of the arachnoid cyst; additional peritoneal shunting of the cyst was performed in one case and of the intramedullary cavity in three. In one patient, the two lesions appeared to have a balancing effect; after drainage of the arachnoid cyst, the syrinx cavity expanded and had to be treated separately. The neurological deficits were reduced in four patients and stabilized in one. Intradural arachnoid cysts and intramedullary cysts may occur together as a late complication of spinal surgery or spinal cord trauma, and either or both lesions may cause delayed neurological deterioration.

Key Words: arachnoid cyst • syringomyelia • magnetic resonance imaging • spinal trauma • intramedullary cyst

Arachnoid cysts, or diverticula, of the spinal canal are rare lesions of the extradural and intradural spaces. They occur most often in the thoracic region dorsal to the spinal cord but may be found ventrally as well. Although asymptomatic cysts may be common incidental findings at myelography, the rare symptomatic cyst causes neurological deficits by compressing the spinal cord or nerve roots. Syringomyelia is a more common spinal lesion that may be idiopathic or congenital. Spinal cord trauma may also lead to the delayed formation of intramedullary cystic myelomalacia or frank syringomyelia. To our knowledge, spinal arachnoid cysts associated with intramedullary cysts have not been described previously.

This report describes the clinical presentation, diagnostic studies, and treatment of five patients in whom intradural arachnoid cysts of the spinal canal and intramedullary cysts were diagnosed 6 to 18 years after spinal trauma or surgery.

Case Reports

Case 1

This 36-year-old woman first had bilateral leg weakness in 1973. Myelography showed a partial subarachnoid block caused by an epidural mass at the T-10 level. A laminectomy was performed, and a hemangioma of the T-10 vertebral body was partially resected. Postoperatively, the patient received spinal irradiation and regained normal leg strength. In August, 1984, her leg weakness recurred along with numbness of the right leg and burning pain in the chest and leg on the left side. Metrizamide myelography and delayed axial computerized tomography (CT) scans showed an intraspinal syrinx extending from T-6 to T-10. The patient underwent placement of a syringoperitoneal shunt, and her symptoms were partially relieved. In March, 1985, she developed spasticity of the legs and incontinence of bowel and bladder. Repeat axial CT scans of the spine showed recurrent compression of the cord by tumor.
Arachnoid cysts and intramedullary cysts

FIG. 1. Case 1. Left: T₁-weighted direct sagittal magnetic resonance (MR) image of the spine showing an abnormal T-10 vertebral body and, inferiorly, a ventral cystic cavity of cerebrospinal fluid density causing dorsal displacement of the spinal cord (open arrow). The collapsed syrinx cavity is also visible (arrowheads). Right: Axial MR image at the T-11 level confirming the marked dorsal displacement of the spinal cord caused by the ventral arachnoid cyst.

within the left pedicle of T-10. In May, 1985, laminectomy was performed with additional resection of the hemangioma, which resulted in partial improvement of leg strength and sensation.

The patient's condition remained stable until April, 1986, when her spastic paraparesis worsened. Magnetic resonance (MR) imaging showed enlargement of the syrinx cavity. The syringoperitoneal shunt was revised, resulting in a temporary improvement of strength and sensation. Four weeks later, she again developed burning dysesthesias, increased paraparesis, and recurrence of her thoracic pain. An MR study showed a collapsed syrinx cavity within the spinal cord and a large ventral intradural cyst with the signal density of cerebrospinal fluid (CSF) extending from T-10 to T-12 and displacing the conus dorsally (Fig. 1). A T-12 laminectomy was performed; at operation, the dura appeared tense and the cord was displaced dorsally by a large arachnoid cyst. Intraoperative real-time ultrasound images clearly demonstrated the relationship of the cord to the ventral cyst. Fenestration and drainage of the cyst resulted in immediate relaxation of the cord. A Silastic T-tube shunt* was implanted in the arachnoid cyst cavity and connected with a Y-connector to the syringoperitoneal shunt catheter already in place.

Postoperatively, the patient showed little neurological improvement. A repeat MR image obtained 3 days after the operation showed collapse of the ventral subarachnoid cyst and enlargement and extension of the syrinx cavity down to the L-1 level (Fig. 2). Surgical reexploration was performed. Intraoperative ultrasound images at T-11 and T-12 showed marked dilatation of the spinal cord by a syrinx cavity (Fig. 3). A dorsolateral myelotomy was performed, and a new syringoperitoneal shunt was placed. Postoperatively, the patient had progressive improvement in leg weakness, paresthesias, and pain. Four months after the last operation, she remains ambulatory and has less spasticity and thoracic pain.

