Surgical Treatment of Post-traumatic Myelopathy Associated with Syringomyelia

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Study Design. Retrospective review

Objective. Evaluate the clinical outcome of surgical intervention for post-traumatic syringomyelia.

Introduction. Progressive post-traumatic cystic myelopathy (PPCM), or syringomyelia, can occur after spinal cord injury. The authors present their surgical treatment protocol and treatment outcome of a series of patients with post-traumatic syringomyelia.

Methods. The medical records of 53 patients with PPCM undergoing surgical treatment were reviewed. Laminectomies and intraopertive ultrasonography were performed. For patients with no focal tethering and only a confluent cyst on ultrasonography, a syringosubarachnoid shunt (stent) was inserted. For patients with both tethering and a confluent cord cyst, an untethering procedure was performed first. When a cyst showed significant size reduction (>50%) after untethering, no shunt was placed. When the cyst size persisted on ultrasonographic images, a short syringosubarachnoid shunt was used. The mean follow-up was 23.9 months for the 45 patients available for follow-up (range 12 – 102 months).

Results. The interval between the causative event and the operation was from 5 months to 37 years (mean 6.5 years). Pain was the most frequent manifestation, followed by motor deterioration and spasticity. Postoperative improvements in >50% of the patients were noted in those presenting with worsening motor function or spasticity. In 19 of 28 patients with associated tethered spinal cord, untethering alone caused significant collapse of the cyst. Postoperative MRI demonstrated cyst collapse in 95% of the patients with untethering alone and 93% of the patients with a syringosubarachnoid shunt.

Conclusion. Post-traumatic syringomyelia can occur with or without cord tethering. Untethering alone for patients with cord tethering and cyst formation can reduce cyst size and alleviate the symptoms and signs of syringomyelia in the majority of these cases. Untethering with expansion of subarachnoid space with an expansive duraplasty may be a more physiologic way of treating a tethered cord with associated syringomyelia, i.e., treating the cause rather than the result. [Key words: syringomyelia, progressive post-traumatic cystic myelopathy (PPCM), progressive post-traumatic myelomalacic myelopathy (PPMM), spinal cord injury] Spine 2001; 26: S119-S127

Editor's Comments:

The authors have written an article having spent considerable time and effort to review both their case experience as well as the literature noting the problems that occur in patients with spinal trauma who develop syringomyelia and cord tethering.

Longer survivals in spinal injured patients have allowed for more complications to occur and be discovered. The authors note that the differential diagnosis of a progressive neurological deterioration can occur in many settings after a traumatic episode: progressive spinal column deformity or instability, tumor infringement, post-radiation myelitis, transverse myelitis, late complications of spinal infections (TB), iatrogenic causes including arachnoiditis, the development of a new disease involving the spine, spinal cord injury complicating a chronic illness, presentation of a congenital disorder (Arnold Chiari, or tethered cord), progressive degeneration of the spine with spondylosis and disc disease. Often, the cause of the progressive deterioration is evident from a

common problem, but once eliminated suspicion may fall on PPCM (progressive post-traumatic cystic myelopathy) or PPMM (progressive post-traumatic myelomalacic myelopathy.

The 53 patients consisted of 2/3 men, average age 45.6 years, with blunt trauma the most common cause and mainly from motor vehicle accidents. The neurologic deterioration or "new pain" syndrome occurred about 11 years after the initial insult with one case 41 years later. Most had a "new pain" syndrome, motor function level deterioration, increased spasticity, and, about half, had sensory function or progressive paresthesias. About half the patients evidenced cord tethering.

MRI scanning, CT scan with intrathecal contrast, cine-MRI, and intraoperative ultrasound are diagnostic studies that permit differentiation of these entities, syrinx with or without tethering, or myelomalacia.

Treatment of a post-traumatic syrinx is usually a shunt, fenestration, or expansile duraplasty, while treatment of PPMM may include release of the tethered cord and/or release of adhesions. Unfortunately, the clinical outcomes in either diagnosis have been disappointing with shunt failures of 50%.

The authors outline their treatment algorithms for post-traumatic syrinxes. Surgery for a syrinx without tethering consisted of laminectomy, ultrasound localization of the syrinx, dural opening, and syringe-subarachnoid shunting. In some cases, extracavitary shunting was done. When tethering was present, adhesions were lysed microscopically and the syrinx collapse followed by ultrasound. If not more than a 50% collapse of the syrinx was achieved, a shunt or stent was placed followed by water-tight duraplasty.

The results in 53 patients included at least one-year follow-up in 43 patients, with 73% having satisfactory results, considered to be resolution of one or more of the presenting symptoms or signs). 7% had worsening of motor or spasticity. Generally, improvement correlated with post-surgical reduction on MRI of cyst size. Surgical complications were few and the authors are to be congratulated in this regard, as this is tricky surgery in an already compromised patient coterie. Complications occurred in the shunt-treated group only: shunt failure requiring revision, ventral cord tethering, transient weakness, and CSF leak with pseudomeningocele.

The authors point out the not uncommon occurrence of tethering with post-traumatic cyst development and observe that it is due to scar and adhesions that change local CSF dynamics. They feel, as others in the literature have noted, that post-traumatic myelomalacia and syrinx formation may be in a continuum. Other factors of causation include traction, ischemia, and cord stretching. The authors allay their better-than-expected results on treatment beyond simple cyst shunting including lysis of adhesions and relief of CSF blockage. They conjecture that prevention of this problem may start with post-injury prompt spinal column realignment and cord decompression followed by prompt mobilization including the use of a RotoRest bed. The authors also note the difficulty of ventral cord adhesion lysis and a need to deal with this problem.

Overall, this study has reviewed this “niche” subject well and provided the reader with a treatment scheme that improves on what has been done. Physicians will have a better understanding of this subject and be alerted to what can cause post-traumatic neurological deterioration in spinal injured patients and recognize what can be done to help them.

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