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Morphofunctional Alterations of the Bladder Secondary to Central or Peripheral Neurological Lesions

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Abstract: ***'Neurogenic bladder' refers to any morphofunctional alterations of the bladder– sphincter complex secondary to central or peripheral neurological lesions. The control of micturition can be impaired by lumbar disc disease: the most frequently involved discs are in fact L4/L5 and L5/S1 and protrusions or herniations at these levels can compromise bladder innervation The term 'disc disease' includes different intervertebral disc structural modifications such as degeneration, protrusion and herniation. Discal degeneration is characterized by dehydration and shortening of the intervertebral disc. Discal protrusion is the distension of the disc beyond the vertebral body edge. It can be uniform and symmetrical or focal and asymmetrical, generally median or monolateral. The nucleus escape, usually asymmetrical, median or monolateral, is called herniation and it can be contained (by the posterior longitudinal ligament) or expelled. The discal damage causes several functional bladder alterations and urinary symptoms that include neurogenic overactivity the in early stage, with progressive underactivity and areflexia in the late stage.***

Keywords: neurogenic bladder, neurological lesions, morphofunctional alterations

1. Introduction

'Neurogenic bladder' refers to any morphofunctional alterations of the bladder– sphincter complex secondary to central or peripheral neurological lesions. The control of micturition can be impaired by lumbar disc disease: the most frequently involved discs are in fact L4/L5 and L5/S1 and protrusions or herniations at these levels can compromise bladder innervation The term 'disc disease' includes different intervertebral disc structural modifications such as degeneration, protrusion and herniation. Discal degeneration is characterized by dehydration and shortening of the intervertebral disc (1). Discal protrusion is the distension of the disc beyond the vertebral body edge. It can be uniform and symmetrical or focal and asymmetrical, generally median or monolateral. The nucleus escape, usually asymmetrical, median or monolateral, is called herniation and it can be contained (by the posterior longitudinal ligament) or expelled. The discal damage causes several functional bladder alterations and urinary symptoms that include neurogenic overactivity the in early stage, with progressive underactivity and areflexia in the late stage. The aim of this review is to underline that disc-disease related neurogenic bladder is a specific condition due to a particular etiology. This implies that disc-disease-related neurogenic bladder requires a different therapeutic approach that can therefore radically modify the management of patients. Furthermore, a timely diagnosis can significantly improve the outcome of patients in terms of quality of life (2). The medical literature on neurogenic bladder secondary to disc disease is reviewed in this article.

2. Material and Methods

The literature search was performed on PubMed, Medline and Google scholar, chosen because they are reliable and easy to read. 'Neurogenic bladder', 'disc herniation', 'disc prolapse', 'disc protrusion', 'cauda equina syndrome', 'treatment', 'surgery' and 'urodynamic' were used as keywords, either alone or in combination using 'AND' or 'OR', to focus the search on the topic without excluding relevant papers. The reference lists of the articles retrieved were examined to capture any other potentially relevant article. The search was restricted to articles published between 2000 and 2022 to acquire all the scientific knowledge about neurologic bladder, which grew fast in this period. Seventy-nine papers were found, but only 42 were reviewed and summarized. The excluded papers were judged neither pertinent nor very useful.

3. Results and Discussion

Micturition is a coordinated event comprising distal sphincter relaxation, detrusor contraction and trigone and bladder neck opening. It is triggered by the minctional reflex activation, integrated with the Pontine Micturition Center (PMC) located in the rostral pons. The distal end of the spinal cord becomes conical at the level of T12 and it is thus called 'conus medullaris' (3). This structure reaches the level of L2 body and ends with the 'cauda equina', that is made of the last nervous roots (including the ones from/to the bladder) and the Filum terminale. The spinal cord segments are named after the vertebral body at which the nerve roots leave the spinal canal. In the adult, all the sacral nerves originating 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. The somatic nerves to the bladder, originating from the III and IV sacral segments, reach the external sphincter and other pelvic floor musculature (4). The parasympathetic pelvic nerves originate from the second to the fourth sacral segments of the spinal cord. They conduct the main excitatory input to the bladder. The sympathetic pathways are provided by the hypogastric nerves arising from I, II and III lumbar

