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ORIGINAL ARTICLE

Risk factors for anatomic pelvic organ prolapse at 6 weeks postpartum: a prospective observational study

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Abstract

Introduction and hypothesis The objective was to identify risk factors for postpartum anatomic pelvic organ prolapse (aPOP) by comparing women with and without aPOP at 6 weeks postpartum with regard to pelvic floor measurements antepartum and obstetrical characteristics.

Methods We carried out a prospective observational cohort study including nulliparous pregnant women in a Norwegian university hospital. Participants underwent clinical examinations, including pelvic organ prolapse quantification system (POP-Q) and transperineal ultrasound at gestational week 21 and at 6 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. The dependent variable was aPOP, defined as POP-Q stage ≥2 at 6 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 6 weeks postpartum.

Results A larger LHarea, a more distensible LAM, a longer distance from the meatus urethra to the anus (Gh + Pb) and a more caudal position of the anterior vaginal wall (Ba) at mid-pregnancy were risk factors for aPOP at 6 weeks postpartum, whereas delivery route and the presence of major LAM injuries were not.

Conclusion Prelabor differences in the pelvic floor rather than obstetrical events were risk factors for aPOP at 6 weeks postpartum.

Keywords Pelvic organ prolapse · Pelvic floor · POP · Postpartum · Pregnancy

Introduction

The etiology of pelvic organ prolapse (POP) is complex, and is shown to be multifactorial with heritage, increasing parity, and age being 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 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 with both POP and childbearing, but whether this is caused by connective tissue weakness, pregnancy, childbirth injury or by the presence of the prolapse itself is debated [9–14].

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The facts that most women who deliver vaginally do 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 aimed at describing the natural history of the pelvic floor from mid-pregnancy until 1 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 have 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 9 % at 6 weeks postpartum followed by a recovery to a prevalence of 2 % at 1 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 has previously made identification of risk factors related to childbearing challenging, we chose to stratify our sample as early as 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 6 weeks postpartum with regard 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 6 weeks postpartum.

Materials and methods

The present study is part of a prospective observational cohort study following 300 nulliparous pregnant women from mid-pregnancy until 1 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 participation. The public healthcare system in Norway is organized in such a way that virtually all pregnant women within the geographic area of a hospital attend the routine ultrasound examination in the second trimester at this hospital, and when due, 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 4,500 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, previous pregnancy of more than 16 weeks' gestation, and missing POP-Q data at 6 weeks postpartum. Continuing exclusion criteria were premature delivery before 32 weeks of gestation and stillbirth. The women included in the cohort have been shown to be comparable with the total population of nulliparous pregnant women who delivered at the hospital during the inclusion period ($n = 2{,}621$) with regard 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, whereas obstetric data were 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 assessed according to standardized methods previously described in detail [17]. Transperineal 3D ultrasound describing LHarea and major LAM injuries was carried out according to the methods described by Dietz et al., defining major LAM injuries as 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 6 weeks postpartum.

Independent variables were:

1. 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).
2. Delivery route, and the presence of major LAM injuries at 6 weeks postpartum.

No a priori power calculation was performed for this study.

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-squared test for categorical variables, and with independent samples t test for continuous variables.

A possible crude association between aPOP at 6 weeks postpartum and the following variables; LHarea at Valsalva, LAM distensibility, Ba, and Gh + Pb at gestational week 21, delivery route, and the presence of major LAM injuries at 6 weeks postpartum, were analyzed using univariate logistic regression models. In addition, each of the models described above was adjusted for maternal age and BMI 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 21 weeks' gestation, 5 women had been excluded, 10 had dropped out, and 1 refused POP-Q examination at 6 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 6 weeks postpartum, 25 women (9%) had aPOP defined as POP-Q stage ≥2, of whom 22 had POP-Q stage 2 anterior prolapse and 3 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 6 weeks postpartum for the total study sample and for the women with and without aPOP at 6 weeks postpartum. There were no statistically significant differences between women with and without aPOP regarding maternal age, height, weight or BMI at 21 weeks' gestation.

