Disc Disease

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Disc disease encompasses a variety of vertebral disorders including disc degeneration and herniation. Up to 80% of people will experience back pain over their lifetime and this can commonly be associated with degeneration of the intervertebral disc [1]. Degenerative disc disease is the clinical syndrome described by manifestations thought to be related to disc degeneration, characterized broadly by fissures, degeneration, and herniation. Disc herniation is the displacement of disc material, such as nucleus pulposus, beyond the disc edge [2]. Disc herniation occurs most commonly during the third and fourth decades of life with the most common levels of disc herniation being L4–L5 and L5–S1 [3]. Although lumbar disc herniation is the most common, cervical and thoracic discs can also herniate. The level and degree of disc herniation will affect patient symptoms.

Voiding abnormalities are present in 27–68% of patients undergoing surgery for disc disease [4]. Direct compression of the spinal cord and lumbar and/or sacral nerve roots, in addition to local inflammatory effects, can result in lower urinary tract dysfunction (LUTD). Specifically, this can lead to functional bladder changes, including neurogenic detrusor overactivity (NDO) in the early stages, progressing to neurogenic bladder later [4]. The objective of this chapter is to review the relevant neuroanatomy, physiology, clinical presentation and diagnosis of disc disease and potential effects on the lower urinary tract. We will also highlight management options for LUTD secondary to disc disease.

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66.1 Spinal and Vertebral Anatomy

The vertebral column is composed of 7 cervical, 12 thoracic, 5 lumbar, and 5 sacral vertebrae and the coccyx. Between each two vertebrae is an intervertebral disc composed of an inner gelatinous nucleus pulposus surrounded by an outer fibrous annulus fibrosus. A disc is named based on the vertebra immediately above and below. For example, the disc between the fifth lumbar vertebrae and first sacral vertebral body is called "L5–S1" [2].

The spinal cord lies within the vertebral canal. At the level of the T12 vertebral body, the spinal cord becomes conical in structure and is termed the conus medullaris. The conus medullaris tapers to form the filum terminale. Distal to the filum terminale, at the level of the L2 vertebral body, are a collection of nerve roots, called the cauda equina.

There are three types of disc herniation based on the location of protrusion: intraforaminal, posterolateral, and central. Disc herniation usually occurs in a posterolateral orientation where the posterior longitudinal ligament is weakest [3]. Central or midline lesions can compress the anterior portion of the spinal cord. A central disc herniation of the lumbar spine may compress the cauda equina causing cauda equina syndrome. Disc herniation at L4–L5 will most often compress the fifth lumbar root and herniation of L5–S1 usually compromises the first sacral nerve root. However, variations in root anatomy as well as the direction (ex. posterolateral or central) of the herniation can affect these relationships [3].

66.2 Micturition Pathophysiology

Micturition is a coordinated event consisting of sphincter relaxation, detrusor contraction, and bladder neck opening, which is initiated by the pontine micturition center and coordinated through autonomic and somatic nervous systems. The parasympathetic pelvic nerves originate from the sacral spine and provide excitatory signals to the bladder. Sympathetic pathways along the hypogastric nerves arise

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from the thoracic and lumbar spine sending inhibitory signals to the bladder and excitatory input to the urethra and trigone. The somatic nerves to the bladder, specifically the pudendal nerve, arise from sacral segments of the spinal cord and control the external urethral sphincter and pelvic floor musculature. Afferent activity from the bladder and urethra travels to the spinal cord along the pudendal, pelvic, and hypogastric nerves.

When considering relevant neuroanatomy and bladder physiology, it becomes clear that disc disease may affect bladder function. Suprasacral lesions can result in NDO with detrusor sphincter dyssynergia (DSD). Disc disease above the lumbar spine may affect sympathetic innervation (T11–L2). Lumbar disc prolapse may specifically affect the sacral cord interfering with parasympathetic and somatic innervation leading to detrusor areflexia and sphincteric dysfunction [5]. Independent of the level of disc involvement, altered detrusor compliance and upper urinary tract damage may result. The resultant LUTD from disc disease can significantly affect patients' quality of life.