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segments. They deliver inhibitory input to the bladder body and excitatory input to the urethra and trigone. Sensitive stimuli from the bladder may travel to the spinal cord along both sympathetic and parasympathetic pelvic nerves. Information from tension receptors and nociceptors in the bladder wall, however, travel via the parasympathetic nerves to the sacral segments of the cord and have the most important role for initiating micturition. The neurogenic bladder is accompanied by anatomical modifications in peripheral nerves and bladder morphology. These modifications are related to the disc disease bulk and duration (5). Disc disease, at any level of the lumbar spine, could interfere with the parasympathetic and somatic innervation of the lower urinary tract, striated sphincter and other pelvic floor musculature. Neurological modifications include autonomic (parasympathetic and sympathetic) and somatic fiber damage. An early stage prolapse can induce an acute nerve root stretching, causing an irritative state that induces overstimulation of the detrusor with neurogenic overactivity. Chronic disc disease causes a prolonged injury to the nervous roots with a prominent compression of the nerves inducing trophic damages and nerve demyelination. The consequence is a progressive atrophy of autonomic and somatic fibers, first involving the sensory fibers. This alteration leads to decreased bladder sensation resulting in detrusor underactivity or areflexia (6). As well as direct compression, a disc protrusion or herniation can damage the sacral nerves by interfering with blood flow. Several studies have shown changes in the intraneural venous blood flow with veins compression, leading to congestion and ischemia of the nerve roots. In an animal model Delamarter et al. demonstrated that bladder dysfunction and detrusor areflexia appear to occur with blockage of axoplasmic flow and early sensory changes occur with neurovenous congestion. The nerve roots damage is connected with bladder muscular fiber modifications. Damphousse et al. analyzed 68 patients with neurogenic bladder secondary to cauda equina syndrome with overactive or acontractile detrusor (7). They found a significant relationship between bladder deformations and overactive detrusor, which turned out to be more significant than the relationship between bladder deformations and acontractile detrusor. The overactive detrusor being an infrequent condition related to an early disc protrusion, correlated bladder deformations could be represented by modest detrusor hypertrophy, similar but not identical to the typical pattern of bladder deformations during obstructive diseases. This hypothesis could be confirmed by ultrasonography in patients with overactive detrusor caused by disc disease. Chronic nervous damage, instead, inducing a progressive reduction of bladder sensitivity and as a result of motor fiber damage, leads to the atrophy of detrusor. These conditions allow a progressive dilatation of the bladder with a global and circumferential thinning of the muscular wall of the detrusor. General disc disease symptoms usually include leg and foot pain (sciatica), which may occur with or without lower back pain. The leg pain is typically worse than the lower back pain. In addition to classic sciatica symptoms, nerve impingement at the L4–L5 level can cause weakness in extending the big toe and potentially in the ankle (foot drop). Numbness and pain can be felt on top of the foot, and the pain may also radiate into the buttock (8). Nerve impingement at the L5–S1 level may cause loss of the ankle reflex and weakness in ankle push off (patients cannot do toe rises). Numbness and pain can radiate down to the sole or outside of the foot. Additional findings on physical examination are sensory loss in the perineum or perianal area (S2–S4 dermatomes) and altered bulbocavernosus reflex (pudendal nerve function index). The prevalence of urological symptoms in patients with disc disease candidates for surgical treatment goes from 20.00% to 67.74% (9). Detrusor areflexia correlates with obstructive voiding symptoms and stress incontinence due to overflow or lack of resistance at the level of the external sphincter. In the case of detrusor overactivity, patients usually complain of urgency and urge incontinence, and in some cases these symptoms are observed even when urodynamic testing is normal. Symptoms appear to be the same in both sexes, but stress incontinence due to perineal floor denervation is mostly observed in women. Female patients can also present variable degrees of cystocele, due to prolonged use of abdominal straining13. As reported by Abid et al.14, Susset et al. and Liu et al., voiding symptoms can support an early diagnosis of disc disease. Cases with no classical painful syndrome, where the only manifestations are LUTS, are reported in literature (10). Urodynamic evaluations of patients with lumbar intervertebral disc disease reveal a prevalence of neurogenic bladder from 26.23% to 74.19%. The most common finding is detrusor areflexia: no detrusor contraction during the voiding phase. In Bartolin's studies areflexia is the only urodynamic alteration observed (11). Some authors distinguish detrusor underactivity as a condition of reduced strength and/or duration of bladder contraction, but its prevalence seems to be lower than complete areflexia. Detrusor overactivity is also found in some studies with a prevalence between 3.75% and 21.25% Decreased bladder sensation seems to be the most probable cause of the onset of detrusor underactivity and areflexia (12). Bartolin reported decreased bladder sensation in all patients with detrusor areflexia. This group of patients voids by abdominal straining, has a bladder capacity of more than 500 mL and a residual volume from 25 to 145 mL11. Sandri et al. studied perineal floor muscle innervation by needle electromyography: a normal or incomplete denervation of perineal floor muscles was frequently associated with detrusor areflexia (82%), though both innervations traverse the same sacral roots. The primary aims for treatment of neurogenic bladder are: Preservation of the upper tract function, ensuring that detrusor pressure remains within safe limits during both the filling phase and the voiding phase; Improvement of urinary continence, to prevent urinary tract infection (13); Restoration of the lower urinary tract functions in patients with detrusor overactivity or detrusor–sphincter dyssynergia; Improvement of patient's quality of life. A flexible approach must be adopted in choosing therapy, taking into account the individual wishes of each patient and family and the practicality of each proposed solution for that particular patient. Noninvasive methods to improve the voiding process can be useful, among which are: third party bladder expression (Crede´), voiding by abdominal straining (Valsalva) (14) and behavioral modification techniques including prompted voiding, timed voiding (bladder training), and lifestyle modification. Pelvic floor muscle exercises, aimed to improve continence, may be helpful in selected patients, and Biofeedback can be used for supporting the voiding pattern modification. Biofeedback results have been demonstrated by reduced number of leaks

