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Voiding Dysfunction in Women with Lumbar Disc Prolapse

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Abstract: A significant proportion of women with lumbar disc prolapse experience voiding dysfunction. The most common finding is detrusor areflexia, frequently associated with impaired sensation. The pertinent neuropathophysiologic findings, clinical features and methods of evaluation and treatment are reviewed.

Keywords: Bladder; Cauda equina syndrome; Female; Intervertebral disc displacement; Urodynamics

Introduction

Voiding dysfunction is a well known complication of lumbar intervertebral disc disease. From 27% [1] to 92% [2] of patients with a prolapsed disc are noted to have such dysfunction, and in most cases detrusor areflexia is found. All physicians who care for women with voiding dysfunction must be able to recognize and treat patients with this condition. This paper reviews the pertinent neuropathophysiology, clinical features, evaluation and treatment of women with voiding dysfunction resulting from lumbar disc prolapse.

Neuropathophysiology

A review of the relevant neuroanatomy is necessary before discussing the neuropathology resulting from lumbar disc prolapse.

Innervation of the lower urinary tract is derived from both the autonomic and somatic nervous systems. The parasympathetic pelvic nerves, which emanate from the second to the fourth sacral segments of the spinal cord, provide the principle excitatory input to the bladder. The somatic nerves originate from the third and fourth sacral segments and provide innervation to the external sphincter and other pelvic floor musculature. Finally, the sympathetic pathways of the hypogastric nerves arise from the lower thoracic and upper lumbar segments, and provide inhibitory input to the bladder body as well as excitatory input to the urethra and bladder base. Afferent activity may travel from the bladder to the spinal cord along both sets of autonomic nerves. However, those that convey information from tension receptors and nociceptors in the bladder wall are the most important for initiating micturition travel via the parasympathetic nerves to the sacral segments of the cord. Sensory nerves from the vagina and clitoris also travel in the somatic nerves to the sacral cord (Table 1) [3].

In the adult, the sacral segments of the spinal cord are at the level of the first and second lumber vertebral bodies. This distal end of the spinal cord is commonly called the conus medullaris. The spinal cord segments are named for the vertebral body at which the nerve roots exit the spinal canal. Thus, although the first sacral segment of the spinal cord is located at L1, its nerve roots run in the subarachnoid space posterior to the vertebral bodies L2–L5 until they reach the first sacral vertebral body, at which point they exit the canal. Thus, all of the sacral nerves which originate at the L1 and L2 levels run posterior to the lumbar vertebral bodies until they reach their appropriate site of exit from the spinal canal. This group of nerves running at the distal end of the spinal cord is commonly referred to as the cauda equina.

The most frequent sites of lumbar disc prolapse are the L4/5 and L5/S1 intervertebral spaces [4–6]. Usually, prolapse is in a posterolateral direction not affecting the majority of the cauda equina. However, in 1%–15% of cases central disc prolapse occurs, and compression of

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Table 1. Innervation of the lower urinary tract

	Spinal cord segment	Function
Efferent		
Autonomic		
Parasympathetic		
(Perineal nerve)	S2–S4	Excitatory to bladder
Sympathetic		
(Hypogastric nerve)	T11–L2	Inhibitory to bladder Excitatory to urethra
Somatic		
(Pudendal nerve)	S3–S4	Control over external sphincter
Afferent		
Tension receptors in bladder wall		
Nociceptors	S2–S4	Micturition initiation
Sensory	S2–S3	Perineal sensation

