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R. Ramnarayan,
Bipin Chaurasia

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The extended post spinal surgery syndrome (EPSS). A narrative review

R. Ramnarayan[1], Bipin Chaurasia[2]

[1] Consultant Neurosurgeon, New Hope Hospital, Kilpauk, Chennai, India
[2] Consultant Neurosurgeon, Neurosurgery Clinic, Birgunj, Nepal

Abstract

Pain can occur after any spinal surgery. Despite this, there are other many signs and symptoms of neurological deficits that can occur in patients with varying severity. Our aim is to find some of the main neurological deficits that can occur after any spinal surgery. We searched the literature based on some of the important keywords like neurological deficits after spine surgery, foot drops, cauda equina syndrome, epidural hematoma, and nerve and dural injury. Based on this we analyzed the most important and widely read articles. The problems associated with spine surgery have been published in the literature but are much more than the failed back surgery syndrome and cause more discomfort to patients with varying degrees of neurological deficits. We have coined a new term "Extended Post-spinal Surgery Syndrome (EPSS)" for these conditions. We propose this to include the other complications after lumbar surgery including nerve injury, dural injury, cauda equina syndrome and epidural haematoma.

Keywords
post spinal surgery,
neurological complications,
management,
foot drop,
cauda equine syndrome

Corresponding author:
Bipin Chaurasia

Neurosurgery Clinic,
Birgunj, Nepal

trozexa@gmail.com

Introduction

Complications following spine surgery do occur very often. These include persistence or recurrence of the pain, dural and nerve root injuries; cauda equina syndrome; and formation of extradural scar tissue. [1] The recurrence of pain also called the post-laminectomy syndrome has been studied very well probably because of the higher incidence. But others like nerve root injury or cauda equine syndromes have not been that much looked into. An attempt is made in this article to review these possible complications. A new term "Extended Post Spinal Surgery Syndrome" is being coined for these complications.

Marquez-Lara [2] looked at a national database to find out the incidence and outcomes of sentinel events in lumbar spine surgery retrospectively. A total of 414 patients had sentinel events out of 53146 patients. Of this vascular and nerve injuries occurred in 2/10000 cases. This caused longer hospitalization, greater costs, and a greater incidence of in-hospital complications, and mortality. Shah et al, [3] looked at wrong level surgery in lumbar spine surgery. This causes friction between the patient and the surgeon and has a lot of medical

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and legal consequences. The commonest reason for wrong-level surgery even with such advanced imaging and other techniques include anatomical variations of the lumbosacral spine including transitional vertebrae, lumbar ribs, butterfly vertebrae, hemivertebra, block/fused vertebrae, and spinal dysraphism. Other causes include tumours, infection, previous lumbosacral surgery, obesity, and osteoporosis.

Nerve injury

Neurological complications of spinal surgery were classified as peroperative and postoperative aetiology by de Loubresse. [4] The author described that neurological structures may be damaged by direct or indirect mechanisms. Direct compression, traction, laceration and avulsion from the direct causes. Indirect causes include Ischemic phenomena produced by elongation or compression of medullary or radicular blood vessels. The main peropertive complications noted were ocular injury causing visual disturbances, elongation of the brachial plexus causing pain and mild weakness of the upper limb and compression of the peroneal nerve causing unilateral foot drop. All these injuries can happen due to defective patient positioning. Following defective patient positioning, the rate of neurological lesions is 0.14% for all procedures taken together.

For lumbar surgery procedures especially high-grade lumbar spondylolisthesis, reducing the slippage and local kyphosis entails neurologic risk, especially to the L5 root. It was found that serious neurological impairment after fusion on a well-aligned spine (0.14%) or after lumbar disc surgery was about 0.03%. The other peroperative complication that can happen especially in implantation is malpositioning of the screws. The incidence of malpositioning is quoted as 4.2%, but is around 15.7% on postoperative imaging. Severe lumbar canal stenosis or large disc prolapse can cause neural injury. The incidence of peroperative root injury is 0.4%.

