REVIEW ARTICLE

Stress urinary incontinence in pregnant women: a review of prevalence, pathophysiology, and treatment

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Abstract

Introduction and hypothesis Stress urinary incontinence (SUI) is the most common type of urinary incontinence (UI) in pregnant women and is known to have detrimental effects on the quality of life in approximately 54.3 %. Pregnancy is the main risk factor for the development of SUI. This review provides details of the pathophysiology leading to SUI in pregnant women and SUI prevalence and treatment during pregnancy.

Methods We conducted a PubMed search for English-language and human-study articles registered from January 1990 to September 2012. This search was performed for articles dealing with prevalence and treatment of SUI during pregnancy. In the intervention studies, we included studies that used a randomized controlled trial (RCT) design or studies comparing a treatment intervention to no treatment.

Results A total of 534 articles were identified; 174 full-text articles were reviewed, and 28 of them met eligibility criteria and are reported on here. The mean prevalence of SUI during pregnancy was 41 % (18.6–60 %) and increased with gestational age. The increasing pressure of the growing uterus and fetal weight on pelvic-floor muscles (PFM) throughout pregnancy, together with pregnancy-related hormonal changes, may lead to reduced PFM strength as well as their supportive and sphincteric function. These cause mobility of the bladder neck and urethra, leading to urethral sphincter incompetence. Pelvic floor muscle exercise (PFME) is a safe and effective treatment for SUI during pregnancy, without significant adverse effects.

Conclusions Understanding these issues can be useful for health-care professionals when informing and counseling pregnant women to help prevent SUI during pregnancy and the postpartum period.

Keywords Stress urinary incontinence · Pregnancy · Prevalence · Pathophysiology · Treatment · Review article

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Introduction

Stress urinary incontinence (SUI), the complaint of involuntary loss of urine on effort or physical exertion or sneezing or coughing [1], is the most common type of urinary incontinence (UI) in pregnant women. It is known to have detrimental effects on quality of life (QoL) in approximately 54.3 % of all pregnant women in four domains: physical activity, travel, social relationships, and emotional health [2]. Pregnant women with UI have statistically significant lower QoL during pregnancy than those without UI [3], and the QoL of incontinent pregnant women worsens with increasing gestational age to term [4]. Pregnant women with urgency (UUI) or mixed (MUI) incontinence had worse QoL scores during pregnancy than those with SUI alone. QoL scores on the physical, social, and emotional functioning domains were low, whereas scores on mobility and embarrassment domains were higher, suggesting a minimal restriction in lifestyle [5]. According to van Brummen et al. [6], the bother of lower urinary tract symptoms (LUTS) occurred most frequently at 36 weeks of pregnancy and were still a bother at 1 year after pregnancy. Interestingly, SUI occurred in the older maternal age groups, and the presence of bothersome LUTS occurred at 12 weeks of pregnancy, both of which were accounted to the predictive factor of bothersome SUI at 1 year after delivery. According to that study, cesarean section seemed to have a protective effect on bothersome SUI at 1 year after delivery.

Mascarenhas et al. [3] reported that UI negatively affects social relationships and particularly interferes with sexual relationships. Dolan et al. [2] found that UI impinges on sexual relationships. Although SUI severity increases with pregnancy progression from the first to the third trimester [7] and during 36 weeks of gestation, 16.9 % of pregnant women report having moderate to greatly bothersome UUI [6]. However, the QoL of pregnant women was found to be mildly affected by UI [8]. This may be because UI symptoms were not severe [9]. Pregnant women tend to consider this to be a common discomfort associated with pregnancy and a consequence of childbirth in the postpartum period [10]. Van de Pol et al. [4] found significantly higher rates of depressive symptoms and SUI during pregnancy than after childbirth. Another important impact of pregnancy-related UI is an increased risk of permanent incontinence in the postpartum period or later in life [11, 12]. Many studies have reported that pregnant women who had UI during pregnancy are at higher risk for postpartum UI than those without UI during pregnancy [13–15].

Pregnancy and delivery-related factors are considered to be the main risk factors for SUI development during pregnancy. The purpose of this review is to explain the pathophysiology leading to SUI in pregnant women and SUI prevalence and treatment during pregnancy. This information can be useful for health-care professionals when informing and counseling pregnant women in SUI prevention during pregnancy and the postpartum period.

