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ORIGINAL ARTICLE

Is the extent of obstetric anal sphincter injury correlated with the severity of fecal incontinence in the long term?

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Abstract

Background Obstetric anal sphincter injury is the most frequent cause of fecal incontinence (FI) in young women. However, the relationship between the extent of anal sphincter defects and the severity of long-term FI (at least 1 year after delivery) has been poorly studied. The aim of the present study was to determine if, in the long term, the extent of anal sphincter defects graded at anal endosonography was linked with the severity of FI.

Methods A retrospective study was conducted on women with a history of vaginal delivery, who presented with FI and had three-dimensional anorectal high-resolution manometry and endoanal ultrasound in our center from January 2015 to 2016. The detailed clinical history of each patient was obtained from the institutional database. The severity of FI was assessed with the Jorge and Wexner continence scale.

Results There were 250 women with a mean age of 60 ± 14 years. Seventy-six (30.4%) had an isolated defect of the internal anal sphincter, 21 (8.4%) had an isolated defect of the external anal sphincter, and 150 (60%) had both internal and external sphincter defects. The extent of IAS and EAS defects was proportionally correlated with the decrease in mean resting anal pressure ($p < 0.01$) and the decrease in mean squeeze pressure ($p = 0.013$) measured by 3DHRAM. No significant correlation was found between the extent and location of the defect (IAS, EAS or both) on endoanal ultrasound and the severity of FI. Menopause was the only independent factor significantly associated with the severity of FI.

Conclusions In our study, no significant correlation was observed between the extent of the anal sphincter defect and the severity of FI. Menopause was the only identified and independent risk factor for FI. These data confirm that, in the long-term, FI is often multifactorial.

Keywords Fecal incontinence · Obstetrical anal sphincter injury · Endoanal ultrasonography · 3D high-resolution anorectal manometry · Delivery

Introduction

Fecal incontinence (FI) is a disabling condition that has a significant impact on quality of life. Although it is probably underestimated, its prevalence varies from 5 to 15% in the general population [1]. The etiological factors are diverse [2]. Obstetric anal sphincter injury is the most common cause of FI in young women and may be associated with stretch-induced neuropathy [1–7]. Sultan et al. reported that the prevalence of these defects among primiparous women is 35% [8], and Snooks et al. showed that in 75% of women with idiopathic FI and in 60% of patients with anal sphincter defects vaginal delivery also induced pudendal neuropathy [9, 10]. Clinically, FI can be observed in the postpartum period in up to 47% of women, but can disappear spontaneously after 3–6 months, probably in part due to the regression of neuropathy and sphincter repair [11–13]. However, if it persists when a defect of the external anal sphincter has been identified, a repair may be proposed to restore, at least partially, the anatomical barrier necessary for fecal continence. The literature recommends this strategy in patients

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whose recent sphincter defect does not exceed half of the sphincter circumference [14–16]. In cases of minimal subclinical disturbance, some authors recommend that sacral nerve stimulation be preferred to sphincter repair because there might be associated pudendal and, therefore FI could be better treated by this technique [17, 18]. The relationship between the extent of anal sphincter injury and the severity of long-term FI (at least 1 year after delivery) has been poorly studied [19–22]. It is important to understand whether FI is mainly due to sphincter injury, stretch neuropathy or some other factor. In addition, in clinical practice, this is a question frequently asked by patients. Therefore, the aim of our study was to evaluate the relationship between the extent of obstetric anal sphincter injury and the severity of FI in the long term.

Materials and methods

Patients

A retrospective study was conducted on consecutive women with a history of vaginal delivery, who presented with FI and underwent three-dimensional anorectal high-resolution manometry (3DHRAM) and endoanal ultrasound (EAUS) in our center from January 2015 to 2016. The inclusion criteria were as follows: age ≥ 18 years, anal sphincter injury identified by EAUS, FI at least 1 year after delivery.

The exclusion criteria were as follows: age < 18 years, any anorectal organic lesion, any history of rectal anal surgery, inflammatory bowel disease, diabetes mellitus, systemic sclerosis, chronic neurological disease or any other potential cause of anal injury other than obstetrical, nullipara and incomplete EUS or 3DHRAM assessment.

