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Navigating the Complexities of Pelvic Organ Prolapse and Stress Urinary Incontinence: A Review of Current Literature

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ABSTRACT

Pelvic organ prolapse (POP) and stress urinary incontinence (SUI) are prevalent conditions that significantly impact women's health and quality of life. This literature review explains current research on the complex association between these disorders, highlighting risk factors and prevention, diagnostic and management, and also recent advances in the molecular mechanism of POP and SUI. Recent studies have identified key risk factors for SUI in women with POP, including age, obesity, genetics, vaginal delivery, and menopause, suggesting a multifactorial pathophysiology that warrants further investigation. In terms of surgical management, there is growing recognition of the need for individualized treatment plans that consider patient-specific factors to optimize outcomes. Additionally, emerging research has explored the role of inflammatory cytokines and oxidative stress in the development of these conditions, providing insights into potential therapeutic targets. Continued research is essential to further elucidate these complex interactions and develop targeted therapies that address both conditions effectively.

Keywords: pelvic organ prolapse; stress urinary incontinence; risk factors; quality of life.

INTRODUCTION

Pelvic organ prolapse (POP) is characterized by the downward displacement of one or more pelvic organs, including the anterior and posterior vaginal walls, cervix, and/or vaginal apex following a hysterectomy [1]. On the other hand, stress urinary incontinence (SUI) involves the involuntary leakage of urine triggered by an increase in intra-abdominal pressure, such as during physical exertion, sneezing, coughing, laughing, or straining [2].

It is often quoted that approximately 50% of all women will experience pelvic organ prolapse. This refers to the anatomical change of pelvic organ descent into or up to the vagina, perineum, and anus, and does not reflect the severity and symptoms associated with prolapse. Therefore, the prevalence of symptomatic POP is actually much lower than that of asymptomatic POP [3].

The complexities associated with POP and SUI present numerous challenges. Both POP and SUI are conditions that can lead to discomfort, pain, urinary issues, reduced sexual function, limitations in daily activities, and social embarrassment due to stigma [4]. These disorders often go undiagnosed and untreated until they significantly impact daily life, as many patients believe they are a natural part of aging and not life-threatening, despite the considerable negative impact they have on women's quality of life [5]. These conditions can lead to significant healthcare costs, as well as reduced productivity and quality of life for affected individuals. Furthermore, the overlapping nature of these disorders complicates diagnosis and treatment, as many women may not report their symptoms due to embarrassment or lack of awareness [4].

Current management strategies for POP and SUI include conservative treatments such as pelvic floor exercises, lifestyle modifications, and the use of pessaries [6], alongside surgical interventions like vaginal mesh surgery or mid-urethral sling procedures [7]. However, the effectiveness of these treatments varies widely among patients, highlighting the necessity for individualized care plans based on comprehensive assessments.

This literature review aims to address the existing gaps in understanding the interplay between POP and SUI by synthesizing current research findings. It will explore recent advances in treatment approaches and identify risk factors associated with both conditions. By doing so, this review will contribute valuable insights for clinicians seeking to optimize patient care. Recent updates from organizations such as the American Urological Association emphasize the importance of individualized treatment plans that consider patient preferences and specific clinical circumstances [8].
In conclusion, this literature review will not only elucidate the complexities surrounding pelvic organ prolapse and stress urinary incontinence but also propose actionable insights aimed at enhancing patient care through updated guidelines and targeted research initiatives.

DISCUSSION

Pelvic Organ Prolapse

Definition of Pelvic Organ Prolapse

Pelvic organ prolapse (POP) is defined as the descent of one or more pelvic organs into the vagina (incomplete prolapse) and possibly out of the vagina (complete prolapse) due to weakness or damage to the supporting structures of the pelvic floor organs [1]. POP is a common condition that can cause urogenital tract dysfunction and decreased quality of life. Symptoms include sensations of vaginal protrusion, pelvic pressure, and pelvic organ prolapse. The type of prolapse depends on the organ involved and its position against the vaginal wall. POP is common in a large proportion of the female population. However, it is considered a disease only when an individual requires surgery or non-surgical therapy to relieve symptoms, restore function, and improve the individual's quality of life [5].

