Spinal arachnoid diverticula

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Four cases of symptomatic arachnoid diverticula are presented to illustrate the diagnosis, radiographic findings, and treatment of this condition. The myelographic demonstration of asymptomatic diverticula is not rare, and only when neurological symptoms are localized to the level of the myelographically demonstrated diverticula is surgical treatment considered. Supine and upright positions during myelography are important to demonstrate the lesions.

KEY WORDS: spinal arachnoid diverticula · myelopathy · myelography · spinal cord

Spinal arachnoid diverticula are intradural cysts of the arachnoid membrane which communicate with the subarachnoid space. Characterized by a narrow rostral ostium, the diverticula may collect cerebrospinal fluid, empty incompletely with positional changes, and produce signs of spinal cord compression. Sensory disturbances, weakness progressing to paraplegia, loss of sphincter control, and pain localized to the level of the lesion are the clinical findings. Neurological deterioration may be gradual or acute requiring decompression of the spinal cord by laminectomy and excision of the diverticula. Although arachnoid diverticula have been observed throughout the spinal canal, they occur more frequently along the dorsal surface of the thoracic spinal cord. They can be demonstrated during myelography, either by direct filling or indirectly as an intradural space-occupying mass, in which case the lesion must be differentiated from a tumor. Demonstration of asymptomatic diverticula is not rare during myelography. It is only when neurological symptoms are localized to the level of the myelographically demonstrated diverticula that treatment by laminectomy and local excision is considered. It is the purpose of this communication to discuss the diagnosis, radiographic findings, and treatment of four such cases recently encountered.

Case Reports

Case 1

An 18-year-old Marine Corps recruit was admitted to the hospital because of spasticity of the legs and inability to walk. He had been absent without leave for 5 months and during that time had noted the onset of urinary and fecal incontinence associated with progressive paresthesias and weakness of the legs. He had recently noted dull pain in the posterior thoracic area relieved by lying down. Three years previously, he had had an episode of mild back pain lasting several days associated with heavy lifting.

Examination. Percussion of the dorsal spine elicited mild tenderness over the region of T-8 through T-10. No gibbus was present. There was bilateral hypalgesia from T-8 through T-12, analgesia from L-1 through

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upon reversing the position of the patient did the diverticulum fill with Pantopaque descending from the cervical region. It could be emptied and filled repeatedly.

**Operation.** Laminectomy was performed from T-7 through T-9, and the laminae appeared normal. Upon opening the dura, a grey slightly opaque cystic structure measuring $1.5 \times 1 \times 4$ cm was demonstrated resting on the dorsal aspect of the spinal cord. The lesion was filled with spinal fluid and Pantopaque and appeared to be a diverticulum of the arachnoid within the subarachnoid space. The spinal cord vascularity, size, and contour were normal. The diverticulum was excised and the dura closed.

**Postoperative Course.** The patient improved rapidly. Three weeks later, he was able to walk without support, although his gait remained unsteady because of residual spasticity. Below T-8, slight hypalgesia persisted, which was less marked in the sacral area. Appreciation of movement, vibration, and position of the toes improved. Sphincter tone remained mildly impaired but the patient became continent of urine. Six months later no further significant improvement had occurred.

**Case 2**

A 40-year-old man for 2 months had experienced occasional dull aching pain in the thoracic area with anterior perithoracic radiation. One morning while bending over to tie his shoes, he suddenly experienced similar but more severe pain which was intensified by maintaining a sitting position and was relieved when lying prone. This was followed by the onset of left-sided numbness and paresthesias below the midthoracic region, and left leg weakness. He was admitted to the hospital later the same day. There was no history of trauma to the back.

