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Characteristics associated with pelvic organ prolapse in women with significant levator ani muscle deficiency

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Abstract

Introduction and Hypothesis—Age is a risk factor associated with symptomatic pelvic organ prolapse (POP) among women with significant levator ani deficiency.

Methods—This cross-sectional study included patients who were referred for varied pelvic floor disorders, had 3D endovaginal ultrasound as part of their evaluation, and were diagnosed with significant levator ani muscle deficiency defined as a score of 12 or more on 3D endovaginal ultrasound. Patients were categorized as control group (stage 0 and 1), or symptomatic prolapse (stage 2–4).

Results—Seventy-six women were available for analysis and found to have significant levator ani muscle deficiency, including 51 with symptomatic POP and 25 without POP. Patients with symptomatic POP were older, mean age 66 (SD± 11.8) vs 48 (SD± 17.3) years; $p < 0.0001$, had greater mean minimal levator hiatus area 19.7 cm^2 (SD± 4.6) vs 17.5 cm^2 (SD± 3.5); $p = 0.048$, and were more likely to be menopausal (91.3% vs 54.5%; $p < 0.001$) compared to those with no POP. In a modified Poisson regression analysis excluding nulliparous women, increasing age (RR=2.39, 95% CI 1.03–5.55) and smoking (RR=1.34, 95% CI 1.08–1.67) remained associated with symptomatic POP after controlling for one another and the MLH area.

Conclusions—Among women with significant levator ani deficiency, older women and smokers had an increased prevalence of symptomatic POP. On average, women without POP but with

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significant levator ani deficiency were 18 years younger than women with POP and significant muscle deficiency.

Brief Summary

age is the main risk factor for occurrence of pelvic organ prolapse in women with significant levator ani deficiency.

Keywords

levator ani muscle deficiency; pelvic organ prolapse; 3D pelvic floor ultrasound; cross-sectional study

Introduction

Approximately 200,000 women undergo inpatient surgery for pelvic organ prolapse (POP) each year in the United States [1]. A report from Kaiser Permanente Group indicated that 29% of the operations performed for incontinence and prolapse are repeated operations [2]. These statistics show that pelvic floor dysfunction is a prevalent and disabling condition with suboptimal treatment. The pelvic floor consists of muscles under neural control, held together by connective tissue arranged in a unique 3-dimensional arrangement [3]. Together, the unique structure formed by these three types of tissue influence pelvic organ support and function. Our ability to understand treatment failure and prevention strategy must therefore arise from understanding these three tissue elements and their structural and functional interactions [3].

Injury to the levator ani muscle as a result of birth has been documented [4–7]. To detect such birth-related injuries to the levator ani muscles, modern imaging techniques such as magnetic resonance imaging (MRI) [8,9], transperineal ultrasonography [10,5,11] and endovaginal ultrasonography (EVUS) [12,13] have been used. Observational studies have shown that there is a direct correlation between the size of the levator ani muscle defect and the symptoms and / or signs of prolapse [14]. Women with bilateral levator ani muscle avulsions are more likely to suffer from uterine prolapse [15]. DeLancey et al. observed an adjusted OR of 7.3 (95% CI 3.9- 13.6, $p<0.001$) for major levator muscle defect associated with pelvic organ prolapse [16].

On the other hand, while all patients with severe prolapse had moderate to severe levator ani muscle (LAM) deficiency, many patients with severe levator ani muscle deficiency (LAD) did not have clinically significant prolapse [17]. Increasing LAD score was correlated with worsening stage of prolapse. In this study population, no subjects with stage 3 prolapse had a score lower than 6; no subjects with stage 4 prolapse had a LA score lower than 9. The high frequency of severe levator deficiency in severe prolapse stages suggests that muscle decompensation is a major part of pathogenesis of severe prolapse [17]. Increasing LA deficiency was correlated with worsening stage of prolapse. In this study population, no subjects with stage 3 prolapse had a score lower than 6; no subjects with stage 4 prolapse had a LA score lower than 9. The high frequency of severe levator deficiency in severe

prolapse stages suggests that muscle decompensation is a major part of pathogenesis of severe prolapse [17]

It is postulated that women with severe LAD who do not demonstrate POP at the time of imaging will develop POP as their connective tissue decompensates with aging. The aim of the current study is to identify risk factors associated with pelvic organ prolapse in women with significant levator ani muscle deficiency.

Methods

Our Institutional Review Board approved the study. This cross-sectional study was conducted among 76 patients referred for evaluation during January 2009 - March 2012. The study included patients with significant levator ani muscle deficiency that underwent a standard pelvic exam, POP-Q examination, and a high-resolution 3D endovaginal ultrasound with the BK Ultrafocus 2052/8838 12 Mhz probe (Peabody, MA, USA) for assessing the levator ani muscles. Women were excluded from the study if they had a history of pelvic reconstructive surgery or if ultrasound quality was determined to be insufficient or data were incomplete. POP stages defined based POP-Q exam were categorized as no POP (stage 0 and 1) or symptomatic prolapse (stage 2–4).

