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GENERAL GYNECOLOGY

Anatomic and functional evaluation of the levator ani muscle after an obstetric anal sphincter injury

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Abstract

Purpose To study the relationship between symptoms of anal incontinence (AI) and the anatomy and functionality of the levator ani muscle in women with a history of obstetric anal sphincter injury (OASI).

Methods This is a cohort study including patients with OASI from 2013 to 2016. Patients were assessed by a physical examination, endoanal ultrasound using Starck Scale, perineometry and 4D transperineal ultrasound. AI in all patients was measured with the Wexner scale. Correlation between variables has been analyzed in these patients.

Results 72 patients were analyzed: 28 with a IIIA degree tear, 26 with a IIIB, 13 with a IIIC and 5 with a IV. 38 patients showed a residual anal sphincter (AS) defect on endoanal ultrasound with an average Starck score of 6.5 ± 3.7. 21 patients expressed AI, with an average Wexner score of 4.1 ± 2.4. In 27 (37.5%) patients, a levator ani avulsion was observed: 17 unilateral and 10 bilateral. Patients with a levator ani defect had weaker pelvic floor muscle (PFM) function. These differences were statistically significant with perineometry ($p = 0.01$ and $p = 0.03$) but not for the Oxford test ($p = 0.08$). Patients with a residual AS defect as well as an injury to the levator ani muscle expressed greater AI symptomatology than patients with residual sphincter injury who maintain the integrity of the levator ani: Wexner 4.9 0.9 vs 3.3 1 ($p = 0.02$).

Conclusions The PFM has correlation with AI symptom development in patients with a history of OASI. Therefore, we suggest a key role of anatomical and functional assessments of the levator ani muscle in these patients.

Keywords Anal sphincter obstetric injury · Anal incontinence · Levator ani

Introduction

According to Sultan's classification, obstetric anal sphincter injuries (OASIs) are defined as tears produced during a delivery that affect the anal sphincter complex (AS), and which are classified according to the affected anatomical structures [1]. This is the main risk factor for the appearance of AI symptoms in young, healthy women [2]. Women with a history of OASI are at a double risk of presenting symptoms of anal incontinence (AI) over the course of their lifetimes compared to those who have not suffered this injury in delivery [3].

OASI occurs in 2–6% [4, 5] of vaginal deliveries. The incidence of AI following an OASI ranges from 9 to 60%, according to the literature, and symptoms can appear many years after the injury [6].

A determining risk factor for developing symptoms, after an OASI primary repair during delivery, is the presence and the degree of residual AS defects [2]. Patients with extensive residual defects are at a higher risk of presenting AI during their lives and the severity of these symptoms would be greater than in women with a minor defect or without residual lesion [7].

Historically, it has been demonstrated that the anorectal complex functionality is also influenced by the pelvic floor muscles, mainly the levator ani [8, 9]. Some studies assessed the link among levator ani structure and its function after delivery. However, there is scarce information on the role of this muscle in the development of AI.

The aim of our study was to determine the relationship between the presence of AI symptoms and the anatomy and

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functionality of the levator ani muscle in women with a history of OASI.

Patients and methods

We aimed to study the presence of symptoms of anal incontinence after obstetric anal sphincter injury (OASI) and specially its relationship with levator ani structure and function. For this purpose, a single-center cohort longitudinal study was conducted in a university hospital from January 2015 to December 2016.

The study was approved by the Comitè d'Ètica d'Investigació amb medicaments (CEIm) in Fundació Sant Joan de Déu (Code AR201602) and an informed consent was obtained from all the participants.

Inclusion criteria

- Patients who had an OASI from January 2013 to June 2016.
- Consented to participate in the study.

Exclusion criteria

- Patients with previous anorectal surgery or sphincter injury.
- Patients that did not want to participate.
- Patients that did not attend to all the follow-up appointments.

Follow-up

All patients were assessed, at minimum 6 months post-partum, with a physical examination, a perineometry and both an endoanal and 4D transperineal ultrasound. Patients from 2013 to 2014 have been clinically assessed using telephone interview and from 2015 to 2016 by face-to-face interview.

