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ORIGINAL ARTICLE

Pubovisceral muscle and anal sphincter defects in women with fecal or urinary incontinence after vaginal delivery

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Abstract

Background Vaginal delivery is the most frequent cause of direct anal sphincter trauma as well as pelvic floor muscle defects in women with corresponding signs and symptoms. The aim of the present study was to identify anatomical and functional abnormalities of the anal canal and pelvic floor in women who had had a vaginal delivery and determine the relationship between such abnormalities and the symptoms and severity of fecal incontinence (FI).

Methods Consecutive female patients with symptoms of fecal and/or urinary incontinence were recruited through the colorectal and gynecological outpatient clinics at two large university hospitals and were eligible if they had had at a vaginal delivery. All women were assessed for symptoms FI by means of the Cleveland Clinic Florida Incontinence Scale (CCFIS) and for urinary incontinence symptoms, including the presence of complaints of any involuntary leakage of urine, leakage on exertion, sneezing, or coughing, and/or leaking or losing urine associated with an urge to urinate. All women underwent anorectal and endovaginal three-dimensional ultrasonography and anal manometry. The extent of the anal sphincter and PVM defects identified by ultrasound was scored from 1 to 6 based on the longitudinal involvement of the external and internal anal sphincter, the radial angle of the anterior external anal sphincter defect and the longitudinal involvement of the PVM.

Results There were 130 women and 89 (68%) had at least one defect of the anal sphincter or the pubovisceral muscle or both (42/32% had a pubovisceral muscle defect with or without sphincter defects, 47/36% women had an intact pubovisceral muscle but sphincter defect); and 41 (32%) had intact anal sphincter and pubovisceral muscles. The mean levator hiatus area at rest in women with anal sphincter and/or pubovisceral muscle defects was 18 (± 4 SD) which was significantly greater than in women with no defects (16 ± 3 SD; $p = 0.01$). Women with PVM defects had significantly higher ultrasound scores (median ultrasound score = 4/range 1–10 vs Intact = 2/range 2–5), indicating more extensive defects ($p = 0.001$). Bivariate analysis revealed a positive association ($p < 0.05$) between increasing FI symptom severity (CCFIS score) and women with PVM defects ($\rho = 0.6913$). Within the group of women with defects mean maximum anal squeeze pressure was significantly lower in women with PVM defect (mean 73 ± 34 SD mmHg vs mean 93 ± 38 SD; $p = 0.04$). Women with PVM defects had significantly higher median CCFIS scores (median score, 7/range 0–16) compared to women with intact PVM (4/range 0–10) ($p < 0.001$). There was a significant positive correlation between the CCFIS and ultrasound scores ($\rho = 0.625$; $p < 0.001$). Bivariate analysis revealed a negative correlations between the CCFIS score and the lengths of the anterior EAS ($\rho = -0.5621$, $p < 0.001$), IAS ($\rho = -0.40$, $p < 0.001$) and the area of the levator hiatus ($\rho = 0.5211$, $p = 0.001$). However, no significant correlations were observed between CCFIS scores and the gap measurement ($\rho = 0.101$; $p = 0.253$) or the resting ($\rho = -0.08$, $p = 0.54$) or squeeze pressure ($\rho = -0.12$; $p = 0.34$) values on anal manometry. The variables associated with worsening FI symptom severity (CCFIS score) that remained significant in multiple linear regression included the shorter lengths of the anterior EAS and/or the lengths of the anterior IAS and increased area of the levator hiatus.

Conclusions The study data demonstrate that half of the women had combined defects of PVM and sphincter. There were correlations between anatomical abnormalities including the anal sphincter and/or pubovisceral muscle defects with decrease in the anal pressures and increased severity of FI.

Keywords Pelvic floor · Fecal incontinence · Sphincter defect · Ultrasound

Extended author information available on the last page of the article

Introduction

Studies using multiplanar, high spatial resolution imaging methods such as three-dimensional (3D) anal endosonography and magnetic resonance imaging have demonstrated the asymmetrical conformation of the anal canal and distribution of the sphincter muscles [1–5]. The anterior part of the external anal sphincter (EAS) is the shortest part and is positioned more distally in relation to the posterior and lateral EAS and puborectalis muscles in both men and women. Furthermore, in the anterior quadrant, the muscles (especially the anterior EAS) are shorter and the gap is longer in women than in men, resulting in a weak area that may explain the higher incidence of pelvic floor dysfunction in women [6].

Vaginal delivery is the most frequent cause of direct anal sphincter trauma in women [7–9]. Anatomical changes can be detected in women who have undergone vaginal delivery, even in the absence of a defect and fecal incontinence (FI) [10]. Furthermore, studies have visualized anatomical abnormalities of pelvic floor muscles, such as traumatic avulsion of the pubovisceral muscle (PVM) (synonymous with the pubococcygeus and puborectalis muscles, as defined by DeLancey) [11] from the pubis after vaginal delivery during the second stage of labor. The PVM becomes detached, or partially detached, from its insertion to the pubis in 15–55% of women [12–15], with corresponding signs and symptoms of pelvic organ prolapse (POP) and enlargement of the levator hiatu [16–18].

In recent studies of women with previous vaginal delivery and FI symptoms, 3D endovaginal and anorectal ultrasonography showed a direct correlation between avulsions of PVM and severity of FI [19, 20]. However, considering the complex asymmetrical conformation of the anal canal, especially in women, it is still unclear whether defects of the pubovisceral and anal sphincter muscles affect the occurrence and severity of FI symptoms [17, 21, 22]. Therefore, the aim of this study was to identify anatomical and functional abnormalities of the anal canal and pelvic floor in women who had had a vaginal delivery and determine the relationship between such abnormalities and the symptoms and severity of FI.