Ultrasound methods

Imaging was obtained at the time of the primary visit using the BK Medical Ultrafocus (Peabody, MA, USA) and a 2052/8838 12 MHz transducer. All ultrasound exams were performed in the office setting, with the patient in dorsal lithotomy position, with hips flexed and abducted. No preparation was required and the patient was recommended to have a comfortable volume of urine in the bladder. No rectal or vaginal contrast was used. To avoid excessive pressure on surrounding structures that might distort the anatomy, the probe was inserted into the vagina in a neutral position with no pressure on the vaginal walls. US volumes were digitally stored for further analysis. High quality images were defined as those that showed the pubic symphysis anterior, levator plate posterior, and the superficial transverse perineal muscle caudad at the edge of the 3-D volume.

Levator ani deficiency scoring

US volumes were evaluated blinded to patient symptoms, and muscles were scored according to LAD scoring system, which has been previously validated [18]. The levator muscle was divided into three subgroups; the puboperinealis/puboanalis [PA], puborectalis [PR], and iliococcygeus/pubococcygeus [PV] [19] (Figure 1). Subgroups were evaluated and scored (0=no defect, 1=minimal defect with < 50% muscle loss, 2=major defect with >50% muscle loss, 3=total absence of the muscle) on each side based on thickness and detachment from the pubic bone. Significant levator ani deficiency (LAD) was defined as a total score within the range of 12–18. Only patients with significant LAD were included in the study. Levator ani muscle thickness in each side at the level of minimal levator hiatus was measured.

Minimal levator hiatus area

The minimal levator hiatus area was defined by measuring the MLH plane, which is the axial plane where the anterior posterior diameter has the shortest distance between pubic symphysis to levator plate (Figure 2).

Anorectal angle (ARA)

The anorectal angle was measured in the mid-sagittal plane as the angle between anal canal and rectum.

Levator plate descent angle (LPDA)

Levator plate descent angle measured as an angle between vertical reference line of plane and shortest line between levator plate and pubic angle in mid-sagittal plan.

Statistical methods

Patient characteristics obtained from a standardized interview and urogynecological characteristics obtained from the examinations were compared between those with and without POP using t-tests, r Wilcoxon rank sum tests, chi-square tests or Fisher's exact texts as appropriate according to the distribution of the data. Demographic and medical history factors examined in relation to POP included patient age, body mass index, smoking status, parity, number of vaginal and cesarean deliveries, menopausal status, the use of hormone replacement therapy and a prior history of hysterectomy, hypertension, or diabetes. Clinical assessments compared between those with and without POP included LAD category, laterality of MLH avulsion, rectum deviation, sphincter injury, atrophy, anal rectal angle, levator plate descent angle and MLH area. Age distributions were compared for combinations of POP and MLH > or = 17 using boxplots and analysis of variance. Modified Poisson regression models were estimated to calculate prevalence portion ratios (PPR) and 95% confidence intervals controlling for age. Due to the small number of nulliparous patients (n=3), the regression analyses were restricted to parous women. Because small sample size limits the number of covariates that regression models can support [20], all covariates were initially examined in separate models controlling only for age. Factors associated with POP in the age-adjusted models were included in the final multivariable model controlling for MLH area. Due to sparse data and zero cells (i.e., no subjects under age 50 with atrophy or sphincter injury), regression models for sphincter injury and atrophy would not converge so age-adjusted associations for these factors were examined using the Cochran-Mantel-Haenzsel method.

Results

Seventy-six women with significant LAD were available for analysis, including 51 with symptomatic POP and 25 without POP. The distribution of patient characteristics by POP status is displayed in Table 1. In bivariate analyses, patients with symptomatic POP had a greater mean age [66 (SD± 11.8) vs 48 (SD± 17.3) years, p<0.0001], and were more likely to be menopausal (82% vs 48%, p=0.0009) compared to those with no POP (Table 1). The majority of patients were non-smokers, but all six patients who reported current or past smoking also had POP.

Of the ultrasound measurements analyzed (Table 2), only MLH area differed by POP status, with greater mean MLH area 19.7 cm^2 ($SD \pm 4.6$) vs 17.5 cm^2 ($SD \pm 3.5$), $p=0.048$ observed among those with POP. When the distribution of age was examined by POP and MLH area, the age distributions for combinations of POP and MLH $> \text{or } 17$ overlapped extensively. Mean age differences across the groupings were statistically significant (ANOVA $p=0.005$), but the boxplots demonstrate younger ages among those without POP regardless of their MLH status.

