RISK FACTORS FOR ANATOMIC PELVIC ORGAN PROLAPSE AT SIX WEEKS POSTPARTUM: a prospective observational study

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Abstract

Objective: Identify risk factors for postpartum anatomic pelvic organ prolapse (aPOP) by comparing women with and without aPOP at six weeks postpartum with respect to pelvic floor measurements antepartum and obstetrical characteristics.

Design: Prospective observational cohort study including nulliparous pregnant women

Setting: Norwegian university hospital

Methods: Participants underwent clinical examinations including pelvic organ prolapse quantification system (POP-Q) and transperineal ultrasound at gestational week 21 and at six weeks postpartum. 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.

Main outcome measures: The dependent variable was aPOP defined as POP-Q stage ≥ 2 at six weeks postpartum. 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: A larger LHarea, a more distensible LAM, 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 were risk factors for aPOP at six weeks postpartum, while delivery route and presence of major LAM injuries were not.

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

KEYWORDS:

Pelvic organ prolapse, Pelvic floor, POP, Postpartum, Pregnancy.

BRIEF SUMMARY

Antepartum pelvic floor differences rather than obstetrical events were risk factors for POP at six weeks postpartum

SHORT VERSION OF TITLE

Risk factors for anatomic POP at six weeks postpartum

Introduction

The etiology of pelvic organ prolapse (POP) is complex, and is shown to be multifactorial with heritage, increasing parity and age to be important risk factors (1-4). The integrated lifespan model presented by DeLancey et al(5) describes predisposing and inciting causal factors for the development of POP where childbirth is considered as an important inciting factor. The delay, often by several decades, from childbearing to the manifestation of POP is explained by an initial recovery of the pelvic floor postpartum. This initial recovery of the pelvic floor after pregnancy and delivery has made identification of risk factors related to childbearing challenging, and to date very few prospective studies exist. Retrospective and cross-sectional studies have suggested that major injuries to the levator ani muscle (LAM) are important risk factors for POP being present in one third of patients with POP, and exclusively in women who have delivered vaginally (6-8). An enlarged levator hiatus (LH) is another feature found to be associated both to POP and to childbearing, but whether caused by connective tissue weakness, pregnancy, childbirth injury or by the presence of the prolapse itself is debated (9-14).

The fact that most women who deliver vaginally will not develop POP, and that most women with POP do not have major LAM injuries, indicate that we are still far from understanding the complex etiology of POP and how childbearing interacts with the pelvic floor.

This study is part of a prospective observational study aiming at describing the natural history of the pelvic floor from mid pregnancy until one year postpartum in a cohort of first time mothers, using the pelvic organ prolapse quantification system (POP-Q) and transperineal three dimensional (3D) ultrasound (15-21). Data presented from this cohort has shown that pelvic organ support changed both during pregnancy and following delivery. Anatomic POP (aPOP) defined as POP-Q stage ≥ 2 reached a peak prevalence of nine per cent at six weeks

postpartum followed by a recovery to a prevalence of two per cent at one year postpartum (17). In the present study we wanted to study risk factors for having aPOP postpartum. To avoid the effect of the initial recovery of the pelvic floor that previously has made identification of risk factors related to childbearing challenging we chose to stratify our sample already at 6 weeks postpartum.

The aim of the present study was to identify possible predisposing and inciting risk factors for aPOP by comparing women with and without aPOP at six weeks postpartum with regards to predelivery characteristics such as pelvic floor measurements including POP-Q variables and LHarea at mid pregnancy, and obstetrical characteristics including delivery route and major LAM injuries identified at six weeks postpartum.

Methods

The present study is part of a prospective observational cohort study following 300 nulliparous pregnant women from mid pregnancy until one year postpartum. The study was conducted at Akershus University hospital in Norway from January 2010 to October 2012, and was approved by the Regional Ethics Committee (REK Sør-Øst D 2009/170) and the hospital Privacy Ombudsman (2799026). The participants gave their written informed consent to participate. The public healthcare system in Norway is organized in such a way that virtually all pregnant women within the geographic area of a hospital will attend the routine ultrasound examination in the second trimester at this hospital, and when due, will deliver at the same hospital. All healthcare related to pregnancy is free. Akershus University hospital is located in the vicinity of Oslo, and is one of the largest obstetric units in Norway with 4500 deliveries per year. During the inclusion period all nulliparous pregnant women aiming to

deliver at the hospital received written information about the study together with the invitation for routine ultrasound examination at 18-22 weeks of gestation. Following the ultrasound examination women with a singleton pregnancy and Scandinavian language skills were invited to participate in the study by the project coordinator. Exclusion criteria were serious illness to mother or fetus, prior pregnancy of more than 16 weeks gestation, and missing POP-Q data at six weeks postpartum. Continuing exclusion criteria were premature delivery prior to 32 weeks of gestation and stillbirth. The women included in the cohort have been shown to be comparable to the total population of nulliparous pregnant women who delivered at the hospital during the inclusion period (n=2621) with regards to age, body mass index (BMI), fetal birth weight and delivery route, but more women in the study sample had university or college education (75.3% compared with 50.8%, P<0.001)(17). The study participants were predominantly of Caucasian ethnicity (96%).