Materials and methods

This review was performed following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [16]. We conducted a PubMed search for English-language and human-study articles registered from January 1990 to September 2012 using the following search terms: epidemiology, prevalence, urinary incontinence, stress urinary incontinence, pregnancy, pregnant women, pelvic-floor-muscle exercise, pelvic-floor-muscle training, and conservative treatment Conference proceedings and abstracts from the International Continence Society annual meetings were also searched. Additional relevant publications were selected from the reference lists of identified articles, and abstracts presented at international meeting were also included. No language restriction was defined. Abstracts and conventional reports were excluded. All articles were screened by title and abstract by BS and NS. Studies reporting on the prevalence of all UI that included SUI, UUI, and MUI in pregnant women both primigravidae and multigravidae were selected. Intervention studies related to pelvic-floor-muscle exercises (PFME), pelvic-floor-muscle training (PFMT), and other conservative treatments

to prevent and treat UI during pregnancy and the postnatal period were included. In intervention studies, we included studies that used a randomized controlled trial (RCT) design or that compared a treatment intervention to no treatment. Noncomparative studies (i.e., pre-post studies, case report, case series) were excluded. A total of 534 publications in PubMed were identified. Titles and abstracts were screened for relevance and applicability, and 360 articles were excluded. The remaining 174 articles were screened in full text. Of these, 26 articles were included: 18 of UI prevalence and eight of UI treatment during pregnancy and the postnatal period met eligibility criteria as defined in Fig. 1.

The prevalence of urinary incontinence in pregnancy

The most common type of UI in pregnant women is SUI. However, the true prevalence of SUI is still unknown. There are many studies on the prevalence of SUI during pregnancy, and results vary depending on study method and design, such as definitions of UI, evaluation questionnaire, and stage of pregnancy [12].

Most studies focused on European or Western counties. Only a few studies determined SUI during pregnancy in Asia, especially in Southeast Asia. Wesnes et al. [17] presented questionnaire data from the Norwegian Mother and Child Cohort Study at the Norwegian Institute of Public Health and found that the most common type of UI was SUI in high prevalence in both nulliparous and multiparous women, 31 % and 42 %, respectively. Morkved and Bø [18]
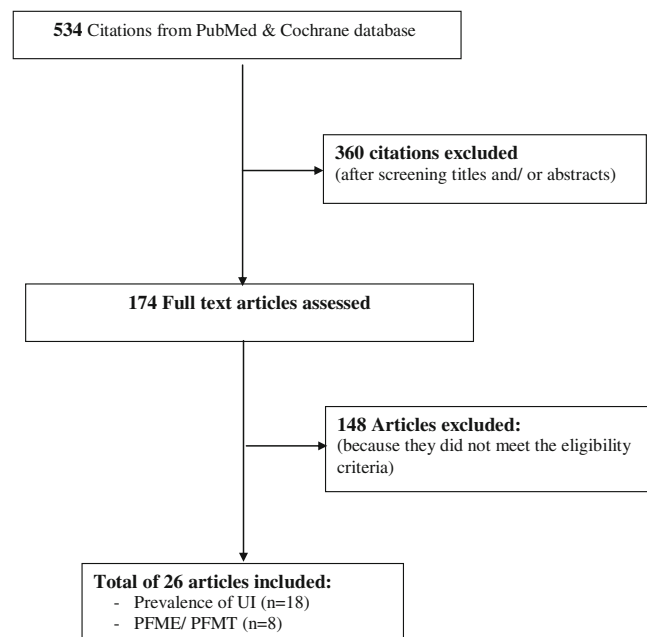

Fig. 1 Selection process for the review of stress urinary incontinence (SUI) prevalence and treatment during pregnancy

from Norway found the prevalence of SUI during pregnancy was 42 %. According to the same study, SUI was 38 % 8 weeks after delivery. Other studies from Europe (Table 1), UK [19], Spain [20], Scotland [21], Germany [22], and Denmark [23] found similarly high prevalence of SUI during pregnancy.

A large population survey conducted in China [24] found a little lower prevalence of UI compare with in Europe, but it was still high among the pregnancy population: 26.7 % of pregnant women presented with UI, including 18.6 % with SUI, 4.3 % with MUI, and 2.0 % with UUI. In Taiwan, there was some discrepancy. One study found a low incidence in primiparous women: a prevalence of SUI, MUI, and UUI of 26.7 %, 6.1 %, and 4.7 %, respectively [25]. Another study found a high prevalence: SUI, 46.1 % [26]. A study conducted in Jordan also found the prevalence of SUI as high as 45 % and UUI at 35 % [27]. In India, the prevalence of SUI, MUI, and UUI during pregnancy was 19.2 %, 3.8 %, and 2.9 %, respectively [28]. The highest reported prevalence of SUI was in the USA. Thomason et al. [29] reported up to 60 %, whereas Raza-Khan et al. [30] found 70 % and 75 % of UI prevalence in nulliparous and multiparous women, respectively, and 32 % pure SUI. A high prevalence was also reported South America [31]. Results reported from Australia were comparable with those in the USA: 64 % of women reported incontinence during pregnancy [32]. The prevalence of SUI and MUI were more common during pregnancy than was UUI alone: 36.9 %, 13.1 %, and 5.9 %, respectively [33]. In a multiethnic population in Norway, Bø et al. [34] conducted their investigation in the first trimester. They investigated the prevalence rate of SUI at 28 weeks of gestation> and found the lowest prevalence in women of African origin: 26 %. The prevalence in the Middle East population was 36 %, slightly higher than in women of African origin but lower than individual investigation. A prevalence of 40 % and 43 % was found in East and Southeast Asian women, which is higher than individual reports. The highest incidence was reported among women of European or North American origin: 45 %. Although the prevalence reported was different worldwide, the concordant feature was the increasing prevalence with gestational age [25, 26, 35]. The authors reported prevalence was highest in third trimester, followed by in the second and then first trimesters [25, 28, 36]. Only 13–19 % was reported in the first trimester [25, 37]. The second trimester was comparable at 19.2 %, but a significantly higher prevalence was found in third trimester: 37.5 % [25]. UI was higher with advance gestational age, as reported by Wijma et al. [36], who found the incidence of UI increased from 30 % at 28–32 weeks of gestation to 35 % at 36–38 weeks of gestation, which is similar to that reported by Kerrebroeck et al. [38]. The number of pregnant women with SUI varied according to this summary,

