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Educational Corner

Cauda Equina Syndrome, a Disease with Various Presentations and Potential for Misdiagnosis: Educational Corner

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Background

Cauda equina syndrome (CES) is a condition in which a bundle of nerves at the end of the spinal cord is injured. Usually, it presents with low back pain (LBP), pain sciatica (radiated down the leg), lower extremity weakness or sensory impairment, numbness around the anus, and bladder and bowel control loss. It could start rapidly or gradually due to disc herniation (most common), spinal stenosis, cancer, trauma, epidural abscess, and epidural hematoma (1). After suspected clinical symptoms, medical imaging is required for confirmation of diagnosis. Laminectomy is needed to relieve the pressure on the cauda equina within 48 hours. Timing to surgery is vital, though the best time to conduct it remains controversial. Permanent sequel such as incontinency, sexual dysfunction, or numbness may still happen. Even despite laminectomy, 20% of patients experience poor outcomes (2). We aimed to review the CES and its symptoms to avoid misdiagnosis as the case introduced in this paper.

Cauda Equina

The bundle of axons at the end of the spinal cord is known as cauda equina, which was first described by Andre du Laurens. It is a Latin term meaning "horsetail." Cauda equina axons innervate lumbar, sacral, and coccygeal spinal cord levels of sensory and motor neurons (1).

Cauda Equina Syndrome (CES)

CES is an impairment and injury of cauda equina followed by numerous symptoms. It has two types, CES-incomplete (CES-I) and CES-retention (CES-R). CES-I has minor urinary consequences (i.e., reduced bladder sensation and difficulty voiding) with other common features. However, CES-R has overflow incontinence and complete urinary retention (1, 2).

Causes

Lumbar disc herniation is the most common cause of CES, as 3% of all lumbar disc herniations result in CES (1). Additionally, CES could occur after spinal surgery, which is triggered by postoperative edema. Hence, series of neurological examinations of spinal roots after surgery is essential. On the other hand, trauma, spinal fracture, tumor, and abscess due to infection are less common causes of CES (3).

Pathophysiology

The spinal cord usually ends at the L1 vertebral body level, and the cauda equina starts following it. Mostly, lower motor neurons are injured in this syndrome, so lower extremity motor weakness, sensory impairment, and decreased or absent reflexes could be the consequences. Bladder dysfunction is another consequence of this syndrome (4). As detrusor urinae muscle and internal sphincter are innervated by parasympathetic S2, S3, and S4 spinal roots, any lesions to these roots cause contraction of detrusor urinae muscle and relaxation of the internal sphincter. At the same time, sympathetic innervation (T11-L3) promotes urinary storage and external sphincter is innervated by the pudendal nerve arising from S2, S3, and S4 nerves (5).

As a consequence of CES, patients lose motor and sensory function, which makes them unable to sense the expansion of the bladder, and contract the muscle, leading to retention followed by overflow incontinence (4, 6). Since nerve roots of cauda equina are only protected with endoneurium, they are susceptible to traumatic injuries. Mechanical compression of cauda equina decreases the nutritional supply of nerves. Moreover, this mechanical compression causes closed compartment syndrome, increased edema, and pressure (4-6).

Clinical Presentation

Patients with CES have signs and symptoms varying from LBP to loss of bladder and bowel control. Bilateral or unilateral sciatica, lower extremity weakness, decreased reflexes, and ensory deficits could also exist (1). Bladder dysfunction, aforementioned, starts with urinary retention and continues with overflow incontinency. The LBP is usually severe, but as time passes, it could disappear. Although bilateral sciatica is strongly associated with CES, it could be absent. Saddle anesthesia (i.e., perineum, buttocks, posteromedial sensory loss) appears in delayed patients associated with poor outcomes. If bladder dysfunction or saddle anesthesia are absent, the patient is at the risk of future CES. By expressing these two symptoms, the bomb clock starts ticking (7, 8).

Comorbidities

Obesity worsens the condition due to reducing the canal size, associated with disc herniation and changes in inflammatory mediators (9).

Patient Evaluation

History of Patient: A complete history is required,

which is essential. The history of onset of symptoms, bladder and bowel dysfunctions, and saddle anesthesia should be included. The time of any symptom's onset is critical for the outcome, time of treatment, and legal assessment (10).

Examination

Physical examination after taking a history of the patients suspected of CES is necessary and includes sacral nerve roots examination. Perineal, perineum, and posterior thigh sensation should be assessed by pinprick test, pressure, and light touch. Tones of anal and urinal sphincters should be evaluated; their low tone is correlated with CES. Anal wink test and bulbocavernosus reflex check anal sphincter tone (10). The palpation of the bladder, measurement of patients' post-void residual volume, is applied to assess urinary problems. Prodromal symptoms, followed by the more classic sign of CES, suggest the syndrome in patients. Noticing prodromal signs in postoperative spine problems is crucial since they may only develop prodromal symptoms, and physicians should suspect CES.

