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The Role of Oral Contraception on Bladder Symptoms

Lauren N. Wood[1] · **Tamara Grisales**[1]

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Abstract
Purpose of Review While the effects of local and systemic estrogen on the pelvic floor have been widely studied in postmenopausal women, little is known about oral contraceptive (OCP) use and the role on bladder symptoms. Estrogen and progesterone have been implicated in both the treatment and etiology of various pelvic floor conditions including overactive bladder, urinary incontinence, recurrent urinary tract infections, and interstitial cystitis/bladder pain syndrome. This article will review the effect of oral contraceptive pills on bladder symptoms.
Recent Findings Oral contraceptives appear to decrease the risk of stress urinary incontinence (SUI). The effect of OCP on recurrent UTI, OAB/UUI is unclear. Combined oral contraceptives may be implicated in interstitial cystitis/bladder pain syndrome.
Summary There appears to be a link between changes in estrogen and progesterone levels and lower urinary tract symptoms. While many studies have evaluated the effect of hormones on the genitourinary tract and bladder function in postmenopausal women menopause, further studies are needed to directly examine the role of OCP in the etiology and treatment of various lower urinary tract conditions. Oral contraceptives are manufactured with a variety of synthetic progestins and with varying dosages of estrogen and progestin, which have been demonstrated to alter endogenous hormone production and hormone receptor expression. Further research how these changes may affect bladder function is warranted.

Keywords Oral contraception · Estrogen · Progesterone · Urinary incontinence · Bladder pain syndrome

This article is part of the Topical Collection on *Overactive Bladder*

✉ Tamara Grisales
tgrisales@mednet.ucla.edu

Lauren N. Wood
lwood@mednet.ucla.edu

[1] Department of Obstetrics and Gynecology, University of California, Los Angeles, 100 Medical Plaza Suite 383, Los Angeles, CA 90095, USA

Introduction

This review aims to evaluate the effects of oral contraceptives on bladder symptoms. Oral contraceptives in the form of the pill is used by 9% of women under age 49, making it one of the most commonly used methods of contraception [1]. Lower urinary tract symptoms are common and, while estimates vary, the prevalence of urinary incontinence (UI) in women is reported to be as high as 45% [2].

The lower urinary tract and genital tract share a common embryological origin. Estrogen and progesterone receptors have been found to be located in the vagina, bladder, urethra, periurethral tissue, and muscles of the pelvic floor, and evidence suggests that hormonal status contributes to bladder function in women [3, 4]. It is known women undergoing menopause often experience changes new lower urinary tract symptoms. These changes have been attributed to changes in circulating estrogens. The deficiency of estrogen during menopause has been implicated in lower urinary tract symptoms, with 70% of women relating the onset of their urinary incontinence to the time of their last menstrual period at the commencement of menopause [5•]. Reports of variation in urinary symptoms at various time points during the menstrual cycle implicate the role of hormones on the lower urinary tract, and 42% of women have reported worsening symptoms prior to menstruation [6].

However, little is known regarding the effect of oral contraceptives on premenopausal women.

Mechanism of Oral Contraception

Background of OCPs

Combined oral birth control pills are commonly prescribed medications worldwide. Combined oral contraceptives (COCs) utilize combinations of synthetic estrogen and progesterone to suppress ovulation and prevent fertilization. While progestin-only pills are also available, we will focus this review on the more commonly prescribed combined oral contraceptive pills (COCs).

Many formulations and combinations of COCs have been developed. In the USA, only two estrogens are commercially available. Ethinyl estradiol, which is the now most common, and mestranol, which is metabolized to ethinyl estradiol and was largely used in early pills. Both compounds are synthetic hormones, which differ from the body's natural estradiol but can alter levels of endogenous hormones. The metabolism of ethinyl estradiol varies widely from among individuals, which results in variations in types and intensity of side effects among users. There are currently eight progestins available in the USA, which all bind to progesterone receptors and vary in their affinity for the androgen receptor. The wide variations among COCs have also made certain side effects difficult to predict and study.

Many studies have demonstrated COCs reduce circulating estrogen, progesterone, and androgen levels by multiple mechanisms. First, COCs suppress the pituitary secretion of gonatotropins, specifically follicle-stimulating hormone (FSH) and luteinizing hormone (LH). Decreased secretion of FSH and LH decreases endogenous ovarian production of estradiol and testosterone. COCs also increase circulating levels of SHBG, which in turn decreases bioavailable estrogen and testosterone [7, 8]. A recent meta-analysis by Zimmerman et al. concluded that both free and total testosterone decreased significantly in COC users compared to non-users, regardless of dose and progestin type. SHBG was also found to be significantly increased in COC users. The increase in SHBG was found to be dependent on ethinyl estradiol dose [9•].