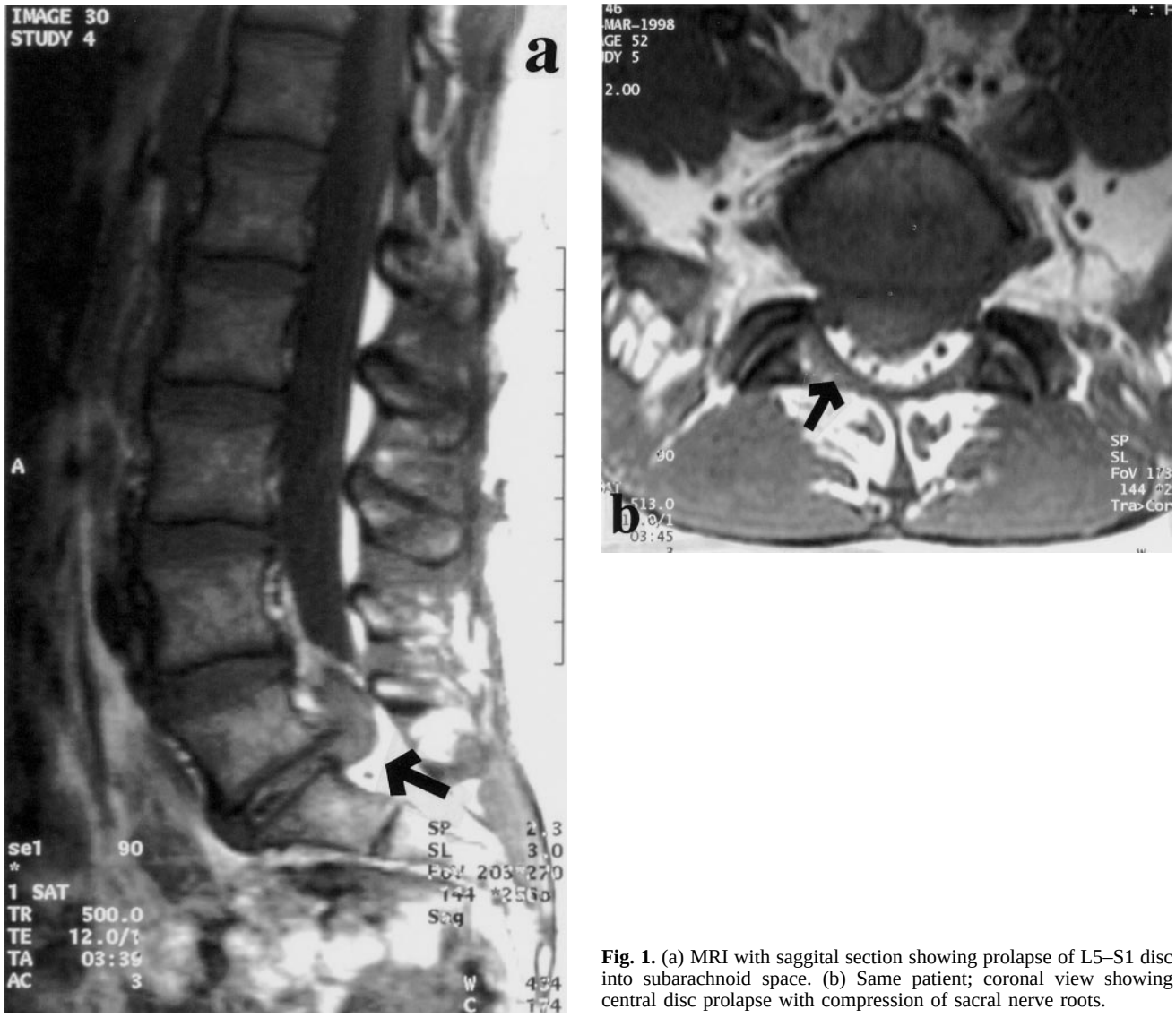

Fig. 1. (a) MRI with saggital section showing prolapse of L5–S1 disc into subarachnoid space. (b) Same patient; coronal view showing central disc prolapse with compression of sacral nerve roots.

the cauda equina may result [4] (Fig. 1). In fact, in some instances a large posterolateral disc prolapse may migrate medially and cause cauda equina compression as well [4]. Therefore, fusing the neuroanatomy with the known neural innervation of the lower urinary tract, one can see how prolapse anywhere along the lumbar spine

would interfere with the parasympathetic (S2–S4) and somatic (S3–S4) innervation, whereas prolapse at the upper lumbar spine only, which is relatively rare, would affect the sympathetic (T11–L2) innervation. As well as interference with parasympathetic and somatic innervation to the bladder and urethra, afferent stimuli from the bladder and perineal sensation, both served via sacral segments, would also face interference.