Shin and coworkers [5] retrospectively analysed 627 patients who underwent surgery for lumbar degenerative pathology. Eight patients (1.3%) had intraoperative spinal root injury. Of this 5 patients had undergone laminotomies for discectomy and 3, for instrumentation and fusion. The causative instrument was the Kerrison punch in 4 cases, pituitary forceps in 2 and rongeur in 1. Four patients had L5 injury, 1 had S1 and another had S2 injury. Two patients had sacral rootlet injuries. Six patients had symptoms including sensory loss in 4, pain in 2, and one patient had both sensory loss and EHL weakness. The prognosis was poor in almost 50 % of patients. In fact, Epstein [6] recorded that minimally invasive procedures have a much higher incidence of nerve injuries compared to conventional open procedures. The rate of injury was 2% for transforaminal lumbar interbody fusion (TLIF) versus 7.8% for posterior lumbar interbody fusion (PLIF) and during anterior lumbar interbody fusions (ALIF: 15.8%) versus extreme lumbar interbody fusions (XLIF: 23.8%), addressing disc disease, failed back surgery, and spondylolisthesis. In comparison Desai et al [7] in a large multicentre trial of 792 patients reported that nerve root injury with open discectomy occurred in 0.13–0.25% of cases, in 0% of laminectomy with or without fusion, and just 2% for open laminectomy with or without fusion.

Ghobrial et al[8] had also described iatrogenic neurologic deficits after lumbar spine surgery. These include complications like radiculopathy, spinal cord compression, motor deficits and new onset radiculitis. Neuromonitoring has been successful in reducing these complications but they still occur. Degenerative spondylolisthesis, spondylosis, scoliosis, and lumbar stenosis were the most common indications for surgery. Fifty six patients out of 2783 reported postoperative neurologic deficit (5.7%). Among this, 4.1% had a new neurological deficit after anterior lumbar and 1.9% after posterior surgery.

Dural injury

Espiritu et al[9] felt that dural tears are among the most commonly seen complications in spine surgery. If the tears are diagnosed early and managed appropriately, long-term outcomes are not negatively affected. Direct suture repair is the best method for durotomy caused during surgery. Cammisa et al[10] retrospectively reviewed patients who underwent spine surgery over a ten-year period for the frequency of incidental durotomy. Seventy-four patients had dural tears during or before surgery out of a total of 2144 patients. Of these 74, 66 happened during surgery. Primary repair was done in 60 out of 66 patients; the rest had pseudomeningocoels repaired surgically later. Long-

term follow-up showed good results for all patients. McMohan et al[11] looked at incidental durotomies and analysed the long-term patient outcomes as well as the major risk factors prospectively. The frequency of incidental durotomy in elective spinal surgery cases was 3.5%, in minimally invasive procedures it was 3.3% and 6.5% in revision surgery. Incidental durotomy was less in cervical surgery compared to lumbar, less when involving instrumentation and less when senior surgeons were operating. In patients with an incidental durotomy, 7.7% had neurological deficits compared with 1.5% of those without.

The overall failure rate of dural repair was 6.9%, and failure was almost 3 times higher in revision surgery. Guerin et al [12] retrospectively reviewed 1326 spinal surgery patients and identified 51 dural tears were identified. This was more with posterior thoracolumbar approach (48/51). Postoperatively, seven patients had CSF leak and two each of wound infection, pseudomemnigocoel and postoperative haematomas. Kamenova et al[13] looked at the management of incidental dural tears during lumbar spine surgery in 64 patients who had a dural tear out of 1173 patients who underwent lumbar spine surgery. The dural closure technique was direct closure or using a patch only or suture with a patch. They realised that the dural closure technique does not affect long-term results. Jankowitz et al [14] looked at the use of fibrin glue to reduce the frequency of CSF leak in dural tear during spinal surgery. This retrospective study had an incidence of 11% of dural tear of which about 50% had fibrin glue used in the dural repair. They concluded that prior surgery significantly increases the incidence of durotomy during elective lumbar spine surgery but the use of fibrin glue for dural repair did not significantly decrease the incidence of a persistent CSF leak.

Foot drop

Foot drop is defined as weakness in dorsiflexion of the foot[15]. The two top most common aetiologies for foot drop include lumbar degenerative disease and common peroneal nerve injury. Foot drop can be unilateral or bilateral. When the foot can no longer be actively lifted against gravity, an abnormal gait pattern arises. This causes the patient to fall down forwards causing injuries, thus reducing the quality of life [16]. But foot drop in lumbar degenerative disease is alike a chicken and egg story. Whether foot drop was present before surgery or is as a result of surgery is always a point of confusion. Bhargava and colleagues [17] reported a retrospective observational study of 26 patients with foot drop who underwent surgery. Of the 26 patients, 88% improved, with complete recovery observed in 61%. It was concluded that preoperative duration of weakness was a significant predictor of extent of recovery. There have been many reports of both unilateral and bilateral foot drop caused by lumbar degenerative pathology [18].