**Examination.** Physical examination was normal except for diminished anal sphincter tone. The dorsal spine curvature was normal, and no tenderness was elicited by percussion of the spinous processes. Strength in the arms and in the right leg was normal. There was weakness graded as 4/5 in all of the muscles of the left leg. Position sense and vibratory appreciation were normal, but perception of pinprick and cotton touch was diminished on the left side below T-8. An ill-
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defined diminished response to pinprick and cotton touch on the right side was elicited. Reflexes in the arm were 1+ and symmetrical, while abdominal reflexes were absent. The knee reflex was diminished on the left side and ankle reflexes were absent. There were bilateral Babinski signs. Straight leg raising to 85° caused discomfort in the back. On the morning after admission, marked weakness in the left leg and very slight weakness in all the muscle groups of the right leg were present. Sensory examination was unchanged with the exception of a more definite loss of pinprick on the right side from approximately T-10 down. Cerebrospinal fluid protein was 46 mg% and 1 lymphocyte per mm³ was present. X-ray films of the chest and thoracic spine demonstrated no abnormalities.

Myelography demonstrated several communicating intradural diverticula along the dorsal aspect of the spinal cord in the thoracic region (Fig. 2). The largest at the T-9 level contained approximately 4 to 6 cc of contrast material. The diverticula could be emptied and filled repeatedly by varying the position of the patient.

Operation. Laminectomy was performed from T-6 through T-9. When the dura was opened, the arachnoid was noted to be thickened and somewhat opaque. The appearance of the lesion and the spinal cord was similar to that in the first case. A 5-cm cystic lesion was excised with the surrounding thickened arachnoid.

Postoperative Course. By 24 hours after surgery the sensory deficit and weakness in the right leg had disappeared. Weakness and sensory disturbance on the left gradually diminished, and 2 months following surgery, strength, sensation, and reflexes in both legs were normal.

Fig. 2. Case 2. Left: Lateral radiograph demonstrating large diverticulum almost completely filled with Pantopaque. Right: Anteroposterior radiograph demonstrating multiple diverticula with closed caudal ends and Pantopaque-CSF interphase.
Case 3

This 24-year-old man was admitted in 1968 for evaluation of back and bilateral leg pain. In 1966, he had developed low back and right leg pain with numbness in the right foot following a parachute jump. At that time a herniated disc was removed from the L-5-S-1 area on the right but because of persistent pain, the area was re-explored and recurrent disc material removed. The L4-5 disc level was also examined and thought to be normal. He had been discharged from the hospital in 1967 with persistent back and bilateral leg pain, weakness of the dorsiflexors of the right ankle, and sensory loss in the L-5 and S-1 dermatomes of the right leg. He improved slightly but in 1968 had a recurrence of severe low back and bilateral pain in the sciatic distribution. Because of the persistent pain and progressive weakness of the right ankle dorsiflexors and mild urinary hesitancy, a myelogram was performed which demonstrated irregularity of the Pantopaque column consistent with arachnoiditis, a probable lateral defect at L4-5 on the right side, and a subdural collection of Pantopaque at L-3. Exploration of the L-5 nerve root demonstrated dense scar tissue compressing the dura with no evidence of disc material compressing the root. Little improvement occurred, and in 1969, he noted severe shock-like pains radiating down both legs when coughing or bending forward. An area of numbness developed on the right anterior lateral thigh. Urinary hesitancy persisted.

Examination. Anal sphincter tone was decreased, and a healed midline operative site in the lower lumbar area when gently compressed caused a shooting pain to go into both legs. Decreased pinprick sensation on the dorsum of the right foot and over the right anterior lateral thigh was associated with mild weakness of the right peroneal, posterior tibial, and gastrocnemius muscles. Reflexes were normal. Cerebrospinal fluid contained no cells, and its protein content was 21 mg%. Lumbar myelogram demonstrated a marked irregularity of the contrast medium column from the L-3 through the S-1 interspaces thought to be secondary to arachnoid and epidural scarring (Fig. 3).
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The most striking finding was the presence of a dorsally located diverticulum-like structure communicating with the subarachnoid space which compressed the cauda equina causing an incomplete block of the spinal canal at the level of L-3. The diverticulum could be emptied and filled with contrast material by altering the patient's position. Review of the 1968 myelogram revealed that the dorsal collection of Pantopaque thought at that time to have been subdural in location was indeed the diverticulum described above.