In a modified Poisson regression analysis omitting nulliparous women, age was entered as a dichotomous variable indicating age $\geq \text{or } < 50$ because the prevalence of POP among the 50–64 and 65+ age groups was similar and two-fold greater than patients under 50. Age 50 and over ($PPR_{\text{adj}}=2.39$, 95% CI 1.03–5.55) and ever smoking ($PPR_{\text{adj}}=1.34$, 95% CI 1.08–1.67) were associated with symptomatic POP after controlling for one another and MLH (Table 3). No other patient or clinical characteristics were associated with POP after controlling for age.

Discussion

Our study showed that among women with significant levator ani deficiency, older women and smokers have an increased prevalence of symptomatic POP. Women with prolapse have a larger MLH area, but POP prevalence does not differ significantly across MLH area groupings after controlling for age. On average, women without POP but with significant levator ani deficiency are 18 years younger than women with POP and significant muscle deficiency.

There is ample evidence that childbirth or LAM injury is an important contributing factor for pelvic floor disorders [21,22,17,4]. Birth related LAM defect can be identified early postpartum with different imaging modalities; however, the impact of LAM injury on the pelvic floor is likely to emerge later in life.

In a study of 10,177,480 adult women who were followed for 24,979,447 person-years, overall lifetime risk of any primary surgery for stress urinary incontinence (SUI) or POP was 20.0% (95% CI 19.9–20.2) by the age of 80 years. Separately, the cumulative risk for SUI surgery was 13.6% (95% CI 13.5–13.7) and that for POP surgery was 12.6% (95% CI 12.4–2.7). For age-specific annual risk for POP surgery, it increased progressively until ages 71 and 73 years when the annual risk was 4.3 per 1,000 women [23]. Swift et al showed that advancing age, increasing weight of infants delivered vaginally, a history of hysterectomy and a history of previous prolapse surgery were found to be the strongest etiologic predictors of severe pelvic organ prolapse [24]. Among 641 Swedish women, age ($P < .0001$), parity ($P < .0001$), and pelvic floor muscle strength ($P < .01$) [and among parous women, the maximum birth weight ($P < .01$)] were significantly and independently associated with presence of prolapse [25].

Regarding MLH area, ultrasound studies had shown the mean MLH area at rest in women with and without pelvic organ prolapse had been 14.98 cm^2 and 17.49 cm^2 respectively [26].

The question remains, what is the association between childbirth related LAM defect to increasing age, larger MLH area, and ultimately POP symptoms?

Our findings demonstrate that increasing age is a major risk factor for POP in women diagnosed with significant LAD. Our study reported an increased prevalence of POP among smokers, although only 8% (6/74) of our patient population reported past or current smoking. Further long-term prospective studies are warranted to elucidate whether major LAM defect needs time to enlarge the levator hiatus and lead to symptomatic prolapse.

Current study is limited with small population number. The main strong point of the study is investigating an important question regarding the risk factors for pelvic organ prolapse when levator ani muscles are deficient. We identified women with severe muscle deficiency without pelvic organ prolapse and could recruit them to the study as control group.

This finding presents novel opportunities for prevention of POP because levator ani muscle deficiency is likely to be a useful intermediate variable for intervention studies. Any change in clinical practice that can result in reduced prevalence of levator ani muscle injury, and early diagnosis of muscle injury would be expected to have a positive effect on the prevalence of significant prolapse later in life. Once levator ani muscles deteriorate to the level of severe levator ani deficiency, it is only a matter of time for symptomatic Pelvic organ prolapse to occur. This sub-group represents a prime target for institution of preventive strategies.

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Figure 1.
axial view of Levator ani muscle on EVUS
LAM; levator ani muscle, PA; puboanalis, PC; pubococcygeus, PR; puborectalis, PS; pubic
symphysis, R; rectum, U; urethra, V; vagina

Figure 2.

Minimal levator hiatus and pelvic floor biometry

A; LP; levator plate, PS; pubic symphysis, U; urethra

Yellow line identifies the shortest distance between pubic symphysis and levator plate in mid sagittal plane.

B; The shortest line in mid sagittal plan is anterior posterior length of minimal levator hiatus.

MLH; minimal levator hiatus, PS; pubic symphysis, R; rectum

Table 1

Distribution of patient characteristics for women with and without pelvic organ prolapse (POP), based on POP quantification examination