Ma et al[19] analysed the risk factors for foot drop in patients with lumbar disc herniation retrospectively in 236 patients. Fifty-two patients had foot drop. They concluded that diabetes mellitus, disc calcification, patients with acute episodes, and far lateral disc prolapse had a bigger risk of foot drop. Also, a canal occupancy rate of more than 50% was at greater risk. For every 1 mm change in canal diameter the risk of developing foot drop changed by around 50%. Liu et al [20] reviewed 135 patients with foot drop due to lumbar degenerative disease. Foot drop was observed in 8.1% of all inpatients of lumbar degenerative disease. L5 nerve root compression was observed in 126 of all 135 patients. The muscle strength of TA was improved in 113 (83.7%) patients after surgery. They concluded that patients with shorter duration of palsy, better preoperative muscle strength of TA and younger age showed a better surgical outcome. Aono et al [21] also showed that palsy duration and preoperative strength were factors that most affected drop foot recovery following surgical intervention for spinal degeneration. Investigating a patient of foot drop after lumbar surgery usually involves repeat MRI imaging and electrophysiology. This usually localises the site of lesion. Daniels et al [22] introduced MR neurography to verify the site of the lesion site. MRN also helps in accurately characterizing the cause of the neuropathy and helps guide treatment. MR neurography can help overcome potential pitfalls in clinical and electrodiagnostic evaluation when tailored and focused.

The biggest problem with foot drop is it affects gait and causes patients to trip and fall on uneven surfaces. So this problem needs proper attention. Macki et al [23] looked at the predictors of improvement in foot drop due to lumbar degenerative disease. They retrospectively reviewed 71 patients undergoing posterior lumbar

decompression for foot drop due to degenerative spinal disease. Fifty-two patients had postoperative improvement. They concluded that the power of the tibialis anterior muscle and the duration of the foot drop were statistically significant predictors of improvement. The average time to improvement was 6 weeks. Takenaka and Aono [24] also tried to predict postoperative improvement through a Bayesian Network. They studied 102 out of 141 patients who underwent decompressive surgery for foot drop. The models showed that weaker muscle power before surgery (≤ 1) and longer duration of neurologic injury before treatment (> 30 days) were associated with a decreased likelihood of return of function by 2 years. Age, herniated soft disc, and leg pain were identified as indirect predictors. The probability estimates of posttibialis anterior muscle strength of 3 or greater and posttibialis anterior muscle strength of 4 or greater were 94% and 85%, respectively, in the most favourable conditions (pretibialis anterior ≥ 2; duration ≤ 30 days) and 18% and 14%, respectively, were the least-favourable conditions.

There are presently many treatment options for foot drop. Repeat surgery offers a realistic chance of restoring nerve function if a compressive element can be demonstrated. In the current DGN guideline on lumbar radiculopathy, a relative and absolute indication for surgery is described for muscle strengths of more than 3/5. Recovery is correlated with the severity of the paresis. There is no clear consensus to what extent the preoperative duration of the paresis correlates with recovery.[16] However, there is a trend for better recovery if the patient undergoes surgery within 48 hours of the onset of the paresis.

Treatment of foot drop is usually a multidisciplinary exercise. In mild cases braces, shoe inserts and other orthotic appliances may be useful. Physical exercises to strengthen the muscles also help. As already mentioned decompressive surgery does have a role. [16, 17] In selected cases, neurolysis of the peripheral nerve help. Repair processes are possible in the peripheral nervous system. As long as the nerve-cell nucleus is intact, axonal sprouting occurs for up to six months after the injury. However, the capacity for regeneration already starts to decline after three months because of a variety of changes in the distal stump [25]. Tendon transfers which help to restore specific movements are another technique used in some patients. The posterior tibial tendon is pulled through the interosseous membrane to the instep where it is anchored or redirected to the front, with the tibialis anterior and the peroneal tendons to create a stirrup. The measured increase in foot lifting strength resulting from a tendon transfer is only about 30% of the full strength, but it produces a significant functional increase and the quality of life is satisfactorily improved [26]. Functional electrical stimulation is another technique used to prevent muscle atrophy but how much it is useful to improve foot drop remains a question. Botulinum toxin (Botox) can be useful if the foot drop is associated with spasticity but in lumbar surgery patients, this is very rare.