Epidemiology of Pelvic Organ Prolapse

According to data from the Women's Health Initiative (WHI), the prevalence of POP types in women aged 50-79 years is 39.46% cystocele, 21.42% rectocele, and 16.3% uterine prolapse [6]. In addition, in post-hysterectomy cases, 6-12% of women will experience vaginal crest prolapse, and in two-thirds of these cases multi-compartment prolapse occurs [9].

Research by Paraton et al. (2016) in a research report written by Khoiriyyah (2023), stated that there were 200 reported cases of POP in the period 2013-2016 with 38.9% of them aged 60-69 years. Research by Khoiriyyah (2023) stated that there were 72 cases of POP in the gynecology clinic of Dr. Soetomo Hospital in the 2018-2019 period. Of the 72 cases, most (68.1%) experienced multi-compartment prolapse [10].

Risk Factors of Pelvic Organ Prolapse

a) Age

The prevalence of POP increases with age. In women aged 20 to 59 years, the incidence of POP doubles every decade (10 years) [5]. POP occurs in women of advanced age (50 years) because at this age there is a decrease in the function of the female body, especially reproductive function, which affects the strength of the pelvic floor muscles. Meanwhile, in women aged <50 years, the incidence of POP can be triggered by several other factors. Increasing age makes the fascia tissue more rigid, easily broken, and unable to hold the organs inside. In addition, at the age of >40 years until before the menopause phase begins, estrogen levels decrease, even though estrogen plays a role in the formation of collagen tissue. Reduced levels of collagen tissue cause pelvic floor muscle weakness which usually occurs 5-10 years after menopause [11].

b) Loose Tissue Disorders

Women with connective tissue disorders are at higher risk of POP. It was reported in one study that one-third of women with Marfan Syndrome and three-quarters of women with Ehlers-Danlos Syndrome had POP [5].

c) Genetic

The development of POP rarely involves genetic factors. For example, African women, with less collagen tissue than Asian women, have a higher risk factor for POP [1]. Some studies have shown that if a woman has a sibling with a history of severe POP, the woman has a five-fold higher risk of POP than other women. Recently, new genomic linkage studies have been conducted to identify specific predisposition genes that will hopefully lead to better prevention strategies for future female populations [5].

d) Birth delivery

Labor can result in trauma to the pelvic floor and loss of support to the pelvic organs. Direct trauma such as tears to the musculus levator ani or ligaments can occur during vaginal delivery. Pelvic nerve damage also plays a role when the nervus pudendus is crushed by the pelvic bones during labor. The longer the labor, the more pelvic nerve damage occurs. Labor using forceps also increases the risk of musculus levator ani damage. Prolonged labor along with a history of macrosomia babies and assisted delivery will increase the risk of prolapse even higher [1].

In a large study conducted 5-10 years after the first delivery, the risk of POP in women with vaginal delivery was five-fold higher than women with caesarean delivery. Theoretically, fewer women would experience POP if most women had cesarean deliveries. However, keep in mind that the woman also has to bear the potential risks of a cesarean section. Therefore, a woman's decision to have a vaginal or caesarean delivery to prevent POP should be based on her specific body condition in discussion with her doctor and the final decision is individualized to the woman [5].

e) Gynecologic Surgery

Surgery can be the cause of some types of prolapse although surgery is often used to treat prolapse itself. For example, colposuspension is used to treat stress urinary incontinence. This procedure lifts the bladder neck behind the pubic symphysis to support

the bladder but affects the gravity of the posterior vaginal wall, causing posterior vaginal wall prolapse in 25% of women who undergo this procedure. In addition, vaginal vault prolapse is a symptom experienced after a woman has a hysterectomy [1].

f) Increased of Intra-Abdominal Pressure

Increased intra-abdominal pressure due to chronic constipation, chronic cough, repetitive heavy lifting, and obesity may contribute to the pathogenesis of POP. In the Women's Health Initiative trial, overweight increased POP rates by 31-39% and obesity increased POP rates by 40-75% [5]. The average POP patient in Spain, Europe has a BMI of 26.6 [12]. Individuals who have repetitive heavy lifting jobs such as laborers, experience more severe POP and have a higher rate of surgical intervention compared to the general population rate. Smoking and COPD are also involved in the development of POP. In addition to chronic cough that can increase intra-abdominal pressure, inhaled tobacco chemicals can cause connective tissue changes and may increase the risk of POP [5].

g) Menopause

Menopause characterized by deficient estrogen levels in the body and loss of connective tissue strength, increases the risk of POP severity. However, POP can also occur in young women before menopause, meaning that menopause is not always involved as a risk factor for POP [1].

