

## Cauda Equina Syndrome (CES) Due to Lumbar Disc Herniation; Correlation between Delayed Decompression and Clinical Outcome

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### **Abstract**

Twenty-six patients presented with fully developed CES with delay of 48 hrs to two months, with average age of 40 years ranging from 20-60 years. The follow-up of patients was for two years post-operatively. All patients underwent surgical decompression. The result is not dramatic, but it takes time, leg and back pain relieved in all patients, nineteen patients regained full control of urination, five patients urinate with straining and two patients remained needing catheterization to treat retention. We concluded that surgical decompression is beneficial for those patients presenting late with fully developed CES. So we recommend to do decompression to all patients with CES in spite of the delay in presentation. Further urodynamic studies and monitoring of the intrathecal pressure preoperatively is required.

### **متلازمة ذيل الفرس بسبب انزلاق الغضروف بين الفقرات القطنية: العلاقة بين التأخر في إزالة الضغط والنتائج السريرية**

#### **الخلاصة**

تمت الدراسة على ستة وعشرون مريضاً مصابين بمتلازمة ذيل الفرس، تم مشاهدتهم متأخرين لفترة تتراوح بين ٤٨ ساعة و شهرين، معدل أعمارهم ٤٠ سنة تتراوح أعمارهم بين ٢٠-٦٠ سنة، تمت متابعتهم لفترة سنتين بعد إجراء العملية كان التحسن في النتائج السريرية يظهر بصورة بطيئة، آلام الظهر والساق أخفقت في جميع المرضى، تسعة عشر مريضاً ظهر عليهم تحسن في السيطرة على الإدرار بصورة كاملة، خمسة مرضى ظهر عليهم تحسن جزئي في السيطرة على الإدرار ويحتاجون إلى ضغوط للمساعدة في التبول، ومريضان لم يستفيدا من العملية ويقوا يحتاجون إلى قسطره بوليه للتبول. استنتج من الدراسة بأن إزالة الضغط جراحياً مفيداً للمرضى الذين يتم علاجهم متأخرين، لهذا ننصح بإزالة الانضغاط جراحياً بغض النظر لفترة تأخر المريض للعلاج، ويحتاج إلى دراسة أبعد بخصوص المتلازمة قبل إجراء العملية من حيث ديناميكية المثانة وقياس الضغط داخل الغراب.

### **Introduction**

Cauda equina syndrome is defined as the compression of the nerve roots distal to L1 most commonly secondary to prolapsed intervertebral disc [1]. This leads to a complex of clinical symptoms and signs differs according to involved nerve roots. The complex of symptoms manifested as a low back pain, bilateral or unilateral sciatica, motor weakness of the lower extremities, loss of bowel or bladder function and sensory

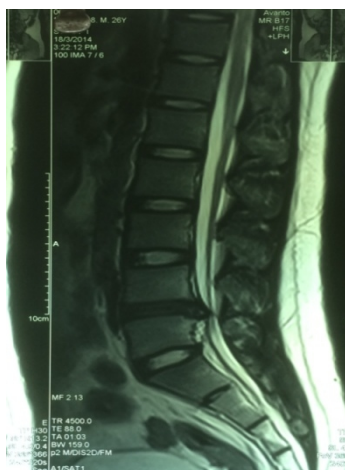
distrbances. CES has been considered the only absolute indication for surgery in cases of lumbar disc disease. CES first described by Mixter and Barr in 1934 [2]. Few reports of cauda equina compression related to lumbar disc prolapsed have been published, and the prognosis for the recovery for the bladder and bowel function postoperatively has been uniformly gloomy [3-5]. The urgency of the diagnosis and treatment of the cauda equine syndrome may be compared with

that for extradural haematoma in head injury [4, 5]. CES due to prolapsed lumbar disc is relatively rare (3%) in the developed countries, but it is more common in the developing countries due to delay in diagnosis and late presentation of patients to the hospitals [6]. The lumbar and sacral nerve roots contain sensation to the perineum and genitals, sensory and motor function for the lower extremities, and they also innervate the pelvic viscera. Voluntary and involuntary functions are also contained within the nerve roots of the cauda equina and are necessary for micturation, defecation, and sexual function. Compression of cauda equina may involve all of the above functions, sensory only, motor only, or only those roots responsible for bowel and bladder function. So there is variability in presenting sign and symptoms of cauda equina. Several authors [5-8] described an acute type of presentation as well as a chronic type. The acute type usually present in a younger patients with no previous history of symptoms. The chronic variety usually presents with a more insidious onset.

