

SS-006 — From Low Back Pain to Paraplegia

Bel Ağrısından Paraplejiye

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Çalışma türü / Manuscript type: Olgu sunumu / Case report

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From Low Back Pain to Paraplegia

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Introduction

Acute bilateral lower limb weakness is a challenging presentation in the emergency department, as it may be associated with several conditions requiring urgent diagnosis and treatment, including spinal cord compression, cauda equina syndrome, spinal vascular disorders, inflammatory myelopathies, and acute polyradiculoneuropathies. We present a patient who was able to walk on arrival but developed rapidly progressive neurological deficits while being observed in the emergency department.

Case Presentation

A man in his forties presented to the emergency department early in the morning because of low back pain that had started the previous evening. He had a history of intermittent low back pain and lumbar disc herniation but no other known medical condition or regular medication use. On arrival, he was alert, cooperative, and oriented, and his vital signs were stable.

The patient was initially able to walk in the emergency department. During follow-up, however, weakness developed first in his left leg and then progressed to involve the right leg. Neurological examination showed normal strength in both upper extremities (5/5), whereas muscle strength was 0/5 in both lower extremities. Patellar and Achilles reflexes were absent. Bilateral lower extremity hypoaesthesia was also present and was more prominent on the left. The patient reported that he could still feel the urge to urinate and defecate. He had no history of an upper respiratory tract infection or diarrhea during the preceding 10 days.

Diffusion-weighted brain MRI performed at another center showed no evidence of an acute neurological lesion. Non-contrast lumbar MRI revealed a suspicious area of increased T2 signal at the L3 level. Neurosurgery was consulted, but emergency surgery was not considered necessary. The patient was subsequently evaluated by the neurology team and admitted to the neurology ward for further investigation.

Discussion

Rapidly progressive paraplegia has a broad differential diagnosis. Spinal compression, cauda equina syndrome, spinal cord ischemia, inflammatory myelopathies, and Guillain-Barré syndrome should all be considered, particularly when neurological deficits evolve over a short period. In such patients, the pattern and progression of weakness, neurological localization, and repeated neurological examinations can provide important clues to the underlying diagnosis.

Conclusion

A patient who is able to walk on arrival may still develop a severe neurological deficit within hours. When neurological symptoms are progressive, a single examination may not reflect the subsequent clinical course. Repeated neurological assessment and timely investigation of potentially serious spinal and peripheral neurological causes are therefore essential in the emergency department.

From Low Back Pain to Paraplegia

Tam Metin

Introduction

Acute or rapidly progressive weakness of both lower extremities is an important neurological presentation in the emergency department (ED), particularly because several potentially serious conditions may present in a similar manner. Depending on the anatomical level involved, the underlying pathology may arise from the spinal cord, conus medullaris, cauda equina, nerve roots, peripheral nerves, neuromuscular junction, or, less commonly, muscle. Among the major conditions that need to be considered are spinal cord compression, cauda equina syndrome (CES), spinal cord ischemia or infarction, inflammatory myelopathies, and acute polyradiculoneuropathies such as Guillain-Barré syndrome (GBS) [1–4]. Distinguishing among these disorders is clinically important because both the urgency of intervention and the diagnostic approach may differ considerably.

Low back pain associated with a newly developing neurological deficit should raise particular concern for structural or vascular spinal pathology. Current recommendations from the World Federation of Neurosurgical Societies emphasize the need for urgent magnetic resonance imaging (MRI) when CES is suspected, as clinical findings alone may not reliably exclude the diagnosis [1]. At the same time, imaging findings should always be interpreted in the context of the patient's clinical course. In some acute spinal cord disorders, particularly spinal cord ischemia, MRI abnormalities may be subtle or even absent during the early phase, with more characteristic changes becoming apparent on subsequent imaging [2,3].

The speed at which neurological deficits evolve may therefore provide an important diagnostic clue. Symptoms that progress over minutes or hours may be seen in vascular events or rapidly developing compressive lesions, whereas inflammatory and peripheral neurological disorders may follow a more variable course [2,4,5]. Nevertheless, overlap is common, and a single neurological examination may not adequately reflect a condition that is still evolving.

We present a patient who arrived at the ED with low back pain and was initially able to walk, but subsequently developed profound bilateral lower-extremity weakness during observation. The case illustrates the importance of repeated neurological examination and of maintaining a broad differential diagnosis when neurological findings change over a short period of time.

Case Presentation

A man in his forties presented to the ED early in the morning with low back pain that had begun the previous evening. He had a history of intermittent low back pain and lumbar disc herniation, but no other known medical condition and no regular medication use.

At the time of admission, he was alert, cooperative, and oriented, and his vital signs were stable. He was also able to walk independently. During follow-up in the ED, however, weakness first developed in the left lower extremity and then progressed to involve the right lower extremity.

