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Review Article

Pelvic floor dysfunction, and effects of pregnancy and mode of delivery on pelvic floor

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A B S T R A C T

Pelvic floor dysfunction (PFD), although seems to be simple, is a complex process that develops secondary to multifactorial factors. The incidence of PFD is increasing with increasing life expectancy. PFD is a term that refers to a broad range of clinical scenarios, including lower urinary tract excretory and defecation disorders, such as urinary and anal incontinence, overactive bladder, and pelvic organ prolapse, as well as sexual disorders. It is a financial burden on the health care system and disrupts women's quality of life. Strategies applied to decrease PFD are focused on the course of pregnancy, mode and management of delivery, and pelvic exercise methods. Many studies in the literature define traumatic birth, usage of forceps, length of the second stage of delivery, and sphincter damage as modifiable risk factors for PFD. Maternal age, fetal position, and fetal head circumference are nonmodifiable risk factors. Although numerous studies show that vaginal delivery affects pelvic floor structures and their functions in a negative way, there is not enough scientific evidence to recommend elective cesarean delivery in order to prevent development of PFD. PFD is a heterogeneous pathological condition, and the effects of pregnancy, vaginal delivery, cesarean delivery, and possible risk factors of PFD may be different from each other. Observational studies have identified certain obstetrical exposures as risk factors for pelvic floor disorders. These factors often coexist; therefore, the isolated effects of these variables on the pelvic floor are difficult to study. The routine use of episiotomy for many years in order to prevent PFD is not recommended anymore; episiotomy should be used in selected cases, and the mediolateral procedures should be used if needed.

Epidemiology and risk factors

Pelvic floor dysfunction (PFD) occurring in women comprises a broad range of clinical scenarios such as lower urinary tract excretory and defecation disorders, including urinary and anal incontinence, overactive bladder (OAB), and pelvic organ prolapse (POP), as well as sexual disorders [1]. In developing countries, the prevalence of POP, urinary incontinence (UI), and fecal incontinence (FI) is 19.7%, 28.7%, and 6.9%, respectively. POP is a major health problem for both developing and developed countries [2]. In the Women's Health Initiative study, varying degrees of POP were observed in 41% of women in the age range of 50–79 years [3]. Despite there being a large number of cross-sectional studies, unfortunately, the number of large-scale longitudinal and prospective studies on the true incidence of PFD is very limited. Nevertheless, there is a consensus that PFD is a major health issue for aging women. In the United States, about 400,000 surgeries are performed every year for UI only. DeLancey [1] stressed this as a hidden epidemic, and has drawn attention to the role of assisted vaginal delivery (VD) by means of episiotomy in the prevention of perineal disease and UI. Age, ethnicity, multiparity, mode of delivery, history of pelvic surgery, pregnancy, chronic cough, obesity, spinal cord disorders, family history, and genetics are among the most common identifiable risk factors for the development of PFD [4]. Reported pregnancy-related risk factors include pregestational body mass index (BMI), BMI at term, weight gain, smoking during pregnancy, duration of the first and second stages of labor, spontaneous or operative delivery, perineal lacerations, weight of the newborn, maneuvers and episiotomy, as well epidural analgesia. The other risk factors that have been reported include past histories

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of previous lower abdominal surgeries such as laparoscopic and hysteroscopic procedures, uterine curettage, and UI surgeries [5].

Impact of levator ani injury, VD, and operative delivery on the pelvic floor

The prevalence of PFD increases significantly with age. Approximately 10% of women between the ages of 20 years and 39 years, compared with 50% of women aged ≥ 80 years, suffer from at least one PFD disorder [6]. The loss of strength of connective tissues may induce PFD formation as a result of hormonal changes, particularly estrogen deficiency related to advancing age and duration of postmenopausal state. However, in the same age group, the prevalence of PFD is more common in multiparous women than in nulliparous women, which again stresses the role of obstetric trauma.

Recently, a relatively large number of studies have been conducted to emphasize the role of levator ani muscle injury (LAMI) in the development of POP, such as uterine prolapse, cystorectocele, and enterocele, as well as vaginal vault prolapse after hysterectomy [7]. Avulsions occurring in levator muscle have been found to play a role especially in the formation of cystocele and uterine prolapse. Furthermore, a direct correlation between POP symptoms and the degree of defect was found. An increased risk of developing uterine prolapse was found in patients with bilateral avulsion compared to those with unilateral avulsion [8]. Despite these findings, not all women who have LAMI present with these compartment defects. Thus, it has been suggested that there may be different factors leading to the development of PFD. Levator avulsions are more common causes of the formation of central and anterior defects than of posterior defects [9]. It is still unclear whether there is an association between different types of LAM defects and specific compartment defects. In a study where 151 patients with POP were compared with a control group of 135, the odds ratio for LAM defects was found to be 7.3 [95% confidence interval (CI) 3.9–13.6]. This indicates that having LAMI increases one's risk of developing POP by 7.3 times. In the literature, there has been a consensus that PFD starts to develop earlier in life in patients with serious LAMI. Moreover, in those patients, despite promising postoperative short-term results, the risk of recurrent POP and cystocele is increased [7,10,11].