Studied variables

Data concerning the clinical history and related to the delivery were also collected in all patients. Anal incontinence symptoms were assessed using the Wexner scale [10].

During physical examination, the levator ani muscle function (strength) was quantified by digital vaginal palpation using the Modified Oxford Grading Scale (from 0 to 5) [11]. Vaginal palpation was performed using two fingers introducing the two distal phalanges. The average from the left and right sides of the vagina was calculated.

Function and imaging test

Perineometry was carried out using a Peritron^{TM} perineometer (Laborie). The peak and average values in squeezing were obtained and analyzed. For both assessments, three consecutive squeezes were recorded with a 10-second interval between efforts.

The endoanal ultrasound was performed by an expert colorectal surgeon (JLL) with the Flex Focus 800 equipment (BK medical), using the 360º rotational, with automatic 3D acquisition probe (model 2052). The presence or absence of residual defects was recorded and these were classified according to the Starck Scale. This scale assesses the length, depth and angle of both sphincter lesions and its score ranges from 0 (no lesion) to 16 (serious defect in the two sphincters). The obtained Starck Scale values were categorized, according to the literature, from 1 to 4 points as a minor defect, 5–7 points as moderate and an 8 or above score as a severe lesion [12].

The 4D transperineal ultrasound was carried out by a pelvic floor expert gynecologist (EMF) using a General Electric Voluson 730 Pro, with the convex 3D/4D probe placed in the vaginal introitus. The images were obtained at rest, in voluntary contraction of the pelvic floor muscles and during a Valsalva maneuver. The presence of levator ani avulsions (unilateral or bilateral) and the hiatal area were assessed using the methodology described by HP Dietz [13]. The muscle contractility was defined as the difference between the hiatal area in contraction and at rest and was expressed in negative numbers. The distensibility was defined as the difference between the hiatal area during Valsalva and at rest and expressed in positive numbers.

Statistical analysis

Continuous variables are presented as absolute numbers or mean ± standard deviation and categorical variables as an absolute number and/or percentage. The Chi-square test was used to compare categorical variables and the Student's T test was used for quantitative variables. The correlation between two numerical variables was calculated using the Spearman Rho (Rs) test. A regression model was built to analyze the impact of the PFM strength on AI symptoms in patients with residual AS defects. The level of statistical significance for all the tests was set at a bilateral p value less than 0.05. Statistical analysis was performed using the SPSS software package (21.0 version, IBM Corp., Armonk, NY, USA).

Results

Ninety-four patients were eligible for the study. Eight patients were excluded because they failed to come to their 6-month control visit and 14 patients with OASI did not come for their scheduled control appointment. Therefore, data from 72 patients were analyzed: 28 (38.9%) with a IIIA degree tear, 26 (36.1%) with IIIB, 13 (18%) with IIIC, and 5 (7%) with IV. Data related to the delivery history are shown in Table 1.

Endoanal ultrasound findings

Thirty-eight (52.8%) patients presented a residual AS defect on endoanal ultrasound with an average Starck score of 6.5 ± 3.7 (3–14). Sixteen (42.1%) of these were minor lesions (Starck 1–4), 10 (26.3%) were moderate (Starck 5–7) and 12 (31.6%) showed a severe defect (Starck 8).

Anal incontinence symptoms

Twenty-one (29.2%) of the patients expressed symptoms of anal incontinence, with a mean Wexner score of 4.14 ± 2.4 [1–9]. Five (31.2%) patients with a minor defect ($n = 16$) reported symptoms of anal incontinence. 4 (40%) patients with moderate lesions ($n = 10$) were symptomatic and 8 (66.7%) of the 12 patients with a severe residual lesion stated AI. A significant positive correlation was observed between AI (Wexner score) post-partum Starck score ($p = 0.001$, $Rs = 0.4$).

4D transperineal ultrasound findings

In 27 (37.5%) patients, the transperineal ultrasound showed a levator ani avulsion: 17 unilateral and 10 bilateral. Six (22.2%) of the avulsions were in patients with spontaneous deliveries, 11 (40.7%) in women who underwent a Kielland forceps delivery, 9 (33.3%) in Naegele forceps and 1 (3.7%) in vacuum deliveries.

