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Review Paper

Etiology, Risk Factors and Pathophysiology of Stress Urinary Incontinence: A Review

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Abstract

Urinary incontinence (UI) is defined as any involuntary loss of urine. It is under-reported, undiagnosed, and often untreated medical situation for women at any age which highly impacts the quality of life. The risk factors for stress urinary incontinence in women are weak collagen, diabetes mellitus, advanced age, chronic obstructive airway disease, ethnicity, constipation, pregnancy and childbirth, obesity, advanced pelvic organ prolapse, hysterectomy, neurological disease, smoking, spinal cord trauma, and pelvic floor injury. The physiopathology is linked to endopelvic fascia, levator ani muscles, and muscular urethra compromise followed by pudendal nerve denervation and urethral complex's loss of ligamentous support. The line of management for stress urinary incontinence is lifestyle change, physiotherapy of the pelvic floor, bulking agent and midurethral sling.

Keywords: Urinary incontinence, Stress urinary incontinence, Urge urinary incontinence, Risk factors, Quality of life.

Introduction

Urinary incontinence (UI) is defined by the International Continence Society Standardization Committee as "a condition in which involuntary loss of urine is a social or hygienic problem and is objectively demonstrable[1]". In addition to the urethra, urine can also leak from an extraurethral source such as fistulas or congenital malformations of the lower urinary tract[2]. According to International Continence Society guidelines, urinary incontinence is "a symptom, sign of vesical dysfunction and/or urethral sphincter mechanism dysfunction,[3] as well as a condition"[4]. UI can considerably impair the quality of life, leading to disturbed social relationships, psychological distress from awkwardness and disappointment, hospitalizations due to skin break down and urinary tract infection and nursing home admission. An incontinent elderly woman is 2.5 times more likely to be admitted to a nursing home than a continent one[5].

Types of incontinence: Stress, urge, mixed, functional, overflow, and reflex incontinence are six different types of UI. The three most common types are stress (SUI), urge (UUI), and mixed (MUI) in which under the age of 60. SUI is the commonest and complaint by 50% of women[6].

The other classification proposed by Clinical Practice Guideline (Agency for Health Care Policy and Research) defines four types of UI: "stress, urge, mixed and overflow. Some authors include functional incontinence as a fifth type of incontinence"[7].

Stress Urinary Incontinence (SUI): In SUI, a patient may complain of involuntary urine leakage on exertion or with sneezing or coughing[2, 4]. Objectively, SUI is confirmed during urodynamic evaluation if involuntary leakage of urine is seen with increased abdominal pressure and absence of detrusor contraction. If the symptom or sign of stress urinary incontinence is confirmed with objective testing, the term urodynamic stress incontinence (USI), formerly known as genuine stress incontinence is used.

Urge Urinary Incontinence (UUI): In UUI, women have difficulty in postponing urination urges and must quickly empty their bladder without delay. If UUI is objectively demonstrated by cystometric evaluation, the condition is known as Detrusor Overactivity (DO), formerly known as Detrusor Instability (DI).

Mixed Urinary Incontinence (MUI): When both stress and urge components are present, it is called MUI[2].

Occult Stress Incontinence: Stress incontinence observed only after reduction of pelvic prolapse is known as latent stress incontinence or stress incontinence on prolapse. Few authors assume that prolapse itself causes kinking of the urethra and provides for at least part of the continence mechanism. Hence, stress incontinence improves and finally resolved as their prolapse worsened[7].

Epidemiology: Most epidemiologic studies in western societies indicate a prevalence of 25 to 55 percent. This wide range is

described due to broad variety of investigative methodology, population characteristics and definitions of incontinence. Furthermore, existing available data are further restricted by the fact that most women do not come forward for medical treatment for this condition. It is anticipated that only one in four women will seek medical advice for incontinence due to mortification, limited access to healthcare, or poor screening by health care providers. Among ambulatory women with urinary incontinence, the most common condition is SUI, which represents 29 to 75 percent of cases. DO accounts for up to 33 percent of incontinence cases, whereas the remainder is attributing to mixed forms[8].