Thus, lumbar disc prolapse interferes with the stimuli from the bladder needed to initiate micturition, excitatory input to the bladder required for detrusor contraction, and somatic innervation to the external sphincter and pelvic floor musculature. The resultant urologic finding is one of detrusor areflexia, frequently with an impaired sensation of filling. This urologic manifestation of compression of the sacral nerves is usually but one aspect of the cauda equina syndrome (compression of the lumbosacral nerves). Classically, saddle anesthesia, bilateral sciatica, lower back pain and, in men, impotence are seen [7]. Although detrusor areflexia is the most common finding in these patients, detrusor hyperreflexia has also been reported [8]. It has been suggested that the mechanism of injury in this group of patients is progressive disc herniation causing irritation and excitation of the sacral nerve roots.

Besides direct compression, a prolapsing disc can affect the sacral nerves by interfering with blood flow to and from the cauda equina [9]. Experimental investigations have shown changes in the intraneural venous blood flow with compression of these veins, leading to congestion and ischemia of the nerve roots. Delmarter et al. [9], in an animal model, constricted the cauda equina to varying degrees and evaluated changes in bladder function. Fifty per cent constriction resulted in no significant cystometric changes but did cause venous congestion of the nerve roots and ganglia; 75% constriction caused detrusor areflexia, increased bladder capacity and overflow incontinence, as well as arterial narrowing and venous congestion of the nerve roots and ganglia.

Complete damage to the conus or sacral roots produces paralysis of the detrusor, such that it is acontractile and must be emptied using external pressure (straining or Credé) or a catheter. Although detrusor contractility is absent, there may still be poor compliance and a steady rise in detrusor pressure during the filling phase, which indicates that the injury to the cauda equina is incomplete. Fortunately, most of these lesions are partial and have a more profound effect on the autonomically innervated bladder than on the somatically innervated external sphincter. Therefore, if the conus medullaris is partially damaged, even without sacral root damage, activity in the pelvic floor and external sphincter remain intact despite the loss of detrusor contractility. The efficiency with which the bladder empties in the absence of detrusor contractility depends on sufficient external force and the absence of any outflow obstruction. Even if detrusor function returns, it is usually inadequate to result in efficient emptying of the bladder, and does not indicate detrusor–striated external sphincter dyssynergia, as it is a form of functional obstruction by the external sphincter because there is no detrusor contractility.

Clinical Features

Recent studies suggest that a minority of patients presenting with lumbar disc protrusion will have voiding dysfunction. Bartolin et al. [1] prospectively studied 114 patients (37 women, 77 men) who complained of low back pain and were found to have lumbar disc protrusion requiring surgical treatment. Of this group, 31 (27.2%) were found to have detrusor areflexia, whereas destrusor activity was normal in the remaining 83. Specifically, 3 of 8 with L3, 10 of 54 with L4 and 18 of 52 with L5 disc protrusion had detrusor areflexia. All of these 31 patients difficulty in voiding requiring straining. Clearly this is a select group, as many patients do not require surgery and probably have less severe disc prolapse. Those with less severe prolapse probably have a lower rate of voiding dysfunction as well. In contrast to this report, Rosomoff et al. [2] reported rates of voiding dysfunction as high as 92%, but their study had far less stringent diagnostic criteria for flexia.

As noted earlier, patients frequently present with a constellation of symptoms representative of the cauda equina syndrome. O'Flynn et al. [4] reported on 30 patients with lumbar disc prolapse and urinary dysfunction. In 23 a long history of lower back pain existed prior to the development of urinary symptoms. However, on occasion voiding dysfunction may be the only or the first symptom of disc prolapse. Sylvester et al. [8] reported on 2 women who presented with painless urinary retention, but no other neurological findings were noted. In both cases a MRI revealed lumbar central disc prolapse and, in retrospect, both patients noted the experience of minor lower back pain prior to presentation.

