

# World Journal of *Clinical Cases*

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## Cauda equina syndrome caused by the application of DuraSeal™ in a microlaminectomy surgery: A case report

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### Abstract

#### BACKGROUND

The management of dural tears is important. While a massive dura can be repaired with absorbable suture lines, cerebrospinal fluid leakage can be attenuated by dural sealant when an unintended tiny durotomy occurs intraoperatively. DuraSeal is often used because it can expand to seal tears. This case emphasizes the need for caution when DuraSeal is used as high expansion can cause complications following microlaminectomy.

#### CASE SUMMARY

A 77-year-old woman presented with L2/3 and L3/4 lateral recess stenosis. She underwent microlaminectomy, foraminal decompression, and disk height restoration using an IntraSPINE® device. A tiny incident durotomy occurred intraoperatively and was sealed using DuraSeal™. However, decreased muscle power, urinary incontinence, and absence of anal reflexes were observed postoperatively. Emergent magnetic resonance imaging revealed fluid collection causing thecal sac indentation and central canal compression. Surgical exploration revealed that the gel-like DuraSeal had entrapped the hematoma and, consequently, compressed the thecal sac and nerve roots. While we removed all DuraSeal™ and exposed the nerve root, the patient's neurological function did not

recover postoperatively.

## CONCLUSION

DuraSeal expansion must not be underestimated. Changes in neurological status require investigation for cauda equina syndrome due to expansion.

**Key Words:** Cauda equina syndrome; DuraSeal; Microlaminectomy; Spinal stenosis; Case report

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**Core Tip:** The number of laminectomies is increasing, and incidental durotomy sometimes occurs intraoperatively. One of the approaches to manage dural tears was using sealants such as DuraSeal™. We present the case of a 77-year-old patient who suffered from incidental durotomy with treatment of using DuraSeal™ when undergoing spine surgery. Postoperative cauda equina syndrome was noted. Surgical exploration revealed thecal sac and nerve roots compression by entrapped hematoma. Our case highlights the potential catastrophic consequences of over-expansion of dural sealant, and demonstrates that cauda equina syndrome should be considered if neurological symptoms develop following application of DuraSeal™.

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## INTRODUCTION

As life expectancy increases worldwide, degenerative diseases of the lumbosacral spine are becoming more common[1]. Debilitating conditions are currently the major causes of morbidity, disability, and lost productivity[2,3]. The current treatment of choice for spine degeneration is laminectomy for nerve root decompression. In laminectomy, managing intraoperative cerebrospinal fluid (CSF) leakage is important because it increases the risk of sequelae such as meningitis or abscesses, as well as the late development of pseudomeningocele[4,5].

When incidental durotomy occurs intraoperatively and causes CSF leakage, primary dural closure is not always feasible or may not be watertight[6,7]. An alternative solution is the use of a polyethylene glycol hydrogel dural sealant, DuraSeal™. This sealant comprises PEG ester and trilycine amine solutions. When these two solutions are mixed, a reaction occurs and covers the ruptured dura. The mixed solution expands and forms a watertight layer, providing sufficient time for the dura to adequately heal following this application. However, this expansion may also be associated with the development of cauda equina syndrome (CES). Here, we report a case of CES occurring following dural closure and consequent compromised neurological function due to DuraSeal™ expansion in the spinal canal.

The study was approved by the Institutional Review Board of Shin-Kong Wu Ho-Su Memorial Hospital (202020708R).

## CASE PRESENTATION

### Chief complaints

A 77-year-old woman with an underlying condition of hypertension for over 15 years who had undergone bilateral cataract surgery 10 years ago presented to the orthopedic outpatient department of our institute with progressive radicular pain, chronic lumbalgia, and right thigh pain. She complained of neurological claudication that had lasted for over five years.

### History of present illness

The patient presented to the orthopedic outpatient department of our institute with progressive radicular pain, chronic lumbalgia, and right thigh pain. She complained of neurological claudication that had lasted for over five years.

### **History of past illness**

She had hypertension for over 15 years, and had undergone bilateral cataract surgery 10 years ago.

### **Personal and family history**

Her family history was unremarkable.

### **Physical examination**

Conservative treatments, such as physiotherapy, administration of muscle relaxants, shockwave treatment, and oral medications including non-steroidal anti-inflammatory drugs, local anesthetic, and steroid injection over the past year had not alleviated the symptoms. Magnetic resonance imaging (MRI) was performed to evaluate disease severity in our hospital.

### **Laboratory examinations**

She did not receive laboratory examinations which related to her spine lesions.