Women with aPOP at 6 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, and a more distensible LAM at gestational week 21 compared with women without aPOP at 6 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. Difference in the vaginal versus abdominal delivery rate was not statistically significant. The subgroups of delivery mode (normal vaginal delivery versus operative vaginal delivery and prelabor versus intralabor cesarean delivery) were too small for meaningful statistical analyses. It is worth noting that one woman with postpartum aPOP had cesarean delivery at 3 cm cervical dilatation owing to failure to progress. There was no difference in the episiotomy rate between the vaginal deliveries in the two groups. The 10 women in this cohort diagnosed with anal sphincter injury were all in the non-aPOP group.

The difference in the prevalence of major LAM injuries was not statistically significant in women with (6 out of 25, 24%) and without (40 out of 259, 15%) aPOP at 6 weeks postpartum.

Table 2 presents crude and adjusted odds ratios (OR) for the association between aPOP at 6 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 6 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 the anus (Gh + Pb) at mid-pregnancy had a higher risk of aPOP at 6 weeks postpartum. Adjusting for maternal age and BMI did not alter these associations. Delivery route and major LAM injuries were not associated with aPOP at 6 weeks postpartum.

Discussion

Main findings

Comparing women with and without aPOP at 6 weeks postpartum with regard to predisposing factors and obstetrical inciting factors, we found that most pelvic floor measurements were different already in pregnancy, whereas there were no statistically significant differences in delivery route or the 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 before, 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 are at 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 6 weeks postpartum.

Table 1 Descriptive statistics of the study sample; maternal characteristics, 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)			
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, mean (SD)			
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), mean (SD)	40.1 (1.5)	40.1 (1.5)	40.2 (1.4)
Fetal birth weight (g), mean (SD)	3,497 (508)	3,493 (514)	3,538 (443)
Maternal height/fetal birth weight-ratio (m/kg), mean (SD)	0.4898 (0.08)	0.4909 (0.08)	0.4787 (0.06)
Vaginal delivery, *n* (%)	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)	–
Cesarean delivery, *n* (%)	43 (15)	42 (16)	1 (4)
Prelabor (cervical dilatation<3 cm)	21 (7)	21 (8)	–
Intralabor (cervical dilatation≥3 cm)	22 (8)	21 (8)	1 (4)
Mediolateral episiotomy, *n* (%)	76 (27)	66 (25)	8 (32)
Obstetric anal sphincter injury, *n* (%)	10 (4)	10 (4)	–
Major LAM injuries 6 weeks postpartum, *n* (%)	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 $p < 0.05$

Interpretation

Finding that prelabor differences in the pelvic floor, rather than obstetrical events, were risk factors for aPOP at 6 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 also agree with those of 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 could 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 with intrapartum characteristics also corresponds with our results [13]. The finding that antepartum differences rather than obstetrical exposure were associated with postpartum aPOP supports the notion that genetic predisposition is an important etiological factor for POP. This is in agreement 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 also concurs with a systematic review by Lince et al., who found that women with POP are substantially more likely to have family members with the same condition than 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 before pregnancy, or whether

Table 2 Logistic regression analysis of association between possible risk factors and anatomic pelvic organ prolapse (aPOP) at 6 weeks postpartum

Variable	aPOP 6 weeks postpartum					
	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
LHarea Valsalva 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 6 weeks postpartum						
Yes	1.73	0.65–4.60	0.27	1.90	0.70–5.15	0.21
No injury (reference)	1					
Delivery route						
Vaginal delivery	4.65	0.61–35.28	0.14	5.69	0.73–44.14	0.10
Cesarean delivery (reference)	1					

CI confidence interval, *OR* odds ratio, *LHarea* levator ani hiatus area, *LAM* levator ani muscle, *GW* gestational week,
In the adjusted OR, each variable is adjusted for maternal age and BMI

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 in very early or before pregnancy could help to 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 that not only precedes the development of POP, it also precedes 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 noncompliant LH [19]. This heterogeneity of causes for enlarged LH area may 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 play 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 6 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 not usually cause anatomic prolapse in the short term after childbirth. A recent study by Thomas et al. shows that the 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 are found in only 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 may be due to lack of statistical power, but the finding that one participant had aPOP after cesarean delivery at 3-cm cervical dilatation demonstrates that (unlike major LAM injuries) vaginal delivery is not a prerequisite for postpartum aPOP.