Prevalence: Female UI is a universal condition with a prevalence that can range between 12.8 and 46% of women[9]. The prevalence of UI in the United States ranges between 3% and 14% with estimates ranging up to 40% for the elderly[10]. UI affects 4% and 28% of men and women respectively of middle-age and younger people, while it affects 17% of men and 35% of women of older age. The incidence of UI is estimated to be 2.79 per 1000 person/years[11]. Though estimates for prevalence varies, yet over one third of adult women suffer from urinary incontinence (UI)[6,12]. A current survey of over 24,000 community-dwelling women in the United States found that 37% of respondents reported UI symptoms during the preceding month and 86% of these were bothered by UI[13]. For UI, the prevalence in the general female population is about 25%[14]. Overall, 50% of the women with UI had SUI, 36% had MUI and 11% had UUI[11].

SUI is a significant clinical problem suffering from any incontinence symptoms at any given time with estimates of between 12% and 44% of women[15]. In general, roughly 50% of all incontinent women are affected by SUI, and this is the major type of incontinence in young and middle-aged women. The incidence of SUI is strongly related to increasing age; this is confirmed when only young and middle-aged women were considered[9].

Cost: The factors such as treatment and diagnosis, absorbent products, lost wages, living or nursing home care, cleaning expenses, direct and indirect loss of work and related medical conditions (urinary tract infections, falls and fractures, depression, skin infections) and vulvovaginitis[11] increases the costs of incontinence, urgency and frequency. Almost 75% women reported using pads, while more than 50% reported additional laundry loads. Eighteen percent needed additional dry cleaning on a weekly basis[16].

Ethnicity and Race: Regardless of ethnic background, UI affects many aging women and frequently results in a diminished overall quality of life (QOL) [17]. However, another study reported that prevalence of SUI differ because of racial and ethnic variation. One large population study of middle-aged women after adjusting for various co-morbidities found that non-Hispanic whites had 60% higher incidence for severe incontinence than compared to both non-Hispanic blacks and Hispanics because of anatomic differences. In a latest analysis of the SWAN (Study of Women's Health Across the Nation) study found that black women without leiomyomata had a decreased risk of UI compared with white women and Hispanic and Japanese women also had a lower risk than white women, while non-Hispanic black women with uterine leiomyomata had a 1.81-fold higher risk of urinary incontinence than white women[18]. Traditionally, Caucasian women were believed to have higher rates of UI than women of other races. In contrast, urge incontinence is believed to be more prevalent among African-American women[2]. In one urodynamic study, leiomyomata, more common in black women, may predispose some to SUI and Caucasian women had lower urethral closure pressures than black women[18]. Though these reports were not population based, and thus were not the best estimate of true racial differences. In addition, existing data on racial differences were largely based on small sample sizes. Of noted trends, it is not yet clear whether these differences were biologic, related to health care access, or affected by cultural expectations and symptom tolerance thresholds[2].

Etiology: SUI obviously requires some degree of weakness of both the proximal and distal urethral sphincter mechanisms. Whilst no single etiological factor exists in all women with SUI, there are a series of predisposing or risk factors such as obesity, prolapse, collagen disorder, menopause and pregnancy[19].

Common causes of SUI include surgical procedures that damage nerves leading to pelvic floor muscles, menopause, pregnancy, and vaginal delivery. The anal and urethral sphincters are innervated by the pudendal nerve that travels along the pelvic sidewall, where it is predominantly susceptible to injury from the compressive forces of labor and delivery[6].

Risk Factors: Risk factors for UI in women are age, obesity, chronically increased abdominal pressure[1,2], chronic cough[2,6], constipation, occupational risk[2], urinary symptoms, childbirth, pregnancy, hysterectomy, menopause, functional impairment, cognitive impairment[1,2], diuretic therapy[1] and smoking[2].

The risk factors for SUI in women are weak collagen, diabetes mellitus[6], advanced age[6,20,11,21], chronic obstructive airway disease[11], ethnicity[6], constipation[6,11], pregnancy and childbirth[11,20,21], obesity[11,20,21], (body mass index>30)[6], advanced pelvic organ prolapse[11], hysterectomy[6], neurological disease, smoking, spinal cord trauma, and pelvic floor injury[6].

