Case Report

L4-5 disk lesion resulting in back pain with bowel, bladder and sexual dysfunction without paraparesis

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This is a case report of a patient with bowel, bladder and sexual dysfunction associated with low back pain. This patient had an essentially normal neurological examination. There was radiological evidence of a disk lesion, and urodynamic findings consistent with lower motor neuron bladder dysfunction. His symptoms are attributed to a L4-5 disk herniation resulting in a partial cauda equina syndrome. The relevant anatomy is reviewed, and the differential diagnosis is discussed.

Keywords: cauda equina lesion; urinary incontinence; fecal incontinence; paraplegia; disk lesion; paraplegia

Introduction

Classically, a cauda equina syndrome on the basis of a disk lesion results in flaccid paraplegia as well as bowel and bladder dysfunction. However, on rare occasions, disorders of micturition can be the only clinical presentation of a cauda equina lesion.¹ The following is a unique case where a patient had an MRI documented L4-5 disk herniation that resulted in bowel, bladder, and sexual dysfunction without any clinical or electrodiagnostic abnormalities in the lower extremities.

Case report

A 30 year old right-handed male custodian of East Indian descent presented to the physical medicine clinic with a 2 month history of low back pain, and occasional intermittent right leg paresthesiae. He had sustained this injury while lifting an 18 kilogram garbage container. Two weeks after his injury, he experienced two episodes of urinary incontinence and one episode of fecal incontinence. In addition, he complained of erectile dysfunction. Past medical history included hypertension which was controlled by beta-adrenergic blockers. The remainder of the history was non-contributory.

On examination, mental status and cranial nerves were intact. Muscle stretch reflexes were symmetrical.

No sensory deficit was identified. Power and rectal tone were normal. Plantar response was normal. One examiner did detect an asymmetrically absent cremasteric reflex. No herpetic skin lesions were identified.

An MRI of the lumbar and thoracic spinal cord demonstrated a moderate size left paramedian L4-5 disk herniation with sequestration of disk fragments (Figure 1). A CT myelogram confirmed the MRI findings. Cerebral spinal fluid revealed a red blood cell count of 147 (believed to be due to a traumatic puncture), normal glucose and protein, and no white blood cells. Somatosensory evoked potentials were unremarkable. CT scan of the abdomen and pelvis was normal. Urinalysis, complete blood count, electrolytes, BUN, creatine, B12, folate, calcium, magnesium, phosphorus and thyroid function tests were all normal. A comprehensive lower extremity electrodiagnostic evaluation was normal.

A neurosurgical consultation was promptly obtained. This case was also presented at the departmental neurosurgical conference. The consensus was that operative treatment was not urgently required. At that juncture, the etiology of the bladder dysfunction was unclear. Furthermore, the pathological insult was probably remote. Surgery may have resulted in increased back discomfort, without clear improvement in bladder dysfunction. In addition, the patient was hesitant to consider surgical intervention.

The initial urodynamic study demonstrated an atonic bladder, and a diminished bulbocavernosus reflex. Concentric needle examination of the anal sphincter demonstrated fibrillation potentials and 'reinnervation'

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Figure 1 The above MRI scan demonstrates the L4-5 disk lesion with sequestered fragments

potentials. These findings are consistent with lower motor neuron bladder dysfunction, with evidence of neuronal regrowth or regeneration. The patient was advised to start a self-catheterization program.

Three weeks after his initial presentation to the clinic, the MRI was repeated. The L4-5 disk lesion was significantly smaller. The patient had no further episodes of sphincteric dysfunction. He was able to empty his bladder with abdominal straining and pressure. He was not consistently following his self-catheterization program. His erectile dysfunction had somewhat improved.

Six months post injury, he was evaluated by a urologist specializing in sexual dysfunction. A repeat electrodiagnostic assessment was ordered. There were no abnormalities in the routine lower limb studies. However, the amplitude of the dorsal penile sensory nerve evoked response was diminished. Penile biothesiometric studies were normal.

This patient still has some residual intermittent low back pain with occasional leg paresthesiae. He has been treated with physical therapy, tricyclic agents and epidural steroids. He has returned to work at a modified level. Intermittent catheterizing has been discontinued. He is able to void with abdominal straining. A subsequent urodynamic study completed 7 months after the injury showed weak detrusor contraction. Renal ultrasound studies were normal.

