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REVIEW ARTICLE

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Postpartum stress urinary incontinence, is it related to vaginal delivery?

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ABSTRACT

A postpartum stress urinary incontinence is highly prevalent in Chinese women. Both pregnancy and delivery can damage muscular, fascial, and neural mechanisms of urinary continence. Elective cesarean section (CS) is not completely protective against postpartum stress urinary incontinence.

Background and purpose

Many women experience urinary incontinence during pregnancy and while some recover postpartum, many develop persistent incontinence. Postpartum stress urinary incontinence (SUI) is a significant medical problem, with prevalence rates of 6.9% at 6 weeks and 5.0% at 6 months among China [1]. At 4 years after the first delivery, the prevalence of SUI is reportedly at a staggering 29% [2]. In the United States, the prevalence of SUI in nulliparous women is 6.5% compared with 9.7% in primiparous, 16.3% in multiparous with 2 births, and 23.9 in multiparous with >3 births [3]. As SUI adversely affects women's quality of life (QoL), more women are seeking treatment for this significant health problem. Specifically, SUI can adversely affect QoL in approximately 54.3% of all pregnant women in four domains including physical activity, travel, social relationships, and emotional health [4]. Multiple studies have supported that vaginal delivery is associated with an increased risk of pelvic floor disorders (PFD) [5,6]. One theory that explains these findings is related to neuromuscular abnormalities in the levator ani muscle. Since vaginal delivery is considered a major cause of pelvic floor damage, we question whether cesarean delivery can prevent the development of postpartum SUI, and is the purpose of this review.

Recent findings

The central organs of the pelvis, including the uterus, vaginal apex, bladder, and rectum, are supported by fascial and muscular components and require an intact nerve supply. Prolapse of these organs is prevented by three mechanisms: constriction, suspension, and flap-valve closure. Constriction of the vaginal by the levator ani occludes the levator hiatus through which prolapse of pelvic structures can occur. Fascia and ligaments suspend the uterus, bladder and bladder neck, and rectum to the pelvic side-walls. Angulation of the vaginal canal over the levator plate allows closure on increasing the intra-abdominal pressure. This intact system of muscles, connective tissue, and innervation is crucial for normal support and function. This complex forms a v-sharped sling running from the pelvic sidewall on one side, posteriorly around the anorectal junction, and back towards the centrally pelvic side-wall. The space between the arms of the v is the levator hiatus which contains the urethra anteriorly, the vagina centrally and the anorectum posteriorly. It will be marked distension and deformation in labor. The mechanism of urinary continence has two major components: structural and neuromuscular, both of which are affected by vaginal delivery.

Structural

Structurally, physical support of the urinary tract is composed of the pelvic floor musculature and connective tissue. Lin et al. [7] revealed a marked decrease of ganglion cells in the neural plexuses posterolateral to the vagina in experimental incontinent rats in which serum CPK and LDH levels were markedly elevated.

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Many c-Fos immunostaining neurons were observed in the L6 to S1 spinal cord segments in incontinent rats, but none were observed in control rats. After 4 weeks, muscle necrosis and degeneration, irregular shape, and size of muscle fibers, and a change in the type I/II ratio were prominent features in the levator ani. In the urethra, a significant decline in urethral wall musculature (both smooth and striated) was noted in incontinent rats. Pathogenesis of urethral tissue gene expression of SUI was shown by the upregulation of the genes involved in inflammation, collagen breakdown, and smooth muscle inhibition. The expression of genes involved in the TGF cellular signaling pathway (Smad2), collagen breakdown (MMP-13), and smooth muscle inhibition (RGS-2) was significantly increased in the incontinent rats. Smad2 protein expression was significantly upregulated in the incontinent rats [8]. During vaginal childbirth, the baby's head distends and stretches the pelvic floor muscles, connective tissue, and nerves, which may then become compromised. The use of forceps, anal sphincter rupture, and episiotomy were found to be the risk factors associated with vaginal delivery [9]. Lenis et al. [10] reported that vaginal distention upregulated urethral expression of CCL7 and CD195 immediately after tissue injury in postpartum rats. Vaginal distention results in the upregulation of the cytokines and receptors expressed during tissue injury, which may facilitate spontaneous functional recovery. Prolonged second-stage labor and vaginal delivery upregulated CD191 and attenuated the expression of hypoxia inducible factor-1α and vascular endothelial growth factor in the setting of vaginal distention, likely by decreasing hypoxia.

