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RISK FACTORS FOR ANATOMIC PELVIC ORGAN PROLAPSE SIX WEEKS AFTER CHILDBIRTH

Hypothesis / aims of study

The aetiology of Pelvic organ prolapse (POP) is shown to be multifactorial with heritage (1), increasing age and parity (2) to be the most consistent risk factors. Finding that parous women have a higher prevalence of POP compared to nulliparous women has led to the perception that POP is a long term effect of an injury acquired during vaginal childbirth. This hypothesis has been supported by the discovery of major injuries to the levator ani muscle (LAM) occurring in one third of women with anatomic POP (aPOP), and exclusively occurring after vaginal deliveries (3). However, the fact that most women who deliver vaginally will not develop aPOP, that most women with aPOP does not have major LAM injuries, and that caesarean delivery is not fully protective for the development of POP indicate that we are still far from understanding the complex aetiology of POP and how childbearing interacts with the pelvic floor.

The aim of the present study was to identify risk factors for postpartum aPOP by comparing women with and without aPOP at six weeks postpartum with respect to pelvic floor measurements antepartum and obstetrical characteristics.

Study design, materials and methods

The present study is part of a prospective observational cohort study aiming at describing the pelvic floor from mid pregnancy until one year postpartum in a cohort of first time mothers. Data already presented from this cohort has shown that pelvic organ support changed both during pregnancy and following delivery, and that aPOP defined as pelvic organ prolapse quantification system (POP-Q) stage ≥ 2 had a peak prevalence of 9% at six weeks postpartum.

Nulliparous pregnant women with singleton pregnancies were followed from mid pregnancy to six weeks postpartum with repeat clinical examinations including POP-Q and transperineal three dimensional ultrasound. Background and obstetrical information was obtained from an electronic questionnaire and from the patient's electronic medical file respectively.

Associations were estimated using logistic regression analyses where the dependent variable was aPOP defined as POP-Q stage ≥ 2 at six weeks postpartum, and the independent variables were mid pregnancy measurements of selected POP-Q variables and levator hiatus area (LHarea), delivery route, and the presence of major levator ani muscle (LAM) injuries at six weeks postpartum.

Results

Of the 300 nulliparous pregnant women included in the cohort at mid pregnancy, 284 women had POP-Q data available for analyses at six weeks postpartum. These 284 women constitute our study sample.

At the first visit the mean gestational age of pregnancy was 21.0 weeks (SD1.4), mean maternal age was 28.7 years (SD 4.3), and mean maternal body mass index (BMI) was 25.8 kg/m^2 (SD 3.9).

At delivery mean gestational age was 40.1 weeks (SD1.5), and mean foetal birth weight was 3497 grams (SD 508). 196 women (69%) had normal vaginal delivery, 41 women (14%) had vacuum delivery and 4 women (1%) had forceps or combined vacuum and forceps delivery. 76 women (27%) had mediolateral episiotomy. 21 women (7%) had prelabour caesarean delivery, and 22 women (8%) had intralabour caesarean delivery (cervical dilatation≥3 cm)

At the follow- up at 6.2 weeks postpartum (SD 1.1), 25 women (9%) had aPOP, all stage 2 prolapses, whereof 22 had anterior prolapse and 3 had posterior prolapse, and major LAM injuries were diagnosed in 46 women (16%).

Comparing women with and without aPOP at six weeks postpartum with independent sample T-test there was no statistically significant difference regarding age, BMI, height, gestational age at birth, foetal birth weight, maternal height/ foetal birth weight-ratio, or use of mediolateral episiotomy.

A larger LH area at Valsalva, a longer distance from the meatus urethra to anus (Gh+Pb), and a more caudal position of the anterior vaginal wall (Ba) at mid pregnancy, gave increased risk for aPOP at six weeks postpartum, while delivery route and presence of major LAM injuries did not. Adjusting for maternal age and body mass index (BMI) did not alter the results.

Variable	aPOP six weeks postpartum					
	Crude OR	**[95% CI]**	**p-value**	**Adjusted OR**	**[95% CI]**	**p-value**
LHareaValsalva at gw 21 (cm^2)	1.13	[1.05-1.21]	<0.01	1.11	[1.04-1.20]	<0.01
Ba at gw 21 (cm)	2.49	[1.33-4.64]	<0.01	2.45	[1.29-4.67]	<0.01
Gh+Pb at gw 21 (cm)	1.65	[1.12-2.44]	0.01	1.58	[1.05-2.38]	0.03
Delivery route						
Vaginal delivery	4.65	[0.61-35.28]	0.14	5.69	[0.73-44.14]	0.10
Caesarean delivery (ref)	1					
Major LAM injury at 6wpp						
Yes	1.73	[0.65-4.60]	0.27	1.90	[0.70-5.15]	0.21
No injury (ref)	1					

Interpretation of results

Finding that prelabour differences in the pelvic floor, rather than obstetrical events, were risk factors for aPOP at six weeks postpartum, supports genetic predisposition to be an important etiologic factor for POP.

An enlarged LH has been attributed either to childbirth injury or to the presence of the prolapse itself, but according to our results, an enlarged LH in women with postpartum aPOP is a trait not only preceding the development of POP, it is also preceding childbirth. Constitutionally enlarged LH area seems to have a dual role; it enables uncomplicated vaginal delivery, but it may also increase the risk of aPOP later in life. The low prevalence of aPOP at six weeks postpartum might increase the risk of type II error in this study.

Concluding message

Prelabour differences in the pelvic floor rather than obstetrical events were risk factors for aPOP at six weeks postpartum.

References

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