Materials and methods

Patient selection

From October 2015 through March 2017, consecutive female patients with a previous vaginal delivery with fecal and/or urinary symptoms were recruited through the colorectal outpatient clinics at the Hospital Walter Cantídio of the Federal University of Ceara and São Carlos Hospital-Fortaleza-CE, Brazil, and those without fecal and/or urinary symptoms were recruited through the gynecological outpatient clinics. The study was approved by the hospital's research ethics committee and all participants gave their informed consent.

All women were assessed for symptoms of fecal and urinary incontinence and had endovaginal and anorectal 3D ultrasonography and anal manometry. Women with anal sphincter and/or PVM defects were compared with those with no sphincter or pubovisceral muscle defects.

Patients with inflammatory bowel disease, human immunodeficiency virus infection, obesity, diabetes, or neurological disorders were excluded, as were patients with a history of previous colorectal, anorectal, or gynecological surgery.

Assessments

3D ultrasonography

3D endovaginal and anorectal ultrasonography was performed by a single colorectal surgeon with experience in 3D ultrasonography (S.M.M.R.). A rectal enema was administered 2 h before the scan. A 3D ultrasound endoprobe was used (Pro-Focus 2052).

Patients were in the dorsal lithotomy position and the endovaginal approach was performed with 12 MHz of frequence and focal distance of 5.2 cm. The transducer in the vagina in the neutral position, and it was introduced as far as the bladder. The scanning identified the PVM at the point where the inferior branches of the pubic bones join at the symphysis pubis as a hyperechoic sling lying posteriorly to the anorectal junction and anal canal and attaching to the pubic bone (Fig. 1a). The ultrasound examination identified the whole length of the PVM attached to the pubic bone in the coronal plane. Defects were defined as detachment (discontinuity) of the PVM from its insertion on the pubic rami. The defect was considered as partial if the length of the muscle was partially compromised and as complete detachment if the whole length of the muscle was compromised (Fig. 2a, b). The detachment was either unilateral (involving just one side) or bilateral (both sides) (Fig. 3a, b).

The area of the levator hiatus was delimited by the internal margin of the PVM and the internal margin of the symphysis pubis in the axial plane, and compared at rest vs during the Valsalva maneuver (Figs. 2a, b, 3a, b). The position of the anorectal junction was measured in the sagittal plane from the anorectal junction (corresponding to the proximal margin of the puborectalis muscles) to the lowest margin of the symphysis pubis at rest and during the Valsalva maneuver [23] (Fig. 4a, b).

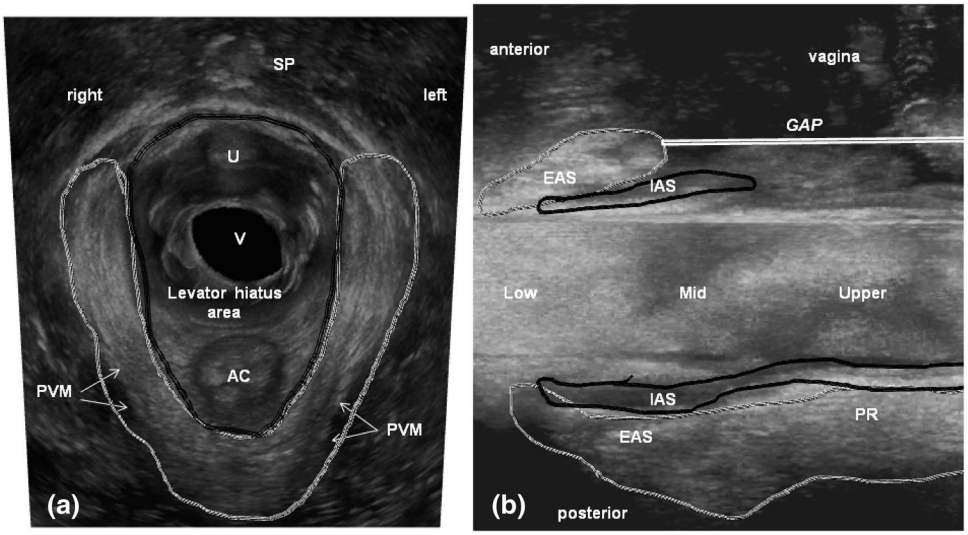


Fig. 1 3D-ultrasound—patient with intact pubovisceral muscle and anal sphincter muscles after vaginal delivery (render-mode). **a** Endovaginal approach showing intact pubovisceral muscle and the area of the levator hiatus (black drawn) (axial plane). **b** Anorectal approach showing intact sphincter muscle (sagittal plane). *U* urethra, *V* vagina, *AC* anal canal, *PMV* pubovisceral muscles, *SP* symphysis pubis, *EAS* external anal sphincter, *IAS* internal anal sphincter, *PR* puborectalis muscle

Anorectal ultrasonography was performed with patients in the left lateral position and the endoprobe with 16 MHz and a focal distance of 3.0 cm was introduced as far as the upper anal canal. The ultrasound examination was used to measure the length of the anterior EAS, the anterior IAS, the posterior EAS plus the puborectalis muscle, the posterior IAS and the gap between the proximal edge of the anterior EAS (Fig. 1b) and the anorectal junction (corresponding to the proximal margin of the puborectalis muscles) as well as identify combined defects involving both the EAS and the internal anal sphincter (IAS) (Fig. 2c–e) and defects of the EAS alone (Fig. 3c–e). The length of each sphincter muscle was measured as the distance from to the proximal to the distal edge of the muscles in sagittal plane.