Radiographic Evaluation

Magnetic resonance imaging (MRI) is the choice of investigation, which determines the presence of pressure on cauda equina and distinguishes the cause, whether it is a tumor, hematoma, or abscess. However, this imaging method is problematic for patients with claustrophobia or individuals for who MRI cannot be used (those having pacemaker or retained metal fragments) (11, 12). For these subjects, the choice imaging is computerized tomography scan (CT-scan). This imaging method should be considered emergently to confirm the diagnosis. For patients who could not undergo imaging investigations and whose strong clinical symptoms are suspected by a clinician, decompression surgery should perform.

Timing of Investigation

There is a hypothesis that earlier investigation and treatment have a better outcome, as it is thought that cauda equina is a vital structure and needs investigation and treatment to access blood supply and venous return and decompress the cauda equina as soon as possible. On the other hand, not numerous patients undergo investigations late, so no findings are reached. Each patient should be treated individually. It is recommended to perform an MRI within one hour if it is available. The secondary classical sign is an urgent necessity of MRI. Those patients presenting only prodromal symptoms have more time to be investigated. Urinary dysfunction and symptoms indicate another urgent need for MRI (13, 14).

Treatment

Decompression surgery of the cauda equina is the treatment. The surgery should provide enough space for the cauda equina to be decompressed; it is hard to determine in practice. This surgery could range from a simple microdiscectomy to a wide laminectomy. The superiority of a surgical approach to another has not been investigated yet. However, it is recommended to go forward step by step, to minimize further neural tissue damages (15).

Timing of Surgery

Sooner decompression is carried out to have a better outcome, proving by insufficient quality studies. These studies were conducted retrospectively with small sample sizes. Many studies revealed that a delay of more than 48 hours for both type CES-I and type CES-R is associated with poorer outcomes (16). However, bladder function is proved a predictor for the outcome. A meta-analysis revealed that patients who undergo surgery within 24 hours after onset are more likely to recover bladder function compared to patients who undergo surgery after 24 hours (17, 18).

Legal Considerations

Sice CES may cause neurological losses such as sexual dysfunction and bowel and bladder complications, these cases should be considered legal. Because it is obligatory to confront patients with CES in practice, surgeons should initially deal with them. Firstly, they should explain the CES to patients thoroughly, from symptoms to permanent complications, in their understanding language. Notably, they should emphasize long-term neurological deficits (19).

Case Presentation

An 18-year old female patient presented to our center with acute LBP that referred to the left lower limb. After two days, she returned to an unskilled bonesetter and after that she had severe weakness of both lower limbs (2/5) and urinary incontinency.

She was first admitted to the neurology department suspicion to Guillain-Barre syndrome or other neurologic disease, which were ruled out. After 20 days from admission, an orthopedic spine consult was required. She complained of lumbar pain radiated to lower limbs, and the numbness and paresthesia of both lower limbs were noticeable. She could flex knees and hips (not full flexion, only 60 degree) without strength of extension. No reflexes were seen in her lower limbs.

In the orthopedic consult, we required a lumbosacral x-ray (Figure 1) and MRI (Figure 2). As seen in the MRI of the patient, L3-L4, L4-L5, and L5-S1 levels had central disc herniation, but a cauda equine block in the L3-L4 level.

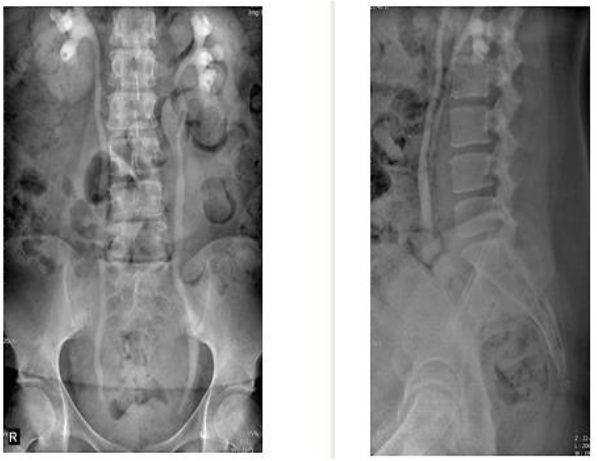

Figure 1. Lumbosacral x-ray [Anteroposterior (AP) and lateral]

She underwent decompression surgery and posterior spinal fusion (L3 to S1 levels), which led to a dramatic relief of the signs and symptoms (not wholly) (Figure 3).

Numbness and paresthesia decreased, and she could move her lower limbs. She could flex and extend her lower limbs (knees and hips), her reflexes returned, and she regained urinary and bowel control.

Conflict of Interest

The authors declare no conflict of interest in this study.

Acknowledgments

None.