and the prevalence of SUI ranged from 18.6 % to 60 %, UUI from 2 % to 35 %, and MUI from 3.8 % to 13.1 %, increasing with gestational age. Many studies of the prevalence of UI are shown in Table 1.

Anatomy and physiology of the lower urinary tract during pregnancy

Normal anatomy and physiology of the lower urinary tract plays an important role in the background of the knowledge of the continence mechanism. Thorough knowledge of the anatomy and physiology provides the concept of the UI. The normal anatomy, the pelvic floor musculature complex, and its normal physiology are beyond the scope of the review.

Pregnancy has significant effect on lower urinary tract function. In uncomplicated pregnancy, micturition frequency is influenced by the physiologic state of the bladder. Frequency has been described as diurnal changes, which may be up to seven times or more of normal, and slight nocturnal changes of one or more times during the night. The incidence is the same in both primigravidae and multigravidae women [39]. The first trimester is the most common time of onset. The uterine weight is the most important factor affecting frequency throughout the pregnancy. Uterine weight not only exerts pressure on the bladder but also irritates the bladder. Normal bladder capacity in the first trimester is 410 ml [39]. In late pregnancy, descent of the presenting part of fetus has an additional effect on bladder irritation. Bladder capacity in the third trimester reduces to 272 ml in conjunction with increased irritability of detrusor muscles [39]. Alternative causes include nervous and hormonal influences. Indeed, the onset of frequency in late pregnancy is a common symptom of engagement of the fetal head [39]. Approximately 80 % of pregnant women, both primigravidae and multigravidae, experience increased micturition frequency at some time during pregnancy [39]. Increased frequency usually begins in early pregnancy but can occur in the later stage; it disappears in mid pregnancy, which may be due to the increase in bladder capacity to 460 ml in the second trimester, and returns in the later weeks. [39]. Once increased frequency has occurred, it is nearly always progressive and becomes increasingly worse until term [39]. Increased frequency during pregnancy results in polyuria and is associated with increased fluid intake. However, which is the cause and which the effect remains an unsolved issue. Average daily excretion, output, and fluid intake are highest in the second trimester and lowest in the third [39].

Uterine position is another issue that plays a significant role. The impacted retrovert gravid uterus causes fluid retention because it interferes with the obliteration of the posterior urethrovesical angle. It does not elevate the urethrovesical junction in the pelvis, nor does it elongate the urethra [39].

Table 1 Prevalence studies of urinary incontinence (UI), stress urinary incontinence (SUI), urgency urinary incontinence (UUI), and mixed urinary incontinence (MUI)