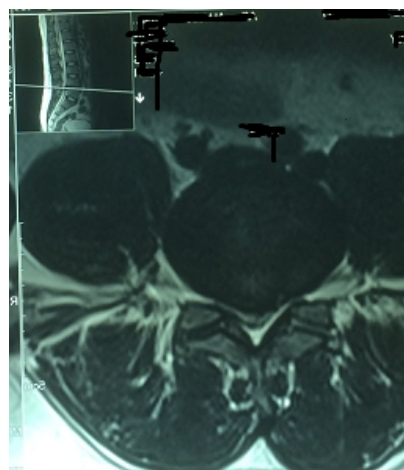
### **Patients and Methods**

Twenty six patients undergoing surgery for CES (20 male and 6 females) with average age of 40 years ranging from 20-60 year. Their presentation had occurred from 48 hrs to two months preoperatively. Clinical examination was done at time of presentation and clinical findings were recorded in details. Backache with urinary incontinence in eighteen patients, complete loss of sphincter control in eight patients, perineal sensation was absent in all patients, absent pulbocavernous reflex in three patients, fecal symptoms in

twenty-two patients, absent anal tone in nine patients, weakness of the lower limb was reported in all patients varying from that sever enough to prevent walking (14 patients) to a moderate weakness (6 patients) and from weakness of one foot (4 patients), ankle reflexes were absent in twelve patients. All patients were underwent plain radiograph and MRI scanning prior to surgery to examine the lumbar spine and to demonstrate any compression on the thecal sac (Figures 1, 2). Four patients received after 48 hrs of developing symptoms and signs of CES, six patients received after 4-6 days with developing signs and symptoms of CES, immediately they sent for MRI, which were done within 24 hrs followed by decompression surgery within 4-24hrs, sixteen patients received within 10 days-2 months with developing signs and symptoms of CES, some of them they had MRI and the other MRI were performed for them within 24 hrs and decompression surgery were done after 1-5 days. All patients' families were informed about the nature of the lesion and the expected results and prognosis. Wide laminectomy and discectomy were done for (14 patients) without fixation or fusion and fenestration discectomy (Figure 3) for (12 patients) . Follow up for all patients at two days ,one week, two weeks, two months, six months, nine months, one year, two years postoperatively and detailed neurological examination was performed and recorded. Results were divided into: 1- complete recovery. 2- near complete recovery (minor bowel, bladder and perineal sensation problem). 3- partial recovery (major bladder and bowel disturbance and persistent perineal anesthesia)



**Figure 1:** MRI shows prolapsed Disc at L4-5



**Figure 2:** Cross-section MRI shows encroachment of the neural canal



**Figure-3:** Removed disc material

## Results

MRI findings revealed that level of herniated discs at L2, 3 (1 patient), at the L3,4 (3 patients), at the L4, 5 (12 patients), and at L5,S1 (10 patients). The greatest canal compromise was 0-25% in 5 patients, 26-50% in 11 patients, 51-75% in 6 patients and 76-100% in 4 patients. The response to decompression surgery was varied in our patients with the response being motor followed by bowel and bladder followed by sensory recovery.

All patients showed variable degrees of improvement in strength of lower extremities within the first six months. In long term follow-up leg and back pain were relieved in all patients. Nineteen patients regained full control of urination (complete recovery). Five patients were

able to urinate by straining (near complete) and two patients showed no benefit regarding urination, meaning that catheterization was regained to treat retention (partial recovery). Only two male patients showed no improvement in sexual function. At late follow-up (two years) there were nineteen patients with complete recovery (73.07%), five patients with near complete recovery (19.23%) and two patients with partial recovery (7.7%).

## Discussion

The term CES describes a wide spectrum of symptoms and signs caused by compression of nerve roots in the lumbar spinal canal. According to the nerve roots involved the patients may presented with low back pain, saddle

anaesthesia, sensory unilateral or bilateral sciatica and/or motor deficit in the lower limbs and evidence of impaired pelvic visceral dysfunction manifesting as urinary and/or fecal incontinence. CES is an uncommon, accounting for 2–6% of all lumbar disc herniations [2,7,9]. The most distressing results of CES lead to loss of bladder and anal sphincter control which can be socially disruptive and emotionally distressing. Our finding demonstrate that urological impairment showed improvement despite only two patients reporting the need for catheterization and five patients urinate by straining. All patients were experiencing urinary incontinence at early follow up and they are unhappy with their urinary pattern, but at a late follow up (two years) only two patients need catheterization and five patients urinate by straining. There are different reports of the recovery of the bladder function in CES with other studies reporting universally poor outcome [10,11]. Few studies reported that the consequences of persistent paralysis may be much less severe than earlier authors have published [12, 13]. Gleave and Macfarlane [14] showed no significant difference in time to surgical decompression. Certain studies, although supporting emergency decompression in CES, have failed to establish any relation between the timing of surgery and clinical outcome [8,12,15-19]. In Shapiro's [20] experience, all patients showed recovery of their urinary or stool continence if the surgery done for them within 48 hrs. of developing symptoms, in comparison with one-third of patients for whom surgery were done after 48 hrs. Kostiuk et al [21] and Loone et al [12] found no relation between neurologic recovery and decompression delay over a range of hrs. to weeks. In spite of this lack of correlation, authors [21] still recommend that surgery should be carried out early, since a partial cauda-equina lesion should not be allowed to progress to complete loss of motor, sensory and sphincter function. The magnitude of compression to be tolerated