Many risk factors have been identified for LAMI; one such risk factor is forceps delivery, which was found to increase the risk of LAMI by 3.4–14.7-fold in different studies [1,12]. LAMI was seen in 35–64% of patients who had forceps-assisted delivery [13,14]. Although a relationship between LAMI and forceps delivery has been demonstrated, it is not clear whether it is solely related to the case itself or application of the device. It is also unclear whether the speed of the fetal head descent during the second stage of labor and/or use of different types of forceps is the cause of injury. Another risk factor for LAMI is the length of the second stage of labor. A study reported that in women who had LAMI confirmed by magnetic resonance imaging, the second stage of labor was 78 minutes longer [12]. In a study investigating the risk factors for LAMI, the use of forceps, anal sphincter rupture, and episiotomy were found to be the risk factors, but surprisingly, vacuum extraction was not. Gestational age, birth weight, and head circumference did not show a statistically significant difference in the development of LAMI. Another study reported that the second stage of labor longer than 110 minutes increased the risk of LAMI by 2.27-fold [15]. Moreover, this strong relationship between the duration of the second stage of labor and LAMI has been emphasized by other investigators [13,16]. Although it has not been defined as a risk factor in Kearney et al's study [12], fetal head circumference has been identified as an independent risk factor in Valsky et al's study [15]. In this study, fetal head circumference in primiparous women was assessed by transperineal ultrasound. The risk of LAMI was increased by 3.34-fold when the fetal head circumference was > 35.5 cm and by 5.32-fold when the duration of the second stage of labor was also increased. The variations in results reported in the literature depend on patient selection bias, demographic and genetic characteristics, and variations in obstetric practice. Based on these findings, many researchers hypothesized that elective cesarean delivery (CD) may prevent LAMI. In a study investigating the effects of fetal head on the vaginal side walls during the second stage of labor, it was found that the maximum head pressure was 31.8 ± 11.0 kPa and 5.5 ± 3.7 kPa during and between uterine contractions, respectively. The average head pressure was 13.34 ± 4.8 kPa during uterine contractions. The pressure of the fetal head during birth was measured to be two-fold more than the amniotic pressure, and this pressure increases toward the end of the birth. Hence, it is stated that fetal head pressure is one of the most important factors for POP development in birth-related injuries [17]. Another study reported that occiput posterior presentation and macrosomia work synergistically, increasing the risk of perineal trauma [18]. Shek and Dietz [19] found that women with a lower BMI were at a higher risk of developing LAMI, but the clinical significance is questionable, because the upper limit was 30.01 kg/m^2. Epidural analgesia has been shown to be protective against LAMI in some studies [13].

An association has been found between advanced maternal age at first delivery and LAMI by some studies [12,20], but not by the others [15,19]. Delayed childbearing has been identified as a risk factor for PFD in several studies. Kuh et al [21] found a strong association between the symptoms of stress UI and the maternal age of ≥ 30 years at the first VD among British women. Foldspang et al [22] found increased risks of UI with increasing age at the time of the last childbirth for women aged 30–44 years. The risk of requiring surgery for stress UI and POP also appears to increase with increasing age at the first childbirth, irrespective of the mode of delivery. For example, in one study, 14% of women aged ≥ 30 years at the first vaginal childbirth required surgery for POP compared with 6% of women younger than 30 years [23]. The trend toward delayed childbearing in developed countries may result in an increased prevalence of PFD in the next decades.

Effect of normal delivery and CD on PFD

One of the key factors causing PFD is the mode of delivery. It is thought that VD may be responsible for the development of PFD by damaging pelvic support tissues such as muscles and connective tissues as well as nervous structures, especially at the second stage of labor. It has been reported that partial denervation in the pelvic floor may occur especially in the first pregnancy, and the risk of PFD increases with the severity of the damage in most women with VD [18]. Damage to the nerves of the pelvic floor and affected pelvic floor muscles has been shown to be more prominent in nulliparous incontinent women compared to nulliparous continent women [24]. Despite all these studies, there are not enough evidence-based data confirming that VD is solely responsible for PFD. Besides, pregnancy itself may be one of the most important risk factors for the development of PFD. Hormonal changes during pregnancy and the mechanical effects that start to increase in the third trimester and reach the maximum level at term are the factors changing the structure of the pelvic floor. It has been suggested that increased intra-abdominal pressure due to growing uterus and the change in the axis of lumbar spine may also be predisposing factors for the development of PFD. It has also been reported in these studies that increased pressure on the bladder during pregnancy causes an increase in the urethrovesical angle, and a decrease in the support of