Study	City/country	Study design	Sample	Sample size	Prevalence of UI during pregnancy
Wesnes et al. 2007; [17]	Bergen, Norway	Cohort	Nulliparous and parous women at 30 weeks' gestation	43,279	SUI 31 % in nulliparous and 42 % in parous women
Morkved and Bo 1999; [18]	Norway	Retrospective	All women in a Norwegian community, delivering at the local hospital during a 1-year period	144	UI during pregnancy 42 %, and 8 weeks after delivery 38 %
Mason et al. 1999; [19]	UK	Prospective	Pregnant women who attended the antenatal clinic at two hospitals in northwest England. A postal questionnaire was sent to a sample of women when they reached 34 weeks' gestation and repeated at 8 weeks postpartum.	572	SUI 59 %
Diez-Itza et al. 2009; [20]	Guipúzcoa, Spain	Observational	Primigravidae women who came to give birth at Donostia Hospital	458	SUI 30.3 % in primigravidae women.
Whitford et al. 2007; [21]	Scotland, UK	Structured cross-sectional interview survey	Nulliparous and parous women over the age of 16 years and >30 weeks' gestation attending antenatal clinics in northeast Scotland.	289	SUI 54.3 % of pregnant women
Huebner et al. 2010; [22]	Berlin, Germany	Retrospective	Primigravidae women who delivered within 1 year (1999) at the Charité Hospital in Berlin	610	UI 26.3 % and significantly increased in the second half of pregnancy.
Viktrup et al. 1992; [23]	Denmark	Prospective	Primiparous women were interviewed repeatedly about SUI before and during pregnancy and after delivery	305	SUI before pregnancy 4 %, during pregnancy 32 %, after delivery 7 %
Zhu et al. 2012; [24]	Beijing, China	Prospective	Primiparous women from the seven regions of China ≥28 weeks' gestation	10,098	SUI 18.6 %, MUI 4.3 %, UUI 2.0 % in late pregnancy
Liang et al. 2012; [25]	Taoyuan, Taiwan	Observational cohort	Primiparous women who delivered at ≥ 36 gestational weeks were recruited in a tertiary hospital	1,501	SUI 26.7 %, MUI 6.1 %, UUI 4.7 %
Sun et al. 2005; [26]	Changhua, Taiwan	Cross-sectional survey	Nulliparous and multiparous women attending the antenatal clinic of a medical center in central Taiwan	799	SUI 46.1 %, significantly higher prevalence in multiparous than in nulliparous women
Al-Mehaisen et al. 2009; [27]	Irbid, Jordan	Prospective	Primiparous and multiparous women at least 36 weeks' gestation admitted in labor suite at three covering hospitals	181	SUI 46 %, UUI 35 % in the late third trimester
Sharma et al 2009; [28]	New Delhi, India	Questionnaire interview survey	Pregnant women >18 years who attended the antenatal clinic of unit III of AII India Institute of Medical Science	240	SUI 19.2 %, MUI 3.8 %, UUI 2.9 % during pregnancy
Thomason et al. 2007; [29]	Michigan, USA	Retrospective	Primiparous women who had one term vaginal delivery and no report of prepregnancy incontinence were recruited 6- to 9-months postpartum	121	SUI 60 %
Raza-Khan et al. 2006; [30]	St. Louis, MO, USA	Prospective	Nulliparous and multiparous women in third trimester receiving at Loyola University Medical Center	113	UI 70 % in nulliparous, 75 % in multiparous women, SUI 32 %
Martins et al. 2010; [31]	Sao Jose do Rio Preto, Brazil	Cross-sectional		500	SUI 46.1 % in primigravidae, 54.0 % in multigravidae women

Table 1 (continued)

Study	City/country	Study design	Sample	Sample size	Prevalence of UI during pregnancy
Chiarelli & Campbell 1997; [32]	New South Wales, Australia	Cross-sectional	Primigravidae and multigravidae women in third trimester who attended at 14 outpatient clinics in Sao Jose do Rio Preto	304	UI 64 %
Brown et al. 2010; [33]	Melbourne, Australia	Multicenter prospective cohort	Nulliparous at >18 years and <24 weeks' gestation who gave birth at six metropolitan public hospitals	1,507	SUI 36.9 %, MUI 13.1 %, UUI 5.9 %
Bø et al. 2012; [34]	Oslo, Norway.	Population-based cross-sectional	All pregnant women in first trimester at three administrative city districts attending the Child Health Clinics	722	UI at 28 weeks' gestation was 26 % for women of African origin, 36 % for women of Middle Eastern origin, 40 % for women of East-Asian origin, 43 % for women of South-Asian origin, and 45 % for women of European/North American origin

Pathophysiology of stress urinary incontinence during pregnancy

PFM weakness causes bladder-neck and urethral mobility, leading to urethral sphincter incompetence [40, 41]. When the pregnant woman coughs, sneezes, laughs, or moves, intra-abdominal pressure increases, and this pressure is transmitted to the bladder. When pressure inside the bladder is greater than urethral closure pressure, incorporated with weakness of the urethral sphincter, SUI is the result. Pregnancy is one of the main risk factors for the development of SUI in young women [42–44]. Studies in pregnant women with SUI have found significantly decreased PFM strength in incontinent pregnant women compared with continent pregnant women [45]. Furthermore, there is a significantly higher prevalence of SUI in pregnant women than in non-pregnant women [46–48]. Pregnancy may also be associated with reduced PFM strength, which can cause SUI. However, the exact causes of pregnancy-related SUI remain unclear [49]. Physiological changes during pregnancy may lead to reduced PFM strength and SUI development in pregnant women due to the following mechanism.

Trauma to the PFM due to maternal weight, the uterus, and the fetus

Physiological weight gain during pregnancy may lead to increased pressure on the PFM and bladder, which may result in greater urethral mobility [50, 51]. Furthermore, excess maternal weight gain may impair blood flow and innervations to the bladder and urethra [52]. Several studies show the association between obesity and SUI. Zhu et al. [24] reported that the risk of SUI increased with increasing prenatal BMI [odds ratio (OR = 1.037)]. Liang et al. [25] reported that women with a prepregnancy BMI >30 kg/m^2 were increased risk of developing SUI during pregnancy. Glazener et al. [53] found that women with UI first occurring during pregnancy had a BMI > 25 kg/m^2 [OR 1.68, 95% confidence interval (CI) 1.12–2.43]. Increased maternal weight correlated with increased intra-abdominal pressure during urodynamic assessments [54, 55]. In addition, increased BMI was not only associated with UI but with pelvic organ prolapse (POP) as well. Gyhagen et al [56] found that symptomatic POP increased 3 % with each unit increase of BMI (OR 1.03; 95 % CI 1.01–1.05). They also found that women with POP more frequently had UI and UI> for > 10 years compared with women without POP. Weakening of PFM is found more often in women with POP [57]. Obesity or high BMI before pregnancy is a potentially modifiable risk factor for SUI; weight reduction may be an effective treatment option [58]. Weight loss by behavioral change can significantly improve SUI [58].