Etiology and Pathophysiology of Pelvic Organ Prolapse

Pelvic organ prolapse is commonly acquired and may worsen as conditions occur that weaken the supporting structures of the pelvic floor organs. The etiology of POP is multifactorial which results in weakness/damage of the pelvic floor support structures [13]. The pathophysiology of POP can be explained through the following mechanisms. Any condition that can chronically increase intra-abdominal pressure can lead to POP. For example, constipation and habitual straining during bowel movements will cause an increase in intra-abdominal pressure, which can damage the connective tissue and nerves around the pelvic floor due to continuous (chronic) strain. Another example is pregnancy and childbirth, which can be one of the risk factors for pelvic floor muscle and fascia damage, which can trigger POP [14].

Changes in collagen structure, for example in women with Marfan Syndrome, there is an increase in collagen degradation resulting in an increase in the ratio of the number of collagen III fibers in the body. This can weaken the tissue supporting the pelvic floor organs. In addition, decreased estrogen levels in post-menopausal women result in a decrease in the ratio of type I collagen to type III and IV collagen. This can weaken the tensile strength of the vaginal wall and increase the risk of vaginal wall prolapse [14].

Classification of Pelvic Organ Prolapse

The Pelvic Organ Prolapse-Quantification (POP-Q) system was recommended by the International Continence Society (ICS) in 1996. This system describes the site-specific components and degree of prolapse. The prolapse at each segment is measured relative to the hymen. The measurement is given a negative sign if the prolapse point is proximal to the hymen and a positive sign if it protrudes past the hymen. The POP-Q system also describes the degree of prolapse in four stages as follows [5].

TABLE 1: Classification of Pop Stage Based on Pop-Q [6].

Stage	Description
0	No prolapse
I	Lowest part of prolapse > 1 cm proximal to the hymen
II	Lowest part of prolapse < 1 cm proximal to the hymen
III	Lowest part prolapsed > 1 cm distal to the hymen but < 2 cm total vaginal length (< 2 cm tvl)
IV	Entire vagina folded out (eversion)

The six prolapse points in reference to the hymen included two points on the anterior vaginal wall (points Aa and Ba), two on the vaginal vault (points C and D), and two on the posterior vaginal wall (points Ap and Bp). In addition to these six points, there are three other points, namely the genital hiatus (gh), perineal body (pb), and total vaginal length (tvl). All these points are measured during patient straining and should reflect maximum protrusion [5].

Karen Guerrero (2019) divides the types of POP into three compartments, namely the anterior compartment (urethrocele/ urethra, cystocele/ bladder), posterior compartment (rectocele/ rectum, enterocele/ small intestine), and superior compartment (prolapse uteri/ uterus and cervix, vaginal vault/ top of vagina) [1].

Signs and Symptoms of Pelvic Organ Prolapse

Signs and symptoms of POP generally depend on the severity and location of the prolapse. Mild prolapse is common in parous women and may be asymptomatic. Symptoms often vary and are related to the nature of the prolapse. Based on the levels of pelvic floor organ support structures created by DeLancey (1992), Rane et al. (2020) summarized the symptoms that appear based on these levels in the following table [15].

Management of Pelvic Organ Prolapse

Not all POP requires therapy as most have no complaints especially in stage I and sometimes stage II. There are principles of POP management which include establishing a diagnosis to determine the stage of prolapse, eliminating or minimizing risk factors, considering the patient's wishes whether to follow conservative or operative therapy, and managing comorbid diseases found [14].

Pelvic organ prolapse without complaints does not need treatment. Stage I and II POP is treated with exercises to strengthen the pelvic floor supporting muscles, namely kegel exercises or with biofeedback equipment. POP stage III and IV if the patient is not able to do surgery, waiting for surgery, does not want to be operated on, still wants to get pregnant, and is pregnant, a pessary ring is installed, given a low dose of estrogen in menopausal women, and regular control to the doctor every month [6].

