Original Article

The Effect of Body Mass Index on Pelvic Floor Support 1 Year Postpartum

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Abstract

Elevated body mass index (BMI) is associated with the incidence, prevalence, and progression of pelvic organ prolapse (POP). This study investigated the effect of peripartum BMI on pelvic floor support 1 year postpartum (PP1y). One hundred eight nulliparous women had their BMI recorded and underwent POP assessments using the Pelvic Organ Prolapse Quantification System at baseline, third trimester (36th to 38th week of pregnancy [G36-38w]), and PP1y. Pelvic organ prolapse was defined as ≥stage II. Women gained on average 1.9 kg between baseline and PP1y. After adjustment, increasing BMI PP1y was associated with increasing anterior wall descent ($P < .0001$) and higher odds of having POP PP1y (odds ratio: 1.41, 95% confidence interval: 1.01-1.97, $P = .045$). Trial of labor compared to unlabored cesarean delivery, POP G36-38w, and decreased fetal weight were independently associated with anterior vaginal wall laxity PP1y. Our finding suggests that postpartum BMI influences pelvic floor laxity 1 year after delivery. Postpartum weight reduction may serve as a strategy for POP prevention in some women.

Keywords

body mass index, trial of labor, cesarean delivery, pelvic organ prolapse, postpartum

Introduction

Pelvic organ prolapse (POP) affects up to 50% of women older than 40 years, leading to decreased quality of life in a large group of women as they transition into midlife.[1-3] Pelvic organ prolapse is implicated as the discriminating diagnosis in over 300 000 gynecological surgeries performed annually.[4] Overall, women have an 11.1% risk of undergoing at least 1 operation for either POP or urinary incontinence by the age of 80 years,[5] and up to 29% of women will require reoperation for POP.[3] Direct costs of POP surgery exceed 1 billion dollars annually in the United States alone, and it is estimated that the rate of women seeking treatment for POP will double over the next 30 years as the elderly population rapidly expands.[6,7]

Factors associated with pregnancy and parturition are known to predispose to POP later in life.[8-15] Most attention has focused on the impact of vaginal delivery which has been found to be the strongest risk factor for clinically significant POP.[8-15] Despite its known detrimental effects to the pelvic floor, vaginal delivery does not account for POP in all women, and cesarean delivery is not fully protective against postpartum pelvic floor laxity.[16-18] Thus, exploring other peripartum factors that may impact pelvic floor support may be beneficial.

Increased body mass index (BMI) has been consistently reported to play a role in the occurrence of clinically significant POP.[19-22] Findings from cross-sectional and prospective studies have identified associations between being overweight or obese and increased incidence, prevalence, and progression of POP.[19-22] Women gain on average between 0.5 and 3 kg[23] in weight from conception to 1 year postpartum (PP1y), and 15% to 20% of women retain more than 5 kg between 6 and 18 months postpartum.[24] We hypothesize that postpartum weight retention increases pelvic floor laxity after delivery. The goal of this study was to investigate whether changes in BMI after delivery affect pelvic floor support at PP1y.

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Methods

Study Population

This is a secondary analysis of data collected for a prospective, longitudinal study evaluating the effects of trial of labor (TOL) and unlabored cesarean delivery (UCD) on the development of POP in primiparous women up to one year after delivery. Between April 1, 2009, and May 31, 2009, 108 women who were in their 36th to 38th week of pregnancy (G36-38w) and planning to undergo a TOL or an elective UCD were recruited for participation during their routine prenatal care visit. Details of the study design, inclusion and exclusion criteria, and results have been reported previously.[15] The study was approved by the Wenzhou Third People's Hospital Institutional Review Board, and written consent was obtained from all participants before enrollment.