66.3 Patient Presentation and Lower Urinary Tract Evaluation

66.3.1 Clinical History

Clinically, the most common complaint of patients with disc disease is pain, which may radiate the length of the affected nerve root (e.g. sciatica). Other symptoms may include paraspinal muscle spasms and/or neurologic sequela such as numbness and weakness and urinary and/or bowel dysfunction. Symptoms will vary depending on the degree and level of spinal cord or spinal root involvement. The onset of symptoms may be acute or gradual and vary with intensity depending on activity. Additionally, patients should be questioned about the presence of voiding or bowel symptoms prior to the onset of their back symptoms. In addition to voiding dysfunction and fecal incontinence, patients with lumbar disc herniation may also experience sexual dysfunction, which should be considered in the overall care of the patients, but is beyond the scope of this chapter [6].

Although there are variations in the definition of cauda equina syndrome (CES) in the literature [7], clinical features of CES include low back pain, saddle anesthesia, motor weakness, and bladder and bowel dysfunction. CES due to lumbar disc herniation is relatively rare with an estimated incidence of 2–3% of all cases of herniated lumbar discs [8, 9]. CES can be classified as complete or incomplete based on the presence of urinary retention and perianal sensory loss in complete CES [10]. Patients with incomplete CES may have voiding symptoms including altered bladder sensation or urgency or may not have any voiding dysfunction. In a study of patients undergoing MRI for back pain, no single clinical feature could predict the presence of CES, highlighting the variable presentation of this syndrome [11].

The prevalence of lower urinary tract symptoms in patients undergoing surgery for disc disease is reported to be 27–68% [4]. Patients should be questioned about overactive bladder symptoms including urinary frequency, urgency with or without incontinence, and nocturia. Patients may have obstructive voiding symptoms or the feeling of incomplete emptying in instances of detrusor areflexia. Patients may complain of urinary incontinence and careful questioning may help to elucidate if this is a stress urinary incontinence due to sphincter dysfunction or an urge urinary incontinence from detrusor overactivity. In some instances, painless urinary retention may be the only presenting symptom of patients with central lumbar disc prolapse [12]. However, more commonly, patients initially experience back pain prior to the onset of voiding dysfunction, and the duration from onset of initial disc disease-related symptoms to voiding dysfunction is variable, ranging from 3 days to 5 years in one study [13].

66.3.2 Physical Examination

In addition to a thorough genitourinary examination, patients presenting with voiding dysfunction secondary to disc disease need a careful neurologic examination. This should include assessment of sensation to both light touch and pinprick. Sensory loss on the lateral foot can be isolated to S1–S2 and sensory loss of the perineum or perianal area involves S2–S3 nerve roots. Also, with sacral root compression, patients may have loss of reflexes. Specifically, if the first sacral root is compressed, patients may have difficulty rising up on their toes due to weakness of the gastrocnemius-soleus muscle complex. Depressed patellar tendon and Achilles tendon reflexes can be caused by compression of L3–L4 and L5–S1, respectively. The bulbocavernosus reflex (S2–S4) and anal sphincter should also be assessed. Additionally, atrophy of involved muscle groups may be noted on exam in instances of chronic nerve compression.

66.3.3 Patient Work-up

Following a thorough history and physical examination, the European Association of Urology Guidelines on neurogenic LUTD recommends a voiding diary, urinalysis, serum chemistry, uroflowmetry, and upper tract imaging in patients with neurogenic bladder [14]. Additionally, urodynamic evaluation is felt to be essential in the documentation of LUTD. If video capabilities are available, videourodynamics is preferred to fully document pathology of the lower and upper urinary tract [14].

A voiding diary, completed over 2–3 days, can help elucidate information on frequency of voids, volumes voided, and severity of incontinence. Urinalysis can identify patients with hematuria or urinary tract infection and blood chemistry will give baseline measure of renal function. On uroflowmetry, intermittent pattern in the flow curve may indicate abdominal voiding. In fact, Bartolin et al. demonstrated an intermittent flow pattern in the majority of patients with detrusor areflexia with lumbar disc protrusion who were able to void spontaneously [15].