Ultrasound score (severity of the defect)

The extent of the anal sphincter and PVM defects identified by ultrasound examination was described by a score based on the longitudinal involvement (none, partial, or total length) of the EAS and IAS, the radial angle of the anterior EAS defect ($\leq 180°$ or $> 180°$), and the longitudinal involvement (none, partial or total length) of the left and right lateral pubovisceral muscles. In this scoring system, 0 denotes an intact muscle, 1 point is allotted for partial length detachment and 2 points for total length detachment, and 2 points are allotted for an angle of the EAS defect $> 180°$, so that combined and extended lesions result in higher ultrasound scores, with a maximum score of 10 [20] as shown in Table 1.

Anorectal manometry

Anorectal manometry was performed using a flexible, water-perfused polyethylene catheter with an 8-channel manometer with ProctoMaster software to calculate the length of the anal canal, anal canal pressure at rest, maximum anal squeeze pressure, and capacity required to sustain squeeze pressure, and to evaluate pressure during straining and the rectoanal inhibitory reflex. Rectal sensitivity (corresponding to the first sensation of rectal filling) and the maximum tolerable volume were also measured.

Fecal incontinence

FIis defined as the uncontrolled passage of feces or gas for at least 1 month in an individual at least 4 years old, who had previously achieved control [24].

Patients with FI were assessed by means of the Cleveland Clinic Florida Incontinence Scale (CCFIS) [25] to quantify the severity of the symptoms.

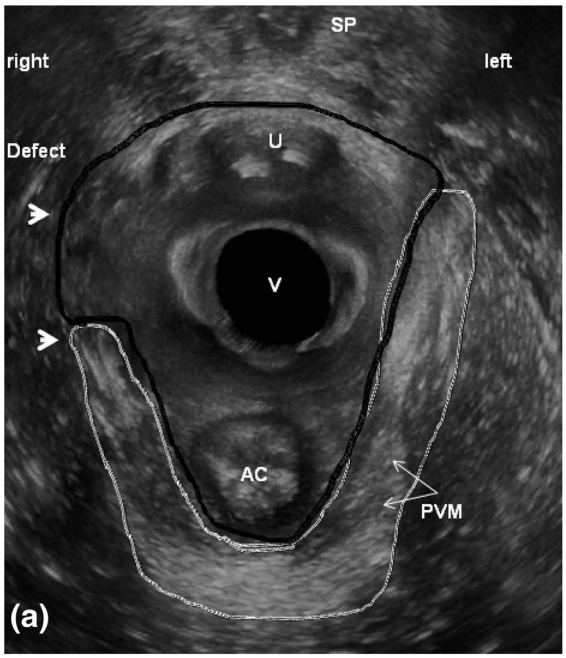
Urinary incontinence

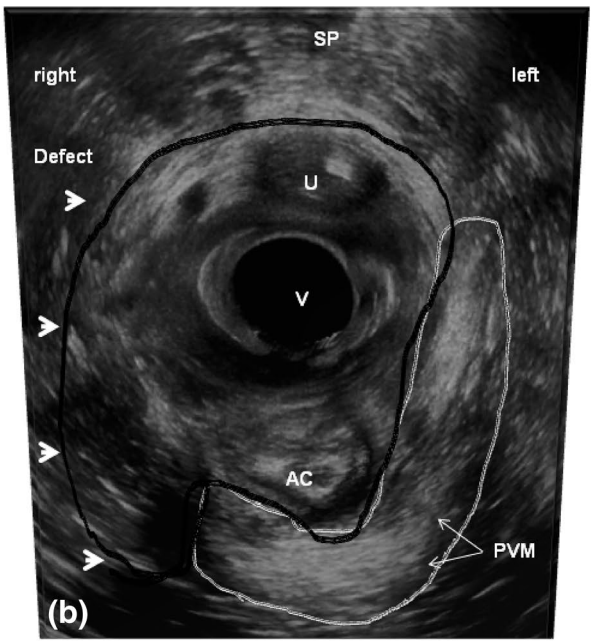
Urinary incontinence was assessed according to standardized terminology proposed by the International Continence Society, [26] including the presence of complaints of any involuntary leakage of urine, leakage on exertion, sneezing, or coughing, and/or leaking or losing urine associated with an urge to urinate.

Statistical analysis

Differences between patients with anal sphincter and/or PVM defects and those with intact sphincter and PVM were assessed with Student's *t* test for continuous data and the Mann–Whitney *U* test for ordinal data.

Bivariate analyses were performed to examine associations between patients who complained of FI symptoms and the patients with anal sphincter and/or PVM defects.


