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Review

Management of Voiding Dysfunction Following Gynecological Surgery

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ABSTRACT

Some operations for gynecological problems, especially prolapse and stress incontinence surgeries, are obstructive and may result in voiding dysfunction. Extreme elevation of the bladder neck, which occurs with over-enthusiastic colposuspension or undue tension being applied to a sling, is the most likely cause of postoperative voiding difficulty. Apart from obstructive causes, other causes are pharmacological, inflammatory, endocrine, over-distension and psychogenic. The true incidence of voiding dysfunction due to obstruction after gynecological surgery is difficult to ascertain. Impaired voiding may be asymptomatic but the majority is symptomatic. The simplest investigations include uroflowmetry and ultrasonography for residual urine. Urinary tract infection should be excluded as it may lead to voiding difficulty. Prevention or early recognition of retention may avoid long-term voiding difficulty. Clean intermittent self-catheterization is the principal treatment for chronic urinary retention. Diazepam used as an anxiolytic may help with postoperative voiding problems. Novel tension-free suburethral sling procedures rarely lead to voiding dysfunction. However, undue tension being applied to the sling is found to be the main cause of postoperative voiding dysfunction, thus necessitating the need to perform a sling take-down procedure. *Key words:* gynecological surgery, voiding dysfunction, treatment

INTRODUCTION

Normal voiding is accomplished when the bladder contraction is initiated with the bladder neck and urethra synchronously relaxed. This event occurs when the 'micturition reflex' is activated. Subsequently, it results in a coordination of relaxation of the striated urethral sphincter, contraction of the detrusor, opening of the vesical neck and urethra, and onset of urine flow. The micturition reflex is integrated in the pontine micturition center, and the contraction of the detrusor is activated through the sacral micturition center independent of the cortical and pontine input [1,2].

Detrusor contraction is initiated through the mechanoreceptors that respond to tension exceeding its threshold. Depending on the relationship between the force of detrusor contraction and the residual urethral resistance, the intravesical pressure may rise to a variable extent. If voiding is not desired, the central nervous system inhibitory pathway will be engaged. This will cause a reflex inhibitory response to the bladder and interruption of the voiding reflex. Storage of urine continues to take place. If voiding is desired, the central nervous system facilitatory pathway will then be involved. Stimulatory messages are sent to the bladder for detrusor contraction while inhibitory messages are transmitted to the hypogastric and pudendal nerve for relaxation of the sphincter mechanism. When the falling urethral pressure equals the increasing intravesical pressure, urine flow will commence. Both the autonomic and somatic nervous systems play a crucial role in lower urinary tract function [1,2].

The act of micturition is governed by a number of contributory factors: control from higher centers, the sacral reflex arc, the innervations of the bladder muscle and sphincter mechanisms, the outflow resistance and the speed of contraction of the detrusor muscle fibers. Abnormalities of any component of this interactive mechanism may result in voiding dysfunction [1,2].

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CLASSIFICATION OF VOIDING DYSFUNCTION

The absence of clear definitions presents difficulty in attempting to classify and clarify voiding dysfunction. The standardization of terminology of lower urinary tract dysfunction, published by the International Continence Society, does not provide clear distinction [3]. In females, voiding dysfunction may occur when the detrusor muscle cannot maintain an effective contraction, the urethra fails to relax to lower the urethral resistance or if there is a failure in synchronizing these actions, all of which lead to detrusor sphincter dyssynergia. Therefore, these problems can be summarized as follows [4]:

1. Failure to store urine.
2. Failure to empty urine.
3. Failure to store and empty urine.

SPECIFIC CAUSES OF VOIDING DYSFUNCTION DUE TO GYNECOLOGICAL SURGERIES

Etiology and pathophysiology

Gynecological surgeries are surgical procedures involving the pelvic organs. These surgeries are usually carried out via the vaginal or abdominal route, or both. Some operations might be obstructive in nature, especially the surgical procedures designed for the correction of prolapse and stress urinary incontinence (SUI) [5,6]. A potential complication of iatrogenic post-surgical obstruction leading to voiding dysfunction can be anticipated. The extreme elevation of the bladder neck by over-enthusiastic colposuspension or undue tension being applied to a sling are the most likely causes of postoperative voiding difficulty [7]. Kinking of the urethra in cases of anterior vaginal prolapse (cystocele) or by direct compression of the prolapsed organ on to the urethra causes obstruction of the bladder outlet [8]. The true

incidence of voiding dysfunction due to obstruction after gynecological surgery is difficult to ascertain because there is a lack of standardization for the assessment. However, the incidence of voiding difficulty after anti-incontinence surgeries ranges between 2.4%-24% [5].