Evaluation of BMI and POP

Enrolled women were followed prospectively. Height was measured using a calibrated, wall-mounted stadiometer and rounded to the nearest centimeter. The weight of participants wearing street clothes, without shoes, was measured at all of the defined time points and recorded to the nearest 1 kg. Body mass index was calculated as weight in kilograms/height in meters squared (kg/m^2) and assessed at the first trimester (baseline), 36 to 38 weeks' gestation (G36-38w), and PP1y. Body mass index gain was defined as the difference between BMI PP1y and baseline BMI. The BMI classification was defined by criteria set by the Working Group on Obesity in China,[25] which take international variations into account. The following reference values for BMI were used: 18.5 to 23.9 kg/m^2 for the normal range, 24.0 to 27.9 kg/m^2 for overweight, and 28.0 kg/m^2 for obesity. The Pelvic Organ Prolapse Quantification System (POPQ) was used to assess pelvic floor support at G36-38w and PP1y. The POPQ measurements were recorded to the nearest 0.5 cm using wooden spatulas marked at 1-cm increments. All POPQ measurements were performed by a single investigator. Pelvic organ prolapse was defined as stage II descent or greater of the most prolapsed compartment. Demographic data, mode of delivery, and newborn infant weights were extracted from patient charts.

Statistical Analysis

The characteristics of the cohort were summarized with frequency, mean, and standard deviation as appropriate. Linear regression models were used to assess the relationships between BMI and POPQ measurements. Multivariable models were constructed starting from a model containing mode of delivery, age, fetal weight, baseline BMI, BMI gain or BMI at PP1Y, and smoking as possible predictor variables. For each POPQ point evaluated, the value of that point measurement at G36-38w was also included in the model. Backward stepwise selection was used to remove unnecessary variables from the model using $P \geq .05$ as the criterion for elimination. Logistic regression analysis was performed to model the dichotomous POP outcome at PP1y.

Table 1. Participant's Characteristics.[a]

Variable	Mean (SD) or N (%)
Age	26.6 (2.6)
Baseline BMI	19.7 (1.9)
BMI at PP1y	20.5 (1.6)
BMI gain	0.7 (1.1)
Type of delivery	
Trial of labor	79 (73%)
Unlabored cesarean delivery	29 (27%)
Newborn baby weight (kg)	3.4 (0.4)
Last completed education	
Primary (grades 1-6)	14 (13%)
Secondary (grades 7-12)	48 (44%)
College	46 (43%)
Smoking status	
Yes	6 (5.6%)
No	102 (94.4%)
POPQ measurements at G36-38w	
Aa/Ba	−1.7 (0.6)
Ap/BP	−2.7 (0.4)
Pb	2.5 (0.6)
Gh	3.0 (0.6)
C	−3.3 (0.9)
D	−6.8 (0.7)
TVL	7.8 (0.8)
POPQ measurements at PP1y	
Aa/Ba	−2.0 (0.7)
Ap/BP	−2.9 (.02)
Pb	2.2 (0.5)
Gh	2.7 (0.5)
C	−3.2 (0.8)
D	−6.8 (0.8)
TVL	6.9 (0.7)

Abbreviations: BMI, body mass index; BMI gain = body mass index 1 year postpartum (BMI PP1y) − BMI baseline; G36-38w = 36 to 38 weeks' gestation; POPQ, Pelvic Organ Prolapse Quantification System; PP1y = Postpartum 1 year.
[a]N = 108.

Results

One hundred eight nulliparous women were included in this analysis. Participant characteristics are included in Table 1. Thirty-seven women had at least 1 prior pregnancy. None of these women had pregnancies extending beyond the first trimester or prior vaginal deliveries. At baseline, the mean age of the participants was 26.6 (range, 20-34) years, and the mean BMI was 19.7 $\pm$ 1.8 kg/m^2 (Table 1). The majority of women were normal weight, both at baseline and at PP1y (98%, n = 106 and 96%, n = 104, respectively). On average, women gained 1.9 kg between baseline and PP1y. Twenty-two (20.5%) women lost weight, 6 (5.5%) women stayed the same weight, and 80 (74%) women gained between 1 and 7 kg between baseline and PP1y. Women with lower baseline BMI gained significantly more weight than women with higher baseline BMI between

Table 2. Multivariable Analysis of Factors Associated With Higher Point Aa/Ba at PP1y.