An electronic questionnaire answered by the participants provided background information, while obstetric data was obtained from the woman's electronic medical file in the hospital (DIPS / PARTUS C). The clinical examinations were performed at mid pregnancy and at 6 weeks postpartum by two trained gynecologists blinded to previous findings and the obstetric history of the participants. POP-Q was done according to standardized methods previously described in detail (17). Transperineal 3D ultrasound describing LHarea and major LAM injuries was done according to the methods described by Dietz defining major LAM injuries by a defect in the muscle evident in at least three consecutive tomographic slices at and/or above the plane of minimal hiatal dimension (22). Reliability for assessments of LH area and major LAM injuries has been tested and found to be good in separate studies from this cohort (20, 21).

The dependent variable was aPOP defined as POP-Q stage ≥ 2 at six weeks postpartum.

Independent variables were:

A) Pelvic floor measurements at mid pregnancy including selected POP-Q variables representing the three vaginal compartments (Ba, Bp, C) and the sum of the two measurements of the external genitalia (Gh and Pb) as well as LHarea at rest and Valsalva. LAM distensibility was estimated by calculating the difference in LHarea from rest to Valsalva (cm^2).

B) Delivery route, and the presence of major LAM injuries at six weeks postpartum.

There was no a priori power calculation done for this study.

Statistical methods

Background variables, pelvic floor measurements and obstetrical variables are presented as mean (SD), or as counts (percentages) as appropriate. Differences between the two groups were tested by the use of chi square test for categorical variables, and with independent samples T-test for continuous variables.

Possible crude association between aPOP at six weeks postpartum and the following variables; LHarea at Valsalva, LAM distensibility, Ba, and Gh+Pb at gestational week 21, delivery route, and presence of Major LAM injuries at six weeks postpartum, were analyzed using univariate logistic regression models. In addition each of the above described models was adjusted for maternal age and body mass index at gestational week 21.

All tests were two-sided and p-values < 0.05 were considered statistically significant.

All statistical analyses were performed using SPSS v 20.0.

Results

Of the 300 women included in the cohort at gestational week 21, five women had been excluded, ten had dropped out and one refused POP-Q examination at six weeks postpartum, leaving 284 women with POP-Q data to constitute our study sample. Background and obstetrical characteristics did not differ between women remaining in the study and women lost to follow up (data not shown).

At six weeks postpartum 25 women (9%) had aPOP defined as POP-Q stage ≥ 2, of which twenty-two had POP-Q stage 2 anterior prolapse and three had POP-Q stage 2 posterior prolapse. Table 1 shows maternal characteristics at gestational week 21, and obstetrical characteristics recorded either at delivery or at six weeks postpartum for the total study sample and for the women with and without aPOP at six weeks postpartum. There were no statistically significant differences between women with and without aPOP regarding maternal age, height, weight or body mass index (BMI) at 21 weeks of gestation.

Women with aPOP at six weeks postpartum had a more caudal position of the anterior vaginal wall (Ba), and a longer distance from the meatus urethra to the anus (Gh+Pb). There were no differences in the position of the cervix (c) or the posterior vaginal wall (Bp) between the two groups.

Women with aPOP had a larger LHarea at rest and at Valsalva, as well as a more distensible LAM at gestational week 21 compared to women without aPOP at six weeks postpartum.

Comparing obstetrical data between the two groups, our data did not reveal any differences in gestational age at birth, fetal birth weight, or maternal height / fetal birth weight ratio. Differences in vaginal versus abdominal delivery rate was not statistically significant. The subgroups of delivery mode (normal vaginal delivery versus operative vaginal delivery and

prelabour versus intralabour caesarean delivery) were too small for meaningful statistical analyses. It is worth noting that one woman with postpartum aPOP had caesarean delivery at 3 cm cervical dilatation due to failure to progress. There was no difference in episiotomy-rate between the vaginal deliveries in the two groups. The ten women in this cohort diagnosed with anal sphincter injury were all in the non-aPOP group.

