Traumatic arachnoidal diverticulum associated with paraplegia

Case report

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A case of delayed paraplegia due to a traumatic arachnoid diverticulum from a traction injury of the brachial plexus is reported. The authors emphasize the necessity of carrying out proper radiological studies for evaluation of delayed weakness of the legs following trauma to the brachial plexus; by this means, a surgically correctable lesion can be identified.

KEY WORDS: paraplegia • trauma • arachnoidal diverticulum • brachial plexus

An intraspinal cystic meningeal diverticulum due to avulsion injury of the brachial plexus has been previously reported. We are reporting here a unique situation in which a traumatic arachnoidal diverticulum was responsible for the delayed onset of paraplegia.

Case Report

This 12-year-old boy was hospitalized on September 3, 1970, for paraplegia and progressive truncal weakness. At the age of six he was unconscious for several months following an auto accident. Subsequent to that time he had had speech impairment and weakness of his left arm. Five years after the accident both legs became progressively weaker.

For six months prior to admission he was unable to walk or move his legs.

Examination. The patient had slow monotonous speech. Marked extensor weakness, contractures, and atrophy were present in the left arm. There was no voluntary movement of either leg and only slight movement could be elicited in response to painful stimulus. Perception of pinprick and light touch was absent bilaterally below T-7 with the exception of pinprick perception of plantar surface of both feet. Position sense was absent in the left foot. The left triceps reflex was diminished in contrast to the right. The left biceps reflex was absent. Deep tendon reflexes were increased in both legs, particularly the right. There were bilateral extensor plantar responses.

After admission a Foley catheter was inserted because of urinary retention. Cystometry was indicative of a spastic neurogenic bladder. Skull films were normal, as was the brain scan. X-ray films of the spine showed a widening of the spinal canal and loss of the pedicles in the lower cervical and upper thoracic region in the anteroposterior projection (Fig. 1). Myelography by the lumbar route was attempted. Only 3 cc of xanthochromic spinal fluid could be obtained; this had a protein content of more than 1.0 gm% and a
Fro. I. Anteroposterior film of the cervical and upper thoracic spine. Note the widening of the interpedicular distance over segments C-5 through T-1 and loss of the pedicles over the same distribution on the left side. The effect of rotation is taken into account.

cell count of 142 WBC and 390 RBC/mm$^3$ with 38% polymorphonuclear and 67% mononuclear cells. There was no manometric response to jugular compression. At fluoroscopy, 3 cc of Pantopaque, inserted into the lumbar arachnoid sac, was found to be broken up into multiple clumps in linear orientation that passed no further cephalad than T-6. It was thought that the patient had chronic adhesive arachnoiditis with a complete block, and lysis of adhesions was undertaken.

First Operation. On September 21, 1970, a total laminectomy was carried out from T-11 to T-6. There was some extradural fibrous tissue and blockage to the passing of the catheter beyond the T-6 lamina. The dura was incised and the arachnoid was

Second Operation. On October 6, 1970, total laminectomy was carried out from T-5 through C-5. A large pulsating, thin-walled saccular mass was adherent to the posterior border of the dura in the inferior part of the exposure. More cephalad, it lay along the left side of the dura and was indented anteriorly by the lateral extensions of the nerve root sleeves. The mass was dissected off the dura in a caudad to cephalad direction. The sac communicated with the dura at the C-5 nerve root sheath and it appeared that the entire sac had grown from this source (Fig. 3). The dura overlying the spinal cord was not opened. The sac was removed in toto after separation from its communication at the C-5 nerve root sheath. No nerve root was found within the sheath. Following removal of the sac, the underlying dura of the cervical and thoracic cord immediately began to enlarge and pulsate. The dural leak was sutured and the wound closed in layers.
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Fig. 2. Cisternal myelogram. **Left:** Anteroposterior view shows a large arachnoid cyst overlying the midline. A fluid level is present in the sac. The lower level of the sac lies opposite the T-4 vertebral body. **Right:** A sequential view shows a cephalad extension of the same cyst communicating near its upper pole with the intact arachnoid sac. Note the lobulated appearance of this portion of the cyst and its projection laterally through the intervertebral foramina at several levels. There is also indentation of the sac by the nerve root pouches lying outside it.

Fig. 3. Artist's drawing showing the communication of intact meninges with the arachnoidal diverticulum through the C-5 nerve root sleeve.
FIG. 4. Anteroposterior view from postoperative fluoroscopic study showing flow of the residual Pantopaque without block. There is no evidence of the preoperative arachnoid cyst. Note the absence of pedicles on the left side.

Postoperative Course. Fluoroscopy of the spine several weeks later showed normal flow of residual Pantopaque without block. The cystic structure and its connection with the extra arachnoid space were no longer demonstrable (Fig. 4). By October 23 the patient was moving his legs voluntarily for the first time, and over the next several days there was dramatic improvement in the strength of both legs. General improvement in the sensory deficit occurred and the sensory level at T-7 disappeared. He was discharged on October 29, 1970, and in less than 1 month he was walking with help. At the latest follow-up examination 6 months after discharge, he was walking unassisted. The Foley catheter was removed just before he was discharged; he has had no difficulty with voiding since that time.

Discussion

The traumatic diverticula seen on myelography have been important aids to confirmation of avulsion injuries of the brachial plexus. They have been given various names including traumatic meningocele, posttraumatic meningocele, pseudomeningocele or false meningocele, traumatic diverticulum or traumatic diverticulum, arachnoidal diverticulum, true diverticulum, false diverticulum, and avulsion pocket. These defects may be multiple, and when the diverticulum communicates with a cystic accumulation in the spinal canal extending axially for one or more segments, it has been termed traumatic arachnoid cyst, arachnoid cyst, or subarachnoid cyst. However, this term is commonly used to describe an encapsulated collection of fluid in the pia arachnoid due to head injury. For the purposes of clarity the large intraspinal sac described in this case is termed a traumatic arachnoid diverticulum.

The rare congenital extradural and intradural arachnoid cyst in some respects resembles this lesion. However, the background of trauma, the lack of associated malformations of the spine, and the origin of the cyst from a cervical nerve root sheath argue against a congenital etiology. The most likely cause of the neurological deficit corresponding to spinal cord compression in this case is from the effect of pressure or traction on the spinal cord exerted by the distended cyst. The progressively increasing neurological deficit is attributed to the fact that the cyst apparently enlarges over a period of time. The only cases of delayed spinal cord compression associated with a brachial plexus injury have been those due to a tethering effect on the spinal cord by fibrous adhesions. However, the presence of a traumatic intraspinal cyst had been postulated as a cause of spinal cord compression.

This is the first documented case report of such a pressure effect from a traumatic cyst. The confirmation of this fact is reflected in a resolution of neurological deficit following removal of the cyst.

References

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