Age: Age is the single largest risk factor for UI[1]. Higher prevalence estimates were associated with advanced age[11,22,23]. The prevalence of incontinence appears to increase steadily during young adult life. A wide peak is noted at middle age and then gradually increases after age 65. Few studies suggest a higher prevalence of SUI in women younger than 60 years and UUI in older women, showing that type of incontinence may vary with age[24]. The causes of these age-related trends were not

clearly understood and only some studies authenticate this finding[25]. Thom reported that between 17% and 55% of older women having experienced urinary incontinence at some point, compared with 12–42% of younger women[4]. The incidence of SUI is strongly related to increasing age especially it was confirmed in only young and middle-aged women. Peyrat et al., found that women more than 40 years of age have a risk ratio of 2.18 affected by SUI in comparison with women less than 40 years of age[9].

Sex: UI is a highly prevalent condition, especially in women. The overall estimated prevalence of UI in women is 30%, while in men aged 15-65 years is 1.5-5%[26]. It seems that UI is at least twice as prevalent in women as men, though there seems to be amore steady increase in prevalence with increasing age in men than in women[11]. SUI is the most common type of incontinence in the female population[1, 11]. In the United States, survey-based study showed that 37% of respondents experienced symptoms of incontinence in the past month and of these women, 86% were bothered by the incontinence episodes[27].

Childbirth and pregnancy: Certainly childbirth is the major factor associated with the occurrence of SUI in young women[9] and has been broadly accepted for the last several decades[6]. High parity, birth weight greater than 4000 g, episiotomy, operative vaginal deliveries (forceps and vacuum extraction), and second stage of labor lasting longer than 60 minutes significantly increase the risk of SUI after first delivery[6, 9]. Viktrup and Lose in their study of 242 primiparae showed that at five years (26.3%) and at 12 years (25.3%) after delivery, symptoms of SUI were the most common urinary disorders. Further, SUI had a lower remission rate than overactive bladder and mixed symptoms. In the literature, the prevalence of SUI related to delivery was reported to range from 3 to 67%[9] . First delivery has a higher association with long-lasting SUI over first pregnancy, and compared to nulliparous women women with two live births have as much as 67% increased odds of UI [6].

Causes of pelvic floor damage dysfunctions are multifactorial, and common that includes anatomical changes, sexual dysfunction, urinary and anal incontinence and perineal pain[2, 6,19, 28]. Many studies disclose a higher prevalence of UI in parous women compared with nulliparous women. Particularly, a higher rate of prolonged pudendal nerve motor latency after delivery has been established in women with incontinence compared with that of asymptomatic women[2]. Reports indicate that poorly supervised childbirth irrespective of parity is the strongest predictor of uterovaginal prolapse[29].

Menopause: SUI is highly prevalent among postmenopausal women and occurrence increases progressively in old age[16]. Studies have unpredictably confirmed an increase in urinary dysfunction after a woman enters her postmenopausal years. Separating hypoestrogenism effects from the effects of aging is difficult in women with symptoms. High-affinity estrogen receptors have been identified in the bladder trigone, pubococcygeal muscle and urethra, however are infrequently found elsewhere in the bladder. It is hypothesized that hypoestrogenic-related collagen changes and decrease in urethral vascularity and volume of skeletal muscle together may contribute to impaired urethral function through a decreased resting urethral pressure. Furthermore, urogenital atrophy resulting from estrogen deficiency is thought responsible in part for urinary sensory symptoms following menopause. In spite of this existing evidence that estrogen plays a role in normal urinary function, it is less clear whether estrogen therapy is useful in the treatment or prevention of incontinence (Estrogen Replacement) [2].

Hysterectomy: Studies have unpredictably shown that hysterectomy is a risk factor for developing UI. However, those studies that showed an association were retrospective, lack appropriate control groups, and were frequently based exclusively on subjective data. Contradictory, studies that include pre- and postoperative urodynamic testing showed clinically insignificant changes in bladder function. Furthermore, evidence does not support avoidance of clinically indicated hysterectomy or the performance of supracervical hysterectomy as measures to prevent UI[2]. Sexual dysfunction is a common condition in women with pelvic floor disorders, especially with SUI[22].