Urodynamic findings of patients with lumbar disc disease requiring surgery include detrusor areflexia in up to 74% [4, 15]. In patients with neurogenic bladder due to disc disease, urodynamic findings can be correlated to the location of disc herniation. In cervical and thoracic disc disease, the majority of patients had detrusor hyperreflexia with dyssynergia. Patients with lumbar disc herniation and neurogenic bladder most commonly had detrusor areflexia [16]. Specifically, in a study of 122 patients with neurologic sequelae from lumbar disc protrusion, 26% of patients had detrusor areflexia. Seventy-three percent had normal detrusor activity, although a subset of these patients (14%) had high bladder capacity (>500 mL) suggesting altered bladder sensation. In patients with no complaints of lower urinary tract symptoms, all had normal urodynamic findings suggesting that urologic evaluation of patients with lumbar disc disease is not likely to reveal abnormalities in patients without voiding complaints [15].

The upper urinary tract should be evaluated in patients with neurogenic bladder and disc disease as up to 40% may have some degree of upper urinary tract damage [16]. Patients should be evaluated for the presence of vesicoureteral reflux and hydronephrosis, which can be factors predisposing to renal failure. The presence of detrusor sphincter dyssynergia may predict damages to the upper tracts, whose preservation is of upmost importance to prevent long-term renal damage and renal failure.

66.4 Patient Management

66.4.1 Treatment of Disc Disease and Effects on Lower Urinary Tract

In cases where lower urinary tract dysfunction is felt to be attributed to disc disease, it is important to treat the underlying pathology. Approximately 95% of patients with uncomplicated disc herniation will respond to conservative therapy with medications including anti-inflammatories, analgesics, and muscle relaxants and bedrest followed by physical therapy [3, 8]. Epidural steroid injections may also be trialed prior to surgical intervention when non-invasive options have failed [17]. Indications for surgical treatment of disc herniation (i.e. discectomy) include failure to clinically improve after an appropriate conservative trial or neurologic sequelae from nerve compression.

In the case of cauda equina syndrome (CES), characterized by saddle anesthesia, motor weakness, and urinary incontinence, urgent surgical decompression is recommended and can help improve the likelihood of bladder function recovery. In a meta-analysis of surgical outcomes of CES secondary to lumbar disc herniation [8], patients undergoing surgery within 48 h had significantly better resolution of their symptoms, including urinary and rectal incontinence, compared to those undergoing surgery 48 h or more after the onset of symptoms. Patients with a history of chronic lower back pain pre-operatively were 11 times more likely to continue having urinary incontinence after surgery and had 25 times the risk of having rectal dysfunction after surgery. Overall, of the 322 patients included in this meta-analysis, reported outcomes revealed post-operative urinary continence in 73% [8].

Additionally, the distinction between complete and incomplete CES is important for pre-operative planning and discussion with patients. In a study of 200 patients with CES, 63% of patients with incomplete CES had normal bladder function post-operatively compared to only 26% of patients with complete CES. Furthermore, patients with incomplete CES had a higher likelihood for normal bladder outcome depending on the timing of surgery (<24 h vs. >48 h) [10]. Similarly, in a smaller study of 25 patients with complete CES, normal post-operative bladder function was achieved in 36% and, again, was more likely in those patients operated on <48 h after onset of symptoms [9].

Despite appropriate surgical management in patients with disc herniation and LUTD, recovery of normal voiding function after surgery is variable. One hypothesis is that recovery of autonomic nerves affecting bladder function is slower than that of somatic nerves controlling external sphincter activity [13]. In a study of eight patients with acute urinary retention from central lumbar disc prolapse, pre-operative urodynamic assessment demonstrated an acontractile bladder with absent bladder sensation in all patients. Also, most patients showed absent or denervated motor potentials on electromyogram (EMG). Follow up urodynamic evaluation was performed (follow-up range: 1 month to 6 years) and all patients persisted with an acontractile bladder, however some patients did demonstrate improvement in EMG activity.