The hiatal area in Valsalva was significantly larger in patients with a levator ani avulsion (27.1 9.3 vs 22.2 6.3 cm^2, $p = 0.01$). A positive correlation between the presence of unilateral or bilateral avulsion and the hiatal area in Valsalva was found, with a $Rs = 0.29$ ($p = 0.01$).

Patients with levator ani avulsion showed a weaker PFM strength (Oxford: 3 1.5 vs 2.5 1; peak perineometry: 29.6 13 vs 21.8 11.5; average perineometry: 22.111.1 vs 16.58.4). These differences were statistically significant for the peak and average values of perineometry ($p = 0.01$ and $p = 0.03$) but not for the Oxford test ($p = 0.08$). Additionally, if we classify the levator ani defect based on one side or two sides affected, we found that the PFM strength was lower in patients with both side defects compared to those having unilateral lesions or no lesions (Table 2).

Relationship between levator ani structure and function and anal incontinence

While 23 (31.9%) of the patients showed no lesion in the sphincter neither in the levator ani, in 11 (15.3%) women, an injury to the levator ani but not in the anal sphincter was observed. In 22 (30.6%) of cases, there was an AS but not a levator ani defect and in 16 (22.2%), the injury was in both structures.

The differences between the incidence of anal incontinence in patients with a residual defect in both levator ani and AS structures and those patients with a residual injury to the anal sphincter but with an intact levator ani muscle were not statistically significant (52.4% vs 47.6%, $p = 0.09$), but the symptoms in the first group are greater than in the second [Wexner 4.9 0.9 vs 3.3 1 ($p = 0.02$)].

Table 1 General data

$n = 72$	Mean ±SD
Age at delivery (years)	31 ± 5
Assessment (months)	31 ± 7.6
Delivery	
Normal vaginal delivery	23 (31.9%)
Kielland forceps	21 (29.2%)
Naegele forceps	18 (25%)
Thierry spatula	6 (8.3%)
Vacuum	4 (5.6%)
Newborn weight	3500 ± 419
Starck score	3.4 ± 4.2 (0–14)
Wexner score	1.2 ± 2.3 (0–10)

Table 2 Correlation between PFM strength and levator ani avulsion

$n = 72$	Mean/SD			Rho
	Normal levator ani ($n = 45$)	Unilateral avulsion ($n = 17$)	Bilateral avulsion ($n = 10$)	
Oxford	3 ± 1.5	2.5 ± 1.1	2.4 ± 0.8	− 0.23 ($p = 0.05$)
Maximum perineometry	29.6 ± 13	23.6 ± 11.5	18.8 ± 11.4	− 0.33 ($p = 0.004$)
Mean perineometry	22 ± 11.1	17.3 ± 7.5	15.4 ± 10	− 0.28 ($p = 0.02$)

We built a regression model to assess whether this greater symptom severity, shown in patients with same residual anal sphincter defect, might be more influenced by the severity of the residual anal sphincter lesion rather than merely by the presence or absence of a concomitant levator ani avulsion itself. These results did not reach the statistical significance, but the regression model showed that the presence of levator ani avulsion seems to be a more important factor to explain the symptoms of anal incontinence ($p=0.12$) than the severity of the anal sphincter residual defect ($p=0.97$).

Discussion

The incidence of OASI, in the center where this study was carried out, has been approximately 2% annually over the past 4 years, which is in line with other published data [4, 5]. In addition, the percentage of residual lesions in the anal sphincter complex (53%) and levator ani avulsions (37%) is also consistent with what we find reported in the literature, as well as the 74% of levator ani avulsions are in patients with a forceps delivery [14, 15]. The repair of the laceration was performed according to the guidelines of the Royal College of Obstetricians and Gynecologists (Green-Top Guidelines) [16]. An appropriate identification, classification and repair of obstetric anal sphincter injuries when they occur are, as well as the later presence of residual defect on endoanal ultrasound, the most important factors to avoid anal incontinence in young women [17].

This does not mean that the anal sphincter is the only factor implicated in anal incontinence development. In addition to the sphincter integrity, there is also the need for a correct pudendal nerve and levator ani muscle function [7, 9].

