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Risk of urinary incontinence symptoms in oral contraceptive users: A national cohort study from the Swedish Twin Register

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Abstract

Objective: To assess the impact of oral contraceptives on lower urinary tract dysfunction in premenopausal women.

Design-Subjects: A cohort study of 10,791 women (born 1959-1985) from the population-based Swedish Twin Register who participated in a web-based survey of common diseases.

Setting: National Register.

Intervention(s): None.

Main Outcome Measure(s): Symptoms of urinary incontinence.

Result(s): For users of oral contraception there was a significantly reduced risk for symptoms of stress urinary incontinence (OR 0.57, 95% CI 0.41- 0.79); mixed urinary incontinence (OR 0.52, 95% CI 0.31-0.89); and urgency urinary incontinence (OR 0.36, 95% CI 0.14-0.92). The reduction remained significant when adjusting for age, body mass index and pregnancy history. A reduced prevalence of symptoms of overactive bladder in oral contraceptive users was also observed although the association was non-significant (OR 0.97, 95% CI 0.79-1.18). There were no significant associations between lower urinary tract symptoms and women using a levonorgestrel-releasing intrauterine device compared to non-contraceptive users, with the exception of nocturia (OR 0.53, 95% CI 0.32-0.89).

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Conclusion(s): Oral contraceptive use reduces the overall risk for symptoms of urinary incontinence.

Keywords

contraception; incontinence; overactive bladder; intrauterine device

INTRODUCTION

It is generally accepted that female sex hormones influence the morphology and physiology of vaginal tissues. Peripheral estrogen affects urogenital connective tissue composition and structure, vaginal blood flow, and thickness of vaginal epithelium.(1) Estrogen and progesterone receptors are also abundantly present in the lower urinary tract, which has the same embryonic origin as the vagina.(2-4) A decline in circulating estrogen levels may result in atrophy of vaginal, urethral and bladder trigonal epithelium, as well as initiate metabolic changes in the subepithelial supportive tissues.(5) This process forms the biological rationale for the common clinical practice of prescribing hormone treatment to menopausal women with lower urinary and genital tract symptoms.(6, 7)

Contrary to widespread belief, the Womens Health Initiative (WHI) randomised trial showed that menopausal hormone therapy with conjugated estrogen alone, or in combination with progesterone, increased the risk of de novo or aggravated urinary incontinence after one year of treatment.(8) In premenopausal women, oral contraception is the most common source of hormone intake but very few studies have endeavored to determine the effects of oral contraception on premenopausal urinary incontinence.(9) The aim of this nationwide cohort study was to assess the influence of contraceptives on the risk for lower urinary tract dysfunction in young female twins.

MATERIALS AND METHODS

Data sources

The Swedish Twin Register contains data on nearly all twins born in Sweden since 1886. In 2005, all twins born from 1959-1985 were contacted with a letter inviting them to participate in a web-based survey in order to screen for common complex diseases and common exposures.(10) The present study is limited to female twins participating in the survey. Those not responding to the web questionnaire were offered to answer the survey through a computer assisted telephone interview. After two to five months, 100 twins were contacted again to assess test-retest reliability. The kappa values for agreement between the web questionnaire and telephone interviews ranged from good to excellent.(10)

The entire questionnaire contained approximately 1,300 questions divided into 34 sections using a branching format, meaning that participants were asked follow-up questions only if they responded positively to key initial questions. Data on contraception were registered in the survey section on current medications and were classified as users of: oral contraception; a hormonal intrauterine device; or non-contraceptive users. The questions of the survey did

not distinguish between oral contraceptives containing estrogen-progestagen combinations and those containing progestagen alone.

