Cauda equina syndrome (CES) is a complex of clinical symptoms, including low back pain (LBP), bilateral leg pain, weakness in the lower extremities, saddle anesthesia, genitourinary dysfunction with overflow incontinence or retention, sexual dysfunction, and loss of rectal sphincter tone, occasionally with fecal incontinence.\(^4,5\) The diagnosis of CES is not difficult in patients with all of these symptoms. However, the diagnosis is difficult when these symptoms develop asymmetrically or incompletely or one or more of these symptoms are absent. Even if the initial presentation is incomplete or atypical, CES can progress and potentially cause severe neurological symptoms and disability.\(^3\) Patients with CES should be referred immediately for surgical consideration, as treatment delay may result in serious morbidity, such as loss of bladder, bowel, and sexual function with potential legal consequences.\(^9\) Therefore, it is important that all clinicians who assess patients with LBP should have a detailed knowledge of the atypical presentation and signs highly suggestive of CES. A few cases of atypical presentation of CES due to lumbar disc herniation have been reported, but atypical presentation of CES following a lumbar spinal fracture have been rarely reported. We report a case of urinary retention and sphincter dysfunction without sciatica or motor weakness following an L3 burst fracture in a 52-year-old male and discuss the atypical presentation of CES and treatment of traumatic CES.

### Case Report

A 52-year-old man presented to the emergency department with LBP. The patient fell from approximately 2 m-container box to the ground 2 hours previously. He complained of no pain and no numbness in either leg. A neurological examination revealed grade 5/5 muscle strength and no sensory changes in the lower extremity. Normal rectal tone was present and there was no saddle anesthesia. Plain lateral radiography and computed tomography (CT) scan...
showed a burst fracture with spinous process fracture at L3 with more than 50% of the vertebral body height loss measured on Picture Archiving and Communication System (π-ViewSTAR, INFINITT Healthcare Co., Seoul, Korea) (Figure 1A, B, and C). Spinal canal diameter was estimated at the level of the fracture on the axial CT image that traversing the level of the pedicle and was defined as the distance between the midsagittal border of the posteriorly dislocated bone fragment and the lamina.\(^{20}\) The normal size of the canal was estimated by the average midsagittal diameter of the levels adjacent to the fracture.\(^ {20}\) Spinal canal diameter at the fractured level was 7.92 mm using this method and about 55% narrowing of the spinal canal was observed compared to the normal level (Figure 1C). Magnetic resonance imaging (MRI) revealed findings suspicious of injury in the supraspinous ligament (Figure 1D). Initially, we thought the patient had no neurological deficits, so internal fixation and posterior fusion without decompression of the thecal sac were planned under the impression of an unstable burst fracture.\(^ {26}\)

He underwent pedicle screw fixation from L1 to L4 and posterior fusion the next day, with internal distraction to reduce the fragment away from the spinal canal. Motor strength and sense in his legs were intact postoperatively. Pain was tolerable 3 days postoperatively, and the patient began to ambulate without difficulty. The Foley catheter was removed. He did not feel the desire to urinate. He was able to expel only a few drops of urine by applying pressure to the anterior abdominal wall when he tried to void at the time of 500 cc of urine in his bladder measured with bladder ultrasonography. This was managed initially by regular intermittent catheterization, but he complained of urethral discomfort.
roots travelling more laterally, and the lower sacral nerve

truded L4

ness, as in the present case, in patients with centrally pro

volvement, have been reported.

weakness, without saddle anesthesia, with only fecal incon

CES, such as unilateral sciatica, without sciatica or motor

sensory changes are found on a neurological

examination. However, these findings can often be missed

because 17 different definitions had been proposed in

the literatures. Definitive symptoms or signs for diagnosing

CES vary among authors. Balasubramanian et al.9 suggest-
ed that saddle sensory deficit has a higher predictive value

than other clinical features when diagnosing CES and a de-

gree of canal compromise greater than 75% is capable of

producing CES. Bell et al.5 recommended an urgent MRI

assessment in all patients who present with new onset uri-

nary symptoms in the context of LBP or sciatica. Domen et

al.6 reported that urinary retention above 500 mL after mictu-

rition measured by bladder scan is the most promising di-

agnostic tool to predict the presence of cauda equina com-

pression on MRI. Gooding et al.7 argued that a digital rectal

examination has no significant value in the diagnosis of

CES and cannot be used as a discriminator to rationalize an

urgent MRI. Fairbank et al.8 noted that there is limited evi-

dence from individual symptoms or signs from a patient’s

history or clinical examination, respectively, can be used to
diagnose CES in their systematic review.