With these observations, we may assume that patients with an AS residual defect after an OASI repair which have a levator ani avulsion or a pudendal nerve disorder are likely to present anal incontinence symptoms due to the presence of more than one cause of dysfunction. In contrast, women with a history of obstetric injury to the anal sphincter with an undamaged levator ani or a normal levator ani function might have fewer symptoms of anal incontinence. This indeed has been the main aim of our study.

Women with levator ani avulsion showed weaker PFM strength than those with an undamaged musculature. This finding is consistent with previously published data [18–20], but we have additionally found that perineal strength decreases as the degree of levator ani defect increases.

This is the first study that uses the perineometry values to evaluate muscular function and correlate functional with anatomical levator ani findings. In previous articles, the Modified Oxford Grading Scale (MOS) was used [18, 19].

Although we find differences in Oxford Scale Score as well as in perineometry, the figures are only statistically significant in perineometry values. We think that this fact may be explained by the fact that the Oxford Scale is subjective and, although widely used, less exact than perineometer measurements [21].

Patients with both a residual lesion in the anal sphincter as well as injury to the levator ani showed more and greater symptoms of anal incontinence than women with a residual AS defect but with an undamaged levator ani. These findings are consistent with our previous study in which we demonstrated that in patients with a history of OASI, PFM strength measured by MOS was inversely correlated with Wexner score. This means that PFM strength may play a role in reducing symptoms of incontinence [22]. It is also in line with Johannessen's [23] publication which showed that patients undergoing post-partum PFM training are in a lower risk of developing AI symptoms.

The number of symptomatic women as well as the Wexner score increased with the severity of the AS defect. We may then assume that the severity of the defect is what explains the severity in symptomatology and not the PFM strength. Related to this supposition, the regression model suggested that the role played by the muscle strength is more important than the defect degree itself. This is an interesting finding; although the presence of residual injury on ultrasound has been demonstrated to be an important factor in anal incontinence development, there is still controversy in demonstrating the correlation between the severity of symptoms with the degree or the residual defect on ultrasound, especially at long-term follow-up [7]. However, the correct identification and appropriate primary repair of OASI are mandatory to avoid AI.

The absence of AS residual defect on endoanal ultrasound is important to avoid AI after an OASI. But if there is a residual lesion, there are also other factors added to the defect ultrasound degree to explain the presence and severity of symptoms. Previous studies about symptoms of anal incontinence following OASI injuries failed to take into account the functional and anatomical assessments of the levator ani muscle as the explanation. However, they were usually focused on correlating the symptoms with the endoanal ultrasound and the anorectal manometry findings [7, 24].

Our study might provide an explanation: anatomical and functional states of the PFM can be the factor involved in the improvement or deterioration of AI symptomatology in patients with the same degree of residual lesion in the sphincter. According to our findings, a very recent new theory about the role of the levator ani function has been published [25]: the puborectal muscle is able to contract involuntarily during rectal dilatation. This involuntary contraction contributes to preserve anal continence and it can help to understand and justify our results. In patients

with same residual sphincter defect, a levator ani defect might result in a weaker involuntary contraction that contributes to the more severe symptoms.

The strength of our study is that we elucidate the role of levator ani in the functional follow-up of patients with sphincter injury. However, the main limitation of our study is that due to the sample size we were unable to reach statistical significance for all the studied parameters. We also think it would be interesting to design a new research including the pudendal nerve assessment, using an electromyography. This will clarify the role in anal incontinence development of every involved factor individually using a multivariate regression model: the residual AS defect, levator ani injury and pudendal lesion in patients with a history of OASI.

Conclusions

- After an OASI, AI is more frequent in patients with residual anal sphincter defects and levator ani muscle avulsion.
- The Wexner score is higher in patients with residual anal sphincter defects and levator ani muscle avulsion.
- These findings strengthen the principle of recommending pelvic floor muscle training in symptomatic patients after an OASI.

The pelvic floor muscles play an important role in AI development in patients with a history of OASI. Therefore, we propose as necessary the anatomical and functional assessments of the levator ani muscle in these patients' follow-up. The anatomical and functional evaluation of the levator ani after an OASI may also be helpful to plan an individualized physiotherapy.

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Compliance with ethical standards

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

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