

Female urinary incontinence during pregnancy and after delivery: Clinical impact and contributing factors

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Abstract

Urinary incontinence (UI) is a common condition affecting adult women of all ages and it could have a negative influence on quality of life. The etiology of UI is multifactorial, but some of the most important risk factors are obesity and ageing, as well as adverse obstetric events. Pregnancy and delivery *per se* have been implicated in the etiology of UI. Although several studies have demonstrated a direct association between UI and vaginal delivery in short, medium and long-term, the role of childbirth on the risk of UI remains controversial. The mechanical strain during delivery may induce injuries to the muscle, connective and neural structures. Vaginal birth can be associated with relaxation or disruption of fascial and ligamentous supports of pelvic organs. Parity, instrumental delivery, prolonged labor and increased birth weights have always been considered risk factors for pelvic floor injury. Also genetic factors have been recently raised up but still there are not appropriate guidelines or measures to reduce

significantly the incidence of UI. The role of pelvic floor muscle training (PFMT) in the prevention and treatment of UI is still unclear. However, PFMT seems to be useful when supervised training is conducted and it could be incorporated as a routine part of women's exercise programmes during pregnancy and after childbirth.

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Key words: Urinary incontinence; Pregnancy; Delivery; Cesarean section; Forceps; Episiotomy; Obesity; Collagen; Pelvic floor muscle training

Core tip: The mechanical strain during delivery may induce injuries to the muscle, connective and neural structures. Vaginal birth can be associated with relaxation or disruption of fascial and ligamentous supports of pelvic organs. Parity, instrumental delivery, prolonged labor and increased birthweight have always been considered risk factors for pelvic floor injury. Also genetic factors have been recently raised up but still there are not appropriate guidelines or measures to reduce significantly the incidence of urinary incontinence.

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INTRODUCTION

Pelvic floor dysfunctions include a wide range of anatomic and functional disorders (*e.g.*, hypo-function: urinary incontinence, fecal incontinence and pelvic organ prolapse; hyper-function: defecatory dysfunction, sexual

dysfunction and voiding dysfunction). In women over 45 years old, prevalence of urinary (urge or stress) incontinence ranges from 17% to 45%, anal (faecal or flatus) incontinence (AI) from 0.5% to 17%, and urogenital prolapse between 20% to 30%. These prevalences could be therefore underestimated since most of the epidemiological investigations are obtained by self-report which could confound the real incidence of pelvic floor dysfunctions^[1-4] (Table 1).

Epidemiological evidences demonstrate that adverse obstetric events are related to pelvic floor dysfunction. Lower urinary tract symptoms (LUTSs) during pregnancy and postpartum have been associated to physiological and anatomical changes of pregnancy. LUTSs were present in 63.8% of Brazilian pregnant women, and the main risk factors were multiparity and pre-pregnancy LUTSs, smoking, constipation, and regular coffee consumption^[5]. However, those symptoms are transient and disappear some months after delivery and they do not request further investigations.

According to the most recent definition of the International Continence Society (ICS), urinary incontinence (UI) is defined as “the complaint of any involuntary leakage of urine”. Although UI is not a life threatening status, it is a common, annoying and expensive condition, and it deeply affects a woman’s quality of life^[6]. In a subset of population, UI during pregnancy has been reported to be 19.9% among nulliparous and 24.1% among primiparous women^[7]. In the last years, clinicians and health researchers have extensively investigated the factors influencing prevalence of UI in order to ameliorate the management and treatment of affected patients. However, accurate prevalence data are difficult to obtain from the literature since noteworthy differences among the studies in terms of methodologies^[8,9]. The fact that UI is more common in women than in men indicates the contribution of factors such as pregnancy to UI.

PREVALENCE OF UI DURING PREGNANCY AND AFTER DELIVERY

It is well known that, during pregnancy, women can experience urogynaecological problems which includes not only urinary incontinence, but also urinary tract infection, filling and voiding disorders, pelvic organ prolapse and AI^[10]. The development of these conditions determinate physiological changes that occur in pregnancy but it can be also linked to previous pregnancies. In fact, during pregnancy, supporting structures are supposed to be overloaded due to the fetus weight and the progressive growth of the uterus, both in weight and size^[11,12]. Additionally, pregnant uterus increases the angle between the bladder neck and urethra, which can participate to urinary symptoms. Hormonal changes due to pregnancy can also cause changes in tissue, in the support and in the continence mechanism^[7,13].