On repeat neurological examination, muscle strength was preserved in both upper extremities at 5/5, whereas motor strength in both lower extremities had deteriorated to 0/5. Patellar and Achilles reflexes were absent bilaterally. Hypoesthesia was present in both lower extremities and was more pronounced on the left. The patient stated that he was still able to sense the need to urinate and defecate. He denied any upper respiratory tract infection or diarrhea during the preceding 10 days.

Diffusion-weighted brain MRI performed at another center did not reveal an acute neurological lesion. Non-contrast lumbar MRI demonstrated a suspicious area of increased T2 signal at the L3 level. Neurosurgical consultation was obtained, but emergency surgical intervention was not considered necessary. The patient was subsequently evaluated by the neurology team and admitted to the neurology ward for further investigation.

Discussion

Rapidly progressive paraplegia presents a considerable diagnostic challenge because compressive, vascular, inflammatory, and peripheral neurological disorders may initially produce overlapping clinical findings. In the present case, the combination of acute low back pain, rapidly evolving bilateral weakness, sensory disturbance, and areflexia required consideration of several possible diagnoses.

CES was among the important initial possibilities. Low back pain accompanied by bilateral neurological deficits may indicate involvement of the lumbosacral nerve roots, and typical manifestations of CES may include lower-extremity weakness, sensory loss, saddle anesthesia, and bladder, bowel, or sexual dysfunction [1]. In our patient, the continued ability to perceive the urge to urinate and defecate did not completely rule out an evolving CES. On the other hand, there was no documented saddle anesthesia, urinary retention, impaired anal tone, or MRI-confirmed compressive lesion in the available information. Moreover, the neurosurgical team did not identify a need for emergency surgery. Taken together, these findings were not sufficient either to confirm or to completely exclude CES.

Spinal cord ischemia or infarction was another important consideration because of the rapid progression of the neurological deficit. Spinal cord infarction can reach maximal severity within a relatively short period, and sensory abnormalities are frequently observed [2]. Early imaging may also be misleading. In the series reported by Zalewski et al., the initial spinal MRI was normal in approximately one-quarter of patients with spontaneous spinal cord infarction [2]. Subsequent studies and reviews have similarly emphasized the importance of clinical progression, lesion morphology, diffusion-weighted imaging, and repeat MRI when the initial study is nondiagnostic but clinical suspicion remains high [3]. The rapid deterioration observed in our patient was therefore compatible with a vascular spinal process, although the available clinical and imaging data were insufficient to establish this diagnosis.

Inflammatory myelopathy also remained within the differential diagnosis. The diagnosis of acute myelopathy requires consideration of symptom progression, neurological localization, spinal MRI findings, laboratory investigations, and, when appropriate, cerebrospinal fluid analysis [5]. In the present case, the available records did not include cerebrospinal fluid findings, contrast-enhanced whole-spine MRI, or inflammatory and autoimmune investigations. For this reason, an inflammatory myelopathy could neither be confirmed nor excluded on the basis of the available data.

GBS and other acute polyradiculoneuropathies also deserve consideration, particularly because the patient developed profound bilateral lower-extremity weakness together with loss of patellar and Achilles reflexes. According to current EAN/PNS guidance, the clinical spectrum of GBS may include weakness, sensory symptoms, pain, autonomic dysfunction, cranial nerve involvement, and respiratory compromise [4]. The areflexic bilateral paralysis in our patient was therefore compatible with a peripheral polyradiculoneuropathic process. However, several features made a straightforward diagnosis of GBS difficult, including the prominent low back pain, the initial asymmetry of weakness, the reported MRI abnormality at L3, and the absence of cerebrospinal fluid and electrodiagnostic data. The absence of a preceding respiratory or gastrointestinal infection did not exclude GBS, but it offered no additional support for the diagnosis.

Overall, the available information did not allow a definitive etiological diagnosis to be established. The principal clinical value of this case lies instead in the speed and extent of neurological deterioration observed during ED follow-up. A patient who was able to walk at presentation progressed to complete bilateral lower-extremity paralysis within a short period. This change in neurological status underscores the importance of repeated examination rather than relying on the initial clinical assessment alone.

The case also highlights the limitations of interpreting early imaging in isolation. MRI is central to the investigation of suspected spinal pathology, but the absence of a definitive lesion on the first study does not necessarily exclude an evolving vascular, inflammatory, or compressive process. Clinical progression should therefore remain a major determinant of whether additional imaging or further neurological investigation is required.

From an emergency medicine perspective, the most important lesson is that neurological findings may change substantially over time. Reassessment of motor strength, reflexes, sensory function, gait, and sphincter-related symptoms is particularly important when the patient reports progression or when new deficits emerge during observation.

Conclusion

Acute low back pain accompanied by progressive neurological findings requires careful observation and repeated neurological examination. A patient who is able to walk at presentation may still develop severe motor deficits within hours. Being ambulatory at the initial evaluation should therefore not be considered reassuring when neurological symptoms are evolving. Prompt reassessment and a broad differential diagnosis remain essential for identifying compressive, vascular, inflammatory, and peripheral neurological causes in the emergency department.

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