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ORIGINAL ARTICLE

Does childbirth play a role in the etiology of rectocele?

Rodrigo Guzmán Rojas · Christian Quintero · Ka Lai Shek · Hans Peter Dietz

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Abstract
Introduction and hypothesis Rectoceles are common among parous women and they are believed to be due to disruption or distension of the rectovaginal septum as a result of childbirth. However, the etiology of rectocele is likely to be more complex since posterior compartment prolapse does occur in nulliparous women. This study was designed to determine the role of childbearing as an etiological factor in true radiological rectocele.
Methods This was a secondary analysis of the data from 657 primiparous women recruited as part of a previously reported study and another ongoing prospective study. Women were invited for antenatal and postnatal appointments comprising an interview, clinical examination and translabial ultrasonography. The presence and depth of any rectocele were determined on maximum Valsalva maneuver, as was descent of the rectal ampulla. Potential demographic and obstetric factors as predictors of rectocele development were evaluated using either multiple regression or logistic regression analysis as appropriate.
Results A true rectocele was identified in 4 % of women antenatally and in 16 % after childbirth ($P<0.001$). Mean rectocele depth was 13.5 mm (10 – 23.2 mm). The mean antepartum position of the rectal ampulla on Valsalva maneuver was 4.39 mm above and it was 1.64 mm below the symphysis pubis postpartum ($P<0.0001$). De novo appearance of true rectocele was significantly associated with a history of previous <20 weeks pregnancy and fetal birth weight. Body mass index and length of the second stage were associated with rectocele depth increase.
Conclusions Childbirth seems to play a distinct role in the pathogenesis of rectocele. Both maternal and fetal factors seem to contribute.

Keywords Childbirth · Rectocele · Transperineal ultrasonography · 3D/4D ultrasonography

R. Guzmán Rojas · K. L. Shek · H. P. Dietz
Department of Obstetrics and Gynecology, Sydney Medical School Nepean, University of Sydney, Penrith, Australia

R. Guzmán Rojas (✉)
Departamento de Ginecología y Obstetricia Clínica Alemana de Santiago, Facultad de Medicina Clínica Alemana, Universidad del Desarrollo, Santiago, Chile
e-mail: Rodrigoguzman.66@gmail.com

C. Quintero
Female Pelvic Medicine and Reconstructive Surgery, University of Nevada School of Medicine, Reno, NV, USA

R. Guzmán Rojas
Departamento de Ginecología y Obstetricia, Hospital Clínico de la Universidad de Chile, Santiago, Chile

Introduction

Pelvic organ prolapse is a prevalent condition, affecting millions of women throughout the world [1]. Up to 76 % of patients have been shown to have defective pelvic organ support of the posterior compartment [2]. Among the various conditions that may give rise to clinical prolapse of posterior compartment including perineal descent, enterocele and rectal intussusception, rectocele seems to be a major cause [3].

A true rectocele is defined as herniation of the anterior wall of the rectal ampulla into the vagina, and is thought to be due to a defect in the rectovaginal septum [4] leading to bulging of the posterior vaginal wall depicted on examination during a Valsalva maneuver. This abnormality of the posterior compartment is associated with symptoms of prolapse (vaginal lump or bulge or dragging sensation) and obstructed defecation [5, 6] such as incomplete bowel emptying, straining at stool, and digitation (i.e. splinting the posterior vagina with one or more fingers in order to assist evacuation of the bowel). True rectoceles may be seen in young nulliparous women, and have been shown to be associated with higher body mass index (BMI) and a history of constipation [7]. It has been

hypothesized that in some women defects in the rectovaginal septum may be congenital. Childbirth, however, seems to also play a role in the etiology of rectocele [8]. In this quoted study in 68 women, 2 women were found having a rectocele antepartum and 6 more women postpartum. However, the small dataset did not allow the authors to further evaluate the role of various maternal, fetal and intrapartum factors in the etiology of rectocele.

In order to fill this knowledge gap we set out to design a study on a larger dataset to determine the prevalence and depth of true radiological rectocele in a cohort of nulliparous women before and after childbirth and to evaluate the etiological role of various demographic and obstetric factors in this condition.

