

A Young Male with Lumbar Pain and Acute Weakness of the Lower limbs

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Case Report

A 29-year-old male was admitted to our hospital in November, 2004, because of acute onset of weakness in both legs. He had been in good health until one week prior to admission, when he developed low-back pain, without trauma, that resolved with over-the-counter analgesics. One day prior to admission, intense lumbar pain reoccurred, followed few hours later by difficulty to stand up and inability to walk. The patient worked as a truck-driver. He did not recall prior trauma, diarrheal or febrile illness, insect or tick bites. He had not been vaccinated for influenza.

On examination, he was overweight, afebrile, diaphoretic, in non-acute distress. His blood pressure was 140/90 mmHg; his heart rate was 110 beats per minute; his oxygen saturation was 97%. The general physical examination was normal. The neurological examination showed areflexic weakness affecting both legs symmetrically. No sensory disturbances were found.

Laboratory results included normal hematologic values, blood chemistry with elevated glucose (230 mg/dL) and triglyceride (747 mg/dL) levels, and proteinuria. Two attempts to obtain cerebrospinal fluid (CSF) were unsuccessful. A chest X-ray was normal; an X-ray of the lumbar spine showed sacralization of the 5th lumbar vertebra. An MRI of the spine showed an extruded L3-L4 disk with complete obliteration of the central canal (Fig. 1). An electromyogram (EMG) found signs of demyelinating neuropathy (marked slowing of nerve conduction velocity), affecting both legs symmetrically. Three days after admission, he underwent surgery for excision of the extruded disk. A CSF sample obtained during surgery showed normal cell-count, glucose, and protein values. The patient's neurological condition remained unchanged. One week after admission, a repeated CSF exam showed 2 cells/mm³, normal glucose, and elevated proteins (100 mg%). A repeated EMG remained unchanged. Additional serologic studies included a negative RPR test, positive anti-

cytomegalovirus (CMV) IgG antibodies, negative anti-CMV IgM antibodies, negative anti-HTLV-I/II antibodies, and negative anti-GM1, anti-GQ1b, and anti-GD1b antibodies. Bacterial and fungal CSF cultures were negative. Treatment with intravenous immune globulin (0.4 g per Kg per day) was given for five days.

Over the following weeks, the patient's clinical condition slowly improved. Six months after admission, the patient is able to walk, climb stairs, and perform his daily activities without limitations. The neurological examination is normal, with the exception of decreased dorsiflexion strength of both feet, and absent knee and ankle reflexes. His hyperglycemia and hypertriglyceridemia have been controlled with a low-fat, low-calorie diet.

This case constituted a diagnostic and therapeutic challenge. Due to the acute motor deficit, the finding of an extruded lumbar disk prompted surgical intervention to avoid neurological damage secondary to the mechanical compromise caused by the herniated disk. However, the clinical presentation, together with the EMG and CSF findings, also supported the diagnosis of Guillain-Barré syndrome (GBS) (1); this prompted the administration of intravenous immunoglobulin.

In the absence of radicular pain, the finding of an extruded lumbar disk on MRI was unexpected (2). In addition, it was difficult to ascertain whether the extruded disk was responsible for the symptoms in our patient because disk extrusions have been found in asymptomatic people undergoing MRI evaluations of the lumbar spine (2). On the other hand, few other conditions besides GBS manifest as acute demyelinating neuropathy (1). The absence of antiganglioside antibodies in our case does not exclude GBS (1, 3)

The diagnostic approach to this case deviates from the Occam's Razor principle of parsimony stating that "one should not make more assumptions than the minimum needed" (4). In our patient, more than one assumption was necessary for appropriate treatment of two unrelated, but coincidental, and potentially severe clinical conditions.

References

1. Hauser SL, Asbury AK. Guillain-Barré syndrome and other immune-mediated neuropathies. In: Harrison's Principles of Internal Medicine, 16th Edition, DL Kasper et al (eds). McGraw Hill, 2005.
2. Jensen MC, Brant-Zawadzki MN, Obuchowski, Modic MT, Malkasian D, Ross JS. Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med 1994; 331: 69-73.
3. Visser LH, Van der Meche FG, Van Doorn PA, et al. Guillain-Barré syndrome without sensory loss (acute motor neuropathy). A subgroup with specific clinical, electrodiagnostic and laboratory features. Dutch Guillain-Barré Study Group. Brain 1995; 118:841-847.
4. Hilliard AA, Weinberger SE, Tierney LM, Jr., Midthun DE, Saint S. Occam's Razor versus Saint's Triad. N Engl J Med 2004; 350:599-603.

Figure 1. MRI of the lumbar spine. *Left panel:* sagittal T2-weighted scan showing the extruded disk (H). *Right panel:* transversal views showing almost complete obliteration of the central canal (arrows); *top:* T1-weighted scan; *bottom:* T2-weighted scan.