A 66-year-old man suffered a back injury in a car accident resulting in paraplegia in 1968. Patient underwent a L1-2 lumbar laminectomy and participated in a prolonged rehabilitation program regaining the ability to walk with minimal assistance. Eight years later he developed intermittent paresthesias in the left lower quadrant area of his abdomen while sneezing. During a period of five years, his paresthesias progressed from intermittent to daily, and were associated with dull pain, which ascended over the anterior and posterior left hemithorax, to left arm, left side of the neck, and left angle of the jaw. The pain often was described as incapacitating and electrical, lasting for seconds, and primarily while sitting or standing.

The pain was refractory to a variety of antiepileptics, tricyclics, and narcotics. He did not have facial paresthesias, dysphonia or dysarthria. In 2009 he underwent a C5-6 anterior cervical discectomy and fusion (ACDF) for intractable neck pain radiating to the left arm which was unsuccessful. The neurological exam showed minimal weakness of bilateral hip flexor muscles, right knee flexor and foot dorsiflexor muscles. Reflexes were absent bilaterally. Vibration, pinprick, and temperature were reduced in both legs extending to his left elbow and right hand. Gait was ataxic. Cranial nerves were intact. Mentation was normal.

Mid sagittal T2-weighted magnetic resonance imaging (MRI) of the cervical (a), thoracic (b), and lumbar (c) spine without gadolinium showed a pathological cavitation (syrinx) extending along the entire spinal cord. Cervical MRI (a) showed C5-6 ACDF. Lumbar MRI (c) showed a fracture of L1 and L2 vertebral bodies with compression of the conus medullaris and syrinx cavity. Axial T2-weighted MRI of thoracic spine (d) showed a left paracentral syrinx.

DISCUSSION

Post-traumatic syringomyelia (PTS) is a fluid filled cavity within the spinal cord resulting from spinal cord injury (SCI). Symptomatic PTS is a rare complication from SCI present in one to four percent of SCI victims, however asymptomatic syrinx is more prevalent and likely represents a focal area of liquefaction necrosis of the spinal cord. In a retrospective review of SCI patients who underwent routine spine MRIs on admission, the presence of symptomatic or asymptomatic PTS was reported to be 51 percent. The majority had cervical syrinx, but no patient had a syrinx involving the entire spinal cord (pan-syringomyelia). In a different review of 21 SCI patients with PTS, only one patient showed pan-syringomyelia.

PTS primarily affects young men and commonly presents with segmental pain and sensory loss. The most common physical finding is spasticity, which highly correlates with the presence of syrinx. Weakness and less likely sphincter dysfunction can also be present. Pain is often aggravated with positional changes or Valsalva maneuvers.
Symptoms may start months to decades after the SCI and progress over time.\(^1,5,6\) PTS is mainly located at extracanicular central or dorsolateral gray matter, watershed region, or less likely in the white matter alone. PTS occurs in proximity to the injury and expands rostrally, caudally or in both directions in 81 percent, 41 percent, and 15 percent of the cases, respectively.\(^1\)

The pathophysiology of PTS is poorly understood. Scarring resulting in obstruction of CSF flow and altered tissue compliance can contribute to expansion of the central canal with compression of the surrounding cord.\(^1\) Spinal cord inflammation, edema, hematoma, liquefaction, intracellular lysosome-content release, ischemia, necrosis, arterial and venous occlusion may all in different degrees participate to form a syrinx. Trauma may cause spinal deformities; spinal stenosis, kyphosis, arachnoid adhesions, and tethering of the spinal cord to the dura also contributing to the syrinx formation. Once a syrinx is formed it is unclear how it expands, but imbalances between fluid inflow and outflow caused by one-way valve effect where CSF can enter, but not exit out of the syrinx plus CSF flow turbulence\(^1,5-7\) may explain the expansion. Several risk factors for progression of syringomyelia include arachnoiditis, spinal stenosis, cord compression, and kyphosis.\(^2\)

Treatment is usually unsatisfactory. Progression or stabilization often occurs with either conservative or surgical therapy. Surgery may be considered in progressive cases or early in the syrinx formation to attempt to arrest or improve clinical findings. The goal of treatment is to reduce the expansile intracystic pressure and improve CSF flow.\(^8\) Surgical techniques used are correction of causative deformity or compressive lesion, arachnolysis, cyst fenestration, dural augmentation, release or lysis of spinal cord tethering, and various shunt placements. Surgery results are mixed and patients may deteriorate in spite of the surgical intervention, independent of whether imaging studies show reduction in cavity size.\(^1,9\) Shunt failure is common. Pain may be more responsive to surgery than motor symptoms.\(^6\)

PTS is a serious complication from SCI associated with severe pain, sensory and motor findings that start months or years after an SCI. The presence of spasticity highly correlates with PTS in SCI victims. PTS may develop only at the site of the lesion or most rarely expand throughout the whole spinal cord. Treatment remains unsuccessful in significant number of cases. Treatment aimed at altering the pathophysiology of cyst formation may be more efficacious than procedures aimed at simply draining the cyst.\(^8\)

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