by the contents of thecal sac may be the fact behind that some patients not improved completely (two patient needs catheterization and five patients urinate by raining). Delamarter et al [22] showed that the time for decompression surgery did not play a role in functional or histological outcome at a constant pressure (75% constriction of cauda diameter), which mean that recovery being more dependent on the nature of the prolapsed disc.

Assad Qureshi et al [23] in their prospective series argued that the duration of symptoms prior to surgery did not appear to influence the outcome. We think that the severity of bladder dysfunction at the time of surgery is the dominant factor in the recovery. Our study is deficit in the pre-operative urodynamic studies which represent the most satisfactory method to distinguish between the initial irritative symptoms with loss of bladder and urethral sensation and the end point of an insensate incontinent bladder (complete cauda equine lesion), the prognosis may be more favorable when the syndrome remains incomplete at the time of decompression. Prompt decompression is still advisable to prevent an incomplete lesion progressing to complete sphincter paralysis [14, 24]. Anal wink was a good predictor of recovery of the bladder and bowel function, the patients with an intact anal wink at the presentation will eventually have satisfactory recovery of the bladder and bowel function. Michael J.H. Mc Caarth et al [25] in their recent study concluded the followings; 1-symptoms duration before operation and speed of onset do not affect the outcome more than two years after surgery. 2-Patients who had CES do not return to normal status. 3-There is no hard evidence that early surgery leads to better outcome. 4-Prospective and well powered studies are needed to address this problem. CES is one of the most common causes of litigation against medical practitioners [26]. The surgery should be undertaken by experienced surgeons to avoid a theoretical increase in the risk of post-

operative complications [27]. Urgent decompression should be performed when the circumstances to be optimal. Rydevik et al [28] developed a porcine model to study a neurophysiological changes that may occur when there is increasing compressive pressures in the cauda equine . They found that a threshold pressure (50-70 mmHg) bellow which full functional recovery could be expected. Pressure greater than 70 mmHg was consistently with residual neurologic deficit. Rydevik et al. data [28], found that a decrease in the functional recovery once a certain threshold pressure was surpassed, may provide insight to the different recovery patterns seen clinically [27,29,30]. So those patients who have neurological recovery with delayed decompression may have never reached the critical threshold pressure and the favorable effect of early decompression may reside in the fact that decompression prevents pressure from reaching the critical level where neurologic squeal are irreversible. Measurement of a compressive threshold in humans may give us clearer guidelines for decompression in the future .Further clinical studies toward intrathecal pressure monitoring as well as development of a technique to measure intrathecal pressure may be helpful.

## **Conclusion**

Our study proved that the results of the operation on patients presenting late are good, and this may be due to patients spectrum of compressive pressures of the cauda equina and showed that recovery of neurological function, particularly the autonomic functions, takes a longer time in patients presenting late in the course of disease, so we advise to perform the decompression in all patients irrespective of the delay in presentation. Further urodynamic and clinical studies to monitor the intrathecal pressure are required.

## **References**

1. Kim JS, Lee SH, Arbatti NJ. Dorsal extradural lumbar disc herniation causing

cauda equina syndrome: a case report and review of literature. *J Korean Neurosurg.* 2010; 47: 217-220.

2. Mixter WJ, Barr JS (1934) Rupture of the intervertebral disc with involvement of the spinal cord. *N Engl J Med* 211:210-214

3. Jennett WB. A study of 25 cases of compression of the cauda equina by prolapsed intervertebral discs. *J Neurol Neurosurg Psychiatry*, 1956; 19(2):109-116.

4. Shepherd RH. Diagnosis and prognosis of cauda equina syndrome produced by protrusion of lumbar disc. *Br Med J.* 1959; 26(2):1434-1439.

5. Jeon CH, Chung NS, Son KH, Lee HS. Massive lumbar disc herniation with complete dural sac stenosis. *Indian J Orthop.* 2013; 47(3):244-249.

