



Late decompression in a patient with post-traumatic syringomyelia

Travma sonrasında siringomiyeli gelişen bir olguda geç dönemde dekompresyon

Tarik YAZAR, Kerem BASARIR, Alihan DERİNCEK

Ankara University Faculty of Medicine, Department of Orthopedics and Traumatology

Siringomiyeli omurilik yaralanmasının nadir görülen, ancak ciddi olabilen bir komplikasyonudur. Bu yazıda, travmadan 14 yıl sonra tanı konmuş ve bundan üç yıl sonra dekompresyon yapılmış bir kadın hasta sunuldu. Dekompresyon anterior yaklaşımla herhangi bir komplikasyon olmadan gerçekleştirildi ve üç yıllık izlem süresi içinde hastanın nörolojik semptomlarında gerileme ile tatmin edici sonuç elde edildi. Dekompresyonun, nöral bası oluşmasından yıllar sonra yapıldığında bile faydalı olduğu görüldü. Semptomlara neden olan durumun doğru saptanması tedavinin doğru planlanması için gereklidir.

Anahtar sözcükler: Nöral ileti; omurilik yaralanması/cerrahi/ komplikasyon/radyografi; siringomiyeli/etioloji/tanı/cerrahi.

Syringomyelia is an uncommon, but disabling complication of spinal cord injuries. The authors presented a female patient with syringomyelia, in whom diagnosis was made 14 years after the initial trauma and decompression was performed three years thereafter. Decompression was carried out via the anterior approach without any complication. The result at the end of a three-year follow-up was satisfactory with improvement in her neurologic symptoms. This case indicates that decompression can be of benefit even though it is performed late after the occurrence of neural compression and emphasizes the need for a thorough examination of the symptoms before planning treatment.

Key words: Neural conduction; spinal cord injuries/surgery/complications/radiography; syringomyelia/etiology/diagnosis/surgery.

Syringomyelia is a progressive cystic lesion of spinal cord which might cause neurological symptoms. Estienne defined syringomyelia in 1546 and 400 years later (at 1973) Barnett and Rewcastle^[1] put forward the classical classification based on etiological measurements. According to this classification there are five types of syringomyelia named: communicant, posttraumatic, tumor related, arachnoiditis related and idiopathic. Most common clinical symptoms are loss of sense, radical pain, spasticity, weakness of certain muscle groups, dysfunctions of urinary bladder and autonomic disorders like hyperhidrosis.

The incidence of posttraumatic syringomyelia is reported as %0.9-3 by various authors.^[2-7]

Posttraumatic syringomyelia is the late-term sequel of spinal cord injuries. It was first observed by Bastian. Although cyst formation is observed in half of the patients with spinal cord injury, fluid retention of the cyst and syrinx formation is observed only 4% of these cases. Posttraumatic syrinx is filled with a fluid similar to cerebrospinal fluid. Its length may vary from 0.5 cm to the whole length of spinal cord.^[1]

The mechanism of syrinx formation after trauma is not well-known. Focal necrosis and fluid gradient at wound site are put forward as etiology.^[4,8,9] Neurological signs and symptoms may be observed months or even years after the trauma in clinical settings. The level of these signs is in consistency with the trauma or the syrinx.^[10, 11] The appearance of

these slowly developing signs and symptoms depends on the activities which increase the intra-abdominal pressure, like coughing, sneezing and injuries causing acute tension.^[10-12] The most frequent symptoms are pain, paresthesia, weaknesses, spasticity or autonomic problems.^[10] The investigation of spinal cord by magnetic resonance imaging (MRI) facilitates the diagnosis of syringomyelia.^[1, 11, 13-16] Similarly myelographic computerized tomography is used for diagnosis.^[4, 10] Electromyography, somatosensory evoked potential and neuronal transmission studies are used to determine the size of neuronal damage.^[17] The need of surgical treatment for symptom relief is a topic which is agreed on.^[1,3,6,7,9,18]

Case report

At 1981, a 25 year-old female who admitted to the hospital due to low back pain consequent to falling from high and a burst fracture at L₂ level was observed by direct radiograms. Paraplegia and urinary incontinence which were observed in the admission healed in one month by posterior approached decompression and fixation with Harrington bar. Sensorial functions were protected partially below the lesion and improved to ASIA D postoperatively from ASIA B. Patient could walk without any tool aid 3 months after the operation. Bars were removed by a second operation 3 years later the first one. No symptoms were observed during following 12 years. Patient gave birth to her 2 children in normal ways, without any peripartal complications.

