



The healing potential in cauda equina syndrome secondary to traumatic posterior L5-S1 dislocation A case report with 16 years follow-up

Oguz CEBESoy, Kamil Cagri KOSE, Tarik YAZAR

From Gaziantep University Faculty of Medicine, Gaziantep, Turkey

Cauda equina syndrome is the result of any lesion that compresses or paralyzes cauda equina roots which are both motor and sensory. It is an uncommon syndrome, which features low back pain, sciatica, variable lower extremity motor and sensory loss with possible bladder and bowel dysfunction. It is an emergency situation as it may cause significant morbidity such as permanent paralysis, impaired bladder and/or bowel control or loss of sexual sensation. We present the case of a patient who was admitted to the emergency department with a traumatic posterior L5-S1 dislocation, low back pain and bladder dysfunction 8 days following an initial trauma. Open L5-S1 reduction and posterior stabilization was performed and the dural sac was decompressed. Most of the patient's neurological deficits resolved over several years, following the initial surgery.

Keywords : cauda equina syndrome ; late decompression ; surgical outcome.

cause loss of sensation and motor functions (1, 14). Cauda equina syndrome is uncommon, and a complete neurologic examination including sensation of the perineal area and sphincter function is very important for early diagnosis. Cauda equina nerve roots are susceptible to injury since they have poorly developed epineurium to protect the nerves against tensile and compressive stresses (1, 14). Its possible complications make cauda equina syndrome a surgical emergency. The timing of surgical intervention is critical in terms of outcome. There are reports in the literature showing the benefit of early surgical decompression for early return of function (1, 14). However the literature lacks information about the results of late decompression in this clinical setting. The clinical picture in cauda equina syndrome commonly involves low back

INTRODUCTION

The cauda equina is formed by the nerve roots caudal to the level of medulla spinalis termination. It contains peripheral nerves, both sensory and motor, below the level of the conus medullaris within the spinal canal. Conus medullaris contains the myelons of the five sacral nerve roots. Injury to any part of this structure may result in cauda equina syndrome. Injury to these nerves may

■ Oguz Cebesoy, MD, Assistant Professor.
Gaziantep University Faculty of Medicine, Gaziantep, Turkey.

■ Kamil Cagri Kose, MD, Assistant Professor.
Afyon Kocatepe University Faculty of Medicine, Afyon, Turkey.

■ Tarik Yazar, MD, Professor.
Ankara University Faculty of Medicine, Ankara, Turkey.

Correspondence : Oguz Cebesoy, MD, Gaziantep University Faculty of Medicine, Gaziantep, Turkey.

E-mail : ocebey@yahoo.com and ocebey@mynet.com.

© 2007, Acta Orthopædica Belgica.

pain, saddle and/or perineal hypoaesthesia or anaesthesia, a decrease in anal tone, absent ankle, knee, bulbocavernous reflexes and bowel and/or bladder dysfunction (9, 13). Careful physical examination and early diagnosis may affect the outcome of this syndrome (1, 10, 14). Several causes of cauda equina syndrome and their outcomes have been reported in the recent literature, including those from traumatic injuries (2-5), central disc protrusion (8), metastatic invasion (9), schwannoma (6), pneumococcal meningitis (7), Paget's disease (11). All are possible causes of cauda equina syndrome. Acute cauda equina syndrome secondary to traumatic injury is very rare in the literature and so the knowledge about the healing potential of this entity is unknown. We report a case of traumatic cauda equina syndrome with sixteen years follow-up after the operation.

CASE REPORT

A 33-year-old male patient was referred to our department from a state hospital 8 days after an injury. A stone block had hit his back during a mine explosion and he was first sent to a local state hospital where he received initial medical care. He did not receive any specific treatment (neither medical, nor surgical) for his spinal problem during that time. Eight days following the injury, he was referred to our center for further treatment. His initial examination revealed severe low back pain and a low back deformity in addition to bilateral paralysis of both lower extremities with bilateral drop foot, urinary retention, faecal incontinence, saddle type anaesthesia and diminished deep tendon reflexes in both lower extremities. His radiographs revealed a traumatic retrolisthesis of L-5 over S-1 (fig 1). Open reduction and stabilization was performed using transpedicular screws and rods from a posterior approach (fig 2 A-B). There were no intraoperative or postoperative complications. The patient was transferred to the department of physical therapy 3 days after surgery with an unchanged clinical picture. During his follow-up, he was able to mobilize freely without crutches at 4 months (fig 3), the urinary sphincter dysfunction resolved in 24 months, saddle anaesthesia in 6 months,