FIG. 2. Case 1. Left: T₁-weighted direct sagittal magnetic resonance (MR) image after fenestration and shunting of the ventral arachnoid cyst showing reexpansion of the syrinx cavity below the T-10 level (arrowheads). Right: Axial MR image at the T-11 level confirming reexpansion of the syrinx within the spinal cord.

* Shunt manufactured by Heyer-Schulte Corp., Goleta, California.

FIG. 3. Case 1. Intraoperative direct sagittal ultrasound image of the spinal cord at T11–12 showing a markedly dilated syrinx cavity (asterisk) and no evidence of a ventral arachnoid cyst. The arrow indicates the ventral vertebral bodies.
Case 2

This 57-year-old man received multiple injuries in a motor-vehicle accident in 1971. At the initial examination, he was paraplegic with a midthoracic sensory level and had spinal fractures at T-7 and T-8. Spinal cord decompression at these levels was performed 3 weeks after the injury. By 1972, the patient was able to walk, but had residual spasticity. In early 1985, he developed progressive leg spasticity, urinary frequency, bowel and bladder incontinence, and loss of rectal sensation. In September, 1985, neurological examination showed spastic paraparesis and an incomplete T-10 sensory level. A metrizamide myelogram demonstrated a complete subarachnoid block at the T8-9 level. A delayed CT scan showed a dorsolateral arachnoid cyst that filled with contrast material at the level of the block; immediately below, at the T-10 level, a small intraspinal syrinx was seen extending inferiorly to the conus (Fig. 4). An MR image confirmed the presence of both lesions. The spinal cord was displaced ventrolaterally and compressed by the cyst, but was not expanded in the region of the syrinx.

In October, 1985, surgical exploration of the spinal cord at the T8-9 level showed a circumferential layer of subdural calcification, which was removed. The arachnoid cyst was partially excised and was fenestrated into the adjacent subarachnoid space above. Postoperatively, the patient noted a reduction in the intensity and frequency of his spasms, improved strength in the lower extremities, return of rectal sensation, and improved bladder and bowel control. At the 6-month follow-up examination, he remains ambulatory and is neurologically stable.

Case 3

In 1969, this 43-year-old man fell and suffered a spinal fracture that resulted in paraplegia and a partial T-12 level sensory loss. Nine months after dorsal spinal fusion, he was ambulatory and neurologically intact. In late 1985, he noted the gradual onset of difficulty in walking, decreased sensation in the legs, and impotence.

In April, 1986, the patient was referred to our institution. The neurological examination showed hypotension below T-12 and a mild spastic paraparesis. Lumbar myelography followed by CT scanning showed an abnormal collection of contrast material (metrizamide) ventral to the spinal cord at the T-12 level (Fig. 5). Axial and direct sagittal MR images demonstrated this cyst and a small intramedullary cystic cavity at the T10-11 level, both with a signal intensity consistent with CSF. A laminectomy was performed; intraoperative real-time ultrasonography showed the small syrinx cavity and the ventral intradural cyst. Incision of the dura revealed dense arachnoidal adhesions; the spinal cord appeared to be displaced dorsally. Exploration of the ventral intradural space disclosed a cyst within the arachnoidal adhesions, which was fenestrated superiorly into the normal subarachnoid space. Repeat ultrasonography then showed collapse of this cyst with respiratory movements and less dorsal displacement of the cord. Nine months after the operation, the patient's neurological condition remains unchanged from his preoperative status.

Case 4

This 61-year-old woman had a history of low-back pain dating to 1962. At that time she underwent Pantopaque myelography and lumbar disc surgery, which resulted in improvement of her symptoms. In 1980, she noted the insidious onset of gait unsteadiness. Myelography showed a T-7 level blockage, and a diagnosis of “arachnoiditis” was made. Her symptoms remained stable until late 1985, when she developed numbness and weakness of the left leg, and bowel and bladder incontinence.