Conclusion

Prelabor differences in the pelvic floor rather than obstetrical events were risk factors for aPOP at 6 weeks postpartum.

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

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Compliance with ethical standards

Ethical approval The Regional Ethics Committee (REK Southeast D 2009/170) and the Akershus University Hospital Privacy Ombudsman

(2799026) approved the study. All participants gave their written informed consent before entering the study.

All authors have revised the drafted article critically for important intellectual content and have approved the submitted article.

Conflicts of interest None.

References

1. Mant J, Painter R, Vessey M. Epidemiology of genital prolapse: observations from the Oxford family planning association study. Br J Obstet Gynaecol. 1997;104(5):579–85.
2. Swift S, Woodman P, O'Boyle A, Kahn M, Valley M, Bland D, et al. Pelvic organ support study (POSST): the distribution, clinical definition, and epidemiologic condition of pelvic organ support defects. Am J Obstet Gynecol. 2005;192(3):795–806.
3. Miedel A, Tegerstedt G, Maehle-Schmidt M, Nyren O, Hammarstrom M. Nonobstetric risk factors for symptomatic pelvic organ prolapse. Obstet Gynecol. 2009;113(5):1089–97.
4. Lince SL, van Kempen LC, Vierhout ME, Kluivers KB. A systematic review of clinical studies on hereditary factors in pelvic organ prolapse. Int Urogynecol J. 2012;23(10):1327–36.
5. DeLancey JOL, Kane Low L, Miller JM, Patel DA, Tumbarello JA. Graphic integration of causal factors of pelvic floor disorders: an integrated life span model. Am J Obstet Gynecol. 2008;199(6):610.e1–5.
6. Albrich S, Rommens K, Steetskamp J, Weyer V, Hoffmann G, Skala C, et al. Prevalence of levator ani defects in urogynecological patients. Geburtshilfe Frauenheilkd. 2015;75(1):51–5.
7. Volloyhaug I, Morkved S, Salvesen KA. Association between pelvic floor muscle trauma and pelvic organ prolapse 20 years after delivery. Int Urogynecol J. 2016;27(1):39–45.
8. Dietz HP, Simpson JM. Levator trauma is associated with pelvic organ prolapse. BJOG. 2008;115(8):979–84.
9. Shek KL, Kruger J, Dietz HP. The effect of pregnancy on hiatal dimensions and urethral mobility: an observational study. Int Urogynecol J. 2012;23(11):1561–7.
10. Shek KL, Dietz HP. The effect of childbirth on hiatal dimensions. Obstet Gynecol. 2009;113(6):1272–8.
11. Andrew BP, Shek KL, Chantarasorn V, Dietz HP. Enlargement of the levator hiatus in female pelvic organ prolapse: cause or effect? Aust N Z J Obstet Gynaecol. 2013;53(1):74–8.
12. Dietz HP, Franco AV, Shek KL, Kirby A. Avulsion injury and levator hiatal ballooning: two independent risk factors for prolapse? An observational study. Acta Obstet Gynecol Scand. 2012;91(2):211–4.
13. Caudwell-Hall J, Kamisan Atan I, Martin A, Guzman Rojas R, Langer S, Shek K, et al. Intrapartum predictors of maternal levator ani injury. Acta Obstet Gynecol Scand. 2017;96(4):426–31.
14. Toozs-Hobson P, Balmforth J, Cardozo L, Khullar V, Athanasiou S. The effect of mode of delivery on pelvic floor functional anatomy. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19(3):407–16.