Materials and methods

This was a secondary analysis of the data from 657 primiparous women recruited as part of a previously reported study [9] and another ongoing prospective study [10] between Nov 2006 and Nov 2011. Women were invited for antenatal and postnatal appointments that comprised a standardized interview including symptoms of prolapse (vaginal lump or bulge or dragging sensation) and obstructed defecation, clinical examination including modified Oxford grading [11, 12], and a translabial 4D ultrasound examination using Voluson 730 expert systems with RAB 8 – 4 MHz transducers (GE Kretz Ultrasound, Zipf, Austria). Assessments were undertaken as previously described with the subject supine and after voiding [13]. Ultrasound volumes were acquired at rest, on Valsalva maneuver and pelvic floor muscle contraction (PFMC). At least three Valsalva maneuvers were performed and the volume acquired on maximum Valsalva maneuver, i.e. showing the furthest descent of the pelvic organs, was used for analysis. Volume datasets were analyzed offline on a personal computer using the software GE Kretz 4DView v.10.0 (GE Medical Ultrasound, Ryde NSW, Australia) by an operator blinded to all other data. All volume datasets stored contained cine loops of 12 – 36 volumes encompassing the entire Valsalva and PFMC maneuvers.

Levator avulsion was diagnosed on tomographic ultrasound imaging on maximum PFMC as described previously [14], with a dataset rated positive for avulsion if the puborectalis insertion of all the central three slices was abnormal. Irreversible over-distension ('microtrauma') was defined as an increase of over 20 % in hiatal area on maximum Valsalva maneuver on antenatal compared with postnatal volume datasets [9]. The presence and depth of a rectocele was determined on maximum Valsalva maneuver, as was the descent of the rectal ampulla (Fig. 1). A true rectocele, i.e. a defect of the rectovaginal septum, was diagnosed if there was a discontinuity in the anterior anorectal muscularis (Fig. 2) measuring ≥10 mm in depth [3].

We investigated demographic and obstetric factors as predictors of rectocele development including age, BMI, delivery mode, birth weight, fetal head circumference, length of second stage, epidural analgesia, oxytocin use, and episiotomy. Statistical analysis was undertaken using Minitab version 13 software (Minitab Inc., State College, PA) and SAS 9.2 software (SAS Inc., Cary, NC). The *t* test was used for continuous normally distributed data. Normality was checked using the Kolmogorov–Smirnov method. The chi-squared test was used for categorical variables. Associations between outcomes and predictors were evaluated using either multiple regression or logistic regression as appropriate. A test–retest series (n=20) for rectal descent, rectocele depth, and presence of rectovaginal septal defect was conducted between the first and second author to determine the reproducibility of these ultrasound parameters. Ethics approval was obtained from Sydney West Area Health Service Human Research Ethics Committee for both parent studies (reference SWAHS HREC 05-004 and 07-022). Written informed consent for this study was obtained from all participants.

We did not perform power calculations specific to the research question addressed in this article, as the study was a subanalysis of the above-mentioned parent projects.

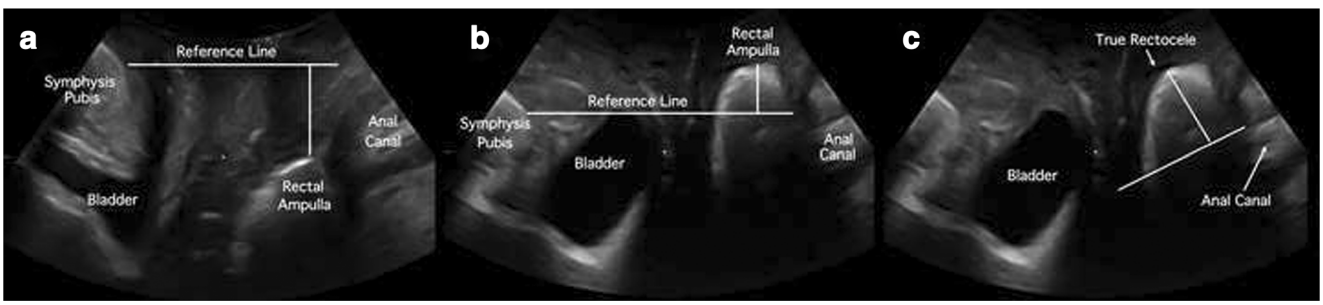

Fig. 1 Midsagittal plane on translabial imaging at rest (**a**) and on Valsalva maneuver (**b**, **c**): **a**; **b** position of the rectal ampulla, **c** rectocele depth