Operation. A complete L-3 and partial L-2 laminectomy was performed and the dura opened. The arachnoid was thickened and contained within it a diverticulum which was closed at its caudal end. This diverticulum was excised and the dura closed.

Postoperative Course. The patient had no sharp back or leg pain and had only minimal aching in the right buttock region. Urinary hesitancy disappeared, strength in the ankle returned to normal, and the area of sensory disturbance in the right thigh disappeared completely. Five months postoperatively, there had been no further improvement.

Case 4

A 59-year-old woman was admitted to Parkland Memorial Hospital complaining of midlumbar pain of 1 year's duration radiating to the left side, occasional pain in her right leg below the knee, and weakness of the right knee and ankle. Six years previously she had injured her back and had been hospitalized for several days with no apparent neurological involvement.

Examination. There was hyperreflexia in the left leg and decreased sensation in the right leg maximally below the knee suggestive of a stocking-type deficit. Thoracic spine films raised the possibility of decreased mineralization of the pedicles on the left at T9-10. Myelography demonstrated at the T7-8 level an arachnoid diverticulum which was not visualized until the patient was placed in the supine position.

Operation. Complete laminectomy at T-7 through T-9 was carried out with the excision of the diverticulum.

Postoperative Course. The patient was free of the radicular pain, and follow-up examination 1 month later revealed normal deep tendon reflexes in her legs with no sensory disturbance.

Discussion

The incidence of arachnoid diverticula is hard to assess. Only 32 cases of symptomatic arachnoid diverticula have been described in the current literature, with 12 of these in a single report, but it is likely that arachnoid diverticula are much more frequent than the number reported. Discussions with several radiologists and neurosurgeons who have seen similar myelographic abnormalities suggest that in many instances the diverticula cause no signs or symptoms of spinal cord dysfunction. We have observed small diverticula on two occasions as incidental findings. However, when clinical findings suggest the presence of an intraspinal space-occupying lesion, the finding of an arachnoid diverticulum must not be dismissed. The etiology of these diverticula is difficult to ascertain. Trauma is one of several possible etiological agents. Three of the four patients presented here had a history of some degree of back injury. The friability of the arachnoid membrane lends itself to easy tearing, and local areas of weakness produced by trauma with an associated inflammatory response may result in formation of diverticula.

Positive contrast myelography is the only method of diagnosing diverticula preoperatively. The examiner stands a better chance of demonstrating these lesions by the use of fairly large amounts of Pantopaque, at least 15 to 20 cc. Since most myelograms are performed with the patient in a prone position and since the diverticula rest on the dorsal aspect of the spinal cord, they may fill incompletely or not at all. In the absence of initial positive myelographic findings in a patient with spinal cord dysfunction, it is imperative that the patient be examined in the supine position. The dependency of the diverticula will allow them to fill readily when the patient is supine, and both anteroposterior spot films and lateral views of the thoracic region will demonstrate their extent. In an upright position, contrast material will fill the most dependent part of the diverticulum and the cerebrospinal fluid-Pantopaque interface will be demonstrated. In the prone position, the diverticula will be seen to empty. This radiographic observation cor-
relates with the partial relief of symptoms some patients experience when changing from upright to recumbent positions.

Review of symptomatic cases in the literature suggests that, in a vast majority, symptoms caused by such diverticula are present for a period of months or even years prior to diagnosis. Three of the cases we are reporting had progressive symptoms extending over a period of months. The other case presented signs of acute spinal cord dysfunction which required immediate diagnosis and treatment.

The treatment of choice is surgical excision of the diverticulum located at the level corresponding with the clinical level of spinal cord involvement. In the second case, there were several smaller diverticula seen myelographically which were not excised. Whether with time smaller diverticula enlarge and cause recurrence of spinal cord dysfunction is not known. The prognosis of symptomatic diverticula treated surgically seems to be dependent upon the duration of loss of neurological function. The sooner the diagnosis is made and surgical treatment instituted, the less chance there is for development of irreversible myelopathy.

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References


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