	POP (n=51) n (%)	No POP (n=25) n (%)	Total (n=76) n (%)	p^a
Age (years)				0.0002
49	5 (10)	13 (52)	18 (24)	
50–64	15 (29)	5 (20)	20 (26)	
65	29 (57)	6 (24)	35 (46)	
Missing	2 (4)	1 (4)	3 (4)	
Mean (std)	66.0 (11.8)	48.0 (17.3)		<0.0001 ^c
BMI (kg/m²)				0.79
24.99	15 (29)	7 (28)	22 (29)	
25–29.99	15 (29)	10 (40)	25 (33)	
30	17 (33)	8 (32)	25 (33)	
Missing	4 (8)	0 (0)	4 (5)	
Mean (std)	27.8 (4.6)	27.9 (4.0)		0.91 ^c
Parity				0.095
0	1 (2)	2 (8)	3 (4)	
1	4 (8)	4 (16)	8 (11)	
2	21 (41)	12 (48)	33 (43)	
3	12 (24)	1 (4)	13 (17)	
4	8 (16)	2 (8)	10 (13)	
Missing	5 (10)	4 (16)	9 (12)	
Median (min, max)	2 (0,6)	2 (0,4)		0.02 ^d
Vaginal Deliveries				0.009 ^b
0	1 (2)	6 (24)	7 (9)	
1	6 (12)	5 (20)	11 (14)	
2	22 (43)	8 (32)	30 (39)	
3	12 (24)	2 (8)	14 (18)	
4	7 (14)	1 (4)	8 (11)	
Missing	3 (6)	3 (12)	6 (8)	
Median (min, max)	2 (0,5)	1.5 (0,4)		0.002 ^d
Menopause				0.0009 ^b
No	4 (8)	10 (40)	14 (18)	
Yes	42 (82)	12 (48)	54 (71)	
Missing	5 (10)	3 (12)	8 (11)	
HRT				0.58
No	19 (37)	13 (52)	32 (42)	
Yes	6 (12)	6 (24)	12 (16)	
Missing	26 (51)	6 (24)	32 (42)	

	POP (n=51) n (%)	No POP (n=25) n (%)	Total (n=76) n (%)	p ^a
Smoking				0.33 ^b
None	43 (84)	25 (100)	68 (89)	
Current	3 (6)	0 (0)	3 (4)	
Past	3 (6)	0 (0)	3 (4)	
Missing	2 (4)	0 (0)	2 (3)	
Diabetes				0.41 ^b
No	45 (88)	24 (96)	69 (91)	
Yes	6 (12)	1 (4)	7 (12)	
Hypertension				0.13
No	32 (63)	20 (80)	52 (68)	
Yes	19 (37)	5 (20)	24 (32)	
Hysterectomy				0.93
No	23 (45)	11 (44)	34 (45)	
Yes	28 (55)	14 (56)	42 (55)	

^a Chi-square test, unless otherwise noted;

^b Fisher's exact test

^c T test;

^d Wilcoxon rank sum tests;

^e All percentages do not total to 100 percent due to rounding.

Table 2

Distribution of pelvic floor characteristics evaluated by ultrasound for women with and without pelvic organ prolapse (POP), based on POP quantification examination ***

	POP (n=51) n (%)	No POP (n=25) n (%)	Total (n=76) n (%)	* p
LAD Category				0.57
LAD score 12–15	25 (49)	14 (56)	39 (51)	
LAD score 16–18	26 (51)	11 (44)	37 (49)	
Left MLH Avulsion				0.68
No	26 (51)	14 (56)	40 (53)	
Yes	25 (49)	11 (44)	36 (47)	
Right MLH Avulsion				0.28
No	28 (55)	17 (68)	45 (59)	
Yes	23 (45)	8 (32)	31 (41)	
Anal Rectal Angle				0.45
< 170	11 (22)	9 (36)	20 (26)	
170	26 (51)	14 (56)	40 (53)	
Missing	14 (27)	2 (8)	16 (21)	
Median (min, max)	180 (150, 180)	171 (145,180)		0.17 **
Levator Plate Descent Angle				0.64
> 9	30 (59)	16 (64)	46 (61)	
9	10 (20)	7 (28)	17 (22)	
Missing	11 (22)	2 (8)	13 (17)	
Median (min, max)	0 (–20.0,20.0))	0 (–17.1,20.9))		0.36 **
MLH Area				0.45
14	4 (7.8)	4 (16.0)	8 (10.5)	
>14	36 (70.6)	19 (76.0)	55 (72.4)	
Missing	11 (21.6)	2 (8.0)	13 (17.1)	
Median (min, max)	19.7 (4.6)	17.5 (3.5)		0.048 **

* Chi-square test, unless otherwise noted;

** Wilcoxon rank sum tests

*** All percentages do not total to 100 percent due to rounding.

Table 3Modified Poisson Regression Models Estimating Prevalence Portion Ratios for Associations with POP^{*}

		Crude PPR (95% CI)	Adjusted PPR (95% CI) ^{**}
Age	50	2.55 (1.22–5.34)	2.39 (1.03–5.55)
Smoking		1.55 (1.29–1.85)	1.34 (1.08–1.67)
MLH area	17 cm ²	1.40 (0.90–2.17)	1.36 (0.87–2.13)

^{*} Analyses excludes 3 nulliparous subjects;

^{**} Adjusted for other factors in table