### **Imaging examinations**

T2-weighted MRI ([Figure 1](#)) revealed collapsed disc height at the L2-3 and L3-4 Levels and a bulging disk compressing the thecal sac and right neural foramen, causing bilateral lateral recess stenosis and neuroforaminal narrowing, especially on the right side, abutting the L3 and L4 nerve roots.

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## **FINAL DIAGNOSIS**

Based on these imaging findings, lumbar stenosis at the L2/3 and L3/4 Levels was diagnosed, accompanied by neurological symptoms.

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## **TREATMENT**

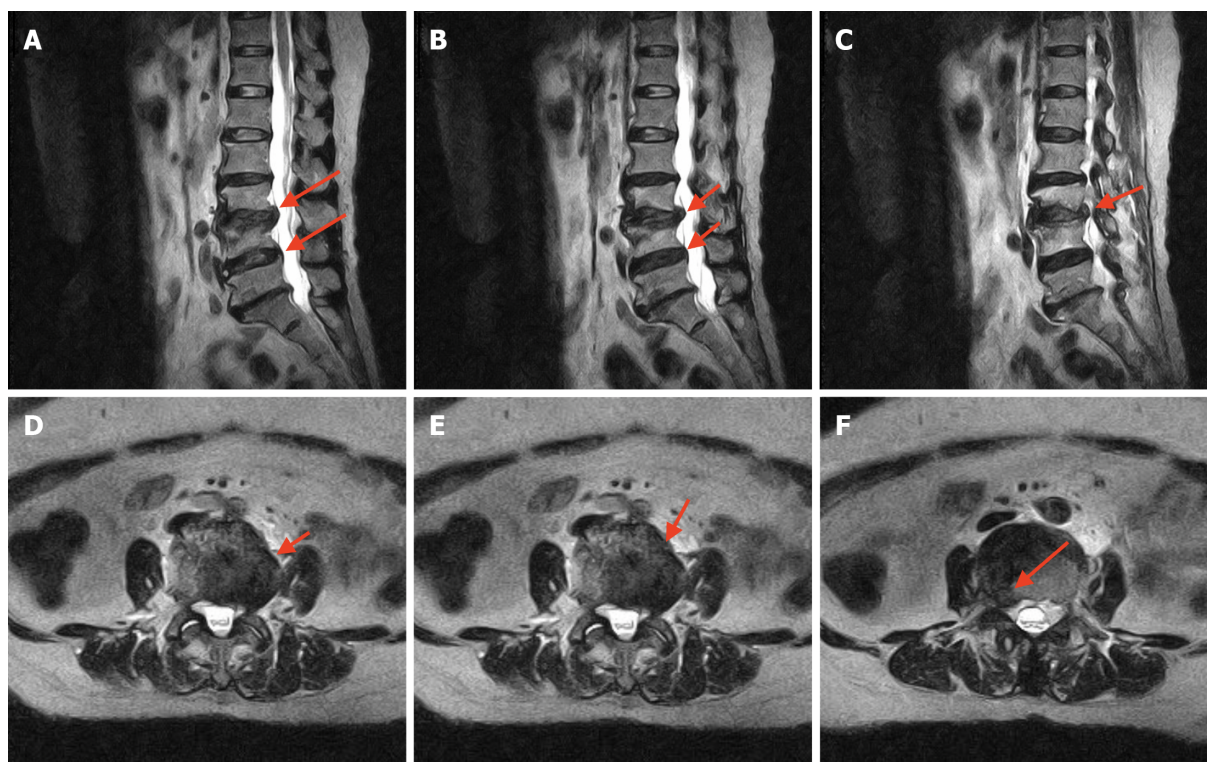
Surgical treatment was suggested and the severity of the disease, risks of surgery, and alternative treatments were discussed with the patient. Microlaminectomy and ossified ligamentum flavum removal followed by foraminotomy were planned; therefore, the traversing and exiting neural structures were free of compression. After adequate microlaminectomy, IntraSPINE®, an interlaminar dynamic spacer, was implanted between the L2-3 and L3-4 Levels of the interlaminar space to restore the disc height.

Microlaminectomy for foraminal decompression was performed at the L2/3 and L3/4 Levels. During microlaminectomy, a tiny unintended durotomy occurred, and CSF leakage was observed during decompressive microlaminectomy *via* an extradural spinal approach. To seal the CSF leakage, we covered the dural defect with oxidized regenerated cellulose, Surgicel, followed by DuraSeal™ (around 2 cc) and a final layer of dry Gelfoam (Pharmacia & Upjohn) before securing hemostasis. This step has been widely used in our past surgical experience in cases of incidental durotomy. After completing lumbar decompression, sealing the dura tear, and implanting the IntraSPINE®, a hemovac was used for blood drainage and the wound was closed.