6. Tandon PN, Sankaran B (1976) Cauda equine syndrome due to lumbar disc Prolapse. *Indeian J Orthop* 1:112-119.

7. Dining TAR, Schaeffer HR (1993) Discogenic compression of the cauda equina: a surgical emergency. *Aust NZ J Surg.*, 63:927-934.

8. Kostuik JB, Harrington I, Alexander D, Rand W, Evans D (1986) Cauda equina syndrome and lumbar disc herniation. *J Bone Joint Surg* 68A:386-391.

9. Shapiro S (1993) Cauda equina syndrome secondary to lumbar disc herniation. *Neurosurgery* 32:743-747

10. Jennett WB (1956) A study of 25 cases of compression of the cauda equina by prolapsed intervertebral discs. *J Neurol Neurosurg Psychiatry* 19:106-116

11. Shephard RH (1959) Diagnosis and prognosis of cauda equine syndrome produced by protrusion of lumbar disc. *Br Med J* 2:1434-1439

12. O'Laoire SA, Crockard HA, Thomas DG (1981) Prognosis for sphincter recovery after operation for operation for cauda equine compression owing to lumbar disc prolapse. *Br Med J* 282:1852-1854.

13. Scott PJ (1965) Bladder paralysis in cauda equina lesions from disc prolapse. *J Bone Joint Surg* 47B (2):224-235

14. Gleave JR, Macfarlane R. Cauda equina syndrome: what is the relationship between timing of surgery and outcome? *Br J Neurosurg.* 2002; 16(4):325-328.
15. Kostuik JP (2004) Medicolegal consequences of cauda equine syndrome: an overview. *Neurosurg Focus* 16(6):39-41
16. Markham DE (2004) Cauda equina syndrome: diagnosis, delay and litigation risk. *J Orthop Med* 26(3):103-105
17. McGrother CW, Donaldson MMK, Shaw C, Matthews RJ, Hayward TA, Dallosso HM, Jagger C, Clarke M, Castleden CM, the MRC Incontinence Study Team (2004) Storage symptoms of the bladder: prevalence, incidence and need for services in the UK. *BJU Int* 93:763-769
18. Ng LCL, Tafazal S, Longworth S, Sell P (2004) Cauda equine syndrome: an audit. Can we do better? *J Orthop Med* 26(2):98-101
19. Nielsen B, de Nully M, Schmidt K, Hansen RI (1980) A urodynamic study of cauda equina syndrome due to lumbar disc herniation. *Urol Int* 35:167-170
20. Shapiro S. Cauda equina syndrome secondary to lumbar disc herniation. *Neurosurgery.* 1993; 32(5):743-747.
21. Kostuik JP, Harrington I, Alexander D ET AL (1986) Cauda Equina syndrome and lumbar disc hrniation. *J Bone Joint Surg (Am)* 68:386-391
22. Delamarter R, Sherman J, Carr J. Cauda equina syndrome: neurologic recovery following immediate, early or late decompression. *Spine (Phila Pa 1976).* 1991; 16(9): 389-391.
23. McCarthy Michael J. H., Aylott Caspar E., Grevitt Michael P., and Hegarty J. Cauda Equina Syndrome. Factors affecting long-term functional and sphincter outcome. *Spine* (2007) Volume 12 ( 2), 207-216.
24. Gleave JRW ,Macfarlane R (2002) Cauda equine syndrome : what is the relationship between timing of surgery and outcome? *Br J Neurosurgery* 16 (4):325-328
25. Shapiro Scott. Medical realities of Cauda equina syndrome secondary to lumbar disc herniation. *Spine* (2000), Volume 25, Number 3, pp 348-352.
26. Kennedy J.G., Soffe K. E., McGrath A., Stephens M. M., Walsh M. G., and McManus F. Predctors of outcome in cauda equine syndrome. *European Spine Journal* (1999) 8:317-322
27. Gleave JRW, Macfarlane R (1990) Prognosis of recovery of bladder function following lumbar central disc prolapse. *Br J Neurosurg* 4:205-210.
28. Rydevik BL, Pedowitz RA, Hargens AR, et al: Effects of acute, graded compression on spinal nerve root function and structure: An experimental study of the pig cauda equine. *Spine* 16:487-493,
- 29- Cifitci H, Ozgonul A, Sogut O, Savas M. Incomplete cauda equina syndrome presenting with acute urinary retention in the emergency department. *Hong Kong J Emerg Med.* 2010; 17:498-501.
- 30- Jalloh I, Minhas P. Delays in the treatment of cauda equina syndrome due to its variable clinical features in patients presenting to the emergency department. *Emerg Med J.* 2007; 24(1):33-34.