During pregnancy and after delivery, both UI and AI are frequent complaints. The cumulative incidence during

Table 1 Pelvic floor dysfunctions

Hypo-function	Hyper-function
Urinary incontinence	Voiding dysfunction
Fecal incontinence	Defecatory dysfunction
Pelvic organ prolapse	Sexual dysfunction

pregnancy was 39.1% and 10.3%, respectively^[14]. According to a recent systematic review, it has been reported a prevalence of UI at the first trimester of 8.3%, at the second of 31.8% and at the third of 34.8%^[14]. The most prevalent type of UI is the stress urinary incontinence (SUI), affecting up to 79.2% at the third trimester which is prompted by a physical movement or activity, such as coughing, sneezing, running or heavy lifting, that puts pressure (stress) on the bladder^[15,16]. In a systematic review of population-based studies, in order to investigate the prevalence of UI within the first year postpartum, during the first 3 mo postpartum, the pooled prevalence of any postpartum incontinence was 33% in all women. The mean prevalence was double in the vaginal delivery group (31%) compared to the cesarean section group (15%)^[17].

RISK FACTORS OF DEVELOPING UI DURING PREGNANCY AND AFTER DELIVERY

Risk factors of developing UI after delivery have been related to the characteristics of mother and baby themselves. Pregnancy per se has been reported to be a risk factor for postpartum UI especially if the incontinence started during the first trimester^[18,19]. That is supported by the increase in the rate of SUI with an increased number of abortions, which suggests that pregnancy by itself may have promoting effects on UI.

It has been found that the main risk factors of UI in pregnancy are maternal age more than 35 years^[20], pregestational maternal body mass index (BMI) and family history of UI^[16]. The prevalence of UI increases with maternal age and there is an annual increase in UI prevalence of 3% per year^[21]. Moreover, the first delivery is considered to exert the greatest increase in risk for UI, even if subsequent deliveries contribute to a further increase in the risk of UI^[22].

The relationship between maternal weight and subsequent development of incontinence has been diffusely investigated. Higher pregestational BMI is known to be associated with postpartum UI^[23]. It has been demonstrated an increased risk of UI (8%) proportionally to the increase of BMI unit, and this risk is not related to the type of delivery, vaginally or by caesarian section^[21]. Pregnant women at term with body weight equal or more than 75 kg appear to double the risk of SUI^[24]. UI during pregnancy and still persistent at 3 mo is usually associated to women with higher BMI^[25]. The Norwegian Mother and Child Cohort Study studied 12679 primiparous

women, continent before pregnancy, at weeks 15 and 30 of pregnancy and 6 mo postpartum. Weight gain greater than the 50th percentile weeks 0-15 was weakly related with higher incidence of UI at week 30 when compared with weight gain less than or equal to the 50th percentile. In addition, weight increase greater than 50th percentile during pregnancy was not associated with higher incidence of UI, 6 mo postpartum^[26].

Most of the interest is given to the impact of obstetric factors on UI after childbirth. In women who delivered vaginally, the risk of incontinence increases with increasing fetal birthweight (especially for children with birthweight ≥ 4000 g^[25,27,28]), and probably in women who received oxytocin^[28]. Shoulder dystocia and associated obstetrical maneuvers for its relief have not detrimental effects on perineum and do not increase UI incidence after delivery^[29]. The vaginal delivery of two successive fetuses does not seem to be a cause of SUI as compared to cesarean, although its rate was higher in the “twin” group (40%) than in the “singleton” group (20%) which appears to be related to total intrauterine weight^[30].

Another important contributing factor in developing UI after delivery is the presence of urinary leakage before pregnancy^[23]. Thus, previous UI was a significant risk factor for period prevalent UI during pregnancy, explaining 34% and 83% of pregnancy UI for nulliparous and primiparous, respectively^[7]. In addition, in nulliparous women prepregnancy UI is a strong herald for the increased prevalence of UI 4-12 years postpartum^[21].

Cesarean section seemed to be followed by less postnatal UI than vaginal delivery^[28,31]. It has been found that the risk is 67%-71% higher after vaginal delivery than after caesarean section^[21], but this advantage given by cesarean delivery seems to disappear after the second cesarean section^[16]. A systematic review reported that cesarean section reduced the risk of postpartum SUI from 16% to 10%, and the number needed to prevent SUI is 15 in 6 cross-sectional studies. In the same report, from the analysis of 12 cohort studies, the incidence of SUI in cesarean section patient decreased from 22% to 10% and the number needed to prevent SUI was 10%^[31]. Nevertheless, a prospective multicenter study do not show a significant difference of risk for bothersome UI between women delivered by one or more vaginal deliveries and women delivered by one or more caesarean sections^[32].

Concerning the type of cesarean sections, it has been found no difference in the prevalence of UI, or UI persisting for more than 10 years^[21], between women delivered by acute or elective caesarean section (elective caesarean is defined as the caesarean section performed before the onset of labour, while caesarean section performed during labour are denoted as acute caesarean sections). That one indicates that it is the later stages of delivery, when the fetus passes through the pelvic floor that leads to the increased risk of UI. However, it should be clear that one has to perform eight or nine caesarean sections to avoid one case of UI. Moreover, operative delivery by caesarean section also involves a degree of

risk for morbidity and mortality over and above that of vaginal delivery.