12 years later the first trauma patient were treated conservatively by bed-rest and analgesics in another center for about one year for back and shoulder pain due to a out-vehicle traffic accident. Urinary incontinence was evaluated as stress incontinence and followed conservatively. At the examination hypoesthesia at L₂ dermatoma, hypoactive deep tendon reflexes at upper and lower extremities, weakness of lower extremities dominant at the right was observed. Weakness and loss of sensation were classified as ASIA C. At computerized tomography, fracture line through posterior was observed at L₂ vertebra. Also, syringomyelia was observed in spinal channel between C₄-L₁ in MRI (figure 1, 2a). It is thought that the clinical features were due to

syringomyelia and multilevel laminectomy between T₁₂-L₂, right lateral myelotomy and catheterization of subarachnoidal space for drainage of the cavity of syrinx were performed. Cerebrospinal fluid fistula formed postoperatively was treated conservatively. Preoperative symptoms did not decreased and also, falling of right foot was added to the symptoms. High dose of steroid given in postoperative period was ended because the pain occurred at hip, knee and ankle was thought to originate from massive osteonecrosis based on the results of MRI. Transchondilar drilling was performed to the left knee for the treatment of osteonecrosis in the following year.

Patient admitted to our hospital at 1999 for the second time. Although neurological symptoms were established, anterior decompression for bone pressure on spinal cord, partial corpectomy and anterior stabilization were performed (figure 2b). During partial corpectomy low frequency digger was used and the space formed was filled with autograft taken from anterior iliac wing.

Motor and sensorial improvements were observed beginning at the first week postoperatively. Patient was mobilized with crutches at the postoperative first week. After the intensive physiotherapy and rehabilitation lasting 6 months, patient could walk without any tool aid. At neurological examinations performed at postoperative 6th month and 3rd year, mild weakness at right lower extremity related to the left one and hypoesthesia at L_{4,5} der-

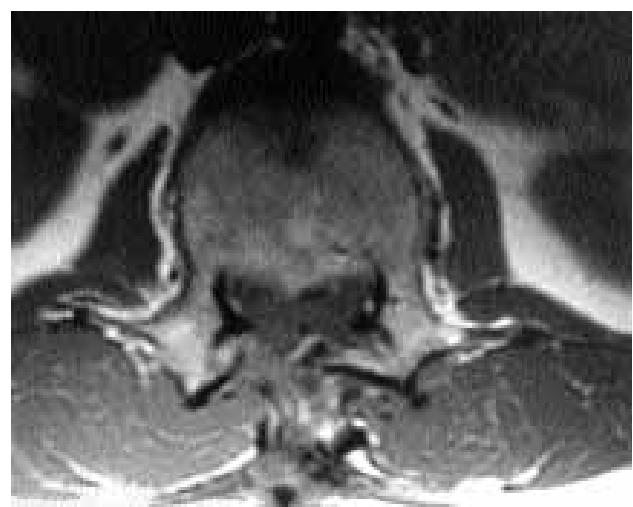


Figure 1. Fragment causing narrowing at spinal channel at axial MR section.

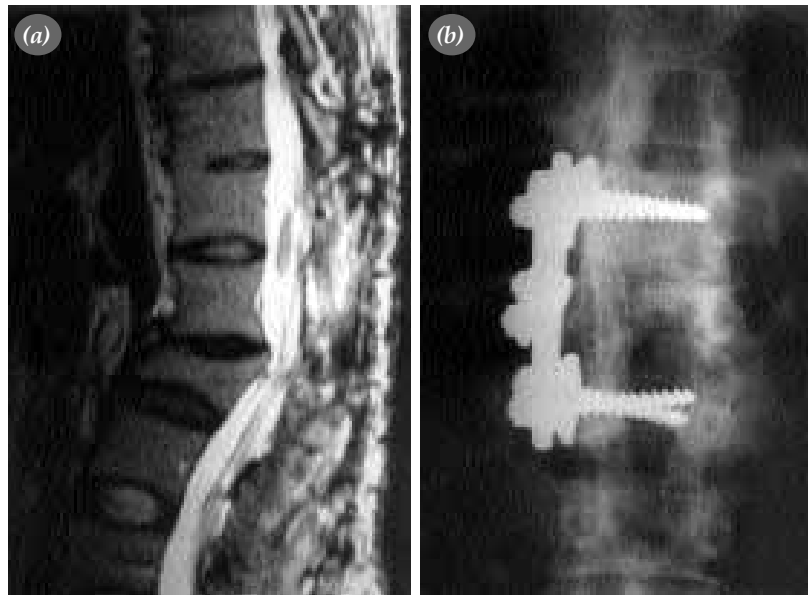


Figure 2. (a) Sagittal MR section showing cystic cavitation
(b) Anteroposterior x-ray image after anterior stabilization.

matomas were observed. Neurological symptoms were classified as ASIA D. In electromyography, chronic C_7 and L_5 lesions were observed. Patient was able to perform daily living activities like sitting, standing up without aid and dressing and was satisfied with the results of the operation.