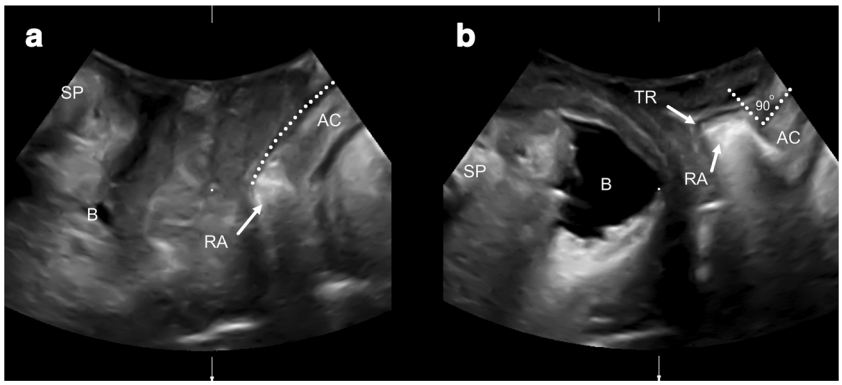

Fig. 2 Midsagittal plane on translabial imaging at rest (**a**) and on Valsalva maneuver (**b**): **a** *dotted line* anterior contour of the anorectal muscularis; **b** *dotted line* 90° disruption on the anterior contour of the anorectal muscularis (*SP* symphysis pubis, *B* bladder, *RA* rectal ampulla, *TR* true rectocele, *AC* anal canal)

Results

A test–retest of 20 ultrasound volume datasets showed strong agreement on ultrasound measurements for descent of the rectal ampulla (ICC 0.712, 95 % CI 0.410 – 0.874) and rectocele depth (ICC 0.749, 95 % CI 0.466 – 0.893), and a Cohen's kappa of 0.71 for the diagnosis of a defect of the rectovaginal septum. Of 657 primiparous women originally recruited, 518 (78.9 %) returned for follow up at 3 – 4 months. In five women we were unable to locate the ultrasound volumes and in one the posterior compartment could not be assessed, and three women were pregnant at the postnatal assessment and one had already delivered a second child, leaving 508 datasets for the subsequent analysis. The average gestational age at first assessment was 36.4 weeks (range 33.2 – 38.3 weeks). The mean age was 29.5 years (range 18.1 – 45.3 years). Of 499 women, 423 (85 %) were of Caucasian ethnicity. Average BMI at antenatal assessment was 29.6 kg/m^2 (range 19.6 – 56.5 kg/m^2). Mean follow-up after childbirth was 5 months (median 4.4 months, interquartile range 3.8 – 5.43 months). Delivery was vaginal in 380 women (75 %; normal delivery in 278, 55 %; ventouse in 66, 13 %; forceps in 36, 7 %). Caesarean section before onset of labor was performed in 26 women (5 %), in the first stage in 76 (15 %) and in the second stage in 26 (5 %). The median length of the first stage was 415 min (interquartile range 270 – 614 min), and of the second stage 60 min. Levator avulsion was diagnosed in 56 women (11 %). The mean postpartum hiatal area on Valsalva maneuver was 21.5 cm^2 (6.7 – 52.9), and by comparing these measurements with antenatal values, 'microtrauma' or irreversible overdistension of the hiatus was diagnosed in 159 patients (31 %).