The section on lower urinary tract function consisted of seven questions on urinary incontinence, six questions on micturition frequency, and five questions on urinary urgency, adopted from a validated epidemiological survey on female incontinence.(11) Participants who answered yes to the question “Do you presently experience involuntary loss of urine?” were classified as having prevalent urinary incontinence. Subsequently, the question “Do you have involuntary loss of urine in connection with coughing, sneezing, laughing or heavy lifting?” were used to define those having stress urinary incontinence, whereas the question “Do you have involuntary loss of urine in connection with sudden and strong urgency to void?” were used to define those with urgency urinary incontinence. Those who responded positively to both alternatives were considered to have mixed urinary incontinence. Overactive bladder was defined as a positive response to the question “Do you experience sudden urgency to void with little or no warning”. Nocturia was defined as one or more micturition episodes per night. This study was approved by the Regional Research Ethics Board at Karolinska Institutet. The study conforms to the STROBE guidelines for reporting observational studies.(12)

Study subjects

The women in this cohort had a response rate of 49.9% to the web questionnaire. An additional 16.0% completed the telephone interview. The overall response rate was thus 66%. The number of female twins with known zygosity and information on at least one symptom of urinary incontinence was 10,791. Data on contraceptive use was available from a total of 8,689 female twins. Three individuals reported both use of oral contraceptives and a hormonal intrauterine device and were categorized as oral contraceptive users.

Statistical analysis

In order to evaluate the association between contraceptive use and symptoms of lower urinary tract dysfunction, logistic regression was used based on generalized estimating equations (GEE), which take into account the correlated (twin) structure of the data. The multivariable analysis was adjusted for age (categorized in quartiles), body mass index (in four categories according to World Health Organisation guidelines),(13) and pregnancy (ever/never). In the co-twin control analysis, healthy co-twins were used as matched controls for the cases. The matched nature of the co-twin control design minimizes confounding by twin pairs sharing: intrauterine exposures; maternal factors; 50% (dizygotic) or 100% (monozygotic) of their segregating genes; and childhood and adolescent environment. Due to insufficient statistical strength for the co-twin control analysis, all incontinence subtypes were merged into a single outcome variable. Odds ratios were estimated with 95% confidence intervals. A p-value less than 5% was considered significant for all analysis. All statistical analyses were performed using SAS software (Cary, NC, U.S.A.).

RESULTS

Characteristics of the female twin cohort are shown in Table 1. Out of 8,689 twins, 2,072 were current users of oral contraception and 118 were fitted with a levonorgestrel-releasing intrauterine device. Information on contraceptive use was missing for 2,111 women.

Oral contraceptive users were significantly younger than women with a levonorgestrel-releasing intrauterine device or non-contraceptive users ($p < 0.001$). Body mass index did not differ between the levonorgestrel-releasing intrauterine device group and the non-contraceptive users ($p = 0.37$), whilst oral contraceptive users had a lower body mass index than non-contraceptive users. More women using a levonorgestrel-releasing intrauterine device had ever been pregnant than both oral contraceptive users and non-users.

Descriptive statistics on symptoms of urinary tract dysfunction are shown in Table 2. In general, there were significantly higher rates of women with no urinary symptoms among oral contraceptive users than non users. In contrast, women with a levonorgestrel-releasing intrauterine device did not differ significantly from non-contraceptive users in prevalent lower urinary tract symptoms except for nocturia ($p = 0.02$).

Risk estimates for lower urinary tract symptoms among users of oral contraceptives or levonorgestrel-releasing intrauterine device compared to non-users are shown in Table 3. Current use of oral contraception was inversely associated with lower urinary tract dysfunction, a finding which remained after adjustment for age, body mass index and pregnancy: stress urinary incontinence (OR 0.57, 95% CI 0.41- 0.79); mixed urinary incontinence (OR 0.52, 95% CI 0.31-0.89); and urgency urinary incontinence (OR 0.36, 95% CI 0.14-0.92). An inverse association between oral contraceptive use and symptoms of overactive bladder was also observed although at a non-significant level. With the exception of nocturia (OR 0.53, 95% CI 0.32-0.89), there were no significant differences in risk estimates for urinary tract dysfunction amongst women using a levonorgestrel-releasing intrauterine device compared to non-contraceptive users. Possible two-way interactions between contraceptives, age, body mass index and pregnancy were tested but were all non-significant.

Table 4 present the results from the co-twin control analysis. Although statistically non-significant, oral contraceptives were consistently associated with a protective effect on symptoms of urinary incontinence overall. The tendency was similar for both mono- and dizygotic twins.

DISCUSSION

In this cohort study of young adult female twins, we report a significantly lower prevalence of stress urinary incontinence, urgency urinary incontinence and mixed urinary incontinence in women using oral contraception compared to non-users. The decreased risk of stress urinary incontinence symptoms, urgency urinary incontinence and mixed urinary incontinence in oral contraceptive users remained after adjustment for possible confounders. In contrast, contraception by use of a levonorgestrel-releasing intrauterine device provided

no significant risk reduction for prevalent symptoms of urinary incontinence compared to non-contraceptive users.