The growing uterus and fetus weigh solely on PFM, which contributes to chronic stress on PFM throughout

pregnancy and results in PFM weakness. Sphincter strength and its supportive function of PFM are jeopardized [41, 47, 59]. Furthermore, multigravidity causes a decrease in PFM strength at a rate of 22–35 % beginning at a gestational age of 20 weeks and lasting until 6 weeks postpartum [40]. Hilde et al. [60], in a cross-sectional study of 300 nulliparous women at 18–22 weeks' gestational age showed that continent pregnant women had significantly higher PFM strength and endurance compared with incontinent pregnant women ($p=0.003$ and $p=0.01$, respectively).

Prenatal issues and childbirth can damage the pudendal nerve, caudal aspects of the levator ani muscle, fascial pelvic organ supports, and the external and internal anal sphincters [61, 62]. This damage reduces PFM strength and may lead to increased bladder-neck and urethral mobility [63, 64] and cause mobility of the urethrovesical junction [11]. Wijma et al. [65] found increased bladder-neck mobility from early pregnancy onward to near term. Jundt et al. [66] found that pregnant women with SUI had more a mobile bladder neck than continent pregnant women.

Collagen changes during pregnancy

Collagen changes included both tensile properties and number. Changes in tensile properties contribute to reduced functional support of PFM, and reduced total collagen content may result in joint laxity and stretching of pelvic ligaments [67]. Several studies have reported a decrease in the total collagen content in women with SUI [63, 68]. Keane et al. [63] showed that nulliparous women with SUI had significantly less collagen content in their tissues than did continence pregnant women ($p<0.0001$). They also concluded that the etiology of SUI in these pregnant women appears due to both quantitative and qualitative reduction in collagen.

Chen et al [69] showed the extracellular matrix (ECM) component is related to increased elastase activity in vaginal tissues obtained from SUI patients. In addition, the breakdown of collagen, smooth-muscle inhibition, and inflammatory processes play an important role in contributing to SUI. Lin et al. found the relevant genes that influence cellular signaling pathways, including matrix metalloproteinases (MMPs), regulator of G-protein signaling 2 (RGS2), and SMAD2 [70]. Skorupski et al. also found more expression of 1G/2G MMP-1 and the 5A/6A MMP-3 promoter single nucleotide polymorphisms (SNPs) in women with POP than in the population with a normal PFM [71]. Genetic and environmental factors contribute to the occurrence of SUI and POP. A study of twins by Altman et al [72] on SUI and POP analyzed 3,376 monozygotic and 5,067 dizygotic female twins and found a greater similarity in outcomes in monozygotic twins. In a study of nulliparous women, Dietz et al. [73] demonstrated the heritability of bladder-neck

mobility. The systematic review by Lince et al. [74] found a substantially greater likelihood of SUI in family members with women with POP compares with women without POP, indicating that genetic predisposition play an important role in the development of POP.

Hormonal changes during pregnancy

Changes in relaxin and progesterone levels during pregnancy may have a significant role in the development of SUI [75]. Relaxin, which plays an important role in maintaining urinary continence during pregnancy [76], could stimulate tissue growth in the lower urinary tract and increase urethral pressure. There is a marked increase in relaxin concentration to peak at a gestational age of 10–14 weeks and then decrease to a stable level of approximately half the peak value at the 17th–24th week of pregnancy, resulting in decreased growth of the urethral epithelium [77, 78]. This may lead to a decrease in urethral pressure [67]. As pregnant women with SUI have lower urethral pressure than continent pregnant women [79], lower relaxin concentrations in late pregnancy, therefore, correlate with a higher prevalence of SUI at the second and third trimesters [76].

Progesterone increases during pregnancy from 24 ng/ml at the 8th week to 150 ng/ml at the 36th week [80]. Increased progesterone may relax smooth muscles in the urinary system [81], result in reduced ureter, bladder, and urethral tone [82]. Values urethral pressure profile parameters below the median value and defective transmission of pressure over the urethra were observed in almost all pregnant women who experienced SUI during pregnancy. These observations suggest that an inherent weakness of the urethral sphincter mechanism plays a key role in the pathogenesis of SUI [80]. However, alterations in hormone levels during pregnancy were not correlated with changes in urethral pressure profile measurements [80].