Fig. 1. — Radiograph showing L5-S1 retrolisthesis

bowel incontinence in 18 months after the operation. Impotence returned to normal 42 months after the operation. The patient got married 6 years after the operation and had a child. Patellar tendon reflexes returned to normal 28 months after operation. The patient had bilateral drop foot initially. The left side returned to normal at 40 months, but the right side did not recover till 16 years of follow up. His low back pain decreased but residual mild low back pain still persists. Instrumentation was removed 6 years after the operation. Plain radiographs were obtained at each control and bony fusion was seen at the level of L-5 and S-1 (fig 4 A-B) with subsequent degenerative changes in the following years.

DISCUSSION

Acute trauma as a cause of cauda equina syndrome is very rare (13). Lumbosacral dislocation is

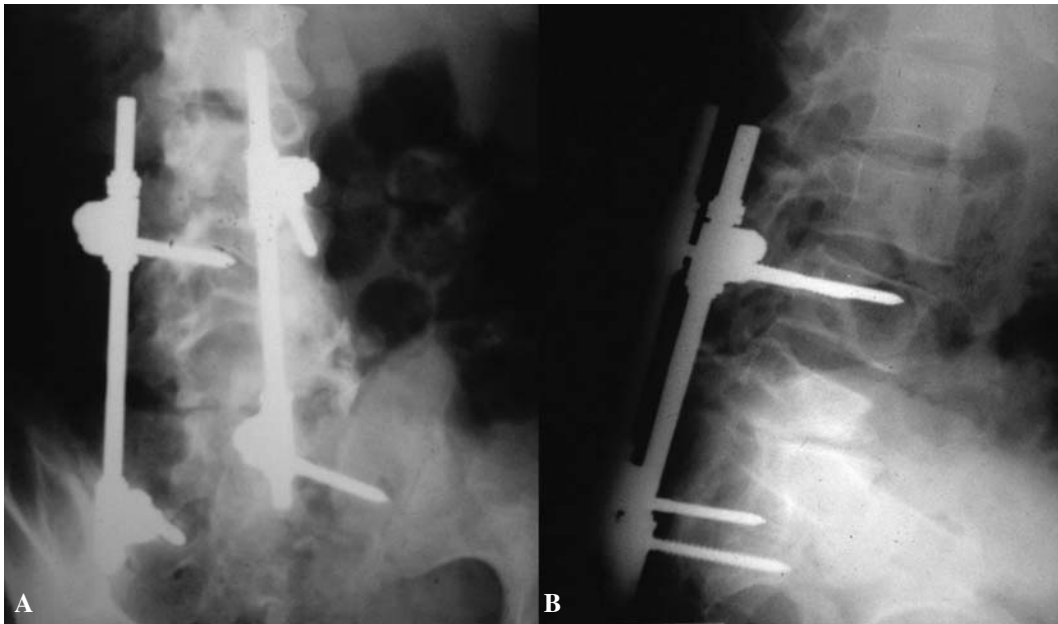


Fig. 2A-B. — Postoperative radiograph following open reduction, decompression and stabilization of the spinal column.