Radical hysterectomy (RAH) for carcinoma of the uterine cervix has an adverse effect on the lower urinary tract, generally described as occurring in over 50% of patients [9,10]. The adverse effect involves both urinary storing and voiding functions. Impairment of bladder sensation, alteration of bladder capacity and bladder compliance, compromise of detrusor function, reduction in maximal urethral pressure and maximal urethral closure pressure, and decrease in the pressure transmission ratio have been reported [9-11]. Postoperative bladder dysfunction after RAH varies with the extension of dissection during radical procedures and the interval of the follow-up period after surgery [9-11]. Neurological damage is considered to be the main cause of the high incidence of lower urinary tract dysfunctions [12,13]. Damaged motor parasympathetic neural innervation can possibly lead to impaired detrusor contraction, resulting in detrusor over-activity or detrusor under-activity [12,13]. In addition, a transection of the cardinal ligaments weakens the bladder neck support, and resection of the upper vagina and parametrium denervates the alpha-adrenergic sympathetic neuron, thus causing a decline in urethral pressure resulting in postoperative SUI [10,13].

In addition to iatrogenic causes for voiding dysfunction following gynecological surgeries, pharmacological, inflammatory, endocrine, bladder over-distension and psychogenic causes are non-surgical factors [14]. Diabetic cystopathy is a condition of impairment in detrusor contractibility. It may be asymptomatic in the early stage or present with urinary retention causing overflow incontinence in the advanced stage. Anti-cholinergic agents used to treat an overactive bladder may result in voiding difficulty, especially when mild impairment is already present preoperatively. However, epidural anesthesia is the most frequent pharmacological cause of voiding dysfunction [14].

Unmanaged surgical pain interrupts the flow of micturition. Painful stimuli are produced when urine is in contact with the inflamed mucosa of a urethral, vaginal or vulvovaginal lesion. Other possible causes of postoperative tenderness include inflammation, infection and edema [14].

Psychogenic causes of urinary retention may correlate with the onset of psychological disturbance, or be a response to psychotherapy or psychopharmacological agents. Psychogenic diagnosis is made only after exclusion of neurological and other significant organic disease. These diagnoses include hysteria and depression [14].

Bladder over-distension usually occurs after failure to detect urinary distension following pelvic surgery or epidural anesthesia. Bladder distension beyond normal bladder capacity may lead to ischemic damage of the detrusor muscle. Continued ischemia will lead to irreversible bladder damage [14,15].

Impaired voiding may be asymptomatic in some patients but the majority present with frequent voiding, poor flow, intermittent stream, incomplete emptying, straining to void and/or hesitancy. Others may present with overflow incontinence and frequency or urinary tract infection due to stasis. Acute retention may present with pain. The simplest investigations include uroflowmetry and ultrasonography for residual urine but cystometry and other investigations, such as radiology, cystourethroscopy and electromyography, may be required to make a more accurate diagnosis.

TREATMENTS

Non-surgical

Urinary tract infection should be excluded as it may lead to voiding difficulty. Prevention or early recognition of urinary retention may avoid long-term voiding difficulty. A pre-emptive need to drain the bladder should be considered for patients after RAH, extensive pelvic reconstructive surgery and epidural anesthesia when their resumption of spontaneous voiding is prolonged or difficult [11,12,14]. If bladder drainage is intended for a short period of time, such as after epidural anesthesia, a urethral catheter is advised. Where a longer-term problem is anticipated, such as surgery after colposuspension, placement of a suprapubic catheter is suggested. This procedure provides a better assessment of voiding and residual volumes as well as minimizing the possibility of urinary tract infection [16].

In patients with evidence of voiding difficulty when spontaneous voiding has not resumed or the post-void residual volume has not recovered within the expected period, it is reasonable to offer counseling and education for clean intermittent self-catheterization (CISC) [17]. Intermittent self-catheterization is now the principal treatment for chronic urinary retention. It allows patients to lead independent lives with efficient bladder emptying and low rates of urinary tract infection. Intermittent self-catheterization can be performed sterilely or cleanly. The former is reserved for patients in a hospital environment to prevent cross infection. Patients are usually encouraged to perform CISC themselves every day. The frequency of catheterization varies, with the aim to avoid incontinence and filling beyond normal bladder capacity.