For surgical intervention, the doctor should explain to the patient the possible side effects that can occur after surgery such as urinary disorders, fistula, urinary incontinence, and overactive bladder. Preoperative estrogen administration in menopausal patients to improve the healing process. The principle of surgery is to make corrections to the tissues that support the position of urogenital anatomy and physiology. The types of surgery that can be performed consist of purandare, uterosacropexy, and hysterectomy [6].

Stress Urinary Incontinence

Definition of Stress Urinary Incontinence

According to the International Continence Society Guidelines, urinary incontinence is a symptom, sign and condition. In patients with stress urinary incontinence (SUI), patients complain of accidental urine loss during exercise, sneezing, jumping, and laughing. SUI occurs due to an increase in intra-abdominal pressure so that intravesical pressure exceeds the maximum pressure of urethral closure and in the absence of detrusor activity [16].

Epidemiology of Stress Urinary Incontinence

The prevalence of SUI varies across different regions and studies. In the United States, the prevalence of SUI from 2005 to 2016 was reported to be 26%, with the highest incidence found in women aged 40–59 years. In China, a study estimated the prevalence of SUI at 12.4%, with the highest incidence occurring in individuals aged 60–69 years. In Australia, up to 50% of women were affected by SUI. In Sub-Saharan Africa, the pooled prevalence of SUI was estimated at 52%. In Europe, the prevalence ranges from 21.4% in Denmark to 24.4% in Germany, with SUI being more common among younger women [17].

A study in 2021 reported that the prevalence of urinary incontinence in Saudi women was around 41.7% [18]. A 2016 study reported the prevalence of SUI in postpartum women in Indonesia to be 8.8% [19]. Another study reported that the prevalence of urinary incontinence was 32.32% in women with POP in Bali, Indonesia [20].

TABLE 2: Pop Symptoms by Level of Pelvic Organ Support Structure [15].

Level	Affected Organs	Type of POP	Symptoms
I – uterosacral and cardinal ligament	uterus, vaginal vault (post hysterectomy), pouch of Douglas	uterine prolapse, *vaginal vault prolapse*, enterocele	vaginal pressure, back pain, "feeling of something coming down", dyspareunia, and vaginal discharge.
II – arcus tendineus fascia pelvis (ATFP)	bladder	cystocele	"feeling of something coming down", stress urinary incontinence, recurrent urinary tract infection.
	rectum	rectocele	"feeling like something is coming down", the difficulty of defecation.
II – anterior/ pubourethral ligament	urethra	urethrocele	"feeling like something is coming down", stress urinary incontinence.
III – posterior/ perineal body	lower third of vagina/ introitus/ anal canal	dilation of genital hiatus	a sense of loosening of the vagina, sexual dysfunction, flatulence, need to press on the prolapsed organ in order to defecate.

Risk Factors of Stress Urinary Incontinence

a) Age

The prevalence and severity of incontinence increases with age. Some age-related physiological changes in the lower urinary tract may be risk factors for incontinence, including bladder overactivity or other micturition difficulties. The total bladder capacity and ability to hold urine decreases, leading to an increase in urinary frequency. Reduced urinary flow rate due to decreased detrusor contractility. Decreased estrogen levels post-menopause result in atrophy of the urethral mucosa and bladder irritation. This leads to a decrease in urethral sphincter strength and the potential for SUI [16].

b) Body Mass Index (BMI)

BMI is a significant risk factor for SUI. The average SUI patient in Spain and Europe had a BMI of 28 [12]. Of 551 SUI women in Northern Jordan, 39.2% were overweight and 46.3% were obese [21]. Obese women are three times more likely to experience urinary incontinence. Theoretically, increased intra-abdominal pressure at a higher BMI gives rise to increased intravesical pressure. In contrast, weight loss by behavior modification or bariatric surgery has significantly reduced the prevalence of urinary incontinence [16].

c) Vaginal delivery and pregnancy

The prevalence of urinary incontinence is higher in parous women compared to nulliparous. The effect of labor on urinary incontinence may result from direct injury to pelvic muscles and connective tissue attachments. In addition, nerve damage from trauma or stretching injury can lead to pelvic muscle dysfunction. A large study identified that fetal birth weight >4000g increased the risk of all types of urinary incontinence. The first vaginal delivery causes maximal damage to the pelvic floor and all subsequent vaginal deliveries may exacerbate the damage. Delivery by caesarean section may provide short-term protection from urinary incontinence. However, the protective effect of caesarean delivery against incontinence may disappear after subsequent deliveries, decrease with age, and cease to exist in elderly women [16]. Pregnancy also causes irreversible pelvic floor damage. Some women experience SUI during pregnancy due to increased intra-abdominal pressure associated with uterine contents and the smooth muscle relaxing effects of progesterone [22].