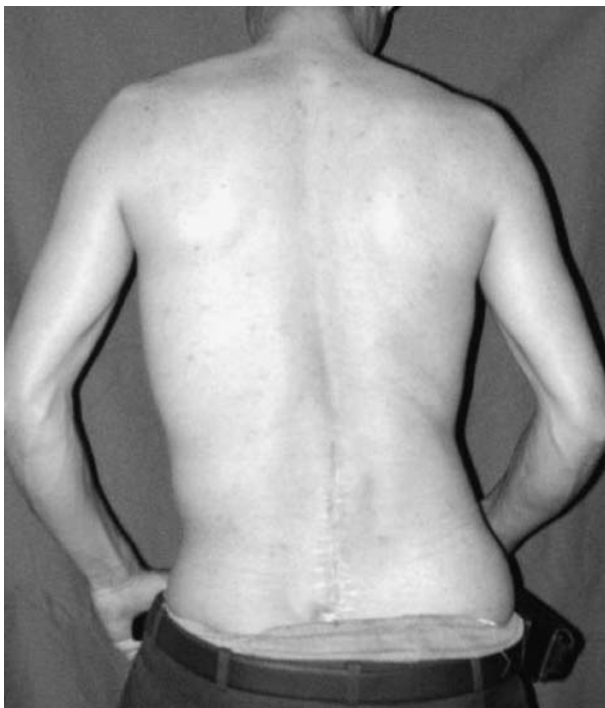


Fig. 3. — The patient stands without any aid of crutches

usually the result of high-energy trauma and there are often associated lesions and fractures (4, 18, 19). Our patient had no associated lesions and this indicates that this problem can be seen with a much milder clinical picture. Cauda equina syndrome is defined as a complex of low back pain, saddle hypoaesthesia, sciatica, motor weakness in the lower extremity and bowel and bladder dysfunction (13-15).

To our knowledge, there is only one case report about traumatic lumbosacral retrolisthesis (3). Different strategies and treatment methods have been used in this type of injury (lumbosacral dislocation) including operative and nonoperative measures (4, 12, 17, 19). Neurological injuries are found in fracture-dislocations of the lumbosacral spine and in approximately 1/3 of the reported L5-S1 dislocations. In this setting, the problem should be treated as an emergency (18).

Prevention of progression of spondylolisthesis and the neurological deficit with back pain remain as the main goal (12, 17). In some reports,

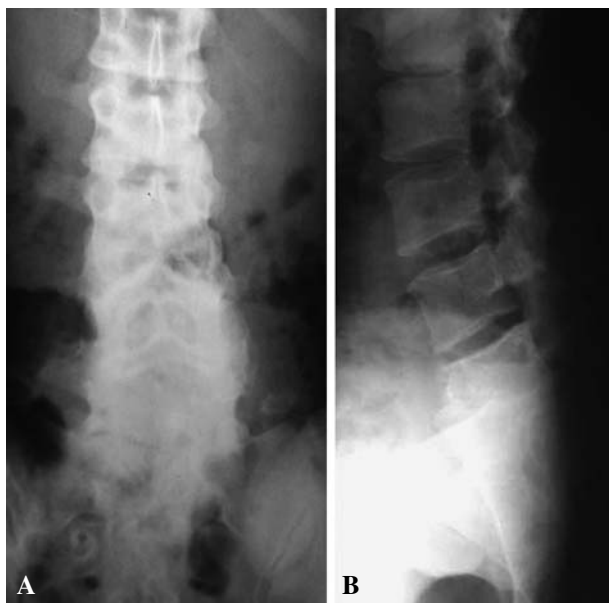


Fig. 4A-B. — Two plane radiograph 16 years after operation. The implants were removed and bony fusion is clearly seen.

360 degrees fusion is advocated to handle this injury properly as this injury involves the three columns of the spine (4, 12, 16, 17). One important point is that, reduction maneuvers may be difficult in cases with fracture dislocation of the articulating facets (17, 19). To address the neurological problems and to relieve nerve roots especially in a case which has a complete retrolisthesis, we believe no attempt at closed reduction should be made. Instead, posterior approach with laminectomy, decompression and fusion should be done to treat the problem. This was our choice of treatment, ending in a satisfactory outcome. Most of the reported literature supports early intervention for return of full neurologic function for cauda equina syndrome (10, 14), but there is no consensus on the optimal time of decompression. The literature about the late presenting cases with neurologic deficits and their outcome following surgical treatment is not clear. Bohlman demonstrated that significant recovery can occur with late decompression (4). Time required for recovery after decompression may vary from months to years (14). This was also the case in our patient, who underwent late decompression because of his late referral to our hospital.

Improvement of bladder function may be seen after many years as in our case (10).