15. Staer-Jensen J, Siafarikas F, Hilde G, Bo K, Engh ME. Ultrasonographic evaluation of pelvic organ support during pregnancy. Obstet Gynecol. 2013;122(2 Pt 1):329–36.
16. Staer-Jensen J, Siafarikas F, Hilde G, Benth JS, Bo K, Engh ME. Postpartum recovery of levator hiatus and bladder neck mobility in relation to pregnancy. Obstet Gynecol. 2015;125(3):531–9.
17. Reimers C, Staer-Jensen J, Siafarikas F, Saltyte-Benth J, Bo K, Ellstrom EM. Change in pelvic organ support during pregnancy and the first year postpartum: a longitudinal study. BJOG. 2016;123(5):821–9.
18. Siafarikas F, Staer-Jensen J, Hilde G, Bo K, Ellstrom EM. Levator hiatus dimensions in late pregnancy and the process of labor: a 3- and 4-dimensional transperineal ultrasound study. Am J Obstet Gynecol. 2014;210(5):484.e1–7.
19. Siafarikas F, Staer-Jensen J, Hilde G, Bo K, Ellstrom EM. The levator ani muscle during pregnancy and major levator ani muscle defects diagnosed postpartum: a three- and four-dimensional transperineal ultrasound study. BJOG. 2015;122(8):1083–91.
20. Staer-Jensen J, Siafarikas F, Hilde G, Braekken IH, Bo K, Engh ME. Pelvic floor muscle injuries 6 weeks post partum—an intra- and inter-rater study. Neurourol Urodyn. 2013;32(7):993–7.
21. Siafarikas F, Staer-Jensen J, Braekken IH, Bo K, Engh ME. Learning process for performing and analyzing 3D/4D transperineal ultrasound imaging and interobserver reliability study. Ultrasound Obstet Gynecol. 2013;41(3):312–7.
22. Dietz HP, Bernardo MJ, Kirby A, Shek KL. Minimal criteria for the diagnosis of avulsion of the puborectalis muscle by tomographic ultrasound. Int Urogynecol J. 2011;22(6):699–704.
23. Sze EH, Sherard GB 3rd, Dolezal JM. Pregnancy, labor, delivery, and pelvic organ prolapse. Obstet Gynecol. 2002;100(5 Pt 1):981–6.
24. Van Veelen GA, Schweitzer KJ, van der Vaart CH. Ultrasound imaging of the pelvic floor: changes in anatomy during and after first pregnancy. Ultrasound Obstet Gynecol. 2014;44(4):476–80.
25. Durnea CM, Khashan AS, Kenny LC, Durnea UA, Smyth MM, O'Reilly BA. Prevalence, etiology and risk factors of pelvic organ prolapse in premenopausal primiparous women. Int Urogynecol J. 2014;25(11):1463–70.
26. Dietz HP, Hansell NK, Grace ME, Eldridge AM, Clarke B, Martin NG. Bladder neck mobility is a heritable trait. BJOG. 2005;112(3):334–9.
27. Abdool Z, Shek KL, Dietz HP. The effect of levator avulsion on hiatal dimension and function. Am J Obstet Gynecol. 2009;201(1):89.e1–5.
28. Oliphant SS, Nygaard IE, Zong W, Canavan TP, Moalli PA. Maternal adaptations in preparation for parturition predict uncomplicated spontaneous delivery outcome. Am J Obstet Gynecol. 2014;211(6):630 e1–7.
29. Laterza RM, Schrutka L, Umek W, Albrich S, Koelbl H. Pelvic floor dysfunction after levator trauma 1-year postpartum: a prospective case-control study. Int Urogynecol J. 2015;26(1):41–7.
30. Thomas V, Shek KL, Guzman Rojas R, Dietz HP. Temporal latency between pelvic floor trauma and presentation for prolapse surgery: a retrospective observational study. Int Urogynecol J. 2015;26(8):1185–9.