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and improvement of pad test. Antimuscarinic drugs are the most useful medications available for neurogenic detrusor overactivity, but there is no evidence of efficacy for underactive detrusor. In patients not responding to the conservative approach, spine surgery should be considered. There are several surgical options when approaching herniated discs, including various microsurgical procedures. The available surgical techniques are: minimally invasive microdiscectomy, minimally invasive percutaneous discectomy and open discectomy with or without foraminotomy (15). The best candidates for minimally invasive approaches are patients with herniation at only one level, occupying less than 50% of the vertebral canal, with no previous surgical treatment. In the presence of lumbar disc degeneration with lumbar pain – with or without irradiation to lower limbs – and X-ray evidence of reduced intervertebral space, prosthetic devices can be useful for preventing articular pain. If the etiology is typically biomechanical (showing for example pain during standing, increasing pain), nucleoplasty through non-thermal ablation (known as coblation, or controlled ablation) can reduce disc volume by 10–20%. Among surgical techniques chemonucleolysis is one of the most conservative, can be used for small herniations or protrusions, but isn't largely applied because of possible complications. The same indications are valid for oxygen–ozone therapy, which is more common. Several studies reporting on surgical and nonsurgical management of herniated discs agree on the pain control efficacy of spine surgery over medical management in the short term, but have some discrepancies when looking at long-term results (16). Controversial results about neurogenic bladder improvement after laminectomy were reported. Shapiro (17) observed continence regain after surgery, and Susset et al.15 reported micturition and cystometric findings normalization. Murayama observed a voiding function improvement in 64% of patient after operation, concluding that orthopedic surgery may improve neurogenic bladder in the majority of patients. Other studies, though, such as that by O'Flynn et al. and that by Bartolin et al., reported a much poorer rate of detrusor recovery, but these authors used different outcome measures: O'Flynn used 'completely normal bladder function' and Bartolin 'normal detrusor activity'. Benefits for neurogenic bladder obtained through disc disease latest surgical techniques are not yet discussed in the literature and should be studied in more detail (18).

4. Conclusions

When approaching neurogenic bladder, all the possible causes should be considered. Discal etiology can be suggested by clinical observation in patients also complaining of lower back pain/sciatica, but cannot be excluded even in the absence of musculoskeletal pain. Neurogenic bladder can indeed be the first discal disease manifestation. Approximately 40% of the patients with lumbar disc protrusion or herniation have abnormal urodynamic testing, and an even larger proportion complain of voiding symptoms. The most common urodynamic finding is detrusor areflexia, but an underactive or overactive detrusor can also be observed. Neurogenic bladder symptomatic treatment is well defined in the literature and the different approaches must be chosen taking into account the individual wishes of each patient and family and the practicality of each proposed solution for that particular patient. Noninvasive methods like third party bladder expression, voiding by abdominal straining, behavioral modification techniques, pelvic floor muscle exercises and biofeedback can be helpful. Antimuscarinic drugs are the most useful medications available for neurogenic detrusor overactivity and their long-term efficacy and safety is well documented. Intermittent catheterization is effective in patients with detrusor underactivity or acontractility and in patients with drug-controlled detrusor overactivity. When conservative treatment fails, nerve stimulation techniques can be proposed and their good outcome is reported even in a long term follow-up. Examples are sacral neuromodulation, dorsal penile or clitoral nerve stimulation, pudendal nerve stimulation and anterior root stimulation. Urological surgery such as bladder augmentation/bladder substitution and supravesical urinary diversion must be taken into account in patients unable to self-catheterize.

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