Obesity: A number of epidemiologic studies have revealed that an increased body mass index (BMI) is a significant and independent risk factor for UI of all types. Evidence suggests that the prevalence of UUI and SUI increases proportionately with BMI[24]. Hypothetically, the increase in intra-abdominal pressure that corresponds with an increased BMI results in a proportionally higher intravesical pressure. This higher pressure overcomes urethral closing pressure and leads to incontinence[30]. Deitel et al., reported a significant decline in the prevalence of SUI from 61 to 11 percent, in 138 morbidly obese women following weight loss after bariatric surgery[31]. Consequently, an increase in the prevalence of UI in the United States has been expected as a greater proportion of population is becoming overweight and obese[32].

Smoking and Chronic Lung Disease: Two large epidemiologic studies have confirmed a significant increase in the risk for UI in women older than 60 years with chronic obstructive pulmonary disease[2]. The pressure from frequent and forceful chronic coughing eventually causes damage to the vaginal supports, urethral sphincter, and perineal nerves, thus worsening symptoms of SUI[6]. Similarly, in several studies cigarette smoking is recognized as an independent risk factor for urinary incontinence[2]. In one of these studies, both current and former smokers were noted to have a two- to threefold risk of incontinence compared with nonsmokers[2,6]. Hypothetically, persistently increased intra-abdominal pressures are generated from a smoker's chronic cough, and collagen synthesis is lessened by the anti-estrogenic effect of smoking[2]. Smoking is associated with decrease in collagen synthesis and may perhaps

weaken pelvic support structures. Smoking-related diseases (such as chronic obstructive pulmonary disease or asthma) may have direct or indirect effects on bladder and urethral function[6].

Sporting Activity and SUI: A higher prevalence of SUI has been reported in women athletes of childbearing age and the vast majority considers only elite athletes. Nygaard found that 28% complained of urinary leakage during their sporting activity, the higher level of catecholamines during competitive activities could stimulate urethral α-receptors leading to UI. Moreover, because of sudden intra-abdominal pressure increases in these subjects, it has been reported that pelvic floor muscles need to be much stronger in elite athletes than in controls to prevent urinary leakage[9].

Collagen Disorder: There is evidence that there are different types of collagen in varying proportions in the pubourethral ligaments of women who become incontinent compared with the pubourethral ligaments of those who do not[19].

Depression: The prevalence of depression among women with UI varies in different studies figures between 6% and 38%. In terms of types of incontinence, 3–14% of women with SUI report depression, compared with 21–42% of those with urgency or mixed incontinence[14].

Pathophysiology: The continence mechanism involves a highly organized series of nerves, muscles, and connective tissue that dynamically influence bladder control. This arrangement permits timely and complete bladder emptying and also maintains continence during remarkable increases in abdominal pressure[33]. Both continence and micturition depend upon a lower urinary tract, consisting of the bladder and urethra, which is structurally and functionally normal[34].

Mechanism of continence: To understand the pathophysiology of incontinence the mechanism of continence must be understood.

Bladder emptying

Innervations related to voiding: As an appropriate time for bladder emptying arises parasympathetic stimulation is triggered, whereas sympathetic stimulation is reduced. Detrusor muscle contraction occurs as soon as neural impulses are carried in the pelvic nerves stimulated by acetylcholine release. Simultaneous to detrusor stimulation, acetylcholine stimulates receptors in the urethra and leads to outlet relaxation for voiding. Acetylcholine receptors within the parasympathetic system are broadly defined as muscarinic and nicotinic. The bladder is densely supplied with muscarinic receptors. Of the muscarinic receptors, five glycoproteins designated M1 through M5, have been identified. The M2 and M3 receptor subtypes have been identified as chiefly responsible for detrusor smooth muscle contraction. Thus, treatment with muscarinic antagonist medications blunts detrusor contraction to improve continence. Specially, continence drugs that target only the M3 receptor maximize drug efficacy however minimize activation of other muscarinic receptors and drug side effects[2].