A detailed clinical history of each patient includes age, sex, duration of symptoms and associated urinary symptoms. The Jorge and Wexner incontinence scale (0–20) was used to assess the severity of FI [23]. Urinary incontinence was considered to be present if the answer to the following question was yes: "Do you have any problems with urinary incontinence (leaking urine)" [22]? The follow-up of patients was defined as the mean delay between the first delivery and onset of FI symptoms.

According to current French legislation on clinical trials concerning retrospective studies, there was no need for patient consent. The data used were anonymized and collected from the APHM computer file, which is in accordance with the Commission Nationale Informatique et Liberté (French National Commission for Data Protection).

*EAUS*EAUS is performed with the patient in the left lateral position. We use a rigid bi-plan transrectal probe with a frequency of 7 MHz (model EUP-U533; Hitachi, Tokyo, Japan). The tip of the probe is covered with a water-filled balloon to maintain the acoustic contact. By slow rotation of the probe through 360°, the various layers of the anorectal wall and adjacent organs can be visualized. A defect of the internal anal sphincter (IAS) is defined as an echogenic interruption of the muscular ring, whereas an external anal sphincter (EAS) disruption is defined as a hypoechogenic interruption. The extent of the defect is measured and expressed in degrees. The radial orientation is determined in relation to adjacent organs such as the puborectalis muscle, bladder, vagina and prostate. The same trained practitioner analyzed all of the described parameters. Anal sphincter defects were classified into three categories: isolated IAS defect, isolated EAS defect, defect of both IAS and EAS. The extent of the defect (IAS, EAS or both) was also classified into three categories: < 45°; 45–90°; ≥ 90°.

*3D HRAM*3D HRAM is performed with the patient in the left lateral position. The probe has a diameter of 10.75 mm and a length of 64 mm with 256 pressure sensors arranged in 16 rows each with 16 circumferential sensors. There is a central lumen for inflation of a balloon and a disposable sheath 3.3 cm long covered by the balloon (capacity of 400 ml). The manometric data are analyzed using the specific ManoViewTM analysis software (Sierra Scientific Instruments, Los Angeles, CA, USA). The other technical characteristics were the same as described by Cheeney et al. [24]. For each procedure, the parameters recorded included the following: anal canal length, resting pressure, squeeze pressure and rectal sensitivity.

The procedures (EUS and 3D HRAM) were performed by two different experienced operators blinded to each other's results.

Statistical analysis

Descriptive data were provided for the whole sample and for the three following subgroups: isolated SAE, isolated SAI, SAE–SAI combination. For each AS disruption, three categories were built: < 45°, [45°–90°] and > 90°. The mean anal canal length, anal resting pressure and voluntary contractions were compared between the three categories using Student's *t* test or the Mann–Whitney test. The Wexner score was correlated to the extent of the defect using Spearman's and/or Pearson's correlation coefficients. To assess variables linked to the severity of FI, linear regression was performed using the Wexner age, BMI, extent of defect, menopause and hormone replacement treatment and manometric data as explicative variables. Results were presented as beta standardized.

Results

From January 2015 to 2016, 250 women with a mean age of 60 ± 14 years were included in the study. Their clinical data are presented in Table 1. The mean Wexner score

was 11.7 ± 5.1 and 61 women were primiparous. There were 126 patients in (50.4%) menopause, 31 of whom were on hormone replacement therapy.

None of the patients had any sphincter repair between delivery and the evaluation in our unit.

Seventy-six (30.4%) had an isolated defect of the IAS, 21 (8.4%) had had an isolated defect of the EAS, and 150 (60%) had both internal and external sphincter defects. The extent of IAS and EAS defects was proportionally correlated with the decrease in mean resting anal pressure ($p < 0.01$) and the decrease in mean squeeze pressure ($p = 0.013$) measured by 3DHRAM. No link between the length of the anal canal and the type of defect was found (Table 2).

No significant correlation was found between the extent and location of the defect (IAS, EAS or both) and the severity of the FI. Detailed data are presented in Table 3.

BMI and urinary incontinence were not related to the severity of FI.