Recent studies have shown that direct peroneal nerve stimulation with an implantable 4-channel peroneal nerve stimulator (ActiGait) allows independent electrode adjustment and leads to better functional results and improved quality of life. The application of this therapeutic option is restricted to patients with a drop foot attributable to a lesion of the first motor neuron caused by stroke, multiple sclerosis, or tumours [27]. Yao et al [28] studied 21 patients with chronic foot drop due to a central lesion who had implantation of this implant. They showed that patients had significant improvement in walking speed, gait endurance and gait performance. Patient satisfaction and improvement in mobility were achieved in 95% and quality of life improved in 90% of patients.

CAUDA EQUINA SYNDROME

Cauda equina is the horsetail-like root of the lumbosacral nerves. So any compression of these roots especially at L1 to L5 vertebral levels can cause neurological deficits like incontinence/ retention of urine, foot drop, pain, weakness and wasting of legs and sensory deficits. Usually, lumbar aetiology includes both lumbar disc prolapses especially L4 L5 and L5S1 levels and lumbar canal stenosis. The cauda equina involvement usually occurs preoperatively but does occur after surgery also. As there is a high possibility of litigation, in both cauda equina syndrome and foot drop a detailed history and clinical examination and recording findings is very important.

Todd and Dickson[29] brought out their recommendations for treatment of this condition.

They recommended next-day surgery for patients with bilateral radiculopathy and large central disc prolapse. For patients presenting with acute cauda equine syndrome (CES), emergency surgery is a must. Again, emergency surgery is indicated if large disc prolapse with uncertain cauda equine syndrome with residual nerve function, If there is prolonged cauda equine syndrome or no residual sacral nerve root function, surgery should be planned on the next day's list. Uckun et al [30] retrospectively studied the effect of surgical timing on improvement of motor function and sphincter function in patients with CES due to lumbar disc herniation. In this series muscle strength improved in 13 and returned to normal in nine patients and sphincter control resolved in five patients. Sensory loss resolved in two patients. So they suggested that patients with CES should be operated within 24 hours. Foruria and coworkers [31] retrospectively studied 18 patients to see whether surgical treatment delayed for more than 48 hours influenced the clinical outcome in patients with cauda equina syndrome. All patients operated within 48 hours had good continence and motor recovery. Three out of five with delayed surgery had residual incontinence. But this result was not statistically significant.

Kaiser et al.[32] prospectively investigated the relationship between postoperative urinary function, preoperative duration of neurogenic lower urinary tract dysfunction and the level of canal compromise in 71 patients. After studying this large series of patients they concluded that there is no correlation between the preoperative duration of urinary dysfunction, the size of disc herniation relative to the size of spinal canal, and postoperative urinary function. Srikandarajah and colleagues [33] categorised CES involvement as CES with retention (CESR) and CES incomplete (CESI). CESI operated within 24 hours had a normal function in 89% but only 48% benefitted when operated after 24 hours. If operated within 48 hours 85% of CESI improved but after 48 hours 56% benefitted. For those with CESR the timing of the surgery did not make any difference to the final outcome.

In contrast, there have been some studies which conclude that the timing of surgery is not that important. Aly and Aboramadan [34] studied 14 patients who underwent surgery one to three months after urinary involvement. All patients were relieved of back and leg pain, 12 regained control of the sphincters and nine improved in motor power. So they concluded that even if surgery is delayed improvement can be seen. Qureshi and Sell [35] tried to determine the factors influencing spine and urinary outcomes in CES due to disc herniation in 33 patients prospectively and assessed at 3 months and one year postoperatively. Seven patients underwent surgery within 2 hours and another 5 within 48 hours. No statistically significant difference in outcome between the patients with respect to the length of time from symptom onset to surgery was made out. Patients who were continent of urine at presentation had a much better outcome. So they concluded that the duration of symptoms prior to surgery had no bearing on the outcome. McCarthy et al[36] studied the factors that influence outcomes after surgery for CES. Acute onset of sphincteric symptoms and the time to operation did not influence the outcomes; also more females were affected. They found that the symptom duration before operation and the speed of onset do not affect the outcome more than two years after surgery.