In addition to the pathology resulting from the disc disease causing bladder dysfunction, consideration must be given to the sequelae of spinal surgery and its potential effects on bladder function. In one series, 60% of patients undergoing discectomy or laminectomy experienced new or worsening urinary symptoms following surgery [18]. Although there are several reasons to explain this finding, such as unmasking of pre-existing urologic pathology, recur-

rence of cord or nerve root compression, worsening of the original disease process, or iatrogenic etiology, it is clear that patients undergoing lumbosacral spinal surgery are at risk for post-operative urinary dysfunction.

In summary, return of urinary continence following surgical decompression is reported to be between 36 and 73% depending on the degree of cord or nerve root involvement and the timing of decompression. EMG activity may improve despite those patients with persistent bladder acontractility. Pre-operative chronic back pain and time to surgical intervention are risk factors for continued urinary dysfunction after surgical decompression. The risk of developing new or worsening post-operative urinary dysfunction following spinal surgery has not been well studied and questions regarding the etiology are not yet answered.

66.4.2 Management of Urinary Tract Dysfunction Related to Disc Disease

For patients with persistent LUTD, the primary goals of bladder management include preservation of upper tract function, maintenance of continence, and restoration of lower urinary tract function while supporting a patient's quality of life. Treatment needs to be individualized based on patient's voiding dysfunction as it relates to their disc disease and other medical comorbidities [14].

Conservative treatment options such as behavioral modifications, pelvic floor muscle exercises, and biofeedback can be tried initially. Behavioral management strategies include timed voiding, controlled fluid intake, and lifestyle changes. Alternative voiding patterns, such as abdominal voiding and crede maneuver may be used, although these are not recommended. Yamanishi et al. showed that despite patients persisting with an acontractile bladder following surgery for disc herniation, all patients could nearly empty their bladder (PVR range: 0–117 mL) with abdominal straining in the absence of detrusor contraction [13]. However, bladder emptying through crede or abdominal straining can have negative effects in the presence of obstruction and should not be employed in patients with high intravesical pressures, ureteral reflux, urethral stricture, or pelvic organ prolapse [19]. Pelvic floor physical therapy may be helpful in selected patients to improve continence and biofeedback can be useful to reinforce voiding pattern modifications [20, 21]. Bladder rehabilitation techniques can aim to restore bladder function through the use of electrical stimulation (e.g. neuromuscular electrical stimulation, intravesical electrostimulation) however high quality evidence is lacking [14].

For patients with detrusor underactivity or areflexia and incomplete bladder emptying, initial management is clean intermittent catheterization. If intermittent catheterization is not possible and the patient is not a candidate for urinary diversion, indwelling catheterization with a suprapubic tube is an option, with patients reporting long-term high satisfaction [22].

To optimize outcomes of patients with voiding dysfunction, often a combination of behavioral management, antimuscarinic medications, and if needed, catheterization is initial therapy for neurogenic LUTD. Antimuscarinic drugs reduce symptoms of overactive bladder and improve bladder capacity and compliance. Specifically, oxybutynin, tropsium chloride, tolterodine, and darifenacin have shown improvement in the treatment of overactive bladder symptoms in neurogenic populations [23]. Titration of drug dosages may be needed for maximum efficacy in this patient population. Newer beta-3 agonist medications, such as mirabegron, have also been shown to have clinical efficacy in urinary frequency and urge urinary incontinence episodes [24, 25] and have shown clinical improvements specifically in patients with NDO from spinal cord injuries [26].

In patients with continued incontinence from NDO despite optimal medical therapy and catheterization, onabotulinumtoxin-A injection into the detrusor is an option and can provide sustained clinical benefits. In a meta-analysis of randomized controlled trials of patients with NDO, intradetrusor injection of 200–300 units of onabotulinumtoxin-A had a significant effect on decreasing urinary incontinence episodes and improving urodynamic parameters, specifically cystometric capacity and maximum detrusor pressure [27].