Discussion

Several reports associate lumbo-sacral disk herniation with sphincteric dysfunction. Love and Emmett² described three patients who had isolated urinary dysfunction that was associated with lumbar disk lesions. However, two of the three patients had other structural pathology such as an epidural lipoma or spinal dysraphism. Sylvester³ described two patients with isolated urinary dysfunction related to a lumbo-sacral disk lesion. Both patients improved after discectomy. Neither of these patients had urodynamic studies. As such, it is not possible to conclude with certainty that these patients had lower motor neuron bladder dysfunction.

The bladder is supplied by sympathetic, parasympathetic and somatic nerve fibers. The sympathetic fibers originate in the lateral horns of the spinal cord at the level of T12–L2. These preganglionic fibers leave the spinal cord and project into the sympathetic ganglia which are located near the aortic bifurcation (ie the inferior mesenteric ganglion). From here, postganglionic fibers innervate the trigone of the bladder. The parasympathetic fibers originate in the sacral spinal cord. These fibers travel through the cauda equina and exit via the S2, 3 and 4 roots. These preganglionic fibers synapse with postganglionic fibers near the bladder wall. The external sphincter is innervated by somatic fibers that originate in the sacral spinal cord. These fibers also travel through the cauda equina and exit through the S2, 3, and 4 roots.

These fibers project through the pudendal nerve to reach the sphincter.⁴

The ability to initiate and maintain a penile erection, is a complex function that requires the interaction between the cortical, subcortical and sacral centers. The following is a summary of the relevant neuroanatomy.

Somatic afferent fibers from the genital region travel through the pudendal and perineal nerves into the sacral spinal cord. Efferent preganglionic parasympathetic fibers originate in the sacral spinal cord. These fibers travel through the cauda equina and exit via the S2–S4 nerve roots. These fibers project through the pelvic nerves and synapse in the prostatic plexus. Postganglionic parasympathetic fibers supply the corpora cavernosa. Activation of the parasympathetic pathways results in a penile erection. Emission, the ability to ejaculate, is primarily controlled by the sympathetic nervous system. Similar to the sympathetic innervation of the bladder, these fibers originate in the thoraco-lumbar spinal cord and travel into the T12–L2 sympathetic chain. These fascicles then travel through the splanchnic nerves into the hypogastric plexus. After synapsing in the inferior mesenteric ganglion, postganglionic fibers travel through the hypogastric nerves to supply the vas deferens, seminal vesicles, and ejaculatory ducts in the prostate.⁴

This patient's disk lesion may have compromised the parasympathetic or somatic fibers innervating the bladder and genital region. The sympathetic fibers exit the spinal column superior to the level of this patient's lesion.

It should be emphasized that many apparently healthy patients have abnormalities on lumbar MRI scans. As such, it is axiomatic that radiological findings must be interpreted in the appropriate clinical context.

Isolated bowel and bladder dysfunction can be caused by a number of medical conditions such as multiple sclerosis, stroke, spina bifida, retroperitoneal lesions and peripheral neuropathy. Generalized dysautonomias such as Shy-Drager syndrome can also result in urinary and fecal incontinence.^{1,4} Many of these

possible causes were excluded on the basis of history, physical exam and/or investigations. Multiple sclerosis is unlikely in this patient as he was a native of a tropical nation, and has no previous history of neurological complaints. Also, he had an unremarkable lumbar puncture and SSEPs. Although this patient had hypertension, stroke is unlikely as he had an essentially normal neurological exam. Furthermore, urodynamic studies were consistent with lower motor neuron bladder pathology. Structural spinal pathology (ie spinal dysraphism, tumors, etc) can be reasonably excluded by MRI. Retroperitoneal lesions are unlikely with a normal CT scan of the abdomen and pelvis. Peripheral neuropathy is equally unlikely as this patient had intact reflexes, normal sensation and normal metabolic indices. As such, this patient's bowel, bladder and sexual dysfunction can be reasonably attributed to a cauda equina lesion secondary to a L4-5 disk herniation.

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