The difference in prevalence of major LAM injuries was not statistically significant between women with (6/25=24%) and without (40/259=15%) aPOP at six weeks postpartum.
Table 2 presents crude and adjusted odds ratios (OR) for the association between aPOP at six weeks postpartum and the following variables; LHarea at Valsalva, LAM distensibility, Ba, and Gh+Pb measured at mid pregnancy, delivery route, and presence of major LAM injuries at six weeks postpartum. Women with larger LHarea at Valsalva, a more distensible LAM, a more caudal position of Ba and a longer distance from the meatus urethra to anus (Gh+Pb) at mid pregnancy had a higher risk of aPOP at six weeks postpartum. Adjusting for maternal age and BMI did not alter these associations. Delivery route and Major LAM injuries were not associated to aPOP at six weeks postpartum.

Discussion

Main findings

Comparing women with and without aPOP at six weeks postpartum with regard to predisposing factors and obstetrical inciting factors we found that most pelvic floor measurements were different already in pregnancy while there were no statistically significant differences in delivery route or presence of major LAM injuries.

Strength and limitations

Major strengths of this study are the comparatively large sample size and the prospective longitudinal design. Given that pelvic organ support changes over time both ante- and postpartum, the fixed time points relative to delivery for the assessments is another strength of this study.

Limitations of this study are the lack of data prior to, and from early pregnancy. A follow up of 10-20 years after delivery would have enabled us to test the assumption that women with transient aPOP after childbearing have a higher risk of manifest aPOP later in life. Another limitation is that the POP-Q system is not validated for women in pregnancy or postpartum.

Despite having a comparatively large cohort followed longitudinally with clinical assessments of the pelvic floor, anatomic POP had a low prevalence, increasing the risk of type II error when evaluating whether LAM injuries and delivery route are risk factors for aPOP at six weeks postpartum.

Interpretation

Finding that prelabour differences in the pelvic floor, rather than obstetrical events, were risk factors for aPOP at six weeks postpartum, are in agreement with a prospective study by Sze et al finding that the proportion of women who had a stage II pelvic organ prolapse 6 weeks after spontaneous vaginal delivery was significantly higher in those with antepartum prolapse than those without antepartum pelvic support defect (23). Our findings are also in agreement with a study by van Veelen et al that found an increase during pregnancy in LH area and LH distensibility that persisted after childbirth independent of delivery mode, and hypothesize that this increased pelvic floor distensibility may play a role in the development of pelvic floor dysfunction later in life(24). A recent study from 2017 finding that enlarged LH area postpartum was not associated to intrapartum characteristics is also coherent with our

results(13). The finding that antepartum differences rather than obstetrical exposures were associated to postpartum aPOP supports genetic predisposition to be an important etiologic factor for POP. This is coherent with a study by Durnea et al(25) stating that the association between uterine prolapse and collagen III level, and the lack of correlation between mode of delivery and uterine prolapse grade is suggestive of an important congenital contribution to POP etiology. It is also coherent with a systematic review by Lince et al finding that women with POP are substantially more likely to have family members with the same condition compared to women without POP(4), and with a study by Dietz et al showing that bladder neck mobility is a heritable trait(26).

Comparing women with and without postpartum aPOP we find that both static and dynamic pelvic floor properties differ between the two groups at mid pregnancy. Whether these differences were apparent already prior to pregnancy or whether the women with postpartum aPOP simply respond differently to the hormones and increased mechanical stress of pregnancy this study is not designed to find out. A longitudinal study including women very early or prior to pregnancy could help explore these questions further.

The role of an enlarged LH area as a risk factor for POP seems to be complex; An enlarged LH has been attributed either to muscle or nerve injury caused by vaginal childbirth (10) or to the presence of the prolapse itself (11), 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. A traumatically enlarged LH following major LAM injury is well documented (27), but seems to represent a different matter altogether, mainly affecting women with an initially small and non-compliant LH (19). This heterogeneity of causes for enlarged LH area might explain why a cross sectional study of urogynaecological patients found that major LAM injuries and enlarged LH were independent risk factors for POP (12).

We suggest that future research should differentiate between enlarged LH area with and without concurrent major LAM injury.

A constitutionally enlarged LH area seems to have a dual role; it is shown to enable uncomplicated vaginal delivery (14, 18, 28), but it may also increase the risk of aPOP later in life. This study shows that an enlarged LH area antepartum increases the risk of transient aPOP at six weeks postpartum, but whether it also increases the risk of manifest aPOP later in life needs to be explored in studies with longer follow-up.

The lack of association between aPOP and LAM injury shortly after delivery is in agreement with a study by Laterza et al (29), and is not contradictory to LAM injuries being an important risk factor for POP, it simply suggests that LAM injuries do usually not cause anatomic prolapse in the short term after childbirth. A recent study by Thomas et al shows that mean latency between first birth and presentation for prolapse surgery in women with avulsion was 33.5 (range 3-66) years (30). The fact that major LAM injuries is only found in one third of women with aPOP(6-8) shows that causal factors other than major LAM injuries are important in the etiology of POP.