SP
right
left
Defect
U
V
AC
PVM
(a)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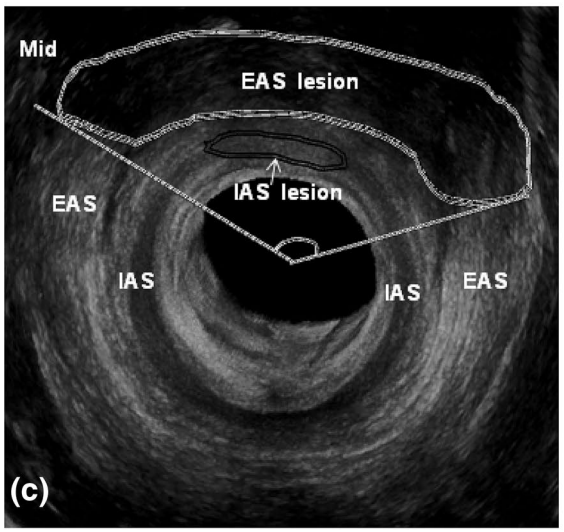
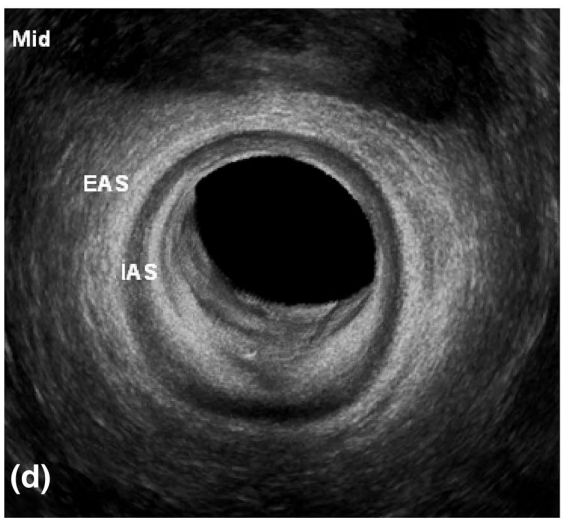
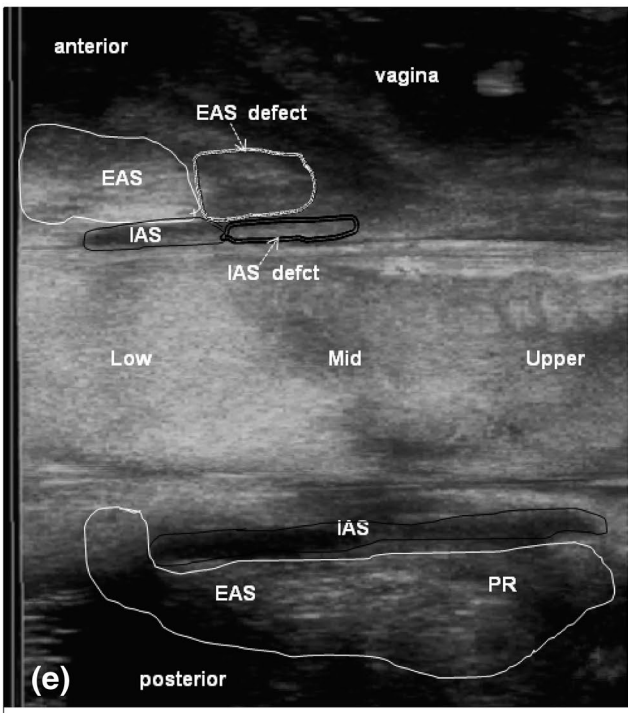

Mid
EAS lesion
IAS lesion
EAS
IAS
IAS
EAS
(c)


Mid
EAS
IAS
(d)


anterior
vagina
EAS defect
EAS
IAS
IAS defct
Low
Mid
Upper
iAS
EAS
PR
(e)
posterior

◂**Fig. 2** 3D-ultrasound—patient with unilateral pubovisceral muscle defect and combined partial external and internal anal sphincter defects after vaginal delivery (render-mode). **a** Endovaginal approach showing right lateral pubovisceral muscle defect (arrows head) and area of the levator hiatus at rest (black drawn) (axial plane). **b** Endovaginal approach—increased area of the levator hiatus in the Valsalva manuever (black drawn). **c** Anorectal approach showing combined sphincter muscle defects in the anterior quadrant and angle of the external anal spincter defect in the mid anal canal (axial plane). **d** Anorectal approach showing intact sphincter muscles in the distal part of the mid anal canal (axial plane). **e** Anorectal approach showing combined sphincter muscle defects and residual external and internal anal sphincter length (sagittal plane). *U* urethra, *V* vagina, *AC* anal canal, *PMV* pubovisceral muscles, *SP* symphysis pubis, *EAS* external anal sphincter, *IAS* internal anal sphincter, *PR* puborectalis muscle

The Spearman rank correlation coefficient (ρ) was used for assessing bivariate analyses to evaluate the relationship between severity of FI symptoms (CCFIS score) and ultrasound findings (score, lengths of the anterior EAS, anterior IAS, gap measurements, area of the levator hiatus, and anal pressures measured by anorectal manometry).

We also conducted multivariable linear regression analysis to evaluate the relationship between severity of FI symptoms (CCFIS score) and all parameters assessed to account for potential confounding factors. Variables with a p value less than 0.10 were retained in the final model. p values less than 0.05 were considered statistically significant. Data were analyzed using SPSS software (version 14.0 for Windows; IBM-SPSS, Chicago, IL, USA).

Results

Patient characteristics

There were a total of 130 women, mean age 59.5 (± 12.5) years. Overall, the number of vaginal delivery was 3.2 (± 2.3) and 98 (75%) complained of FI and 18 (14%) complained of urinary incontinence symptoms. Of the 130 women, 88 (68%) had at least one defect of the anal sphincter or the PVM or both, while 42 (32%) had intact anal sphincter and pubovisceral muscles. As shown in Table 2, 42 (32%) women had a PVM defect with or without anal sphincter defects, and 47 (36%) women had intact an PVM plus a defect of the EAS alone ($n = 31$) or defects of both the EAS and the IAS ($n = 16$).

Patients with anal sphincter and/or PVM defects were significantly older than those without defects ($p = 0.02$; Table 3). The two groups did not differ in the number of vaginal deliveries ($p = 0.97$), episiotomies, or forceps deliveries.

3D anorectal and endovaginal ultrasonography findings

The results of 3D anorectal and endovaginal ultrasonography are summarized in Tables 4 and 5. The median ultrasound score was 3 (range 1–10) in the group with anal sphincter and/or PVM defects. Regardless of whether they had a sphincter defect, women with PVM defects had significantly higher ultrasound scores (median ultrasound score = 4/range 1–10 vs intact = 2/range 2–5), indicating more extensive defects, compared with women with intact PVM ($p = 0.001$).

The mean levator hiatus area in women with anal sphincter and/or PVM defects was significantly greater than in women with no defects, both at rest and during the Valsalva maneuver (Table 4). Furthermore, the mean area of the levator hiatus in women with at least one defect was significantly larger during the Valsalva maneuver than at rest ($p = 0.01$), but no significant difference was seen in patients without defects ($p = 0.22$). The anorectal junction position was significantly lower in patients with defects compared with those with no defects, both at rest and during the Valsalva maneuver (Table 3).