The patient had no neurological discomfort one day after surgery; however, the following day, bilateral lower-extremity numbness and weakness occurred. The Medical Research Council (MRC) scale of muscle power decreased from 5 to 2 (5: Normal muscle power, 2: Active movement with gravity eliminated) on the distal muscles in the bilateral lower extremities and deteriorated gradually[8]. We attempted to remove urine from the Foley tube, but urinary retention was observed. The residual urine volume was > 200 cc. In addition, the bulbocavernosus and anal reflexes were absent. The neurological dysfunction might not have been related to the intraoperative decompression and disk height restoration using an IntraSPINE® device because the symptoms did not appear immediately but rather 2 days after the surgery. Based on these observations, CES was suspected.

An emergent T2-weighted phase MRI examination revealed regional fluid collection at the surgical bed, protruding anteriorly at the junction of L2 and L3 to L4 Levels. This fluid caused thecal sac indentation and narrowing of the central canal ([Figure 2](#)). Therefore, emergent exploration and decompression of the thecal sac were performed. Intraoperatively, a large amount of gel-like DuraSeal™ had formed a layer around the thecal sac and entrapped the extradural hematoma, resulting in spinal cord compression. We removed all DuraSeal™, exposing the bilateral L3 and L4 nerve roots, and ensured that no DuraSeal™ material was compressing the nerve roots ([Figure 3](#)).





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**Figure 1 Preoperative T2-weighted magnetic resonance imaging.** The lateral and sagittal views of the collapsed disc height at the L2-3 and L3-4 Levels and a bulging disk compressing the thecal sac and right neural foramen are visible. A-C: Lateral views; D-F: Sagittal views.

## OUTCOME AND FOLLOW-UP

Unfortunately, although the patient was undergoing rehabilitation and physical therapy was initiated, muscle power and urinary and stool incontinence persisted for four months postoperatively. Her American Spinal Injury Association score was A.