d) Menopause

Decreased estrogen levels in the body can weaken the urethral sphincter complex and reduce maximal urethral closure pressure (intrinsic sphincter deficiency) resulting in SUI [22]. High-affinity estrogen receptors are found in the urethra, pubococcygeal muscle and urogenital trigonum, but rarely elsewhere in the bladder. Hypoestrogenic conditions are associated with changes in collagen, decreased urethral vascularization, and decreased skeletal muscle volume. These are thought to collectively contribute to impaired urethral function by lowering urethral pressure during the resting phase. In addition, estrogen deficiency resulting in urethral atrophy is associated with urinary sensory symptoms after menopause [16].

Etiology and Pathophysiology of Stress Urinary Incontinence

Stress urinary incontinence is experienced when there is a disruption in the proximal and distal urethral sphincter mechanism due to detrusor inactivity (unable to contract) and/or disruption of the connective tissue around the urethra. If no etiologic factors are found in the patient, this condition can be explained by the patient's SUI risk factors [22]. SUI is theoretically described as reduced urethral integrity (sphincter deficiency). The integrity of the urethra and bladder is supported by several structures: (1) ligaments along the lateral part of the urethra (pubourethral ligaments); (2) the vagina and its lateral fascia condensations; (3) arcus tendineus fascia pelvis; and (4) levator ani muscles. Under normal conditions, increased intra-abdominal pressure is equally distributed to the bladder, bladder base, and urethra balanced by the tone of the supporting structures, which are the levator ani muscles and vaginal connective tissue. If the tone of the supporting structures is reduced either due to mucosal abnormalities, vascular plexus, epithelium, innervation, and contraction of the surrounding muscles, the ability of the sphincter at the proximal urethra and bladder neck to close and resist pressure from the overlying structures is also reduced. As a result, when intra-abdominal pressure increases, urine is involuntarily voided [16].

Sign and Symptoms of Stress Urinary Incontinence

Stress urinary incontinence is usually characterized by increased intra-abdominal pressure such as during coughing, sneezing, valsalva maneuver, or sexual intercourse. In patients with SUI, the volume of urine released is small and the patient can still contract the levator ani muscle to stop the flow of urine. Some symptoms can also provide information regarding the diagnosis. If it starts at the time of menopause it indicates that the likely etiology is hypoestrogenism. Obstetric trauma may be associated with damage to the supporting structures of the pelvic floor organs which can lead to SUI [23].

Management of Stress Urinary Incontinence

Conservative (non-surgical) management is performed if SUI is mild, delaying surgery, SUI with a combination of detrusor instability, and there are complications if surgery/anesthesia is performed. The success of conservative therapy in urinary incontinence is 90% if mild, 40-80% if moderate, 20-40% if severe, and only to wait for surgery [2].

a) Pelvic floor strengthening

Pelvic floor strengthening is initial management in most patients with urinary incontinence. Kegel exercises performed using isotonic contractions (contracting and maintaining for a certain period of time) aim to increase pelvic floor muscle tone to anticipate a sudden increase in intra-abdominal pressure in SUI [23]. Kegel exercises are performed for at least 3 months with the aim of training the levator ani muscle (so that, the vaginal angle becomes 45 degrees distally and 120 degrees proximally), not all pelvic floor muscles. The success of kegel exercises depends on patient knowledge such as understanding which muscles to contract, how to contract them, and being willing to provide special time to do kegel exercises [2].

b) Behavioral and environmental modification

By eliminating the underlying disease DIAPERS by stopping drinking coffee, alcohol, foods containing carbon dioxide, sour fruits and drinks, spicy foods, smoking, losing weight, stress management, preventing and treating chronic or systemic diseases [2]. Regarding to diet, food groups with high acidity or containing caffeine may increase urinary frequency and urgency. Meanwhile, supplements such as calcium glycerophosphate can reduce urinary frequency and urgency [23].

c) Scheduling micturition

Initially, micturition is scheduled every half hour, interspersed with kegel exercises when there is an urge to micturate. Bladder training is done based on the daily micturition list by taking the shortest micturition interval, and then asking the patient to hold the fastest micturition at that interval. For example, the first week, micturate every 1 hour. Second week, micturate every 1.5 hours.