The lengths of the anterior EAS, anterior IAS, posterior EAS plus puborectalis muscle, posterior IAS, and the gap between the proximal edge of the anterior EAS and the anorectal junction were measured by means of anorectal ultrasonography in 35 women (20 with anal sphincter and/or PVM defects and 15 without defects). The lengths of the anterior EAS, anterior IAS, posterior EAS plus puborectalis muscle, and posterior IAS were significantly shorter, and the gap was significantly longer, in women with anal sphincter and/or PVM defects than in women without defects (Table 5).

Anal manometry findings

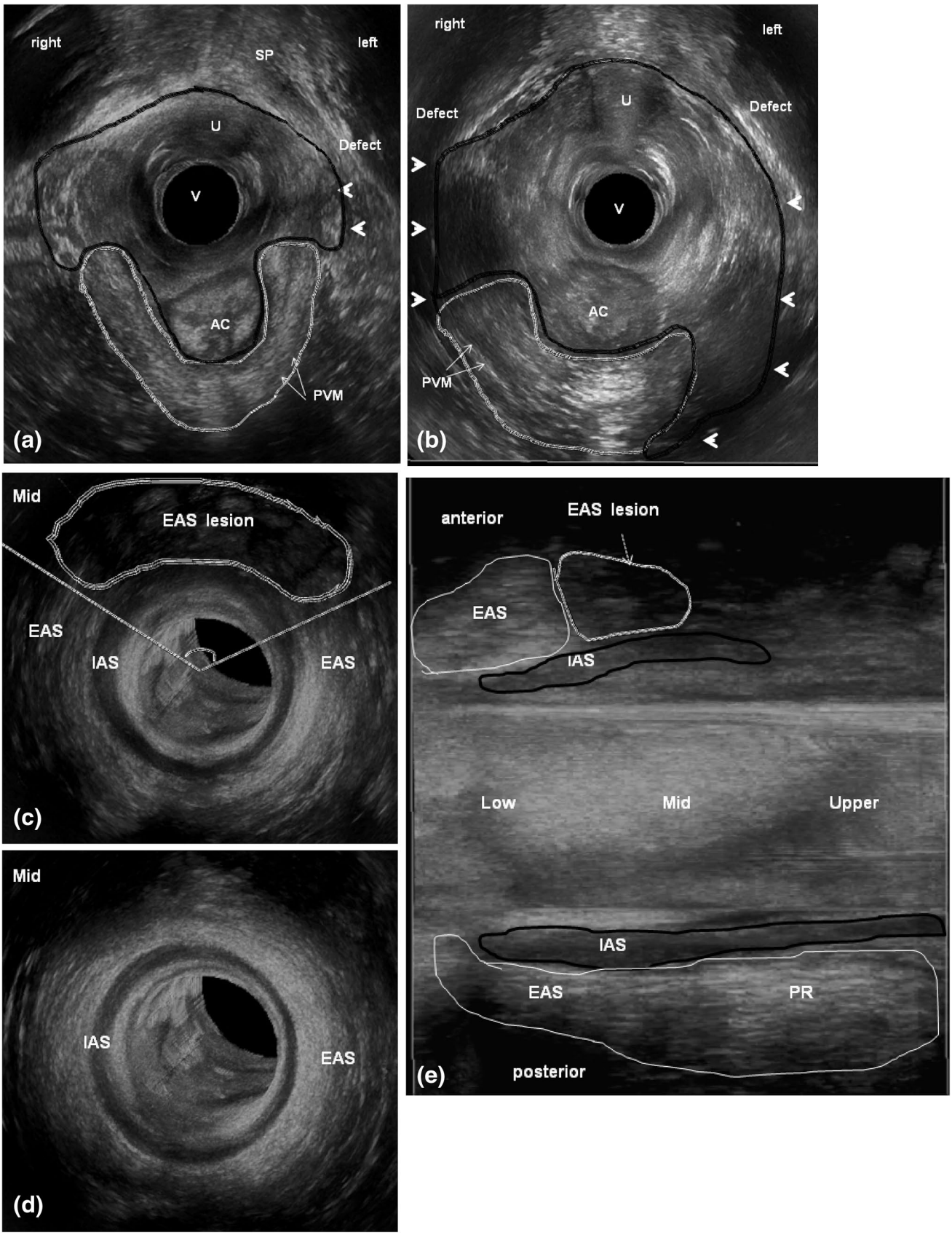
The resting and squeeze anal pressures were significantly lower in women with anal sphincter and/or PVM defects than in those without defects (Table 6).

Within the group of women with defects, women who had a PVM defect were compared to women with an intact PVM (women with sphincter defect only), and it was observed that the mean maximum anal squeeze pressure was significantly lower in women with PVM defects: mean = 73 (34) mmHg vs mean = 93 (38), $p = 0.04$.

The mean resting pressure was not significantly different in women with a PVM defect vs those with an intact PVM: mean = 32 (16) mmHg vs mean = 35 (14), $p = 0.51$.