The expansion of the uterus and fetal weight

Two major factors—expansion of the uterus and increment increase in fetal weight with gestational age, especially at the third trimester—influence the incontinence mechanism. They put direct pressure on the bladder, which may lead to changing the bladder-neck position [66] and reducing bladder capacity, contributing to bladder pressure that exceeds urethral pressure [65, 83]. This results in urine leakage.

In summary, commonly encountered prenatal physiological changes such as increasing pressure of the growing uterus and fetal weight on PFM throughout pregnancy, together with pregnancy-related hormonal changes in progesterone, estrogen, and relaxin, may lead to reduced strength and supportive and sphincteric function of PFM. PFM muscle weakness causes bladder-neck and urethral

mobility, leading to urethral sphincter incompetence. Hence, when intra-abdominal pressure is increased with coughing, sneezing, laughing, or moving, the pressure inside the bladder becomes greater than the urethral closure pressure and the urethral sphincter is not strong enough to maintain urethral closure. Urinary leakage will be the result. In particular, SUI is common during pregnancy and puerperium. After delivery, SUI symptoms resolve in the vast majority of cases [84]. The healing process may take some time after the delivery [85], but in a significant percentage of women, it can persist in subsequent stages of life [84]. In primiparae women, SUI symptoms tend to resolve within 3 month after delivery [86].

The relevant literature reports that the prevalence of UI decreases in the postpartum period [85, 87]. In general, UI can affect 27.4 % of parous women for 6 months postpartum, with only a small portion of women experiencing spontaneous resolution [88]. Thomason et al. [29] found that only 8 % of pregnant women who developed UI during pregnancy had resolution, but in 47 % of those who had UI, incontinence had not resolved at 6 months postpartum. Glazener et al. [53] found that 13.5 % of primigravid women had SUI 3 months after delivery, whereas Wesnes et al. [55] showed an SUI rate around 14 % at 6 months after delivery. Groutz et al. [89] found that 1 year after the first spontaneous vaginal delivery, 10.3 % of women had SUI.

Despite this clear association, the mechanism involved in SUI remains unclear. Similarly, the pathophysiology of the development of SUI during pregnancy is not clearly understood. However, it has been suggested that it could be caused by both hormonal and mechanical changes that occur during this period [90, 91]. After pregnancy, most women recover their prepregnancy hormonal levels, and pressure of the enlarged uterus on the bladder and PFM resolves. Postnatal remission of SUI may be explained by the resolution of hormonal and metabolic changes associated with pregnancy and spontaneous healing of traumatic lesions due to vaginal childbirth [92]. The compensation may be due to PFM, which play a urethral support role, and it may be aided by pelvic floor training [92]. Improvement in PFM strength in women during the postpartum period showed that pregnancy, with its hormonal and mechanic effects, is a very important risk factor for UI during pregnancy [87].

Treatment for stress urinary incontinence during pregnancy

During pregnancy and postpartum, conservative therapy or perineal rehabilitation by PFME is the first-line intervention to treat SUI [35]. Pregnant women are often instructed to perform PFME to prevent the development and treat symptoms of SUI during pregnancy [93]. Other treatments, whether

medical or surgical, must not be proposed at as the first intervention during pregnancy or during the immediate postpartum period [35].

Pelvic floor muscle exercise

PFME, the repetitively selective voluntary contraction and relaxation of specific PFM [1], also known as Kegel exercises, are the most popular method of treatment for SUI. In the late 1940s, an American gynecologist named Arnold Kegel first introduced this method for treating postpartum UI and improving the function and tone of PFM following childbirth. PFME have been successful because postpartum women who have performed these exercises have reported cure rates as high as 84 % and improvement rates as high as 100 % [94]. PFME is the most commonly recommended conservative therapy for pregnant women with SUI. PFME under the direction of a therapist (re-educator or midwife) reduces the prevalence of UI in the short term compared with simple advice about individual PFME [35]. Many investigators have assessed the effectiveness of PFME, as shown in Table 2.

PFME is used to strengthen the pelvic floor and periurethral muscles to improve the continence mechanism [44, 87, 95]. As a result, this method can either cure or relieve SUI by reducing incontinence severity, including frequency and volume of urine leakage [45, 96]. PFME prescribed during pregnancy improves pregnancy UI and reduces the prevalence of UI in late pregnancy and early postpartum [35, 97]. Dinc et al. [87] found that pregnant women with PFME had a significant decrease in UI episodes during pregnancy and in the postpartum period. Undoubtedly, their PFM strength increased to a larger extent. Sangsawang and Serisathien [96] investigated the effects of a PFME on the severity of SUI in 66 pregnant women who had SUI at gestational ages of 20–30 weeks. After the PFME program, women in the experimental group had significantly reduced frequency, volume of urine leakage, and score of perceived SUI severity compared with those in the control group. They concluded that the 6-week PFME program was able to decrease symptom severity in pregnant women with SUI. Sampselle et al. [98] studied the effects of PFME on SUI and PFM strength during pregnancy and postpartum in 72 primigravid women with gestational ages of 20 weeks. This study suggested that PFME can reduce the incidence of SUI at gestational ages of 35 weeks and at 6 weeks and 6 months postpartum by significantly increasing PFM strength compared with women who did not perform PFME. Morkved et al. [99] found that pregnant women who performed PFME reported 32 % UI at 36 weeks of gestational ages compared to 48 % in the control group ($p=.007$). The number of urinary leakage episodes at gestational age 36 weeks was also significantly lower in the experimental group ($p=.014$).