This will continue for 8-12 weeks. The treatment is successful if the patient can hold micturition for >3 hours. The success rate of this therapy reaches 80-90% [2].

d) Hormone replacement therapy
Estrogen increases urethral blood flow and alpha-adrenergic receptor sensitivity at the bottom of the bladder and urethra thereby increasing urethral coaptation and urethral closure pressure. Estrogen also increases collagen deposition and vascularity of the periurethral capillary plexus. Estrogen is generally given topically but can be given orally [23].

For surgical intervention, there are various procedures could be performed.

a) Sling for midurethra
A synthetic sling is placed on the midurethra to provide tension free support. The sling is formed from tissue derived from the fascia lata or fascia rectus abdominis. Sling options for the midurethra include transobturator sling, retropubic sling, single-incision sling, and adjustable sling [7].

b) Open and laparoscopic surgery
The gold standard of surgical intervention for SUI is colposuspension. Options for open surgery include open colposuspension, otology fascial sling, and laparoscopic colposuspension. SUI can be performed anterior colporaphy with success between 45-60% either with Kelly Plication or Kelly Kennedy [2,7].

c) Bulking agents
Bulking agents or transvaginal neddle bladder neck suspension is an intra- or periurethral injection of colloids or silicone under the submucosal layer which then forms an artificial cushion that will increase resistance to urine flow and facilitate continence [2,7].

d) External compression device
If SUI recurs after previous surgery (recurrence) or recurs due to intrinsic sphincter failure or low urethral closure pressure, an alternative that can be used is external compression devices such as adjustable compression therapy (ACT) and artificial urinary sphincter (AUS) [7].

Association between Pelvic Organ Prolapse and Stress Urinary Incontinence

The association between POP and SUI is explained based on a holistic view of pelvic floor anatomy and the comprehensive integral theory of Petros and Ulmsten (1990). Both symptoms share a common etiology of injury or weakness in the supporting structures of the pelvic floor organs [12]. POP is commonly acquired and may worsen with the occurrence of conditions that can weaken the supporting structures of the pelvic floor organs. In addition, any condition that increases intra-abdominal pressure can lead to prolapse [13]. Similarly, when the tone of the urethral support structures is reduced, the ability of the sphincter at the proximal urethra and bladder neck to close and resist pressure from the overlying structures is also reduced. As a result, when intra-abdominal pressure increases, urine may escape involuntarily [16].

Besides having the same etiology, POP and SUI are affected by some of the same risk factors. These risk factors include age (aging), overweight or obesity, vaginal delivery, menopause, collagen/connective tissue abnormalities, family history, chronic diseases that trigger a continuous increase in intra-abdominal pressure, and gynecological surgery such as hysterectomy [12].

The systematic review and meta-analysis by Fitri et al. (2023) explored the risk factors for stress urinary incontinence (SUI) among women with pelvic organ prolapse (POP). Utilizing data from 16 studies involving 47,615 participants, the analysis identified 27 risk factors, highlighting obesity (OR = 1.15), a history of hysterectomy (OR = 2.01), and diabetes mellitus (OR = 1.85) as significant contributors to SUI in this population. The study underscores the interplay between these conditions due to shared anatomical and physiological factors. Increased in intra-abdominal pressure due to obesity, causes weakness of nerve supply and pelvic musculature, and subsequently leads to an increased intravesical pressure and urethral mobility so that SUI occur. SUI after hysterectomy may occur due to prolonged injury of pelvic pressure lead to impaired anatomic support of the bladder neck and urethra. Microvascular damage in diabetes mellitus, causes weakened connective tissue support and dysfunction of the pudendal nerve endings resulting in weakness of the urethral sphincter, so that SUI occur. Despite limitations like a small pool of eligible studies for meta-analysis, this research provides valuable insights for early identification and management of SUI in POP patients [17].