Muscular Activity with Voiding: Smooth muscle cells within the detrusor fuse with one another so that low-resistance electrical pathways exist from one muscle cell to the next. Consequently, action potentials can spread rapidly throughout the detrusor muscle to cause rapid contraction of the entire bladder. Additionally, the plexiform arrangement of bladder detrusor fibers permits multidirectional contraction and is ideally suitable for quick concentric contraction during bladder emptying. During voiding, all components of the striated urogenital sphincter relax. Importantly, for effective voiding, bladder contraction and sphincter relaxation must be synchronized. Infrequently, tonic contraction of the detrusor may be dyssynchronous with urethral relaxation. With detrusor sphincter dyssynergia, the urethra fails to relax during detrusor contraction and retention follows. Seldom, women with this situation may be treated with pharmacologic agents such as muscle relaxants. These drugs supposedly relax the urethral sphincter and levator ani muscles to improve coordinated voiding[2].

Continence Theories: Many theories on continence are put forth and include concepts relating to pressure transmission, anatomic support, and urethral integrity. At present, these serve as the keystone for current urogynecologic treatment. Accurately dissecting the mechanism behind incontinence is intricate; therefore artificial separation of etiology may provide little value to the general practitioner. Thus, simplistically, continence can be conceptualized in terms of urethral support and urethral integrity.

Therapies to improve urethral support include pelvic floor muscle training (Kegel exercises) and vaginal pessary use. Again, urethropexy procedures such as Burch and Marshall-Marchetti-Kranz (MMK) colposuspensions attempt to re-establish this anatomic support of the urethrovesical junction and proximal urethra [2].

The physiopathology of UI is linked to levator ani muscles, endopelvic fascia, and muscular urethra comprise followed by pudendal nerve denervation and urethral's complex loss of ligamentous support. Obesity, paravaginal defects, hyperglycemia, neurological defects, microvascular innervations impairments, chronic bacterial colonization, urinary retention, and urinary tract infections are associated with suboptimal urethral compression pressure of the levator ani muscles to facilitate sphincter closure[35]. The existing theory explaining SUI is that leakage from the urethra occurs when the intra-abdominal pressure exceeds the urethral pressure. Urethral sphincter muscle and nerve integrity, bladder neck position, urethral smooth muscle and vascular plexuses, and surrounding tissue support are the factors that affect the urethral pressure. Bladder pressure remains very low as the bladder fills and

accommodates. Spinal sympathetic reflex activation inhibits detrusor muscle contraction and activation of α adrenergic receptors in the smooth muscle of the urethra and increases the outlet resistance as filling increases. Further, efferent pudendal nerve activity increases tone in the muscles of the pelvic diaphragm and striated urethral sphincter. A stable base is provided by vagina on which the urethra and bladder neck rest. This stable suburethral layer of vaginal wall and endopelvic fascia prevents urethral and bladder neck descent, such that the urethra compresses shut with straining[18].

Urethral hypermobility is major cause of SUI that is caused due to impaired support from pelvic floor while intrinsic sphincter deficiency (ISD) is less common cause, usually secondary to pelvic surgeries. However, urethral sphincter function is impaired in either case that results in urine loss at lower than usual abdominal pressures. Both the internal and the external sphincter mechanism function loss results in ISD and the only cause of SUI in males in women with SUI, either one or both mechanisms may be present, although some authors hold that SUI does not develop in patients with poor pelvic support unless intrinsic sphincter deficiency is also present [7].

Urethral Hypermobility: Impair neuromuscular functioning of the pelvic floor together with injury, both remote and ongoing, to the connective tissue supports of the urethra and bladder neck are cause of urethral hypermobility. When this occurs, at times of increased intra-abdominal pressure, the proximal urethra and the bladder neck descend to rotate away and out of the pelvis. More pressure is transmitted to the bladder as the bladder neck and proximal urethra move out of the pelvis. During this process, when intrinsic sphincter deficiency is also there, the posterior wall of the urethra shears off the anterior urethral wall to open the bladder neck. In women without urethral hypermobility, the urethra is stabilized during stress by three interrelated mechanisms[7].