Neuromuscular

The second component is neuromuscular and consists of the pudendal nerve (PN) and external urethral sphincter (EUS) mechanism [11]. During childbirth, PN injury occurs and the resultant dysfunction is associated clinically as SUI. In the study by Song et al. [12], although continence function recovered 9 weeks after simulated childbirth injury, innervation of EUS was not complete at this time point, suggestive of persistent neurogenic deficiency which, when compounded by the effects of aging, may lead to a delayed recurrence of SUI in an animal model with increased age. After vaginal childbirth, trauma might affect the pudendal nerve or its branches, the pelvic floor muscles, such as anal sphincter, the puborectalis–pubococcygeus complex, and fascial structures.

Previous studies have found that one of the key factors causing postpartum SUI is the mode of delivery. A higher prevalence or incidence of SUI has been observed in women who had at least one vaginal delivery than in women who underwent cesarean delivery [13–18]. There is 20-fold over expression of macrophage chemoattractant protein 3 in the urethra immediately following vaginal delivery, which has been postulated to be secondary to a shear effect on the pelvic muscles [19]. Vaginal delivery has been correlated with SUI development, likely via injury of the pelvic floor structures: muscles, nerves, organs, and extracellular matrix (ECM) responsible for continence [20]. Antenatal and postpartum neurophysiological recordings demonstrate PN injury resulting from vaginal delivery but not cesarean section (CS) or pregnancy itself [21–23]. Vaginal delivery is the most significant risk factor for postpartum stress urinary SUI, but it is not clearly the modifiable surface electromyography of pelvic muscle after different model of delivery.

CS

CS is requested for some complex medical indications involving both maternal and neonatal factors which need to delivery as soon as possible. It is associated with increased risk of neonatal respiratory morbidity and can cause complications in subsequent pregnancies, such as uterine rupture, placenta previa, and placenta accreta. Electrical CS and emergency CS are common types in clinic. Electrical CS is in the absence of any obstetric indications. While the other, compared planned CS, is always with trial of labor. It is quite possible that pelvic floor injury in obstructed labor is already too extensive to be prevented by surgical intervention. The prevalence of postpartum SUI is similar following vaginal delivery and CS performed for obstructed labor. Hence, not all CS deliveries can reduce the likelihood of postpartum urinary incontinence. The key lies in whether the CS is performed before labor.

Discussion

Review of the available evidence allows the assumption that prolonged second-stage labor and vaginal delivery can both damage the pelvic floor [24,25]. One study shows that nearly one-third of women have levator ani denervation after their first delivery, although many recover by 6 months postpartum [26]. A recent study showed that continuous targeted neurotrophin therapy accelerates continence recovery

after simulated childbirth injury, likely through stimulating neuroregeneration and facilitating EUS recovery and re-innervation. Neurotrophins or other therapies targeting neuromuscular regeneration may be useful for treating SUI [12]. PN crush could be a recoverable neurogenic after vaginal delivery. Increasingly more published studies have claimed that CS has not been found to significantly reduce pelvic floor morbidity compared with vaginal delivery in the long term [27,28]. CS was not completely protective against postpartum SUI if second-stage labor was prolonged. Emergency CS after full dilatation can have a significant negative effect on the PN much the same as would vaginal childbirth if second-stage labor is prolonged. Therefore, prolonged second-stage labor may be the real concern associated with an increased likelihood of postpartum SUI.

Physical treatment such as biofeedback (re-training) therapy and pelvic floor electrical stimulation have had a major impact in the reconstruction of local anatomy and in eliminating symptoms of SUI, as demonstrated in many reviews [29,30]. A randomized controlled trail including100 primiparous women indicate that home-based pelvic floor muscle training is effective [31]. Another study showed that increasing awareness of pelvic floor muscle re-training in postpartum women with a weakened PFM may potentially reduce SUI and, as result, increase QoL [32].

Summary

Pelvic floor neuromuscular damages in women are mainly observed in the second stage of labor. To date, studies have shown that elective CS seems to be a good preventative measure. However, CS still carries significant health risks for both mother and child, including increased incidences of postpartum hysterectomy, infection, adhesions, ileus, and placental implantation problems in future pregnancies. Pelvic floor rehabilitation is prescribed as first-line treatment for women with SUI and may actually be a viable protective measure against SUI during the postpartum period. Elective CS should be discussed with the patient only if there is an increased risk for labor functional loss rather than for the purposes of solely purpose of avoiding pelvic floor damage and possible impairment of postpartum SUI.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this article.

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