Before delivery 109 women (21 %) reported symptoms of obstructed defecation (incomplete bowel emptying and/or straining at stool and/or digitation) and 18 (4 %) of prolapse (dragging sensation and/or a vaginal lump). A ¨true rectocele¨ was identified in 18 women (4 %) antenatally; it was symptomatic in 6 (5 obstructed defecation and 1 prolapse and obstructed defecation symptoms). There was a significant increase in the prevalence of true rectocele after childbirth (18, 4 %, vs. 83, 16 %; $P<0.001$, chi-squared test). In five women a true rectocele was detected antenatally but not at the postpartum assessment. Most of the rectoceles were small (mean 12.8 mm, range 10 – 18.1 mm, antepartum; mean 13.45 mm, range 10 – 23.2 mm, postpartum). Just 4 antepartum rectoceles (1 %) and 27 postpartum rectoceles (5 %) were above a cut-off value of 15 mm for a 'clinically relevant' rectocele. Of the patients in whom a postpartum rectocele was diagnosed, 24 were symptomatic (17 with symptoms of obstructed defecation only, 6 with prolapse symptoms only, and 1 with both), and 59 were asymptomatic. True rectocele was not significantly associated with symptoms of obstructed defecation, either antepartum ($P=0.38$, chi-squared test) or postpartum ($P=0.84$, chi-squared test).

The average position of the rectal ampulla antepartum on Valsalva maneuver was 4.39 mm (39.6 to −29.9 mm) above the symphysis pubis, and postpartum was 1.64 mm (37.6 to −35.5 mm) below the symphysis pubis ($P<0.0001$, paired t test). Table 1 shows the association between de novo rectocele and increase in rectocele depth (any increase in depth of a true rectocele diagnosed antepartum) with demographic and obstetric variables and levator avulsion. After controlling for confounders on multivariate logistic regression, a history of previous pregnancy (before 20 weeks) and fetal birth weight remained significantly associated with de novo rectocele. Increase in rectocele depth was significantly associated with BMI and length of the second stage.

Discussion

To date the role of childbirth in the etiology of rectocele remains unclear. In this large observational series, a true rectocele was found in a small minority of pregnant nulliparous women (4 %), but the prevalence increased markedly by the time of postpartum appointment at a mean of 5 months after a first birth (16 %, $P<0.0001$). These findings confirm those of a previous smaller study [8] suggesting that childbirth

Table 1 Associations between de novo rectocele and increase in rectocele depth and demographic and obstetric variables in univariate and multivariate analyses

Predictors	De novo rectocele (n=70/508)				Increase in rectocele depth (n=82/508)			
	Univariate odds ratio (95 % CI)	*P* value	Multivariate odds ratio (95 % CI)[a]	*P* value	Univariate estimate (95 % CI)	*P* value	Multivariate estimate (95 % CI)[a]	*P* value
Age	1.04 (0.99 – 1.09)	0.14			0.03 (−0.05 – 0.11)	0.49		
BMI	1.05 (1.00 – 1.1)	0.04	1.03 (0.99 – 1.08)	0.17	1.08 (1.02 – 1.13)	0.02	1.06 (1.01 – 1.10)	0.03
Previous pregnancy	1.96 (1.12 – 3.31)	0.01	1.88 (1.11 – 3.19)	0.02	0.87 (0.11 – 1.84)	0.08	0.77 (−0.42 – 1.95)	0.2
Birth weight	1.08 (1.02 – 1.15)	0.01	1.07 (1.01 – 1.13)	0.03	0.12 (0.03 – 0.22)	0.01	0.07 (−006 – 0.20)	0.3
Head circumference	1.11 (0.92 – 1.35)	0.28			0.11 (−0.22 – 0.44)	0.51		
Gestational age	1.17 (0.93 – 1.46)	0.19			0.25 (−0.19 – 0.63)	0.18		
Epidural anesthesia	1.05 (0.63 – 1.77)	0.84			0.24 (−0.63 – 1.11)	0.58		
Second stage length	1.003 (0.99 – 1.01)	0.14			1.21 (1.11 – 1.86)	0.002	1.16 (1.08 – 1.67)	0.006
Delivery mode (vaginal/nonvaginal)	1.41 (0.75 – 2.62)	0.28			0.75 (−0.24 – 1.75)	0.14		
Episiotomy	1.22 (0.67 – 2.24)	0.51			0.48 (0.61 – 1.57)	0.39		
Levator avulsion (yes/no)	2.01 (1.06 – 4.13)	0.03	1.99 (0.98 – 4.01)	0.08	−1.25 (−2.74 – 0.24)	0.1		

[a] Includes variables with $P<0.1$ in the univariate analysis

may play a role in the etiology of rectocele. Demographic and obstetric variables were, however, poor predictors of de novo rectocele development in multivariate analysis. De novo rectocele was weakly associated with a history of pregnancy of <20 weeks and fetal birth weight. An interesting finding is the association between a history of previous pregnancy and de novo rectocele development, and this may suggest a hormonal effect of pregnancy, which needs to be studied further.