By multivariate analysis (age, BMI, menopause with or without hormone replacement therapy, extent of the abnormality), menopause was the only independent factor significantly associated with the severity of FI. Hormone replacement therapy did not influence the results ($p = 0.084$) (Table 4).

Discussion

Our study showed that in the long term, there is no significant correlation between the extent of sphincter defect and the severity of FI. In addition, there was no difference according to the location of the defect (IAS, EAS, or both), but the decrease in mean resting anal pressure and in mean squeeze pressure was significantly correlated with the extent of the IAS and EAS defects.

Causes of FI may be perineal lesions (sphincter defect, pudendal neuropathy, impaired rectal function) or general pathologies (diarrhea, chronic inflammatory bowel disease, neurological and systemic diseases) [16]. In most studies, the same risk factors for obstetric anal sphincter injury were identified: instrumental delivery (including forceps), a second stage of prolonged labor by epidural analgesia, vacuum extraction, pregnancy > 40 weeks, episiotomy, high birth weight and increased head size, mainly in primary vaginal delivery [25–27].The incidence of obstetrical anal sphincter injuries appears to be increasing and is as high as 19.3% in primiparous women in the USA [28]. In addition, although it is generally considered that obstetrical anal sphincter injuries are more frequent in primiparous women (35% of anal sphincter disruption after delivery, 13% symptomatic [8]), Abramowitz et al. showed, in a prospective study, that the frequency of injuries may be the same after a first or second delivery [8, 25]. However, in our study, since this is not an early postpartum study, we did not use the Sultan's

Table 1 Demographic data

	Overall population	EAS disruption	IAS disruption	EAS + IAS disruption	Statistical results
General data					
Mean age (years ± SD)	60 ± 14	52 ± 14	62 ± 11	60 ± 15	$p = 0.835$
Number of patients (*n*)	250	26	74	150	$p > 0.05$
Menopause [*n* (%)]	126 (50.4%)	8 (3.2%)	39 (15.6%)	79 (31.6%)	$p > 0.05$
Mean duration of FI symptoms (months ± SD)	53 ± 80	83 ± 109	50 ± 77	49 ± 57	$p > 0.05$
Mean delay between first delivery and onset of Symptoms (years ± SD)	34.5 ± 14.2	33.4 ± 14.9	35.3 ± 14.3	34 ± 15	$p = 0.4$
Mean Wexner score	11.2	10.05	11.71	11.85	$p = 0.61$
Urinary symptoms [*n* (%)]	141 (56.4%)	15 (10.6%)	43 (30.5%)	83 (58.9%)	$p = 0.375$
Mean BMI (kg/m^2) (BMI ± SD)	40.1 ± 7.9				$p = 0.705$
Obstetrical data					
Mean number of vaginal delivery per patient (n ± SD)	1.9 ± 1.2	2.3 ± 1.3	2.2 ± 0.9	2.1 ± 1.1	$p = 0.810$
Mean number of instrumental manoeuvers [*n* (%)]	33 (13.2%)	2 5 (0.8%)	10 (4%)	21 (8.4%)	$p = 0.696$
Mean number of direct tear(s) observed during childbirth [*n* (%)]	103 (41.2%)	9 (3.6%)	29 (11.6%)	65 (26%)	$p = 0.196$
Mean number of episiotomy [*n* (%)]	83 (33.2%)	8 (3.2%)	18 (7.2%)	57 (22.8%)	$p = 0.876$
Mean natal weight (kg ± SD)	3.5 ± 0.6	3.6 ± 0.5	3.5 ± 0.5	3.5 ± 0.5	$p = 0.644$

BMI body mass index, *EAS* external anal sphincter, *IAS* internal anal sphincter