Table 2 Details of studies of pelvic floor muscle exercise (PFME) to prevent and treat stress urinary incontinence (SUI) during pregnancy and the postpartum period

Study	Study design	Sample	Sample size	Intervention	Timing and method of assessment	Findings
Dinc et al. 2009; [87]	Prospective, randomized trial	Pregnant women with UI at gestational age 20–34 weeks	80	Intervention group:3 sets of PFME, each including contracting and relaxing PFME repeated 10 times; control group: usual care	Urine leakage episodes, number of voidings, pad test and examiner-assessed PFM strength at 36–38 weeks' gestational age and 6 to 8 weeks postpartum	Decrease in the number of UI episodes, amount of urine in the pad test during pregnancy in the intervention group; PFM strength increased to a larger extent.
Sangsawang and Serisathien 2012; [96]	Quasiexperimental with comparison group	Pregnant women with SUI at gestational age 20–30 weeks	66	Intervention group: 6-week PFME program with weekly training session led by a nurse; control group: usual care	Severity of SUI: frequency, amount of urine leakage, and VAS score of perceived SUI severity after the intervention period	Lower frequency, amount of urine leakage, and score of perceived SUI in the intervention group
Sampselle et al. 1998; [98]	Prospective randomized trial	Primigravid women with US at gestational age 20 weeks	72	Intervention group: standardized instruction in PFME; control group: usual care	UI symptoms by questionnaire, PFM strength by speculum at 20 and 35 weeks' gestation and 6 weeks, 6 months, and 12 months postpartum	Decreased UI symptoms in the intervention group at 35 weeks' gestation (p=.043), 6 weeks postpartum (p=.032), and 6 months postpartum (p=.044); greater PFM strength in the intervention group at 6 weeks and at 6 months postpartum
Morkved et al. 2003; [99]	Single-blind RCT	Nulliparous women with UI at gestational age 18 weeks	301	Intervention group: 12-week intensive PFMT with weekly training session led by a physiotherapist; control group: usual care	Self-report of UI and examiner-assessed PFM strength at 36 weeks' gestational age and 3 months postpartum	At 36 weeks' gestational age: prevalence of UI 32 % in the intervention group; 48 % in the control group (p=.007); lower number of urinary leakage in the intervention group (p=.014); higher PFM strength in the intervention group (p=.008) and 3 months after delivery (p=.048)
Ko et al. 2011; [100]	RCT	Nulliparous women with UI at gestational ages 16–24 weeks	300	Intervention group: 12- week intensive PFME with weekly training session for 45 min led by a physiotherapist; control group: usual care	Urinary symptoms measured by UDI-6, IIQ-7, and self-reported UI	Lower total UDI-6, IIQ-7 scores and self-report rate of UI in the intervention group during late pregnancy and postpartum period
Stafne et al. 2012; [101]	RCT	Pregnant women with UI at gestational age 20 weeks	855	Intervention group: 12-week PFMT, conducted between 20 and 36 weeks of gestation, with one weekly training session led by a physiotherapist and home exercises at least twice a week.; control group: usual care	Self-reported UI and FI after the intervention period	Fewer reported any weekly UI and FI in the intervention group (p=0.004 and p=0.18, respectively)
Glazener et al. 2001; [102]	RCT	Women with UI 3 months after childbirth	747	Intervention group: advice on PFME and bladder training at 5, 7, and 9 months after delivery by nurses; control group: usual care	Persistence and severity of UI at 12 months after delivery, performance of PFME, FI, well-being, anxiety, and depression	Lower UI in the intervention group of both UI and severe UI (p<0.037 and p<0.002, respectively). Lower severity of UI and less FI in intervention group (p=.007 and p=0.012, respectively).
Reilly et al. 2002; [103]	Single-blind RCT	Primigravid women with UI at gestational age 20 weeks with bladder-neck mobility	268	Intervention group: antenatal PFME at a 20 weeks' gestation until delivery, with monthly training session led by a physiotherapist; control group: usual care	Self-report of UI, pad test, and examiner-assessed PFM strength and bladder-neck mobility at 3 months postpartum	Prevalence of UI at 3 months: 19.2 % in intervention group; 32.7 % in control group (p=0.023). No difference in pad test, PFM strength, and bladder-neck mobility

RCT randomized controlled trial; *UI* urinary incontinence; *SUI* stress urinary incontinence; *FI* fecal incontinence; *PFME* pelvic floor muscle exercise; *PFMT* pelvic floor muscle training; *PFM* pelvic floor muscle; *VAS* visual analog scale; *UDI-6* Urogenital Distress Inventory-6; *IIQ-7* Incontinence Impact Questionnaire-7

Furthermore, PFM strength was significantly higher in women with PFME at gestational age 36 weeks and 3 months after delivery than in the control group ($p=.008$ and $p=.048$, respectively). During late pregnancy and the postpartum period, Ko et al. [100] found that women in the PFME group had significantly lower total Urogenital Distress Inventory-6 (UDI-6) and Incontinence Impact Questionnaire-7 (IIQ-7) scores; their self-report rate of UI was also less than in the control group. This study concluded that PFME applied in pregnancy was effective in treating and preventing UI during pregnancy, and this effect may persist to postpartum period.