Olivero et al [37] reported 28 patients with CES due to herniated discs and followed up for an average of up to 5 years. Twenty-seven of these patients regained continence not requiring catheterization. There was no correlation between the time to surgery and recovery of bladder function. The majority of the patients were adequately treated without the need for a complete laminectomy. Dhatt and others [38] studied patients who were admitted and operated up to 35 days after CES. There was no statistically significant difference in time of delay in surgery between the recovered and non-recovered groups as tested by Student's t-test. But there was a statistically significant positive correlation between duration taken for total recovery and delay in surgery. Anal wink as a predictor of bladder and bowel recovery also showed statistical significance. The result of surgery in CES is not as dramatic and fast as seen after routine disc surgery. Some improvement can be expected with decompression even in those patients presenting late and results are not universally poor as previously thought. The treating physicians of such patients should be aware that the recovery in this group of patients can take an exceptionally long time and hence should be involved in constant reassurance and rehabilitation of the patient.

A study by Korse and others[39] evaluated the association of MRI features with clinical presentation and outcome of CES. They also compared the lumbar spinal canal diameters of lumbar herniated disc patients with CES and those without CES. They found no association between MRI features and clinical features and outcome. They also found that patients with CES had a significant smaller AP diameter of the lumbar spinal canal in CES patients compared with those without CES. So they warned that patients with lumbar herniated disc patients with a relatively small lumbar spinal canal should be approached differently. Chang, Nakagawa and Mizuno [40] reported 4 patients of 144 consecutive surgical series of lumbar disc herniation, whose presenting symptom was classic cauda equina syndrome. The patients were on follow-up for up to 6.4 years. They reported that in all these patients the voiding function improved to normal even though it took many years.

There are some studies which describe that CES can occur postoperatively. McLaren and Bailey [41] reported six cases that developed CES after lumbar discectomy. Of these five had associated lumbar canal stenosis which was not tackled at time of surgery. They opined that motor recovery was poor if a severe deficit had developed before decompression and bladder and sensory deficits recovered well if decompressed early. Similar reports were made by Jensen [42] and Henriques [43]. The possible mechanism for this could be relative spinal stenosis causing postoperative oedema and triggering venous congestion causing nerve root ischaemia. Extended decompression within 48 hours seems to help patients to improve. We also have an example of a lady who had foot drop and retention of urine for more than 8 years and improved by open L4L5 laminectomy discectomy.

There have been some reports of CES following decompression for lumbar canal stenosis. Duncan and Bailey [44] reported an incidence of 2.8% of CES in patients with lumbar canal stenosis. All patients who developed cauda equina syndrome improved over 3 to 9 months, but none completely resolved. Three cases underwent further decompression with no apparent improvement. Comer and coworkers [45] reported on older adults with lumbar canal stenosis and said the diagnosis could be challenging. The degenerative changes in the elderly can lead to lumbar canal stenosis and gradual compromise of the cauda equina. The very slow onset of symptoms may be overlooked. So careful assessment and vigilance are needed to manage this potentially vulnerable patient group.

Sacral nerve stimulation is an excellent method of treating all types of neurogenic bladder and also pelvic pain [46]. It is effective in urinary and bowel involvement in CES causing bowel and bladder involvement and in severe pelvic pain[47,50-54].

Epidural haematoma

Postoperative lumbar spinal epidural haematoma is another complication of lumbar surgery but it is very rare. Aono et al [48] studied 26 patients who had spinal epidural haematoma (SEH) evacuation and looked at frequency of evacuation, symptoms, time to SEH evacuation, comorbidities, and neurological recovery. The frequency of SEH evacuation was 0.41% which included 0% lumbar discectomy, 0.50% in lumbar laminectomy and 0.67% in posterior lumbar interbody fusion. About 50% of the patients had symptoms like leg pain or bladder dysfunction after the suction drain was removed. The study concluded that neurological recovery was better when the evacuation was earlier. Yi and others [49] looked at the risk factors and clinical outcomes in postoperative spinal epidural haematoma. They observed that patients with coagulopathy and highly vascularized tumour were more vulnerable to spinal epidural hematoma. The postoperative outcome was related to the preoperative neurological deficit and the time interval to the decompression; the earlier and complete the evacuation the better the results. There are also a few other case reports of epidural haematoma causing neurological deficits[55-59].

Conclusion

Extended Post-spinal surgery Syndrome (EPSS)" is the proper term to describe complications after lumbar surgery including nerve injury, dural injury, cauda equina syndrome and epidural haematoma.

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