Table 2 EAUS and manometric data

	All	Isolated IAS	IAS < or = 45°	IAS > 45 and < or = 90°	IAS > 90°	Isolated EAS	EAS < or = 45	EAS > 45 and < or = 90	EAS > 90	Both IAS and EAS
EAUS/type of rupture										
Number of patients	250	74 (29.6%)	5 (2%)	42 (16.8%)	27 (10.8%)	26 (10.4%)	7 (2.8%)	15 (6%)	4 (1.6%)	150 (60%)
Extent of rupture(degree)	–	103 ± 33	45	88 ± 7.6	130.3 ± 23.1	90 ± 27	45	82.5 ± 13	120	–
Anal canal length (cm)	31.3 ± 5.9	31.2 ± 4.3	35.2 ± 7.6	31.3 ± 4.4	30.2 ± 4.7	33.3 ± 4.1	33 ± 4.4	33.9 ± 4.5	31 ± 1.7	31.4 ± 5.4
3D HRAM										
Anal resting pressure (mmHg)	53.1 ± 27.5	53.9 ± 27.4	60.9 ± 26	60.1 ± 25.4	42 ± 28.1	80.7 ± 22	69.5 ± 36.1	80.6 ± 13.8	104 ± 25.5	48.7 ± 25.2
Voluntary contraction (mmHg)	122.4 ± 57.3	142.2 ± 66.3	158.8 ± 52.2	151.3 ± 74.5	123.6 ± 49.7	144 ± 50.2	153.6 ± 86.8	134.5 ± 31.3	181.7 ± 71.3	107.4 ± 46.2

EAUS endoanal ultrasound, *3D HRAM* three-dimensional anorectal high-resolution manometry, *IAS* internal anal sphincter, *EAS* external anal sphincter

Table 3 Wexner score and location of tear

	Patients Wexner (*n*) (mean value)
Extent of IAS tear	
< 45°	5 10.4 $p = 1.81$
Between 45° and 90°	42 11.71 $p = 0.94$
> 90°	27 12 $p = 0.83$
Extent of EAS tear	
< 45°	5 12 $p = 1.48$
Between 45° and 90°	12 9.75 $p = 1.53$
> 90°	3 10.67 $p = 4.09$
Location of tear	
IAS	76 11.71 $p = 0.61$
EAS	21 10.05 $p = 1.13$
Both IAS and IAS	150 11.85 $p = 0.41$

IAS internal anal sphincter, *EAS* external anal sphincter

Table 4 Results of multivariate analysis

	p
Age	0.294
Body mass index	0.487
Menopause	0.013
With replacement therapy	0.084
Extent of tear	0.260

classification. Although we are aware that this may be confusing, we have used a description of the sphincter defect as usually described in gastroenterological practice. Indeed, the description includes the affected sphincter as well as the extent of the circumferential defect [29].

In our patients, anal sphincter defects were mainly combined (60%), while 30% concerned only the IAS and 8% only the EAS. These data are consistent with most of the results in the literature with frequent combined defects and a variable prevalence of IAS or EAS defects [8, 29, 30], although some authors report a higher prevalence of EAS than IAS defects [12, 31]. There were no differences in symptoms depending on the sphincter involved or in case of a combined or isolated defect. The fact that data were not collected by observers blinded to the ultrasound results may lead to bias. However, we believe our study is valuable since very little data are currently available regarding the relationship between the damaged sphincter (IAS or EAS) and the severity of FI. Mahony et al. have demonstrated that the presence of an IAS defect (and no EAS defect) was predictive of FI [32]. In addition, most studies have considered only the postpartum period [32–35]. Nordeval et al. showed a positive correlation between the extent of sphincter damage and the degree of FI after primary sphincter repair [33]. In contrast,

Voyvodic et al. found no relationship between muscle damage and FI severity in the 330 adults studied [36]. It is very interesting to note that Starck et al. and then Nordeval et al. proposed a scoring system including the length and depth of the IAS and EAS defect to assess the relationship between the extent of the anal sphincter defect after primary repair and the severity of FI [24, 33]. In both studies, there was a positive correlation between the rating system and the severity of FI, highlighting the importance of adequate anal sphincter reconstruction during primary repair. However, in these two studies, as in the other studies published, the data concern the management of early anal sphincter defects, the follow-up periods do not exceed 4 years and little is known about long-term follow-up [13, 34, 37]. Moreover, in both studies, a BK 3D probe was used which does not allow the same analysis. In addition to the fact that we do not use a combined score in our study, not using the same probe may explain why our results are inconsistent with those of Nordeval and Starck and may be a limitation in the analysis of our results. Indeed, some data suggest that 3D EAUS may be better for the investigation of obstetrical anal sphincter injury after primary repair [38]. In our study, the use of a two-dimensional (2D) probe is a limitation since it does not allow simultaneous assessment of the thickness of the sphincter in all planes, but only in the axial plane, which can induce evaluation bias. Interesting results were reported by Soerensen et al. [20] They demonstrated, in a prospective study in patients who had third- or fourth-degree obstetric sphincter injury reconstruction, that the anterior sphincter length was significantly correlated with increased severity of FI. Our results do not include this data because if the thickness was normal, the sphincter was considered as normal. However, the absence of this data may be a bias.