Stafne et al. [101] assessed the effect of a 12-week pelvic floor muscle training (PFMT) to reduce self-reported UI and fecal incontinence (FI) in late pregnancy in 855 women. After the intervention, the authors found that fewer women in the intervention group reported any weekly UI and FI (11 % vs. 19 %, $P=0.004$ and 3 % vs. 5 %, $P=0.18$, respectively). They indicated that pregnant women who practice PFMT can prevent and treat UI in late pregnancy. Glazener et al. [102] studied the effects of PFME on the severity of UI at 12 months after delivery in 747 mothers who still had UI 3 months postpartum. They found that mothers in the intervention group had significantly less UI and severe UI than the control group (59.9 % vs 69.0 %, $p<0.037$ and 19.7 % vs 31.8 %, $p<0.002$, respectively). FI was also less common in the intervention group than the control group (4.4 % vs 10.5 %, $p=0.012$). The authors concluded that PFMT provided by nurses seems to reduce UI and FI that persists for 12 months postpartum. Reilly et al. [103] studied the effects of antenatal PFME at a gestational age of 20 weeks until delivery for the prevention of postpartum SUI in 268 primigravid women with bladder-neck mobility. The authors suggested that antenatal PFME are effective in reducing the risk of postpartum SUI in primigravid women with bladder-neck mobility at 3 months postpartum. However, there were no changes in bladder-neck mobility and no difference in PFM strength between groups after exercise.

PFME is a specific exercise for PFM and is different from exercise of other muscles in the body. Thus, PFME requires a strong commitment from women. Moreover, this form of exercise requires dedication, endurance, and effort on the part of women in order to result in effective SUI treatment [104].

Women without symptoms of SUI may not want to invest their time in PFME [93]. Women with moderate to severe symptoms of SUI may achieve more improvement in their incontinence than women with mild symptoms. Alewijnse et al. [105] found that women with severe symptoms were more likely to maintain a higher adherence to PFME than women with mild to moderate symptoms. Burns et al. [106] argued that women with mild symptoms may not perceive their incontinence as a serious problem, whereas patients with severe symptoms recognized the incontinence as an ongoing problem and view adherence to exercise as positive training to affect a cure. Findings of that study are in accordance with the study of Sangsawang and Serisathien [96], who found that more than half (54.3 %) and nearly half (45.7 %) of pregnant women in the intervention group had severe and moderate symptoms of SUI, respectively, before participating in the program. Thus, pregnant women may have perceived their incontinence symptoms as a serious problem that could affect their QoL. After completing the program at the sixth week, 100 % of pregnant women in the intervention group were able to perform PFME every day during participated in the program [96]. Women with SUI symptoms were encouraged to perform this exercise continually, resulting in alleviation of SUI symptoms and contributing to the continence mechanism. Morkved et al. [99] found that 19 % of pregnant women had some noncompliance (i.e., skipping classes) in the training schedules of a 12-week antepartum PFME, which is a high rate compared with compliance in drug trials of similar duration. Goode et al. [107] found that 11 % of women dropped out of the 8-week PFME class because the treatment required too much effort and time. In a 15-year follow-up, long-term adherence to PFME was found to be low, with no difference between intensive and home training programs [108].

In summary, PFME is the first-line conservative treatment for women with SUI before consideration of other treatments [97]. PFME is more a effective treatment for SUI during pregnancy because it is a safe treatment without complications, inexpensive, simple to perform, does not require instruments, and can be done anywhere and anytime [97, 109].

Conclusions

The most common type of UI in pregnant women is SUI. The number of pregnant women with SUI in our literature review was variable, with prevalence ranging from 18.6 % to 75 % [16–32, 34] and increasing with gestational age. The increasing pressure of the growing uterus and fetal weight on PFM throughout pregnancy, together with pregnancy-related hormonal changes, may lead to reduced strength of the supportive and sphincteric function of PFM. PFM weakness causes bladder-neck and urethral mobility, thus leading to urethral sphincter incompetence. Hence, when intra-abdominal pressure increases, pressure inside the bladder becomes greater than the urethral closure pressure, and the urethral sphincter is not strong enough to close the urethra, resulting in urine leakage. PFME is an effective treatment for SUI during pregnancy and has no significant adverse effects. Continence can be improved when incontinent pregnant women adequately perform PFME.

Conflicts of interest None.

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