A prolapsed intervertebral disc may produce a cauda equina syndrome with obstructive voiding symptoms on a permanent or intermittent basis. Therefore, the clinical presentation depends on the extent of injury to the autonomic parasympathetic nervous input to the lower urinary tract. Clinically, the patient describes pain in the lower back, radiating in a girdle-like fashion along the lumbar dermatome involved; physical examination may reveal reflex and sensory changes consistent with nerve root compression [10]. Voiding symptoms, when present, are obstructive in nature [11] and include compromised urinary flow rate, interrupted stream due to abnormal straining to void, residual urine, and incontinence. The obstructive symptoms are secondary to the degree of detrusor denervation. The incontinence, however, may be due to overflow or lack of resistance at the level of the external sphincter due to pelvic floor denervation, which is more commonly seen in patients with recurrent or repetitive intervertebral disc problems and spinal stenosis [11].

The most characteristic findings on physical examination are sensory loss in the perineum or perianal area

(associated with the S2–4 dermatomes), sensory loss on the lateral foot (S1–2 dermatomes), or both [12]. A unilateral or mild sensory disturbance indicates a better prognosis, as prolonged sensory deficits imply that the bladder will not recover because its normal function requires an intact visceral reflex arc of the sacral roots [6,13]. The bulbocavernosus reflex (BCR) should be checked because it reflects pudendal (somatic) nerve function. Although this reflex is absent in all patients with complete lower motor neuron lesions of the sacral cord, care must be taken not to assume this is the case, as 19% of healthy female subjects do not have a detectable reflex on physical examination alone [14]. In one series, which included patients with injuries to the conus medullaris and cauda equina from various causes, the BCR reflex was absent or significantly diminished in 84% of the patients and perineal sensation and muscle stretch reflexes were comprised in 77% [15]. These findings correlated well with a diagnosis of pelvic floor denervation.

Evaluation

When other features of the cauda equina syndrome are present it is easy to suspect lumbar disc prolapse as the cause of voiding dysfunction. It is a more difficult diagnostic dilemma when other signs are absent. One's suspicion should be aroused when faced with a woman who is straining to void or having difficulty emptying her bladder. A thorough history and physical examination with a focused neurological evaluation is mandatory. Other disease states, such as diabetes mellitus, multiple sclerosis and vitamin B_{12} deficiency, or infectious neuropathies such as herpes simplex, should be noted. Careful questioning concerning lower back pain, gait disturbances, or any other symptoms that were present when the voiding dysfunction began, can be helpful. For example, the presence of flu symptoms and gait disturbance in the past may be related to an infectious neuropathy. Sensory examination of the perineal area and lateral foot (both associated with sacral segments) may give clues to a neurologic origin. The BCR and anal sphincter tone can also suggest a lesion involving sacral segments or nerve roots. Assessment of the patient's postvoid residual using either a straight catheter or an ultrasound bladder scan device can also give information on the efficiency of bladder emptying.

Further urodynamic testing is frequently required to document the voiding dysfunction. Urodynamically, detrusor motor dysfunction and weakened external sphincter activity are the primary findings, even in urologically asymptomatic patients [11]. A cystometrogram (CMG) with simultaneous pelvic floor electromyography (EMG) is sufficient for urodynamic investigation, and the predominant finding is usually detrusor areflexia associated with sphincter neuropathy [5,10,11,15]. As discussed previously, in cases in which the somatic innervation remains intact the external sphincter may appear to be incapable of relaxing when the patient strains to void, and actually constitutes an obstructive factor that may require pharmacologic manipulation or intermittent catheterization. More sophisticated urodynamic testing is occasionally needed. Confirmation that intravesical pressure elevation on the CMG is due to abdominal straining and not a strue detrusor contraction can be obtained by using subtracted pressure measurements (intravesical pressure minus intrarectal pressure equals true detrusor pressure), or by synchronous perineal floor and rectus abdominal EMG [16]. Sacral evoked potentials, specifically the bulbocavernosus reflex latency time, can be used to study the integrity of the sacral reflex arc further, in this set of patients the response is usually absent or prolonged [17]. Table 2 summarizes the neurological features of patients with cauda equina syndrome [12].