The patient was referred to our institution in January,
Arachnoid cysts and intramedullary cysts

1986. The neurological examination revealed weakness and hyperreflexia of the left leg and a dissociated sensory loss to pin-prick below the T-7 level on the left side. A metrizamide myelogram of the lumbar spine revealed a T-9 level block; the conus and the spinal cord appeared to be widened at this level. An MR image showed a syrinx cavity extending from T-9 down to the conus. The patient underwent a T9–L1 laminectomy; dense arachnoidal adhesions and residual Pantopaque were encountered. The dilated conus was opened, and a large syrinx cavity was shunted into the adjacent normal subarachnoid space below. Postoperatively, the patient was initially paraparetic, but subsequently her strength and sensation improved to better than the preoperative state.

In June, 1986, the patient again noted increasing spastic paraparesis. Repeat MR imaging revealed persistence of the syrinx cavity and a new dorsally located area of CSF density above the syrinx at the T-9 and T-10 levels (Fig. 6). During surgical reexploration, ultrasound images through the dura identified both the syrinx cavity and the dorsal collection of CSF. The previous syringosubarachnoid shunt was removed, and a T-tube shunt was used to drain the syrinx cavity to the peritoneum. Dense arachnoidal adhesions superiorly and a loculated dorsal arachnoid cyst that compressed the spinal cord were encountered. The dorsal wall of the cyst was removed, but the ventral arachnoid was left in place because of its adherence to the cord. A Silastic shunt catheter was placed from this site into the normal-appearing subarachnoid space superiorly. Postoperatively, the patient experienced rapid improvement in her leg strength and sensation, but recovery of bowel and bladder function was slower. Four months after the last operation, she can walk with assistance and has normal bowel and bladder function; however, she still has pain and numbness in the right side of the trunk and the right leg.

Case 5

This 62-year-old woman developed bilateral leg weakness in 1980. Pantopaque myelography demonstrated a tumor dorsal to the spinal cord at the T-4 level. A laminectomy was performed, and a meningioma was completely resected. Postoperatively, her leg strength returned and she did well until late 1985, when she noted recurrent leg weakness and numbness. Mye-
logy showed a total subarachnoid block at the T-4 level. Surgical reexploration at this level at another hospital revealed an arachnoid cyst at the site of the previous tumor. Despite partial resection of the cyst, her symptoms continued to progress slowly.

In August, 1986, the patient was referred to our institution. Neurological examination revealed a spastic paraparesis and an incomplete T-4 sensory level. Axial and direct sagittal MR images showed a large syrinx cavity extending from T-5 down to the conus, and adjacent ventral displacement of the spinal cord at the T-3 and T-4 levels.

Laminectomy of T3–5 was performed. Intraoperative ultrasound images through the dura confirmed the presence of a syrinx cavity and, superiorly, an adjacent ventral deviation of the spinal cord. Incision of the dura revealed dense arachnoid adhesions enveloping the spinal cord; inferiorly, the cord was dilated. A dorsolateral myelotomy disclosed a large syrinx cavity, which was shunted with a T-tube shunt into the peritoneum. Superiorly, the cord was displaced ventrally by a 5 × 1 cm dorsal cyst formed by layers of thickened arachnoid. This cyst was partially resected, leaving some arachnoid attached to the spinal cord. During removal of the cyst, droplets of Pantopaque were seen in the CSF within the cavity. Postoperatively, the patient was initially unchanged neurologically. Six months after the operation, however, she has less spastic paraparesis and sensory loss, but requires assistance in walking.

Discussion

Arachnoid cysts of the spinal canal are rare benign lesions that are lined by a single layer of normal arachnoidal cells and filled with CSF. There may be an adjacent connective tissue stroma or fibrosis. Arachnoid cysts can be entirely asymptomatic or can produce neurological symptoms by compressing the nerve roots or the spinal cord. Syringomyelia may cause a wide range of neurological manifestations. The clinical course is highly variable; patients may improve or stabilize without surgery or may continue to deteriorate despite treatment. Posttraumatic cystic myelomalacia or frank syringomyelia is most often diagnosed at the time of delayed neurological deterioration in patients with spinal cord injury.