Loss of urethral and bladder neck support can impair urethral closure mechanisms during increased intra-abdominal pressure. This fact can be observed in several ways. When the urethra is hypermobile, pressure transmission to the walls of the urethra may be diminished as it descends and rotates under the pubic bone. Intraurethral pressure falls below bladder pressure, resulting in urine loss[7].

Another theory of the mechanism of SUI stems from research that noted 93% of patients with SUI displayed funneling of the proximal urethra with straining, and funneling at rest was revealed in 50% of those individuals[7].

Intrinsic sphincter deficiency: In Intrinsic sphincter deficiency, the anatomic support of the urethra may be normal however; urethral sphincter is not able to coapt and produces sufficient resting urethral closing pressure to hold urine in the bladder. Devascularization and/or denervation of the bladder neck and proximal urethra cause ISD. After pelvic surgery (e.g., failed bladder suspension surgery) nearby nerve damage or excessive scarring of the urethra and surrounding tissues occurs that leads to urethral sphincter weakness. Neurologic injury or pelvic radiation and myelomeningocele are other causes of urethral dysfunction. Women with severe intrinsic sphincter deficiency do not always have the usual urethral hypermobility during a Valsalva maneuver. The urethra remains open at rest and the urethra appears well supported, which results in lead pipe urethra. Whenever intra-abdominal pressure exceeds proximal urethral pressure, involuntary urine loss occurs. The patient experiences almost continuous UI as the urethra cannot remain closed [7].

Symptoms of UI: Urinary frequency without excessive fluid intake may indicate UUI, urethral pathology, interstitial cystitis urinary tract infection and calculi should prompt additional evaluation. Incomplete emptying can result in incontinence associated with either stress or urgency. Large volumes are typically lost with UUI, whereas women with SUI usually describe smaller volumes lost. Hematuria is a common sign of UTI and may also indicate underlying malignancy and can cause irritative voiding symptoms[2]. A positive cough stress test is helpful to confirm the diagnosis of SUI. On vaginal or rectal examination, the pelvic floor muscles should be palpated with the examining finger to check muscle quality for noting any pelvic masses[18]. Few questions asked to distinguish UI are summarized in table 1[36].

Table-1
Distinguishing Incontinence Etiology by History

Question	Genuine stress incontinence	Urge incontinence	Overflow incontinence
Description of incontinence episodes	Loss with cough, sneeze or activity	Sudden urgency with inability to reach toilet	Continues slow loss
Precipitating factors	Cough, physical exercise, strain	Full bladder, sensory triggers (e.g. Running water)	None, stress may exacerbate
Urinary frequency	Normal	Often increased	Urinary hesitancy, inability to void
Nocturia	<1	Variable	Nocturnal Enuresis
Vol. of urine loss	Small amounts, pad sufficient	Large amounts, soaked clothing, runs down leg	Continuous dribbling

The investigations required for UI at level I are urinalysis, voiding dairy and postvoid residual urine measurement (PVR), cough stress test. At level II filling cystometry and Q tip test are done. At level III complex urodynamic tests like multichannel cystometry, pressure flow voiding studies, urethrocystoscopy, rethral pressure profile and stress leak point pressure (SLPP)[37].

Management: In conventional medicine, a range of treatment modalities for UI are medical management, Kegel's exercises, electrical stimulation, biofeedback, pharmacotherapy and surgical intervention. Table-2 summarizes the management of reversible conditions that cause or contribute to UI[38].

Recommended algorithm for treatment of SUI in women: Changes in lifestyle (weight loss, smoking cessation, regular exercise, etc.) Physiotherapy of the pelvic floor (under adequate supervision) If no associated prolapse and absent or moderate bladder neck mobility (BNM): bulking agents (BA), BA failure, associated prolapse, major BNM: midurethral sling.

Recurrent SUI: Redo midurethral sling or BN fascial sling is used[39].