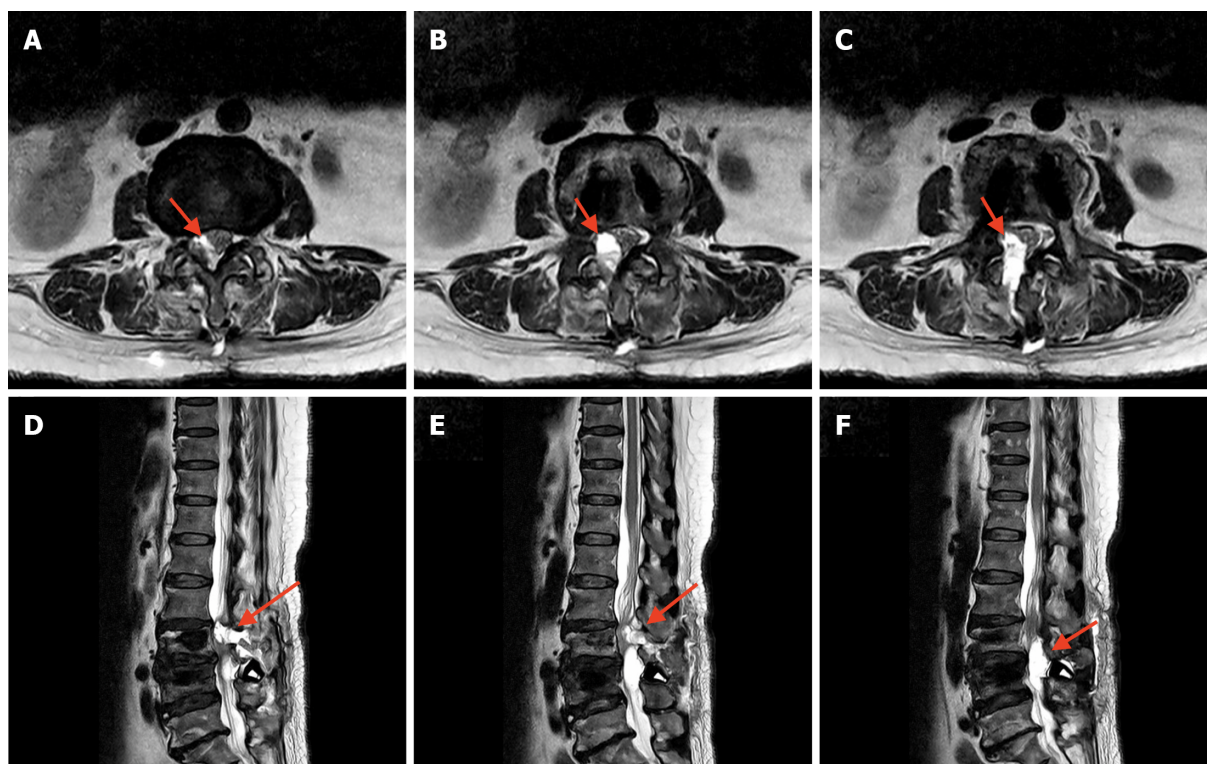
## DISCUSSION

Intraoperative incident durotomy is one of the most common complications of spinal surgery, especially revision surgery. This adverse event is always related to CSF leakage from the subarachnoid space *via* dural defects[9]. The incidence of incident durotomy ranges from 0.1% to 15.9%, depending on the difficulty of spinal surgery[9,10]. Despite the fairly low incidence, the risks and associated costs of these adverse incidents cannot be ignored[11]. Longer admission lengths, higher postoperative infection rates, and lower postoperative satisfaction were also noted in these patient groups.

If the CSF leakage is not sealed, further complications can develop, including nausea, vomiting, vertigo, tinnitus, postural headache, meningitis, and fistula formation[12]. The methods to control CSF leakage include direct dural repair. This can be achieved using an absorbable suture line or by grafting fat, muscle, or fascia to the tear[13,14]. However, direct repair may not be an ideal or feasible solution depending on the position of the tear[15]. DuraSeal™ provides a useful alternative treatment for dural tears because the material can expand to reduce CSF leakage[16]. Furthermore, the non-toxicity, bioabsorbability, and accessibility of this material have led to its widespread use[17]. DuraSeal™ can swell by up to 50%, reaching peak expansion within 3-14 d, and persisting for approximately four weeks[18].

Our study is not the first to report neurological complications associated with DuraSeal™ use. The first reported case was that of a 13-year-old girl who underwent cervical decompression and fusion for Chiari malformation in 2007[19]. In 2009, a case of postoperative cauda equina compression syndrome caused by the use of DuraSeal™ before spinal decompression surgery was reported[15]. Similarly, DuraSeal™-related CES was reported after total laminectomy and transforaminal lumbar interbody fusion in 2012[16]. All these cases involved nerve compression due to DuraSeal™ expansion.

Currently, microlaminectomy is preferred over traditional laminectomy for decompression to reduce the breakdown of bony structures and blood loss, as well as the length of hospital stay and healthcare costs[20]. However, microlaminectomy reduces the extradural space more than total laminectomy, with



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**Figure 2 Postoperative T2-weighted magnetic resonance imaging.** A-C: Sagittal views; D-F: Axial views. Regional fluid collection at the surgical bed, protruding anteriorly at the junction of L2 and L3 toward the L4 Level are visible. The fluid has caused an indentation of the thecal sac and narrowing of the central canal.



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**Figure 3 Removal of the gel-like Duraseal™.** The gel-like Duraseal™ is compressing the thecal sac and narrowing the central canal.

a consequent increase in the possible mass effects. Applying an expandable agent such as Duraseal™ dramatically increases the risk of CES compared to total laminectomy alone because the small extradural space provides limited space for Duraseal™ expansion. Furthermore, it is difficult to predict the nature of the expanding material in the epidural space, with potentially serious consequences.

It is important to achieve adequate hemostasis before dural closure. Duraseal™ is a self-polymerizing agent that can rapidly produce a watertight hydrogel layer over the dural surface. If hemostasis is not under control before applying Duraseal™, the hematoma can be entrapped, contributing to the development of complications[6].

There have been several reports of neurological complications following the intraspinal application of absorbable gelatin sponges, such as Gelfoam, or oxidized cellulose products, such as Surgicel, a loosely woven fabric of cellulose[21,22]. In 2015, a CES case was reported to have been caused by the



application of Surgicel after lumbar microdiscectomy and foraminal decompression[23,24]. Thus, except for DuraSeal™, excess intraspinal sealants should be removed when hemostasis is achieved[25].

## CONCLUSION

The present case highlighted that the potential postoperative expansion of DuraSeal™ should never be underestimated when this product is used in locations sensitive to compression, such as microlaminectomy. The publication of this case will raise awareness of the possibility of DuraSeal™ expansion and the concomitant, potentially irreversible, postoperative complications.

## FOOTNOTES

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