One area of considerable controversy is the role of episiotomy and spontaneous perineal lacerations^[33]. Almost 80 years ago, episiotomy was proposed as a strategy to prevent spontaneous lacerations and to thereby reduce “pelvic relaxation”. However, more recent studies have suggested that episiotomy may increase the odds of pelvic floor disorders. Thus, the role of episiotomy is uncertain. Episiotomy is not significantly associated with any of the pelvic floor disorders considered. In contrast, women who had experienced multiple spontaneous perineal lacerations were significantly more likely to have prolapse to or beyond the hymen, and were significantly less likely to have overactive bladder (OAB)^[34]. In 2005, a systematic review concluded that the effect of episiotomy on the development of pelvic floor disorders remains unknown^[35].

Also controversial is the association between operative delivery and pelvic floor disorders. While some research has suggested that operative delivery substantially increases the odds for pelvic floor disorders^[23,28,36], other research suggests that operative birth is not a strong predictor of urinary incontinence^[19,37-39] or pelvic organ prolapse^[40]. Recently, it has been demonstrated that women with at least one forceps delivery are more likely to report stress incontinence, OAB, AI, prolapse symptoms and prolapse to or beyond the hymen on examination, although this association is statistically significant only for OAB^[34]. Preventing obstetric trauma needs changes in current obstetric practice: reduction in the episiotomy rate, use of vacuum extractor in preference to forceps.

Anal sphincter injuries showed a significantly higher risk of fecal incontinence 10 wk after delivery in women with these injuries, as well as in women with a second-stage labor of more than 50 min^[41].

MOLECULAR AND GENETIC MECHANISMS

Pelvic floor disorders, such as SUI and pelvic organ prolapse, may have common pathophysiological processes related to pelvic floor tissue laxity and loss of support. Those changes could be relevant most of all during period of important modifications, such as pregnancy and delivery. However, the molecular mechanisms responsible for tissue changes in UI are poorly understood yet. Lin *et al.*^[42] have studied 22000 genes from the urethral tissue of a parturition-induced stress urinary incontinence (SUI) rat model. The expression of 42 urethral genes was different between continent and incontinent rats. Genes important in inflammation, collagen breakdown, and smooth muscle inhibition are upregulated in the urethras from rats with parturition-related incontinence.

Using the same model, muscle, collagen I / III and reticular fibers in the urethra of SUI rats were also significantly decreased, besides fragmentation and disorganization. Transforming growth factor (TGF) beta 1, metal-

loproteinase (MMP) 9, and phosphorylated Smad2 were expressed significantly higher in parturition-associated SUI than in continent rats^[43]. Birth appears to activate elastin expression by TGF-beta 1 signals while estrogen interferes with this mechanism, resulting in improper assembly of elastic fibers. The TGF-beta family also contributes in the regulation of myometrial activation at term integrating mechanical and endocrine signals for successful labor contraction^[44].

In humans, there is a three-fold prevalence of SUI among first-degree relatives of female patients with SUI^[45] which suggests a genetic factor involved in the predisposition of connective tissue injury. Allen-Brady *et al*^[46] have studied the relationship between predisposing gene and pelvic floor disorders, including UI, on chromosome 9q21. In a large sample of twins, it has been demonstrated a genetic component for the aetiology of SUI, although environmental factors equally contributed to variation liability^[47]. Further research into the genetic basis of UI may provide a comprehensive understanding of the biological basis of the disorder.

PREVENTION OF URINARY INCONTINENCE DURING PREGNANCY AND AFTER DELIVERY

Most pregnant women had no information about pelvic floor, and a little number of women could only localize to the region. Several studies have demonstrated that antenatal pelvic floor muscle training (PFMT) taught in a general exercise class, during pregnancy, could be helpful in the prevention of postpartum UI in primiparous women without UI during pregnancy^[48]. The utility of regular pelvic floor muscle exercises is due to the ability of the muscles that support the pelvic organs, to become stronger and to help to use the muscles more effectively. Pregnant and postpartum women who do PFMT have significantly less urine leakage^[49], even if PFMT does not affect labor and birth outcomes or complication rates^[50,51]. In addition, PFMT applied in pregnancy is effective in the treatment and prevention of urinary incontinence during pregnancy, and this effect may persist to postpartum period^[50,52].

On the other hand, an Australian prospective randomised controlled trial, among women 3 mo after delivery, has compared women which had PFMT or a usual postpartum care^[27]. At 3 mo after delivery, the prevalence of UI was respectively 31% and 38%. After one year, there was no significant difference in continence status between both groups^[53]. Even if there was no significant difference in continence status, women in the intervention group were more motivated than those in the control group in practicing pelvic floor exercises at adequate frequencies.

In conclusion, there are no specific techniques or treatments to prevent the development of postpartum UI but there are correct behaviors to follow that severely

limit the incidence of postpartum UI. A good management of pre-pregnancy BMI and weight gain during pregnancy, proper management especially of the second and third stage of labor, fetal weight < 4.0 kg, and finally a good awareness of their own pelvic floor and its training to strengthen those muscles, constitute the elements on which we can work to significantly reduce the possibility of developing UI after pregnancy.

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