Cauda equina syndrome is an uncommon entity following spinal column trauma. Early intervention is required for return of neurological functions. Even in case of late referrals, the orthopedic surgeon should not hesitate to do the necessary operation including the decompression of the neural elements and stabilization of the spine. If disc debridement is done properly, and segmental spinal fixation is done using pedicle screws, 360 degrees fusion may still be achieved without anterior surgery. Recovery may take months to years after the surgical intervention. Long term follow-up is needed before deciding that the neurological picture has settled.

REFERENCES

1. Ahn UM, Ahn NU, Buchowski JM, Garrett ES, Sieber AN, Kostuik JP. Cauda equina syndrome secondary to lumbar disc herniation: a meta-analysis of surgical outcomes. *Spine* 2000 ; 15 ; 25 : 1515-1522.
2. Benzel EC, Hadden TA, Coleman JE. Civilian gunshot wounds to the spinal cord and cauda equina. *Neurosurgery* 1987 ; 20 : 281-285.
3. Cohn SL, Keppler L, Akbarnia BA. Traumatic retrolisthesis of the lumbosacral junction. A case report. *Spine* 1989 ; 14 : 132-134.
4. Cruz-Conde R, Rayo A, Rodriguez de Oya R, Berjano P, Garate E. Acute traumatic lumbosacral dislocation treated by open reduction internal fixation and fusion. *Spine* 2003 ; 1 ; 28 : E51-53.
5. Cybulski GR, Stone JL, Kant R. Outcome of laminectomy for civilian gunshot injuries of the terminal spinal cord and cauda equina: review of 88 cases. *Neurosurgery* 1989 ; 24 : 392-397.
6. Kagaya H, Abe E, Sato K, Shimada Y, Kimura A. Giant cauda equina schwannoma. A case report. *Spine* 2000 ; 15 ; 25 : 268-272.
7. Kikuchi M, Nagao K, Muraosa Y, Ohnuma S, Hoshino H. Cauda equina syndrome complicating pneumococcal meningitis. *Pediatr Neurol* 1999 ; 20 : 152-154.
8. Kostuik JP, Harrington I, Alexander D, Rand W, Evans D. Cauda equina syndrome and lumbar disc herniation. *J Bone Joint Surg Am* 1986 ; 68 : 386-391.
9. Nather A, Bose K. The results of decompression of cord or cauda equina compression from metastatic extradural tumors. *Clin Orthop* 1982 ; 169 : 103-108.
10. O'Laoire SA, Crockard HA, Thomas DG. Prognosis for sphincter recovery after operation for cauda equina

- compression owing to lumbar disc prolapse. *Br Med J (Clin Res Ed)* 1981 ; 6 ; 282(6279) : 1852-1854.
11. **Poncelet A.** The neurologic complications of Paget's disease. *J Bone Miner Res* 1999 ; 14 Suppl 2 : 88-91.
 12. **Reinhold M, Knop C, Blauth M.** Acute traumatic L5-S1 spondylolisthesis : a case report. *Arch Orthop Trauma Surg* 2006 ; 126 : 624-630.
 13. **Robinson RG.** Massive protrusion of lumbar disks. *Br J Surg* 1965 ; 52 : 858-865
 14. **Shapiro S.** Medical realities of cauda equina syndrome secondary to lumbar disc herniation. *Spine* 2000 ; 1 ; 25 : 348-351.
 15. **Todd NV.** Cauda equina syndrome : the timing of surgery probably does influence outcome. *Br J Neurosurg* 2005 ; 19 : 301-306.
 16. **Tsirikos AI, Saifuddin A, Noordeen MH, Tucker SK.** Traumatic lumbosacral dislocation : report of two cases. *Spine* 2004 ; 15 ; 29 : E164-168.
 17. **Vialle R, Court C.** Traumatic lateral lumbosacral dislocation : one case and review of literature. *J Spinal Disord Tech* 2005 ; 18 : 286-289.
 18. **Vialle R, Wolff S, Pauthier F, Coudert X, Laumonier F, Lortat-Jacob A.** Traumatic lumbosacral dislocation : four cases and review of literature. *Clin Orthop* 2004 ; 419 : 91-97.
 19. **Veras del Monte LM, Bago J.** Traumatic lumbosacral dislocation. *Spine* 2000 ; 15 ; 25 : 756-759.