Further, different food groups that may have high acidity or caffeine content may lead to greater urinary frequency and urgency. Certain supplement such as calcium glycerophosphate when added to the diet has been shown to decrease urgency and frequency symptoms. This is a phosphate-based product and is thought to buffer urine acidity. Scheduled voiding in UUI leads to an empty bladder during a greater percentage of the day. Because some women will leak urine only if bladder volumes surpass specific threshold volumes, frequent emptying can significantly decrease incontinent episodes. Incontinent women of postmenopause age with atrophy, administration of exogenous estrogen is reasonable. Estrogen is commonly administered topically. Many different regimens are appropriate, conjugated equine estrogen cream administered daily for 2 weeks, then twice weekly thereafter. Alternatively, oral estrogen may be prescribed if other menopausal symptoms for which estrogen would be beneficial co-exist. Pharmaceutical treatment plays a minor role in the treatment of women with SUI. Tricyclic antidepressant has α-adrenergic effects and the urethra contains a high content of these receptors. The only drug licensed for use for SUI is duloxetine, a serotonin and noradrenaline re-uptake inhibitor[2,40]. Certain pessaries have been designed to treat incontinence as well as pelvic organ prolapse. Incontinence pessaries are designed to reduce downward excursion or funneling of the urethrovesical junction. This provides bladder neck support and thereby helps to reduce incontinent episodes[2].

Surgery: For those who are not adequately improved with or do not desire conservative management, surgery may be an appropriate next step for successful treatment of stress incontinence symptoms[2]. There have been several Cochrane reviews on the different operations for SUI including anterior repair, open colpo-suspension, laparoscopic colposuspension, needle suspensions, urethral injectables, pubovaginal slings, midurethral slings and sub-urethral slings[40].

Table-2
Management of Reversible Conditions that Contribute to Urinary Incontinence

Condition	Management
Excess fluid intake	Reduction in intake of especially diuretic fluids (eg, caffeinated and alcoholic beverages)
Urinary tract infection	Antimicrobial therapy
Atrophic vaginitis/urethritis	Oral or topical estrogen may be considered, but effect on UI is controversial
Stool impaction	Disimpaction, stool softeners, bulk-forming agents, and laxatives if necessary, high fiber intake, adequate mobility and fluid intake
Metabolic (hyperglycemia, hypercalcemia)	Better control of diabetes mellitus. Therapy for hypercalcemia depends on underlying cause.
Venous insufficiency with edema	Support stocking, leg elevation, sodium restriction, diuretic therapy
Chronic congestive heart failure	Medical therapy
Chronic or intermittent cough or sneeze	Intensify, if possible, treatment of cold, asthma or allergy
Chronic illness, injury, or restraint that interferes with mobility	Regular toileting, use of toilet substitutes, environmental alterations (e.g., bedside commode, urinal)
Psychological	Remove restraints if possible. Administer appropriate pharmacologic and/or nonpharmacologic treatment.
Delirium	Diagnosis and treatment of underlying cause(s) of acute confusional state
Medications	Suspend or modify use of diuretics, alpha blockers, and ACE inhibitors (the latter of which can exacerbate cough)

Surgical Tr1atment for Intrinsic Sphincteric Deficiency: A recent clinical study has documented that bulking therapy is more effective than conservative treatment in terms of pelvic floor training[41]. Agents are injected submucosally and elevate the urethral mucosa to improve coaptation. A number of available bulking materials are available for injection. These materials can be injected periurethrally or transurethrally, and the location of the injections can vary. Some recommend two locations on either side of the urethra, whereas others advocate injections in three or four quadrants. It is usually inject at the level of the urethra-vesical junction at sites of apparent urethral mucosal defects. However, if a global defect is noted or if a discrete defect is absent, then a two- to four-quadrant approach is used[2].

Conclusion

SUI is the most common type of UI in women. Pregnancy and childbirth, menopause, obesity, advanced age, and race and ethnicity are some common risk factors of SUI. Pudendal nerve denervation and loss of ligamentous support of the urethral complex leads to dysfunction of the levator ani muscles, endopelvic fascia, and muscular urethra causing UI. The treatment for SUI is changes in lifestyle, physiotherapy of the pelvic floor, bulking agent and midurethral sling.

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