Few data are available on long-term follow-up of obstetric anal sphincter injury. Four studies with a 10- to 30-year follow-up observed a link between obstetrical anal sphincter damage and the presence of FI, vs. controls or vs. cesarean sections and episiotomies [28, 39–43]. In contrast, a study of a large cohort of 890 patients with 18-year follow-up did not find any significant difference in the prevalence of FI between patients with obstetric anal sphincter defects and controls [44]. However, none of these series evaluated the impact of the extent of the sphincter defect. Our study has one of the longer follow-ups currently available and, despite its limitations, provides new data on this topic. Linneberg et al. reported a 5-year clinical follow-up in women with obstetric anal sphincter abnormalities that included the following findings: FI, urinary incontinence (UI) and sexual dysfunction [22]. In this study, 74% of patients with post-obstetric anal sphincter defect had FI (44% with UI and 50% with sexual dysfunction) and the grade of obstetric anal sphincter defects was significantly related to an increased frequency of FI, but not to its severity. In addition, in some of the studies cited above, as in our study, information about possible primary sphincter repair was also missing due to difficulties in collecting historical data. Identification of the impact of a sphincter defect on symptoms is especially important for the choice of therapeutic strategy, in particular the decision to repair the anal sphincter. In the postpartum period, it is known from the literature that women may have not only an anal sphincter defect, but also frequent denervation [9, 10]. In our study, no electromyography assessment was available. Indeed, electromyography is no longer recommended in the investigation of FI. In addition, although it has been demonstrated that a primary repair may reduce the risk of FI, there is debate about late sphincter repair because the onset of FI is probably due to multiple and cumulative factors increasing with age [13, 33, 34]. In addition, many studies have reported that obstetric anal sphincter injury can be occult in a significant number of women [27, 31].

In our study, the mean age was 60 years, with a mean duration of symptoms of 53 ± 80 months. The long interval (34.5 ± 14.2 years) between the first delivery and onset of FI confirms the multifactorial nature of FI. Unlike some authors, we did not find that the presence of UI was related to FI symptoms [4, 45, 46]. Similarly, BMI was not associated with the severity of FI. However, the average BMI of our patients was normal or at borderline overweight, whereas the literature data establish a potential link with obesity. Finally, and quite surprisingly, on multivariate analysis, menopause was the only independent factor significantly associated with FI severity, with no difference between patients with or without hormone replacement therapy. This result is in disagreement with what was described by Mous et al. Indeed, in their study comparing long-term effects of obstetric anal sphincter injury, whereas FI was more frequent in the anal sphincter defect group than in the control group, the postmenopausal state was not significantly associated with anorectal complaints. However, data from literature regarding the link between menopause, hormone replacement therapy and FI remain contradictory.

Conclusions

In our study, no significant correlation was observed between the extent of the anal sphincter defect and the severity of FI in long-term follow-up, and menopause was the only identified and independent risk factor for FI. These data confirm that, in the long term, FI is often multifactorial. Further large studies evaluating multiple parameters and scores and utilizing the 3D probe are needed.

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Compliance with ethical standards

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

Ethical approval For this type of study (retrospective) formal consent is not required.

Informed consent This is a retrospective study and according to French legislation on clinical trials at the time of the study, there was no need for patient consent. The data used were anonymized and collected from the APHM computer file which is declared to the Commission Nationale Informatique et Liberté (French National Commission for Data Protection).

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