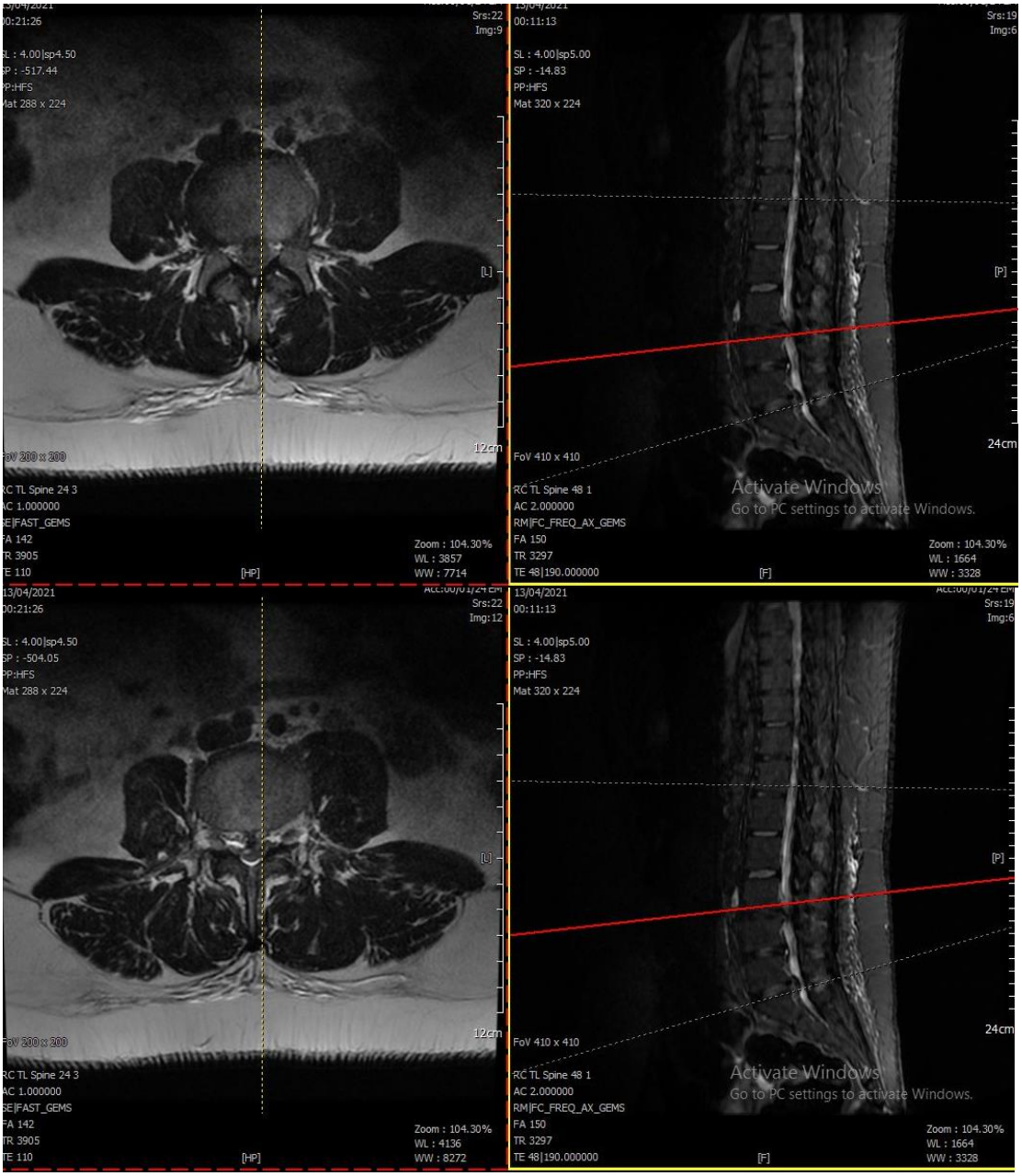

Figure 2. Lumbosacral magnetic resonance imaging (MRI) in axial and sagittal view showed L3-L4 central disc herniation that blocked the cauda equina.

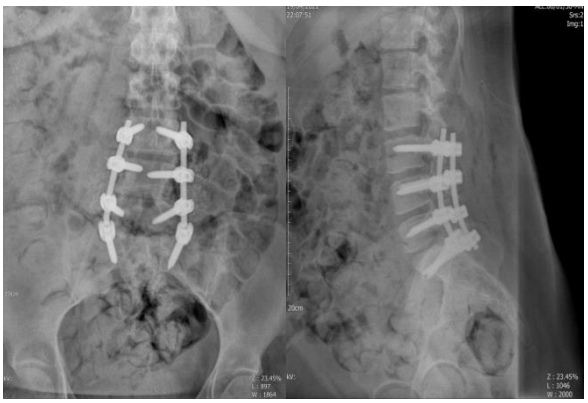

Figure 3. The patient underwent posterior spinal fusion and decompression surgery

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