To overcome these discrepancies, Fraser et al.7 proposed
a single definition of CES after reviewing 105 articles and,
one or more of the following must be present for a diagnosis
of CES: 1) bladder and/or bowel dysfunction, 2) reduced
sensation in the saddle area, or 3) sexual dysfunction, with
possible neurological deficits in the lower limb (motor/sen-
sory loss or reflex change). Based on the above mentioned
proposal, the presence of mild saddle anesthesia or urinary
retention are important findings for diagnosing CES, par-

ticularly when patients have no leg pain and when no motor

weakness or sensory changes are found on a neurological

examination. However, these findings can often be missed

in a trauma patient or during the postoperative period.8,9

Routine placement of a Foley catheter during the initial re-
suscitation after trauma or during the postoperative period
can make urinary dysfunction difficult to detect.10,12 In the

Discussion

A prompt and correct diagnosis of CES is sometimes dif-

cult. When symptoms or signs of CES develop asymmetric-

ally or incompletely or one or more of symptoms are absent,
the diagnosis is difficult. Cases with atypical presentation
of CES, such as unilateral sciatica, without sciatica or motor
weakness, without saddle anesthesia, with only fecal incon-
tinence and perineal hypesthesia, and without bladder in-

volvement, have been reported.5-7,13,19 CES presenting with
only urinary dysfunction without sciatica and motor weak-

ness, as in the present case, in patients with centrally pro-

truded L4–5 lumbar disc herniation has also been report-
ed.20 The cauda equina is arranged with the higher nerve

roots travelling more laterally, and the lower sacral nerve

A Foley catheter was placed again the next day. He also

complained of constipation, but this improved with kinetic
agents (mosapride 10 mg three times a day) and laxatives
(magnesium oxide 500 mg three times a day). Plain lateral

radiography and CT scan confirmed restoration of sagittal
alignment and appropriate positioning of the screws (Figure

2A and B). Although spinal canal diameter increased slight-
ly to 9.29 mm, significant thecal sac compression was de-
tected (Figure 2C). We recommend MRI and additional an-
terior decompressive surgery, but he refused because of
uncertainty of recovering from voiding difficulty after the
surgery and his poor economic status. The Foley catheter
was removed again 1 week later, but he did not void as be-

fore. Electromyography (EMG), nerve conduction study
(NCS), and urodynamic study (UDS) were performed 3
weeks after the surgery. EMG and NCS showed no abnor-
malities in L2–S1 roots. However, abnormal spontaneous
activity and decreased motor action potential amplitude in

the abductor hallucis muscle were detected, which did not
exclude the possibility of S2 root denervation. Further elec-
drodiagnostic evaluation for pudendal somatosensory evoked

potentials and anal sphincter EMG were recommended, but
he refused. No bladder detrusor muscle activity and areflex-
ic neurogenic bladder were found in the UDS. He was man-
aged with suprapubic cystostomy for 3 months. Voiding im-
proved gradually during that time. CT scan of the lumbar
spine at that time revealed a more resolved fractured bone

fragment, and the spinal canal diameter increased to 10.94
mm (Figure 2D). After removing the cystostomy, he was
able to void by himself, but had a weak stream and residual
urine. He voided with abdominal straining or sitting on the

toilet seat to void fully. Voiding has not returned to normal

at the 15-month follow-up.

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The present case, an indwelling Foley catheter was used in the emergency department after initial neurological examination and radiologic evaluation of plain film and CT. But the volume of urine drained immediately after inserting the catheter, which could be the clue for voiding difficulty, was not checked because no abnormal finding suggestive of CES, such as weakness or saddle anesthesia, was found in the neurological examination. The patient had Foley catheter for 3 days after the surgery. Therefore, it was too late to detect the voiding difficulty and this may have made the voiding difficulty long-lasting or perhaps permanent.

The effect of surgical treatment on recovery of neurological deficits in patients with a thoracolumbar or lumbar spinal fracture remains controversial. Some authors have found no association between initial canal encroachment, final spinal canal area, the extent of decompression, treatment technique, or spinal level of injury and neurological recovery. Although nonsurgical care of traumatic CES likely results in some neurological improvement, many authors advocate that the vast majority of these injuries should be treated with surgical stabilization and, when necessary, concomitant decompression. Not only is this likely to reduce the duration of hospital stay and facilitate nursing and rehabilitation, but it is clearly safe from a neurological perspective and may optimize neurological recovery. Evaluating data from 59 patients with lumbar fractures and incomplete neurological deficits and noted that patients had statistically greater motor improvement, regardless of whether the decompression was anterior or posterior, compared with patients who underwent posterior fusion alone at a mean 19 month follow-up. Kaneda et al. retrospectively reviewed 69 patients with traumatic CES. Nearly 75% of patients had complete neurological recovery and complete recovery was found in 9 of 12 patients with voiding difficulty after anterior decompression and stabilization. Bradford and McBride retrospectively examined data from 59 patients with thoracic and lumbar fractures and 17 had CES. There were highly significant rates of bowel and bladder recovery between the posteriorly (11.7%) and anteriorly (70%) treated groups. The timing of surgery in patients with traumatic CES also remains controversial. Thongtrangan et al. reported that 14 of 17 patients had satisfactory outcomes and recommended that surgery be performed within 48 hours of CES onset. Although controversies remains, early decompressive surgery can increase the chance of recovery from neurological deficits including bladder or bowel dysfunction. However, in the present case, missing CES during the early period of trauma led us to fixation and fusion of fractured site without decompression of the nerve roots, and this may have reduced the chances of recovering bladder dysfunction.

**Conclusion**

Symptoms of CES may be atypical. Suspicion of bladder or bowel dysfunction is very important particularly when no motor or sensory changes are evident. A high index of suspicion of CES is essential when encountering patients with LBP or a history of trauma to the lumbar spine to prevent missing a diagnosis of CES and avoiding severe neurological symptoms and disabilities.

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