Table 2. Major neurological features of lumbar disc disorders

Symptoms	Obstructive voiding symptomatology
Signs	Perineal or perianal sensory loss
	Absent or diminished bulbocavernosus reflex
Tests	CMG – detrusor areflexia
	EMG – neuropathic changes

CMG, cystometrogram; EMG, electromyography

When lumbar disc disease is suspected, MRI of the lumbar spine should be performed. MRI defines the anatomical detail much more precisely than myelography (which was formerly the diagnostic test of choice). If lumbar disc prolapse is revealed, neurosurgical or orthopaedic consultation should be obtained.

Treatment

Treatment consists of correcting the underlying cause of the problem, usually requiring laminectomy. However, in some instances laminectomy may not improve the voiding symptoms. Shapiro [7] reviewed 14 patients who presented with cauda equina syndrome secondary to lumbar disc herniation, 13 of whom had incontinence. When evaluating variables to predict the resolution of incontinence, he noted that the time between onset of symptoms and laminectomy was important. All of 7 patients operated on within 48 hours regained continence, whereas only 2 of 6 operated on at lengths greater than 48 hours from presentation regained continence. Other studies, though, such as that by O'Flynn et al. [4], report a much poorer rate of detrusor recovery. They noted that only 1 of 26 patients with prelaminectomy incontinence regained completely normal bladder function. This disparity between the reports of Shapiro [7] and O'Flynn et al. [4] may be due to the use of different outcome measures: Shapiro used 'continence', whereas O'Flynn used 'completely normal bladder function'. Thus it is likely that although many patients became continent and can void by abdominal straining, few actually regain normal bladder function. The sequelae of laminectomy, such as spinal stenosis or

archnoiditis, can also affect bladder function. O'Flynn et al. [4] reported that 4 patients who had no urinary symptoms prior to surgery had voiding dysfunction postoperatively.

The challenge for the clinician is to attain symptomatic relief for the patient by allowing adequate bladder emptying without incontinence while preserving upper tract function. Patients with lower motor neuron lesions do not develop reflex vesical activity, and storage function may or may not be normal. If bladder compliance is adequate, a trial of cholinergic stimulation with bethanechol chloride alone (50 mg up to four times daily) or in combination with metaclopramide (5–10 mg up to four times daily) may be given, but should not be continued if no significant response is achieved by the time medication has been prescribed for a month [18].

Hypertonicity or decreased compliance with a concomitant fixed outlet resistance can impair ureteral function and place the upper tracts at risk. In these patients a regimen of anticholinergic medications, usually combined with self-catheterization, should be utilized. When this is unsuccessful bladder augmentation may be necessary to lower detrusor pressure and protect the upper urinary tracts. Emptying, again, is by self-catheterization. When incontinence results from poor sphincter function the options are similar to those for any woman with intrinsic sphincter dysfunction: sling procedures or collagen injections. A sling is created whenever the patient may require self-catheterization postoperatively, as repeated catheterization may displace the collagen which is injected and render the patient incontinent again.

Finally, it is important for physicians who treat women with incontinence to carefully note any history of lumbar disc disease. These women may actually have some element of detrusor areflexia in addition to ISD or urethral hypermobility, and may empty by pelvic floor relaxation and abdominal straining. Postoperatively they may develop difficulty in emptying or even retention, as their bladder cannot empty against the increased urethral resistance.

Conclusion

Voiding dysfunction may present in conjunction with other neurologic symptoms or as an isolated event in women with a prolapsed intervertebral disc. Damage to the parasympathetic, somatic and sensory nervous innervation of the lower urinary tract is the usual mechanism of injury. In most cases detrusor areflexia results. A careful history and examination, in conjunction with urodynamic testing and MRI, will establish the diagnosis. Although surgical intervention may not always give complete bladder recovery, it can frequently allow for improvement. Various bladder management techniques (of which self-catheterization is the mainstay) will help achieve good bladder emptying and preservation of the upper urinary tracts.

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