An increase in rectocele depth was weakly associated with BMI and length of the second stage. A weak association between BMI and rectocele has also been noted in a previous study in nulliparous women [7] and in the Women's Health Initiative Hormone Replacement Therapy Clinical Trial in menopausal women [1]. However, the link between BMI and pelvic floor dysfunction appears to be more complex than expected. A raised BMI has been negatively associated with levator trauma [15] and may be protective against persistent denervation of the levator ani at 6 months postpartum and anal sphincter laceration [16, 17] . Future studies should evaluate the association between BMI and various forms of pelvic floor disorder more closely.

Based on the findings of the current study it is plausible that the etiological mechanisms in rectocele are similar to those in other forms of pelvic floor disorder, in which mechanical and connective tissue abnormalities may play a role. Some defects were found to have enlarged and others appeared de novo after a first birth. The theory that the birth process could open up defects of the rectovaginal septum or Denonvillier's fascia provides the basis for surgical management in the form of defect-specific repair [18], and presupposes traumatic stretching of posterior vaginal wall structures during passage of the fetal head. The significant effects of birth weight and length of the second stage argue in favor of this concept, but it is surprising that we did not document any significant impact of delivery mode and that levator avulsion was not statistically significant in the multivariate analysis. This may have been due to insufficient power, but even then one would have to postulate a small effect size. Episiotomy, which has been acknowledged as a risk factor for anal sphincter trauma [19], but not necessarily for pelvic organ prolapse and urinary incontinence [20], was not predictive either. Other risk factors that have been associated with rectocele include previous colposuspension [21] and prolapse surgery [22]. This may be explained by changes in the forces (pressure) exerted on the vagina as a result of alteration in its axis after these procedures.

There was no association between rectocele and symptoms of obstructed defecation, either antenatally or postnatally. This was not surprising, since most rectoceles observed by us were small and therefore more likely to have been asymptomatic. At the postpartum appointment only 27 out of 508 women showed a rectocele of 15 mm or more in

depth, which has been suggested as a cut-off value for a 'clinically relevant' rectocele, i.e. a rectocele that is likely to be associated with incomplete bowel emptying/vaginal digitation as has been shown using the receiver operator curve analysis [23].

The strength of the present study includes its prospective design and the large study population, which allowed evaluation of various demographic and obstetric factors in the etiology of rectocele. Furthermore, diagnosis of a true rectocele was made on ultrasound examination rather than on clinical examination, which has been shown to have limited success in differentiating the various conditions leading to posterior compartment prolapse, and therefore may have reduced the power of the study. Furthermore, both ultrasound assessment and evaluation of ultrasound data were performed blinded to clinical information, therefore avoiding observer bias.

Several limitations of the study have to be acknowledged. This was a secondary analysis of datasets from two prospective studies; power calculation was therefore not performed to address the research question of the current study. While to our knowledge this is the largest series in which this topic has been addressed, a follow-up rate of only 79 % may be considered a weakness. As the vast majority of the study population were Caucasians, the results may, not be applicable to other ethnic groups. In addition, one could argue that the use of late third trimester data rather than pre-pregnancy data may have prevented evaluation of the potential role of pregnancy itself on rectocele. Hormonal effects of pregnancy may induce neurological and biomechanical changes to the pelvic floor [16, 24], affecting the mechanical anatomy of the rectovaginal septum. This is supported by the observation in this study that a history of previous pregnancy to <20 weeks was associated with de novo true rectocele. Suboptimal Valsalva maneuver, e.g. Valsalva maneuver for less than 5 s [25], and levator coactivation [26] may explain the detection of rectocele antenatally but not postnatally in five women.

In conclusion, childbirth seems to play a distinct role in the pathogenesis of rectocele. Both maternal and fetal factors seem to contribute.

Conflict of interest H.P. Dietz and K.L. Shek have received unrestricted educational grants from GE.

No other conflict of interest is disclosed.

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