Discussion

Postrumatic syringomyelia was defined by Bastian for the first time at 1867 and the first clinical study was performed by Holmes at 1915.^[1] Important information about its physiopathology was obtained from the experimental studies of Freeman and Wright and same authors reported about the successfully treated patient at 1959.^[7] Williams et al^[7] explained the syrinx formation due to the prevention of cerebrospinal fluid circulation by the posttraumatic scars and attachments, resulting continuous pulses at epidural venous system. Same authors mentioned that coughing (like Valsalva maneuver) even in healthy subjects might cause increase in pressure on the epidural venous net. The sudden increase in symptoms due to coughing may be explained by this mechanism. As surgical procedures do not cause serious morbidity, there is a consensus on the need of surgical interventions for the patients having resistant pain and neurological symptoms.^[1,3,6,9,18]

Reported case is important because it shows that late surgical treatment can also significantly improve the symptoms. For the decision of operation, syringomyelia was differentiated as the reason for the clinical features and multilevel laminectomy which was performed before for treating syringomyelia was evaluated as a cause for possible kyphosis and narrowing at the spinal channel. After decompression at L_{1-2} by anterior approach, symptoms were improved.

As a result, posttraumatic syringomyelia is rarely symptomatic. We think that the determination of the origin of the symptoms is the most important factor for the most appropriate approach for the treatment.

References

1. Barnett HJ, Rewcastle NB. Syringomyelia and tumours of the nervous system. In: Barnett HJ, Foster JB, Hudgson P, editors. Syringomyelia. Major problems in neurology. London: W. B. Saunders; 1973. p. 261-301.
2. Griffiths ER, McCormick CC. Post-traumatic syringomyelia (cystic myelopathy). Paraplegia 1981;19:81-8.
3. Rossier AB, Foo D, Shillito J, Dyro FM. Posttraumatic cervical syringomyelia. Incidence, clinical presentation, electrophysiological studies, syrinx protein and results of conservative and operative treatment. Brain 1985;108(Pt 2):439-61.
4. Watson N. Ascending cystic degeneration of the cord after spinal cord injury. Paraplegia 1981;19:89-95.
5. Rossier AB, Foo D, Shillito J, Naheedy MH, Sweet WH, Dyro F, et al. Progressive late post-traumatic syringomyelia. Paraplegia 1981;19:96-7.

6. Shannon N, Symon L, Logue V, Cull D, Kang J, Kendall B. Clinical features, investigation and treatment of post-traumatic syringomyelia. *J Neurol Neurosurg Psychiatry* 1981; 44:35-42.
7. Williams B, Terry AF, Jones F, McSweeney T. Syringomyelia as a sequel to traumatic paraplegia. *Paraplegia* 1981;19:67-80.
8. Doyle K, Wilmot C, Hall KM, Cooper PR, Fadden AI. Trauma of the spine and spinal cord. In: Joynt RJ, Griggs RC, editors. *Baker's clinical neurology*. Vol. 3, Philadelphia: J. B. Lippincott; 1994. p. 41-2.
9. Vernon JD, Silver JR, Symon L. Post-traumatic syringomyelia: the results of surgery. *Paraplegia* 1983;21:37-46.
10. Kramer KM, Levine AM. Posttraumatic syringomyelia: a review of 21 cases. *Clin Orthop* 1997;(334):190-9.
11. Milhorat TH, Johnson WD, Miller JI, Bergland RM, Hollenberg-Sher J. Surgical treatment of syringomyelia based on magnetic resonance imaging criteria. *Neurosurgery* 1992;31:231-44.
12. Barnett HJ, Jousse AT. Post-traumatic syringomyelia (cystic myelopathy). In: Vinken PJ, Bruyn GW, editors. *Handbook of clinical neurology*. Vol. 26, Amsterdam: North-Holland Publishing; 1976. p. 113-57.
13. Kokmen E, Marsh WR, Baker HL Jr. Magnetic resonance imaging in syringomyelia. *Neurosurgery* 1985;17:267-70.
14. Pojunas K, Williams AL, Daniels DL, Haughton VM. Syringomyelia and hydromyelia: magnetic resonance evaluation. *Radiology* 1984;153:679-83.
15. Sherman JL, Barkovich AJ, Citrin CM. The MR appearance of syringomyelia: new observations. *AJR Am J Roentgenol* 1987;148:381-91.
16. Vaquero J, Martinez R, Arias A. Syringomyelia-Chiari complex: magnetic resonance imaging and clinical evaluation of surgical treatment. *J Neurosurg* 1990;73:64-8.
17. Dyro FM, Rossier AB. Electrodiagnostic abnormalities in 15 patients with posttraumatic syringomyelia: pre- and postoperative studies. *Paraplegia* 1985;23:233-42.
18. Lyons BM, Brown DJ, Calvert JM, Woodward JM, Wriedt CH. The diagnosis and management of post traumatic syringomyelia. *Paraplegia* 1987;25:340-50.