Sacral neuromodulation is a well-established treatment option for patients with medication refractory urge urinary incontinence and non-obstructive urinary retention. Although it is not approved by the Food and Drug Administration (FDA) for patients with neurogenic bladder, studies suggest that it is a safe and effective option in carefully selected patients with neurogenic bladder. A meta-analysis of studies evaluating sacral neuromodulation for patients with neurogenic LUTD, demonstrated the test phase success rate was 68% and the success rate of subsequent permanent implantation was 92% [28]. However, the definition of success was not specifically defined in this meta-analysis. In a recent, small study of patients with multiple sclerosis and neurogenic LUTD, 94% of patients had a successful test phase. At 3 years follow-up, patients had statistically significant improvements in mean voided volume and post void residual, urinary frequency, and number of incontinence episodes [29].

Posterior tibial nerve stimulation (PTNS), a minimally invasive neuromodulation technique, has shown benefit in management of neurogenic overactive bladder in patients with multiple sclerosis. Specifically, in a study of patients undergoing 12 weeks of PTNS therapy followed by 14 day intervals for 3 months, 21 day intervals for 3 months, and 28 day intervals for 3 months, there was improvements in daytime frequency, nocturia, and urge incontinence episodes over the 1 year period [30].

Another type of neuromodulation, sacral anterior root stimulation with rhizotomy, has been studied in patients with spinal cord injury. Anterior root stimulation is thought to activate parasympathetic pathways to induce detrusor contraction and somatic nerves controlling external urethral sphincter to improve bladder emptying. Rhizotomy can suppress detrusor overactivity and detrusor sphincter dyssynergia. In one cross-sectional study, anterior root stimulation and rhizotomy has been shown to improve quality of life, continence, and urinary tract infection rate [31].

When more conservative measures fail, bladder augmentation and/or urinary diversion may be considered in appropriate patients with small capacity bladder and elevated bladder pressures to restore continence and protect upper tracts.

Newer therapies continue to be developed for patients unresponsive to conventional options. Dorsal penile-clitoral nerve electrical stimulation has demonstrated efficacy in suppressing detrusor contractions in patients with NDO [32, 33]. Ongoing research using human embryonic stem cells injected into the lumbar spine of mouse models demonstrated improvements in bladder dysfunction related to spinal cord injury [34]. This could have future implications in management of patients with bladder dysfunction due to cord compression from disc disease.

66.4.3 Follow-up of Patients with Lower Urinary Tract Dysfunction Related to Disc Disease

Careful and consistent follow-up is required in patients with LUTD to ensure stability of the bladder and protection of upper tracts. Patients should be monitored for urinary tract infections and development of hydronephrosis or nephrolithiasis with upper tract imaging. Additionally, any signs of symptoms indicating a change in bladder function (e.g. worsening urinary incontinence, recurrent urinary tract infections, renal function deterioration) warrants further evaluation as appropriate. Specifically, the European Association of Urology recommends urinalysis and bladder-renal ultrasound every 6 months, annual physical examination with serum and urine testing, and videourodynamics every 1–2 years for patients with neurogenic lower urinary tract dysfunction [14].

66.5 Conclusions

The location and type of disc pathology will influence the extent of bladder dysfunction, but the complexity and variations of the sacral nerve root anatomy can lead to variable clinical presentations. The prevalence of voiding symptoms in patients undergoing surgery for disc disease is 26–74%. Physicians should have a high clinical suspicion of disc disease in patients presenting with back pain and voiding dysfunction. In patients with cauda equina syndrome, prompt diagnosis and surgical decompression are paramount to minimize neural damage and increase likelihood of complete recovery of voiding. Urodynamic evaluation with EMG should be considered for diagnosis of LUTD and to help guide bladder management. Given the high likelihood of persistent voiding dysfunction even after surgical treatment of disc disease, patients should be followed post-operatively. Patients with persistent neurogenic bladder need to be followed long-term given the risk of upper urinary tract damage. There are several management strategies available for neurogenic LUTD with the goal of preserving upper tract function and maintaining continence and quality of life.

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