Fecal and urinary incontinence

Table 7 summarizes the findings regarding fecal and urinary incontinence. Both the rate of FI ($p < 0.001$) and the median


right
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(a)
right
left
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U
Defect
V
AC
PVM
(b)
Mid
EAS lesion
EAS
IAS
EAS
(c)
anterior
EAS lesion
EAS
IAS
Low
Mid
Upper
IAS
EAS
PR
(e)
posterior
Mid
IAS
EAS
(d)

◂**Fig. 3** 3D-ultrasound—patient with bilateral pubovisceral muscle defect and partial external anal sphincter defect after vaginal delivery (render-mode). **a** Endovaginal approach showing a bilateral pubovisceral muscle defect (arrows head) and area of the levator hiatus at rest (black drawn) (axial plane). **b** Endovaginal approach—increased area of the levator hiatus in the Valsalva manuever (black drawn) (axial plane). **c** Anorectal approach showing an external anal sphincter muscle defect in the anterior quadrant and angle of the external anal sphincter defect in the mid anal canal (axial plane). **d** Anorectal approach showing intact sphincter muscles in the distal part of the mid anal canal (axial plane). **e** Anorectal approach showing external anal sphincter muscle defect and residual external anal sphincter length (sagittal plane). *U* urethra, *V* vagina, *AC* anal canal, *PMV* pubovisceral muscles, *EAS* external anal sphincter, *IAS* internal anal sphincter, *PR* puborectalis muscle

CCFIS score ($p < 0.01$) were significantly higher in women with anal sphincter and/or PVM defects than in women without defects. In contrast, urinary incontinence symptoms were similar in both groups, 11 (12%) women in the group of with anal sphincter and/or PVM defects and 3 (7%) in the group of women without defects ($p = 0.55$).

Regardless of whether they had a sphincter defect, the 42 women with PVM defects had significantly higher median CCFIS scores (median score 7/range 0–16) compared with the 88 women with intact PVM (median score 4/range 0–10) ($p < 0.001$). Bivariate analysis revealed a positive association between increasing FI symptom severity (CCFIS score) and women with PVM defects ($p < 0.001$) ($\rho = 0.6913$).

There was a significant positive correlation between the CCFIS and ultrasound scores ($\rho = 0.625$; $p < 0.001$). Bivariate analysis revealed a negative correlation between the CCFIS score and the lengths of the anterior EAS ($\rho = -0.5621$, $p < 0.001$), IAS ($\rho = -0.40$, $p < 0.001$) and the area of the levator hiatus ($\rho = 0.5211$, $p = 0.001$). However, no significant correlations were observed between FI scores and the gap measurement ($\rho = 0.101$; $p = 0.253$) or the resting ($\rho = -0.08$, $p = 0.54$) or squeeze pressure ($\rho = -0.12$; $p = 0.34$) values on anal manometry.

The variables associated with worsening FI symptom severity (CCFIS score) that remained significant in multiple linear regression included the shorter lengths of the anterior EAS and/or the lengths of the anterior IAS and increased area of the levator hiatus (summarized in Table 8).

Discussion

This study demonstrated sphincter and/or PVM defects in 68% (89/130) of women with previous vaginal delivery who were examined with 3D endovaginal and anorectal ultrasonography. Pubovisceral muscle defects (with or without sphincter defects) were observed in 32% (42/130). These results are comparable to those of previous studies using nuclear magnetic resonance imaging or ultrasound, which have shown PVM injury after vaginal delivery [16–20]. In our study, FI symptoms were significantly more frequent and the median CCFIS was significantly higher, especially, in patients with combined PVM defects. Of 42 patients with associated PVM defects, all complained of FI and had a higher CCFIS than those with an intact PVM and 12 patients had only an isolated PVM defect. On the other hand, the risk factors associated with the obstetric trauma such as episotomy, forceps and number of vaginal deliveries were similar in both groups (with and without defects) as previously reported in the literature [27, 28]. However, other results in the literature show a correlation between those risk factors and abnormalities of anal sphincter and pelvic floor muscles [29–31].

In our study, we, therefore, used 3D ultrasound to measure the length of the residual muscles in the partial sphincter defects and identify the complete defects. The majority of women (68%) had an isolated sphincter muscles defects or one associated with PVM defects and 91% complained of FI. Previous studies have detected by two-dimensional (2D)- and 3D ultrasound sphincter defects after vaginal delivery in up to 65% of incontinent females [7–9].

In the group with anal sphincter and/or the PVM defects, the ultrasound examination demonstrated that the length of the sphincters muscles was shorter in the anterior and posterior quadrant. Additionally, the gap (the weak area and without any striated muscle in the anterior circumference) was longer. These results demonstrated that the vaginal delivery increased the asymmetrical conformation of the anal canal and the anterior quadrant lost resistance. There was a correlation with the presence of defects with FI as well as CCFIS and ultrasound score. Additionally, there was a correlation between shorter length of anterior EAS and IAS muscles and higher CCFIS. Several studies have compared different ultrasound measurements, like, presence of the sphincter defect in relation to the anal circumference and angle lesion or volume of residual sphincter muscles [22, 32] and correlated them with the presence of FI symptoms instead of the length of the residual sphincter muscles. Thus, controversy still exists regarding ultrasound measurements but the length of the sphincter muscles is simple and reproducible, especially using the 3D with automatic scanning so that the sequence of images are obtained without any interference from probe movement and high-resolution images are obtained [10].

The PBM supports the pelvic organs (bladder, urethra, vagina, uterus, anal canal and inferior rectum) and borders on the lateral and posterior parts of the levator hiatus and the anterior part of the symphysis pubis. Studies have shown the correlation between PVM avulsion, POP and cystocele [12, 13, 18]. However, there was no specific correlation between the PVM avulsion and urinary incontinence symptoms in our