There is conflicting evidence on the effects of hormone intake on lower urinary tract symptoms in postmenopausal women. While some consensus reports and reviews have provided support for the use of menopausal hormone therapy in women with urinary incontinence,(14) recent findings from the WHI randomized trial suggest the opposite.(8) In their conclusion, Hendrix et al.(8) suggest that oral administration of conjugated estrogen alone, or in combination with medroxyprogesterone acetate, increases the risk for de novo or aggravated urinary incontinence in postmenopausal women. However, in comparison to women of reproductive age, expression of estrogen receptors in the anterior vaginal wall and periurethral supportive tissues markedly declines after menopause.(15) Thus, it is unlikely that findings on postmenopausal women can be generalized to women of reproductive age and it seems biologically plausible that intake of female reproductive hormones influences periurethral connective tissue structure and metabolism, differently before and after menopause.

In the present study of premenopausal women we found a general risk reduction for urinary incontinence symptoms in oral contraceptive users when compared to women not using any contraception. This finding was corroborated also when considering confounding factors which are unique for twins. Even though estrogen receptor concentrations are higher in the urethra than the bladder trigone,(16) the paraurethral tissues and bladder outlet are both targets for gonadal steroid hormones.(17) It is therefore suggested that the observed protective effects of oral contraceptives on urinary incontinence in the present study, reflects an influence of estrogen on urethral function and morphology.(18) This assumption is corroborated by experimental studies where estrogen increases urethral sphincter muscle contractility and urethral closing pressure.(19, 20) Estrogen has also been shown to have a positive influence on periurethral vascularisation, urethral neuronal control, and Nerve Growth Factor expression,(21, 22) which may convey secondary beneficial effects on urethral function.

In contrast to the noticeable risk reduction for urinary incontinence, symptoms of isolated bladder overactivity and nocturia were not improved in contraceptive users when compared to non-users. These results are in concurrence with most clinical trials which fail to show that postmenopausal estrogen supplementation is superior to placebo when used in the treatment of irritative bladder symptoms such as urinary frequency and urgency.(23, 24) Experimental data have also suggested that bladder function in young rats is less susceptible to exogenous estrogen than in mature animals and that the effects of estrogen on bladder function depends on the age at which it is administered.(25) Even though high concentrations of estrogen has the potential to inhibit bladder contraction in the female pig bladder,(26) it nonetheless seems that therapeutic doses of estrogen when used for contraception is unlikely to mediate a clinically relevant effect on bladder contractility in premenopausal women.

When compared to non-users, current use of a levonorgestrel-releasing intrauterine device had neither an effect on symptoms of stress urinary incontinence, nor overactive bladder.

Progestagen receptors are found in the subepithelial vaginal stroma and are scattered throughout the lower urinary tract.(27, 28) In clinical studies, orally administered progestagens have been associated with unfavorable effects on urinary incontinence and overactive bladder symptoms.(29) When comparing oral administration to a levonorgestrel-releasing intrauterine device, pelvic tissue concentrations of levonorgestrel are similar.(30) It is therefore assumed that the observed protective effects of oral contraceptives on the lower urinary tract were mediated by the contraceptive estrogen rather than the progestagen component.

In a previous study, the prevalence of urinary incontinence was unaffected by duration of oral contraceptive use.(9) A limitation of our study is that we cannot elucidate upon the influence of duration of contraception use, and time since last use, since these data were not included in the survey. We recognize that using oral contraceptives as a single measure of exposure is a source of misclassification. However, the vast majority of prescribed oral contraceptives involved low-dose preparations of combined oral contraceptives type, as other studies in Sweden have shown that no more than 5% of oral contraceptive users were using progestagen only pills at the time of this study.(31) This indicates that the effect and risk reduction attributed to estrogen containing oral contraceptives was underestimated.

Strengths of our study include the large scale collection of population based data, a well characterized cohort of twins, use of validated questionnaires specifically developed for epidemiological surveys on incontinence and the ability to adjust for known effect modifiers and confounders. A relatively high response rate among eligible women reduces the risk for selection bias. The relatedness of twins was corrected for by statistical methods which incorporated the within-pair correlations in the multivariable analysis.