Patients with anterior POP have a 2.5 times higher prevalence of urinary incontinence than women without anterior POP [12]. Anterior POP is a prolapse of the urethra, bladder, or both, so it is often called urethrocystocele [6]. Anterior POP involves the pubocervical fascia and pubourethral ligament which are between the anterior vaginal wall and pubis and support the bladder and urethra [24]. This type of prolapse can cause urethral hypermobility, which can disrupt the continence mechanism and lead to urinary incontinence [1]. Meanwhile, one of the data in a journal states that the prevalence of urinary incontinence is not only in anterior prolapse, but 58% in women with anterior prolapse, 55% in women with posterior prolapse, and 72% in women with superior prolapse [25].

Patients with POP often have urinary tract symptoms such as urinary incontinence or difficulty micturition. SUI symptoms are often complained along with POP and there is a significant risk of SUI progression when measures are taken to repair prolapse. However, most advanced stage POP (stage III-IV) do not complain of SUI symptoms but may be at risk [27]. Only 33% of patients with stage IV POP complain of SUI. This is because POP increases pressure on the urethra, causes obstruction, and

maintains continence [28]. Eventually, after measures such as pessary ring insertion and POP surgery, these SUI symptoms appear [27].

Prevention of Pelvic Organ Prolapse and Stress Urinary Incontinence

Early-stage of POP is rarely given medical attention, even though it is often identified in young and active women who often complain of decreased quality of life especially with regard to sexual life, work, and physical activity [4]. SUI is also a quality of life issue as it can cause discomfort and negatively impact physical, psychological, social, sexual activity, work, and decrease social and interpersonal interactions. However, both conditions often go undiagnosed and untreated before symptoms appear that interfere with daily activities [2]. The etiology of both conditions is multifactorial. The cause of the weakening of the connective tissue and ligaments of the pelvic floor remains unclear, but answers can be found when looking more closely at the risk factors [3].

Prevention of POP and SUI can be done as early as possible by reducing preventable risk factors such as BMI and stage of POP, which can be done among others: basic pelvic muscle exercises (kegel exercises), lifestyle modification by eating high-fiber foods, improving defecation habits, and lowering BMI with lifestyle modification or bariatric surgery, treating chronic cough disease, and reducing activities or jobs that carry heavy items repeatedly [14]. To inform this prevention program to be carried out by the community, of course, activists are needed who go down to the community to provide counseling, training, and support so as to reduce the prevalence of SUI and POP, and the end result is an improvement in the quality of life of women in the world.

Recent Advances

Recent research has significantly advanced our understanding of the molecular mechanisms involved in pelvic organ prolapse (POP) and its association with stress urinary incontinence (SUI). A comprehensive review highlighted the role of cytokines in the pathogenesis of both conditions, particularly focusing on pro-inflammatory cytokines that contribute to tissue remodeling and inflammation within the pelvic floor. For instance, the interaction of interleukin-1 (IL-1) family members has been shown to promote collagen deposition in the uterosacral ligament, suggesting a direct link between inflammatory processes and the structural integrity of pelvic support tissues [29].

Moreover, a study identified reactive oxygen species (ROS) as key mediators of pelvic floor damage. Mechanical injury activates the NLRP3 inflammasome, leading to chronic inflammation and impaired fibroblast function, which ultimately disrupts extracellular matrix (ECM) metabolism [30]. This suggests that targeting oxidative stress could be a potential therapeutic strategy for managing POP and SUI.

Additionally, researchers have begun to explore the role of microRNAs in regulating gene expression related to POP. For example, miR-138 has been implicated in enhancing collagen synthesis while reducing inflammatory cytokine levels, indicating its potential as a therapeutic target for improving pelvic floor integrity [31].

CONCLUSIONS

The etiology of POP and SUI is multifactorial. To prevent the occurrence of POP and SUI, it must be done as early as possible by reducing preventable risk factors, such as doing Kegel exercises and maintain healthy lifestyle. Regarding the management of POP and SUI patients, individualized treatment plans are needed that consider patient preferences and specific clinical circumstances. For this reason, the success of therapy and prevention education depends on how effective communication between doctors and health workers with patients and women in the general population. The recent advances in understanding the molecular mechanisms underlying POP and SUI, along with innovative surgical techniques and materials, hold promise for improving patient outcomes. Continued research is essential to further elucidate these complex interactions and develop targeted therapies that address both conditions effectively.

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