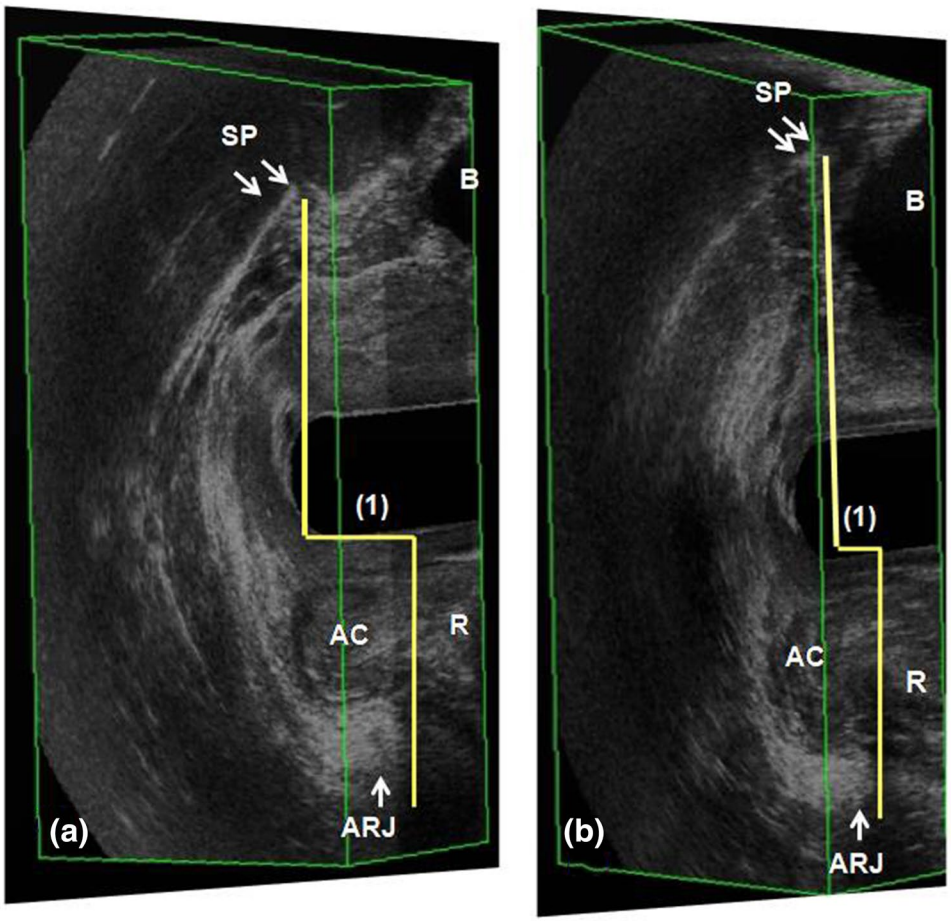

Fig. 4 3-D endovaginal ultrasound. Measurements of anorectal junction (ARJ) position in mid-saggital plane. Line 1 = Distance from the ARJ to the lowest margin of the symphysis pubis (SP). The displacement of the ARJ in the Valsalva maneuver. **a** At rest, **b** Valsalva maneuver. *B* bladder, *AC* anal canal, *R* rectum

Table 1 Scoring system for anal sphincter and pubovisceral muscle defects detected with anorectal and endovaginal three dimensional ultrasound

Defect	Score		
	0	1	2
External anal sphincter			
Length of defect	None	Partial	Whole
Size of defect (angle)	None	≤180°	>180°
Internal anal sphincter			
Length of defect	None	Partial	Whole
Pubovisceral muscle			
Length of left lateral defect	None	Partial	Whole
Length of right lateral defect	None	Partial	Whole

Table 2 Anal sphincter and pubovisceral muscle defects after vaginal delivery

	PVM intact *n* (%)	PVM defect *n* (%)	Total *n* (%)
Sphincter intact	41 (31.5)	12 (9.2)	53 (40.8)
EAS defect	31 (23.8)	12 (9.2)	43 (33.1)
EAS + IAS defect	16 (12.3)	18 (13.8)	34 (26.2)
Total	88 (67.7)	42 (32.3)	130 (100)

EAS external anal sphincter, *IAS* internal anal sphincter, *PVM* pubovisceral muscle

study population. We did not investigate the quantification of pelvic organ prolapse (POP-Q), as well as, identification of cystocele in the women including in this study.

Various studies describe the enlargement of the hiatus after vaginal delivery, as well as, the correlation with the POP [12, 13, 16, 18, 20, 21]. In our study, the enlargement of the levator iatus area was increased significantly in the group of PVM avulsion when compared at rest and with Valsalva. Additionally, the area at rest and with Valsalva was significantly higher than in the group without defects. There were a positive correlation with CCFIS and this finding may indicate that the PVM has a role in maintaining continence. Furthermore, in the women with defects (sphincter and/or PVM) the anorectal position was lower at rest and during Valsalva compared with the group without defects due the major displacement of the anorectal

Table 3 Characteristics of women with and without anal sphincter or pubovisceral muscle defects after vaginal delivery ($N=130$)

	Defect (≥1)[a], $n=89$	No defect[b], $n=41$	p
Age, years			
Mean (SD)	61 (12)	55 (10)	0.02
Range	19–79	31–75	
Number of vaginal deliveries, median (range)	3 (1–11)	3 (1–9)	0.97
Number of forceps deliveries, median (range)	11	04	0.77
Number of episiotomies, median (range)	14	05	0.79

SD standard deviation

[a]Women with one or more anal sphincter and/or pubovisceral muscle defects

[b]Women with intact anal sphincter and pubovisceral muscles

Table 4 Three dimensional anorectal and endovaginal ultrasound findings in women with and without anal sphincter or pubovisceral muscle defects after vaginal delivery ($N=130$)

	Defect (≥1)[a], $n=89$	Intact[b], $n=41$	p
Ultrasound score, median (range)	3 (0–10)	0	< 0.01
Levator hiatus area (cm^2)			
At rest			
Mean (SD)	18 (4)[c]	16 (3)[d]	0.01
Range	11–29	12–21	
During Valsalva maneuver			
Mean (SD)	20 (6)[c]	17 (4)[d]	0.01
Range	11–40	11–30	
Anorectal junction position (cm)			
At rest			
Mean (SD)	1.2 (0.9)	1.6 (0.8)	0.01
Range	− 0.9 to 3.2	− 0.5 to 3	
During Valsalva maneuver			
Mean (SD)	0.2 (0.8)	0.7 (1.1)	0.01
Range	− 1.6 to 2.3	− 2.4 to 2.9	