Explanatory Variable	Coefficient Estimate (β)	Standard Error	*P*
BMI PP1y	.1329	0.032	<.0001
Points Aa/Ba at G36-38w	.5724	0.083	<.0001
Trial of labor	.5211	0.115	<.0001
Fetal weight, kg	−.3100	0.127	.02

Abbreviations: BMI, body mass index; BMI PP1y, body mass index 1 year postpartum; G36-38w, 36 to 38 weeks' gestation.
Note. Multivariable models were constructed using variable selection of all covariates listed in Table 1 (excluding BMI gain) as explanatory variables. Significant covariates retained in the final model are listed and adjusted for the other covariates in this table.

Table 3. Multivariable Analysis of Factors Associated With Genital Hiatus Length.

Explanatory Variable	Coefficient Estimate (β)	Standard Error	*P*
Genital hiatus at G36-38w	.544	0.055	<.0001
BMI gain	.083	0.030	.009

Abbreviations: BMI, body mass index; BMI Gain, BMI PP1y (body mass index 1 year postpartum) − BMI first trimester; G36-38w, 36 to 38 weeks' gestation
Note. Multivariable models were constructed using variable selection of all covariates listed in Table 1 (excluding baseline BMI) as explanatory variables. Significant covariates retained in the final model are listed and adjusted for the other covariates in this table.

baseline and PP1y. The amount of weight gained between baseline and G36-38w was not associated with BMI at PP1y.

On linear regression analysis, increasing BMI PP1y was associated with increasing anterior wall descent (higher point Aa and Ba measurements) PP1y both before and after adjustment (Table 2). Specifically, a 1-unit (1 kg/m^2) increase in BMI PP1y was associated with a 0.13-cm increase in point Aa and Ba measurements PP1y. The G36-38w point Aa and Ba measurements, TOL (compared to UCD), and decreased fetal weight were independently associated with increased anterior wall laxity PP1y ($P < .001$; Table 2).

Increasing BMI gain was associated with increasing genital hiatus length (GHL) PP1y both before and after adjustment (Table 3). Genital hiatus length G36-38W was the only significant covariate associated with GHL PP1y after adjustment ($P < .0001$; Table 3). An increased GHL at G36-38w was strongly associated with an increased GHL PP1y. Body mass index PP1y and BMI gain were not significant predictors of any other POPQ measurements.

Twenty-three women (21%) had POP PP1y. To evaluate for factors associated with the presence of POP at PP1y, a logistic regression analysis was performed. Higher BMI PP1y was associated with an increased odds of having POP PP1y (OR: 1.41, 95% CI: 1.01-1.97, $P = .045$) before and after adjustment (Table 4). Trial of labor was the most significant risk factor for POP PP1y (Table 4).

Table 4. Multivariable Logistic Regression Analysis of Factors Associated With POP at 1 year Postpartum.

Factors	Odds Ratio	95% Confidence Interval	P_r (>|z|)
Trial of labor	7.95	1.49-42.50	.02
POP at G36-38w	4.02	1.46-11.1	.007
BMI PP1y	1.41	1.01-1.97	.045

Abbreviations: BMI, body mass index; BMI PP1y, BMI at 1 year postpartum; G36-38w = 36 to 38 weeks' gestation; POP, pelvic organ prolapse.
Note. 's' = variables.

Discussion

Factors associated with pregnancy and parturition have been consistently found to be associated with pelvic floor laxity. Consistent with our prior findings,[15] TOL was the biggest risk factor for anterior wall descent and POP 1 year after delivery. Here, we also show that after controlling for mode of delivery, increasing BMI at PP1y was associated with increasing anterior wall descent and increased odds of having POP at PP1y. A 1-unit increase in BMI 1 year after delivery was associated with a 41% increased odds of having POP PP1y. In addition, the net increase in BMI between the first trimester and PP1y was significantly associated with an increased GHL at PP1y. A weak but significant association between lower fetal weight and increased anterior vaginal wall descent was also noted, and the third trimester GHL was associated with GHL PP1y.