The present study suggests that in premenopausal women, oral contraceptive use has the potential to influence bladder and urethral function whereas use of a levonorgestrel-intrauterine device conveys neither beneficial, nor adverse effects on continence status. Oral hormonal intake may come to play a role in the pharmacological treatment of urinary incontinence although it remains to be decided whether or not oral contraceptives may actually improve urinary incontinence symptoms in women with manifest disease.

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Table 1

Characteristics of the female twin population born 1959-1985

	No contraceptive users	Oral contraceptives	Levonorgestrel-releasing intrauterine device
N	6,499	2,072	118
Age	34.3 (7.4)	28.7 (6.8)	38.1 (5.4)
Body mass index	23.6 (7.9)	22.5 (4.9)	23.6 (4.6)
<18.5	237 (3.6)	89 (4.3)	2 (1.7)
18.5-24.9	4,531 (69.7)	1,634 (78.9)	83 (70.3)
25-29.9	1,145 (17.6)	252 (12.2)	26 (22.0)
30	398 (6.1)	56 (2.7)	5 (4.2)
missing	231 (3.6)		
Pregnancy			
No	1,864 (28.7)	1271 (61.3)	5 (4.2)
Yes	4,598 (70.7)	787 (38.0)	113 (95.8)
missing	51 (0.8)	-	-

Figures are frequencies (%) or mean values (\pm standard deviations).

Descriptive statistics of urinary incontinence symptoms in a female twin population born 1959-1985

Table 2

	No contraceptive use	Current use of oral contraceptives	p-value	Current use of a levonorgestrel- releasing intrauterine device	p-value
Stress urinary incontinence					
No	6,050	2,026		107	
Yes	447	44	<0.0001	11	0.30
Urgency urinary incontinence					
No	5,100	1,634		100	
Yes	55	5	0.004	0	0.29
Mixed urinary incontinence					
No	6,321	2,056		116	
Yes	178	16	<0.0001	2	0.49
Overactive bladder					
No	5,925	1,914		108	
Yes	574	158	0.09	10	0.89
Nocturia					
No	4,537	1,490		96	
Yes	1,514	450	0.10	18	0.02

Risk estimates for lower urinary tract symptoms in users of oral contraceptives or a Levonorgestrel-releasing intrauterine device in a female twin population born 1959-1985.

Table 3

	Current use of oral contraceptives			Current use of a levonorgestrel-releasing intrauterine device		
	N	Crude OR	Adj OR	N*	Crude OR	Adj OR
Stress urinary incontinence	8,678	0.29 (0.21-0.40)	0.57 (0.41-0.79)	8,388	1.39 (0.74-2.61)	0.95 (0.49-1.84)
Urgency urinary incontinence	6,791	0.28 (0.11-0.71)	0.36 (0.14-0.92)	6,635	-	-
Mixed urinary incontinence	8,682	0.28 (0.17-0.46)	0.52 (0.31-0.89)	8,405	0.61 (0.15-2.51)	0.50 (0.12-2.05)
Overactive bladder	8,682	0.85 (0.71-1.02)	0.97 (0.79-1.18)	8,405	0.95 (0.49-1.84)	0.90 (0.47-1.74)
Nocturia	8,100	0.90 (0.80-1.02)	1.07 (0.94-1.22)	7,870	0.56 (0.34-0.93)	0.53 (0.32-0.89)

All comparisons made with non-contraceptive users as reference i.e OR 1.0.

N*: is the total number used in the adjusted analyses.

Overactive bladder includes urinary urgency both with and without incontinence.

Table 4

Co-twin control analyses oral contraceptive using twins discordant for urinary incontinence.

	N pairs	Crude OR (95% CI)	N pairs	Adjusted OR (95% CI)
All	231	0.81 (0.50-1.31)	230	0.83 (0.50-1.38)
DZ	111	0.73 (0.36-1.48)	110	0.67 (0.30-1.52)
MZ	120	0.89 (0.46-1.72)	120	0.94 (0.47-1.86)

DZ= dizygotic twins, MZ= monozygotic twins.

All urinary incontinence subtypes merged into one outcome variable due to insufficient statistical strength for specific variables.

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