In this review, we will examine the literature evaluating the effect of COCs on bladder function. We will explore proposed mechanisms due to their changes in serum hormone levels and hormone receptor expression and function within the genitourinary tract.

Role of Estrogen in Genitourinary Tract

Bladder storage and voiding are dependent on interaction between parasympathetic, sympathetic, somatic, and sensory nerves. Estrogen has been proposed to affect continence by a number of mechanisms, including ß-3 mediated detrusor relaxation, changing the bladder's threshold for sensation, affecting α receptors in urethral smooth muscle, and increasing urethral resistance [10]. Estrogen has been shown to directly affect detrusor function through modifications in muscarinic receptors and by inhibition of the movement of extracellular calcium ions into muscle cells [11, 12]. As a result, estradiol reduces the amplitude and frequency of spontaneous contractions and may increase the sensory threshold of the bladder in some women [13–15]. Estrogen has also been shown to promote collagen synthesis and appears to be involved in collagen metabolism within the urinary tract [16]. Centrally, estrogen receptors have been found in the cerebral cortex, limbic system, hippocampus, and cerebellum while androgen receptors have been found in the pontine micturition center [17].

The urinary and genital tracts are sensitive to female sex steroid hormones, which are associated with bladder function. The actions of several of these steroid hormones (estrogen, progesterone, and androgens) on the lower genitourinary tract have been studied. Steroid hormones, such as estrogen, progesterone, and testosterone, activate their respective specific steroid receptors within the genitourinary tract. A number of studies have characterized these steroid receptors within steroid-sensitive tissues [18–24]. Estrogen receptors (ER) and progesterone receptors (PR) have been found in both pre- and postmenopausal women throughout the urogenital tract [18, 21, 25–31]. Estrogen receptors are expressed in the squamous epithelium of the urethra, the trigone of the bladder, and the vagina [5•].

Two types of estrogen receptors (ERs), ER-alpha and ER-beta, have been identified are expressed throughout the squamous epithelium of the lower urinary tract [32]. Alpha ERs are found in higher density in the urethral sphincter and are thought to contribute urethral tone when sensitized by estrogen [17]. Though progesterone and androgen receptors are expressed in the genitourinary tract, their role in bladder function is less clear than that of estrogen receptors.

A randomized trial of postmenopausal women concluded that either oral or vaginal estrogen increased blood flow to the bladder neck and urethra and could alleviate symptoms of OAB and stress urinary incontinence (SUI). This further suggests the relationship between circulating levels of estrogen and other steroid hormones and bladder function.

Effect of OCP on Urinary Tract

The hormonal effects of menopause on the genitourinary tract have been studied extensively. Menopause-induced decrease in estrogen has been shown to result in changes in the genitourinary tract tissue and its function. In premenopausal women, urinary function has been related to the menstrual cycle. Studies have suggested longer functional urethral length around ovulation as measured by urethral pressure profile associated with peak levels of estrogen in the middle of the

menstrual cycle [17]. Studies have also found detrusor contractions to be more frequent during the late luteal phase [5•]. While changes in hormonal levels related to menstrual cycle and menopause have been shown to result in changes in lower urinary tract symptoms, the effect of COCs on bladder tissue and function has not been well studied. It can be hypothesized that the changes in bioavailable endogenous hormone levels (estrogen, progesterone, and testosterone) and hormone receptor modulation in the genitourinary tissue may result in changes in lower urinary tract function.

As previously discussed, many of the effects of COCs on hormonal function have been well established. Studies have demonstrated lower levels of circulating 17-beta estradiol in COC users compared with non-users [33]. COCs increase levels of SHBG which decreases circulating levels of free estradiol and testosterone, while also suppressing endogenous estrogen production [8]. Estrogen, progesterone, and androgen receptor expression has also been found to vary, with up- and down-regulation in response to SHBG levels [33]. Changes in the ratio of ER-alpha and ER-beta are thought to contribute to tissue changes related to menopause. These changes may apply to the use COCs as well. The mechanism is unclear, though it is postulated that such alterations in ER ratios may result in changes in the genitourinary tissue uptake of bioavailable estrogen and other hormones, which may consequently alter bladder and voiding function.

Review of Current Literature

Few studies have addressed the effect of contraceptives on bladder symptoms. Of those that address contraceptives overall, even fewer specifically address oral contraceptives.