SD standard deviation

[a]Patients with one or more anal sphincter and/or pubovisceral muscle defects

[b]Patients with intact anal sphincter and pubovisceral muscles

[c]Significantly greater during the Valsalva maneuver than at rest ($p = 0.01$)

[d]No difference between the Valsalva maneuver and at rest ($p = 0.22$)

Table 5 Three-dimensional anorectal ultrasound measurements of the anal canal in women with and without anal sphincter or pubovisceral muscle defects after vaginal delivery

3-D AUS measurement, cm	Defect (≥1) $n=20$[a], mean (SD)	Intact $n=15$[b], mean (SD)	p
Anterior EAS length	1.0 (0.5)	1.7 (0.2)	< 0.01
Anterior IAS length	1.6 (0.8)	2.4 (0.4)	< 0.01
Posterior EAS + PR length	3.1 (0.5)	3.5 (0.5)	< 0.01
Posterior IAS length	3.0 (0.5)	3.3 (0.5)	< 0.01
Gap length	2.5 (0.5)	2.3 (0.5)	0.03

3-D AUS 3-dimensional anorectal ultrasound, *EAS* external anal sphincter, *IAS* internal anal sphincter, *PR* puborectalis muscle

[a]Women with one or more anal sphincter and/or pubovisceral muscle defects

[b]Women with intact anal sphincter and pubovisceral muscles

Table 6 Anal manometry findings in women with and without anal sphincter or pubovisceral muscle defects after vaginal delivery ($N=130$)

Anal manometry, mm Hg	Defect (≥1)[a], $n=89$	Intact[b], $n=41$	p
Resting pressure			
Mean (SD)	34 (14)	46 (18)	< 0.01
Range	12–67	18–80	
Maximal squeeze pressure			
Mean (SD)	86 (49)	129 (40)	< 0.01
Range	20–212	43–222	

SD standard deviation

[a]Women with one or more anal sphincter and/or pubovisceral muscle defects

[b]Women with intact anal sphincter and pubovisceral muscles

Table 7 Fecal and urinary incontinence in women with and without anal sphincter and/or pubovisceral muscle defects ($N = 130$)

	Defect (≥1)[a], $n = 89$	Intact[b], $n = 41$	p
Fecal incontinence, n (%)	81 (91)	17 (41)	< 0.001
CCFIS, median score (range)	5 (2–16)	0 (0–7)	< 0.01
Urinary incontinence, n (%)	11 (12)	3 (7)	0.55

CCFIS Cleveland Clinic Florida Incontinence Scale, *SD* standard deviation

[a]Women with one or more anal sphincter and/or pubovisceral muscle defects

junction in relation to the symphysis pubis and may lead to excessive perineal descent [33].

The prevalence of FI with no defect in women with a previous vaginal delivery was 19% and in those with defects was 62%. So, our results show a difference in severity of CCF incontinence scores between patients with and without defects of the anal sphincter and PVM, as well as a difference in functional findings (low resting and squeezing pressure in group with defects). On the other hand, as in other studies in the literature we found no correlation between the severity of FI symptoms measured by CCFIS and anal pressure [22, 32]. However, anal pressure was significantly lower in women with PVM or sphincter defects and squeeze pressure was lower in women with PVM defects. Because of that, the complete assessment of symptoms and severity, as well as assessment of anal function with anal manometry and anatomy with ultrasound images of the sphincter muscles and pelvic floor muscles are necessary to manage the patient and choose the appropriate treatment.

We recommend that even patients without FI symptoms or low CCFIS after vaginal delivery undergo a complete anatomical and functional evaluation including combined evaluation of the anal sphincters and PVM since other risk factors (such as anorectal and colorectal surgery and clinical conditions) may have an additive effect and lead to the subsequent development of continence disorders. Oberwalder et al. reported that 71% of women with late-onset FI after vaginal delivery had occult sphincter defects [34].

This study has several limitations: the type of the methodology, prospective data concerning the risk factors of vaginal delivery to identify the main factor (s) that determine the pubovisceral muscle defect like the first delivery, birth weight the forceps used and the episiotomy. Further studies are necessary to assess the primiparous patients before and after delivery with regard to symptoms, anal function and using anal/endovaginal ultrasound to evaluate the sphincter and PVM muscles.

Table 8 Variables analyzed of factors associated with fecal incontinence scores by multiple linear Regression

Variables	r^2	p
Fecal incontinence scores ($n = 130$) vs.		
Anterior EAS length	0.2810	< 0.0001
Anterior IAS length	0.3315	< 0.0001
Llevator hiatus area	0.1236	0.0002
Gap measurement	0.0092	0.9517
Resting pressure	0.0008	0.4578
Squeeze pressure	0.0120	0.4578

EAS external anal sphincter, *IAS* internal anal sphincter

Conclusions

Our results demonstrated that the majority of women with a previous vaginal delivery had at least one anal sphincter or pubovisceral muscle defect and half of them had a combined defect. There were correlations between anatomical abnormalities including the anal sphincter and/or pubovisceral muscle defects with FI and severity of symptoms as well as reduced anal function. Vaginal delivery increases the asymmetry of anal canal and pelvic floor as demonstrated by the shorter length of the anal sphincter muscles in anterior and posterior quadrants as well all the enlargement of the levator hiatus area in women with defects and incontinence symptoms.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval The study was approved by the hospital's research ethics committee.

Informed consent All participants gave their informed consent.

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