The lack of association between delivery route and postpartum aPOP might be due to lack of statistical power, but the finding that one participant had aPOP after caesarean delivery on 3 cm cervical dilatation demonstrates that (unlike major LAM injuries) vaginal delivery is not a prerequisite for postpartum aPOP.

Conclusion

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

To further increase our understanding of the natural history of POP, larger studies comparing dynamic properties of the pelvic floor during pregnancy to prolapse status both in the short and the long term postpartum are needed.

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Table 1: Descriptive statistics of the study sample;

Maternal characteristics and pelvic floor measurements at gestational week 21 and obstetrical characteristics.

	Total study sample N=284	Women without aPOP 6 weeks postpartum N=259	Women with aPOP 6 weeks postpartum N=25
Maternal characteristics at gw 21	Mean (SD)	Mean (SD)	Mean (SD)
Maternal age (years)	28.7 (4.3)	28.6 (4.3)	30.1 (4.4)
Maternal height (m)	1.68 (0.6)	1.68 (0.1)	1.67 (0.1)
Maternal BMI (kg/m^2)	25.8 (3.9)	25.7 (3.9)	26.7 (3.7)
Pelvic floor measurements at gw 21			
Levator hiatus area at Valsalva (cm^2)	15.4 (4.9)	15.1 (4.7)*	18.8 (5.3)*
Levator hiatus area at rest (cm^2)	11.7 (2.2)	11.6 (2.2)*	12.9 (2.0)*
LAM distensibility (cm^2)	3.7 (3.7)	3.5 (3.7)*	5.9 (4.1)*
Ba	-2.7 (0.5)	-2.7 (0.5)*	-2.4 (0.7)*
Bp	-2.9 (0.4)	-2.9 (0.4)	-2.8 (0.4)
C	-7.6 (1.0)	-7.6 (1.0)	-7.7 (1.2)
Gh+Pb (cm)	7.2 (1.1)	7.2 (1.1)*	7.8 (1.1)*
Obstetrical characteristics			
Gestational age at birth (Weeks)	40.1 (1.5)	40.1 (1.5)	40.2 (1.4)
Foetal birth weight (gram)	3497 (508)	3493 (514)	3538 (443)
Maternal height / foetal birth weight-ratio (m/kg)	0.4898 (0.08)	0.4909 (0.08)	0.4787 (0.06)
	N (%)	N (%)	N (%)
Vaginal delivery	241 (85%)	217 (84%)	24 (96%)
• Normal vaginal delivery	• 196 (69%)	• 177 (68%)	• 19 (76%)
• Vacuum delivery	• 41 (14%)	• 36 (14%)	• 5 (20%)
• Forceps or combined vacuum and forceps delivery	• 4 (1%)	• 4 (1%)	• -
Caesarean delivery	43 (15%)	42 (16%)	1 (4%)
• Prelabour (cervical dilatation<3cm)	• 21 (7%)	• 21 (8%)	• -
• Intralabour (cervical dilatation≥3 cm)	• 22 (8%)	• 21 (8%)	• 1 (4%)
Mediolateral episiotomy	76 (27%)	66 (25%)	8 (32%)
Obstetric anal sphincter injury	10 (4%)	10 (4%)	-
Major LAM injuries 6 weeks postpartum	46 (16%)	40 (15%)	6 (24%)

aPOP= anatomic pelvic organ prolapse, gw= gestational week, SD= standard deviation, BMI= Body mass index, LAM= levator ani muscle.

* Statistically significant difference with $p< 0.05$

Table 2:

Logistic regression analysis of association between possible risk factors and anatomic pelvic organ prolapse (aPOP) at six weeks postpartum

	aPOP 6 weeks postpartum					
Variable	**Crude OR**	**[95% CI]**	**p-value**	**Adjusted OR**	**[95% CI]**	**p-value**
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
LHareaValsalva at gw 21 (cm^2)	1.13	[1.05-1.21]	<0.01	1.11	[1.04-1.20]	<0.01
LAM distensibility at gw 21, rest to Valsalva (cm^2)	1.14	[1.04-1.25]	<0.01	1.13	[1.03-1.23]	0.01
Major LAM injury at 6wpp • Yes • No injury (ref)	1.73 1	[0.65-4.60]	0.27	1.90	[0.70-5.15]	0.21
Delivery route • Vaginal delivery • Caesarean delivery (ref)	4.65 1	[0.61-35.28]	0.14	5.69	[0.73-44.14]	0.10

CI= confidence interval, OR= Odds Ratio, LHarea= Levator ani hiatus area, LAM= Levator ani muscle, gw= gestational week, wpp= weeks postpartum.

In the adjusted OR each variable is adjusted for maternal age and body mass index.