A variety of causes have been proposed to explain the pathogenesis of intradural arachnoid cysts. Cysts dorsal to the spinal cord may arise congenitally from the septum posticum, the membrane dividing the midline dorsal subarachnoid space of the cervical and thoracic spinal canal. Arachnoid cysts may also be formed as a result of arachnoidal adhesions caused by meningitis, instillation of drugs, or spinal trauma. Some cases of syringomyelia are also thought to be caused by trauma and arachnoiditis. It is generally held that posttraumatic syringomyelia develops from hematomyelia or myelomalacia that becomes cystic at the level of maximal spinal cord injury. In two of our cases, the cysts were located at the site of previous spinal cord trauma and evident myelomalacia. The occurrence of an intradural arachnoid cyst in association with syringomyelia or posttraumatic spinal cord cysts may indicate a common etiology for these lesions. In the cases described in this report, arachnoidal adhesions leading to impairment of CSF dynamics after spinal trauma, previous spinal surgery, or Pantopaque myelography may have been the common pathogenic mechanism for both the extradural and intramedullary cystic cavities.

The diagnosis of these associated spinal lesions was made by myelography, delayed CT scanning, and MR imaging. In patients with intradural arachnoid cysts, myelography typically shows a total or partial subarachnoid block caused by an extradural mass lesion. Teng and Papatheodorou cautioned that myelograms obtained when patients are in the prone position may not show dorsal arachnoid cysts and suggested that supine positioning may be necessary to demonstrate such lesions. In our five cases, myelography showed a total or subtotal block that was probably caused by the extradural cyst and adjacent arachnoid adhesions. Computerized tomography with contrast enhancement allows better definition of both arachnoid cysts and syrinx cavities than does myelography alone. Recently, MR imaging has proven valuable in delineating intracranial arachnoid cysts and syringomyelia. In our patients, MR imaging appeared to be the single best method for identifying the presence of both lesions. Intraoperative real-time ultrasonography has also proved useful in localizing the cystic cavities, as reported here in four of our cases and as described by Wilberger et al.

The treatment of spinal arachnoid cysts has included the complete removal of cysts that are located dorsal or lateral to the spinal cord, fenestration into the adjacent normal spinal subarachnoid space, and shunting of the cyst to the peritoneum or atrium. Complete or partial resolution of symptoms has been reported with each of these treatments. Three of our patients had partial resection and fenestration of their cysts, and one also had shunting of the cyst to the peritoneum. Syringomyelia has been treated with a variety of techniques, including posterior fossa decompression with or without closure of a patent central canal, laminectomy and syringostomy, terminal ventriculostomy, syringoatrial shunting, and syringoperitoneal shunting. Barbaro et al. reported that syringoperitoneal shunt procedures were more effective than other methods in relieving symptoms. Of 15 patients treated in this manner, 12 (80%) had either improvement or stabilization of their neurological deficits, including five of six patients with posttraumatic syringomyelia. In one of our patients (Case 4), no neurological improvement was achieved after implantation of a syringosubarachnoid shunt; only after placement of a syringoperitoneal shunt did the patient improve.
Arachnoid cysts and intramedullary cysts

In our two patients with previous spinal cord injury (Cases 2 and 3), the intramedullary cysts were small and caused little apparent mass effect on the remaining cord. In three patients, the syrinx appeared to cause significant compression of the spinal cord and therefore was treated with a shunting procedure. In Case 1, the syrinx appeared to be in a dynamic balance with the adjacent arachnoid cyst; after fenestration and shunting of the cyst, the syrinx cavity expanded and the symptoms persisted. Only after adequate drainage of both lesions were the symptoms relieved.

It is important for clinicians to recognize the association between extramedullary arachnoid cysts and intramedullary cysts as a consequence of previous spinal trauma or surgery. Either or both lesions may cause delayed neurological deterioration. When both are identified in a patient with delayed deterioration after spinal cord trauma, it may be reasonable to treat the larger or more compressive cavity first. Because the lesions may be in a dynamic balance, treatment of one may lead to symptomatic dilatation of the other. Persistent or recurrent symptoms may therefore necessitate treatment of both cavities. Alternatively, if both lesions appear to exert a significant mass effect, they should probably be treated during a single operation.

Acknowledgment

The authors thank Stephen Ordway for editorial assistance.

References


Manuscript received January 10, 1987.
Accepted in final form September 21, 1987.
Address reprint requests to: Brian T. Andrews, M.D., Department of Neurological Surgery, 1360 Ninth Avenue, Suite 210, San Francisco, California 94122.