Previous authors have identified the relationship between POP progression in overweight and obese postmenopausal women.[20] Among their cohort of women, weight loss was not significantly associated with POP regression, and the authors concluded that damage to the pelvic floor related to weight gain might be irreversible.[20] Our data indicate that for normal-weight women, higher BMI postpartum affects the development of objective POP and, more specifically, anterior wall descent 1 year after delivery. Although the effect size appears small, it must be noted that the centimeter increase in point measurement is per unit change in BMI. Although the average BMI is normal in our patient population PP1y (mean: 20.5), our findings suggest that the value for points Aa and Ba would be at least 5 and 10 times greater (0.65 cm and 1.3 cm greater descent) for overweight and obese women, respectively. This is particularly concerning since current statistics suggest that in nearly 50% of the 34 countries represented by the Organization for Economic Co-operation and Development (OECD), 1 in every 2 persons is overweight or obese, with rates projected to increase over the next 10 years.[26] Longitudinal evaluations in overweight and obese women are needed to help clarify the relationship between BMI and anterior wall laxity among these populations.

Pelvic organ prolapse is a multifactorial condition, and our study focuses on the independent contribution of BMI 1 year after delivery. The remodeling process of the tissues is occurring in this time period,[15] and our data highlight the negative effect of postpartum weight retention on restoration of normal

pelvic anatomy postpartum. Studies characterizing the combined effects of postpartum weight retention and other known risk factors for pelvic floor laxity, such as aging and menopause, may help elucidate the impact of this modifiable risk factor on the development of symptomatic POP later in life.

Increased GHL is associated with the development of POP and poor long-term outcomes.[27,28] In women with POP, higher GHL correlates with more advanced POP and higher rates of POP recurrence following surgery.[27] Consistent with our findings, increased GHL in the third trimester of pregnancy has been found to be associated with worsening POP postpartum.[28] Our data implicate postpartum weight retention as a plausible factor that may be responsible, at least in part, for the differential increase in GHL size and development of POP postpartum. Our study was not designed to determine the interrelationship between pelvic floor laxity and GHL; however, postpartum weight retention appears to influence the biomechanics of the pelvic floor.

The strengths of our study are its prospective design, the use of objective outcome measures, and the implementation of multivariable models to adjust for relevant confounders. The 100% compliance rate of every participant at baseline, G36-38w, and PP1y further enhanced our ability to separate the influence of postpartum weight retention from pregnancy-associated weight gain on pelvic floor laxity PP1y. One of the main limitations of our study was our lack of subjective outcome assessments. However, in order to implement preventative strategies, it is imperative that we begin to understand factors that may contribute to the development of early pelvic floor laxity that typically develops in advance of symptoms; our study was appropriately designed to accomplish this. Another limitation was our lack of prepregnancy POP-Q measurements to serve as a true baseline to which to compare the observed changes in pelvic floor laxity. Hence, our study was not designed to assess for a causal relationship between BMI and incident POP. The inclusion of predominantly normal-weight Asian women limits the generalizability of the findings, however, the use of a more homogeneous population also provides a good model for isolating the effects of postpartum weight retention on postpartum pelvic floor support. As previously reported by Chen et al,[15] approximately 80% of women who delivered vaginally received a mediolateral episiotomy that was evident during postpartum evaluation. This may have led to bias in outcomes assessment. Additionally, there were only 2 forceps deliveries among the women studied. Larger studies, controlling for episiotomies, operative vaginal deliveries, and other labor and delivery elements known to impact the pelvic floor, are needed.

Much attention has been focused on how being overweight and obese leads to the development and progression of POP.[19-22] Our findings show that even small differences in BMI at PP1y can be among the initial insults that lead to pelvic floor laxity in normal-weight women. Our findings of an association between BMI and POP PP1y shed light on a potential critical, time-dependent, opportunity to modify the risk of developing POP for some women.

Author's Note

This secondary analysis was conceived, analyzed, and synthesized into a manuscript at Yale School of Medicine.

Declaration of Conflicting Interests

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