OCP and UI

There are few studies evaluating the impact of oral contraception on urinary incontinence (UI). An epidemiologic study using validated questionnaires found the rate of UI among nulligravid young women to be 12.6%. Incontinence subtypes were also evaluated. The rate of SUI was found to be 6.2%, urge urinary incontinence (UUI) 4.5%, and mixed urinary incontinence (MUI) 1.4%. The same study found that women using combined oral contraception reported lower rates of incontinence, though the few other studies evaluating COCs and UI have been conflicting [34].

OCP and SUI

Stress urinary incontinence (SUI) is defined as involuntary loss of urine on effort, physical exertion, or on sneezing or coughing [35]. Unopposed estrogen has not traditionally been considered a treatment for SUI [36]. The largest and most notable study regarding SUI and oral contraceptive users examined over 10,000 premenopausal women and concluded that those using oral contraception had a reduced risk of SUI (OR 1.57) after adjusting for confounding factors [37]. There was also a reduction in risk of mixed urinary incontinence (OR 0.52) [37]. The same reduction in SUI was not seen in women with a hormone-releasing intrauterine device [37].

OCP and OAB/UUI

Urgency incontinence is the involuntary loss of urine associated with urgency [35]. Urgency incontinence is part of a larger symptom complex known as overactive bladder syndrome, defined as urinary urgency, usually accompanied by frequency and nocturia, with or without urgency urinary incontinence, in the absence of urinary tract infection or other obvious disease [35]. In animal models, estrogen has been shown to increase the ratio of collagen to bladder smooth muscle, potentially reducing compliance and contributing to symptoms of OAB [38]. In contrast, estradiol has been shown to reduce both the amplitude and frequency of detrusor contractions in rabbit bladders [15]. In oophorectomized rats, combination treatment of estrogen plus progesterone was shown to decrease bladder muscarinic activity when compared to estrogen alone [39]. Systemic estrogen has been shown to be superior to placebo in reducing incontinence episodes and the first sensation to void in a meta-analysis of RCTs [40]. A Cochrane review concluded that estrogen could improve or cure UUI in premenopausal women [41]. Townsend et al. found a 27% increased risk of UI in women taking oral contraceptives when compared with those who had never used oral contraceptive pills [42]. The OR increased to 1.48 in women taking the pill for 10 or more years, and this association was only seen in urgency incontinence, not stress incontinence [42]. In contrast, in a large population-based study of over 10,000 women, oral contraceptive users had a reduced risk of urgency urinary incontinence (OR 0.36) [37]. Additionally, there was a trend toward reduction in symptoms of OAB in OCP users; however, this did not reach statistical significance [37]. Solifenacin was found to be well tolerated in a population of young healthy women without pharmacologic interaction with oral contraceptives, and this data supports the use of anticholinergics in young women with overactive bladder [43].

OCP and Recurrent UTIs

It is known that estrogen deficiency can cause a wide array of voiding complaints, in addition to reduction of vaginal lactobacilli [44, 45]. This reduction can lead to an increased risk of developing a urinary tract infection (UTI) [45]. While it

is well understood that oral and vaginal estrogen can help in the setting of recurrent UTI in postmenopausal women, little is known regarding oral contraception and UTI. One study demonstrated that in women with recurrent UTIs taking contraception, the addition of vaginal estrogen decreased risk of recurrent UTI. [46].

OCP and IC/BPS

Estrogen may be implicated in the pathophysiology of interstitial cystitis (IC)/bladder pain syndrome (BPS) as well. In women with IC, estrogen receptors have been found in their bladder mast cells [47]. This has led some to propose that this finding could explain the increased prevalence of IC in women over men and the worsening of bladder symptoms when estrogen levels are highest during the middle of the menstrual cycle [48]. It has been demonstrated that women with IC are more likely to be currently taking or have previously taken OCP [49, 50]. A systematic review of contraception and the effects on the pelvic floor concluded that IC had a statistically significant association with OCP use (OR 2.31) [51••].

Conclusions

Estrogen and progesterone appear to play a role in the lower urinary tract and may have a noticeable effect on bladder symptoms. However, the role of combined estrogen/progesterone OCP on lower urinary tract symptoms has been evaluated in only a few studies. Oral contraceptives appear to decrease the risk of SUI. The effect of OCP on recurrent UTI, OAB, or UUI is unclear. Combined oral contraceptives may be implicated in interstitial cystitis/bladder pain syndrome. While the effect of hormones on the genitourinary tract and bladder function has been extensively studied in the setting of menopause, further studies are needed to directly examine the role of OCP in the etiology and treatment of various lower urinary tract conditions. Changes in SHBG, endogenous estrogen, progesterone, and testosterone levels, as well as hormone receptor expression, may be areas of further exploration.

Compliance with Ethical Standards

Conflict of Interest Drs Wood and Grisales declare they have no conflicts of interests.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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