A cholinergic agent, such as bethanechol chloride, has been advocated for urinary retention due to detrusor underactivity. However, there is no real evidence to show its clinical benefit. An alpha-adrenergic blocking agent has no proven benefit in women. Nevertheless, an anxiolytic agent, such as diazepam, may help in postoperative voiding problems [18].

Surgical

Treatments for iatrogenic persistent bladder neck obstruction following gynecological surgeries are many. The role of urethral dilatation for postoperative obstruction is not known. However, using Hegar's dilator to push the proximal urethra downwards is the frequently adopted method for urinary retention after a tension-free vaginal tape procedure. Wang and Lo reported a 17% incidence of immediate postoperative voiding difficulty that was easily corrected by insertion of a Hegar's dilator into the urethra with downward traction [19]. All patients subsequently resumed normal voiding. Lo et al objectively examined the tape using ultrasound and found that tape under undue tension could be taken down by pushing downward on the Hegar's dilator [20].

Surgery by urethrolysis or sling incision (take-down) is the most definitive treatment for iatrogenic persistent obstruction. Various approaches have been described for the 'take-down' of the previous repair utilizing retropubic, infrapubic and vaginal routes [21]. The approach route chosen for urethrolysis depends on the type of incontinence procedure performed, patient presentation and surgeon preference. A transvaginal procedure is usually performed for urine retention after a sling-type procedure. Retropubic urethrolysis is reserved for a previously failed transvaginal procedure or retropubic Burch colposuspension. The effect after various approaches appears to have similar success ranging from 65% to 93% [21]. Following a

vaginal or retropubic urethrolysis or take-down of a synthetic suburethral sling, obstructive symptoms are likely to improve, irritative symptoms may remain unchanged and almost half will develop recurrence of SUI. In order to prevent recurrence of SUI, the transvaginal lateral excision of the tape is described. The suburethral tape was simply transected 1-2 cm lateral to the urethra and left in place forming a J-shaped sling in situ. Recurrent SUI is less likely to occur if the transvaginal lateral excision is performed more than 14 days after the initial tension-free vaginal tape operation [22]. Furthermore, Klutke et al reported that over-tensioned mid-urethral mesh can be released by pulling the sling down via a small vaginal incision to expose the suburethral sling [23]. All of the patients required tape relief and all could void to completion within 24 hours of sling release. Of 17 patients who underwent sling release, 16 remained dry subjectively.

Transurethral endoscopic bladder neck incision is the treatment option for patients with high post-void residual urine due to impaired detrusor contraction. Wang reported that 24 patients who had developed persistent high post-void residual urine following RAH underwent a transurethral bladder neck incision [24]. A coagulating electrode was employed to incise the bladder neck at 3-, 9- and 12-o'clock positions. The postoperative bladder neck resistance, post-void residual and total bladder capacity were decreased compared with the preoperative condition. The overall improvement rate was 71%. The incidence of post-incision induced urinary incontinence was rare (4%). Moloney and Fenster performed a single incision on the bladder neck of patients with persistent bladder neck obstruction observed after repeated anti-incontinence surgeries. Similar good results were obtained [25].

The clinical use of botulinum toxin for treatment of detrusor external sphincter dyssynergia, dysfunctional voiding, detrusor overactivity and chronic pain syndrome has been investigated [26]. Usage of botulinum toxin for different causes of bladder outlet obstructions and to decrease outlet resistance in patients with an acontractile detrusor has been reported [27]. Phelan et al used a cystoscopic injection needle for delivery of botulinum A toxin into the external sphincter at the 3-, 6-, 9- and 12- o'clock positions in patients with voiding dysfunction due to neurogenic detrusor sphincter dyssynergia, pelvic floor spasticity, acontractile detrusor and multiple sclerosis [27]. Postoperative post-void residual decreased by 71% and voiding pressures decreased on average by 38%. Of the 21 patients, 14 (67%) reported significant subjective improvement in voiding. Since follow-up of botulinum toxin studies have been relatively brief and the number of patients small, further studies with longer follow-up and more patients are necessary.

CONCLUSIONS

Voiding dysfunction after gynecological surgeries is hard to predict preoperatively. However, prompt treatment of acute retention and pre-emptive management to avoid over-distension are essential. CISC remains the most effective therapy for chronic retention, although newer modalities are being investigated. Relief of sling tension is the most effective method for iatrogenic obstruction after sling surgeries.

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