

THE RELATIONSHIP BETWEEN ONSET OF SYMPTOMS OF FAECAL INCONTINENCE AND THE STRUCTURAL AND NEUROLOGICAL ABNORMALITIES OF THE ANAL SPHINCTER

Hypothesis / aims of study: Fecal incontinence is often due to multiple pathogenic mechanisms and is rarely attributable to a single factor. Vaginal delivery is an independent risk factor for AI and obstetric trauma is the most common cause of anal sphincter (AS) disruption, that may involve the external and / or internal anal sphincter (EAS, IAS) , and also the pudendal nerves, singly or in combination with AS disruption.

The aim of this study was to compare two groups of women with FI, according to their perception about the onset of their symptoms: patients with symptoms in the immediate postpartum and patients with the onset of their symptoms years after the deliveries and to analyse the associate factors, with structural and neurological abnormalities of anal sphincter.

Study design, materials and methods: 326 consecutive women, with symptoms of FI referred to a specialized Unit in a tertiary referral hospital were assessed clinically and the severity of their symptoms were evaluated with Wexnex score. Endoanal ultrasound (EUS), anal sphincter electromyography (EMG), pudendal nerve terminal motor latency (PNTML) and anorectal manometry (AM) were performed in all patients. For this study we excluded 78 patients: 7 nuliparas, 43 patients who when asked they consider the onset of their symptoms was after anal surgery (26) or other pelvic surgery (17) and 28 patients with medical problems that could be considered a cause of FI. We included 248 women in the final analyses. Group 1 with 49 patients who initiate their symptoms immediate postpartum and the group 2 with 199 women, who initiate FI symptoms many years after delivery and that also these patients could not associate the onset of their symptoms with any medical problem. Clinical, manometric, structural and neurological differences, were analysed between the 2 groups.

Results: Patients from group 1 had lower age (39.2+-8.4 vs 62.3+-10.4), higher frequency of use of forceps (69.4% vs 30.2%), episiotomy (87.8% vs 74.9%) and "tear" in the anal sphincter (63.3% vs 41.2%) and lower vaginal parity (1.7+-1.1 vs 2.6+-1.4), lower frequency of hysterectomy (8.2% vs 36.2%) and surgery for urinary incontinence and/or prolapsed (10.2% vs 32.7%) than those of group 2 (all p<0.05). No differences in severity, measured with Wexner score, were found, but FI to solid was more frequent in patients of group 2 (Table 1).

	Group 1. Onset of FI symptoms after delivery n=49	Group 2. Onset of FI symptoms late in life n=199	P
Fecal urgency	38 (77.6)	156 (78.4)	0.517
FI to only liquid stool	21 (42.9)	35 (17.6)	<0.001
FI to liquid and solid stool	28 (57.1)	164 (82.4)	
Urgency FI	40 (81.6)	121 (60.8)	0.063
Passive FI	3 (6.1)	25 (12.6)	
Mixed FI	5 (10.2)	43 (21.6)	
Patient's awareness without urgency	1 (2)	9 (5.5)	
Stress FI	19 (38.8)	54 (27.1)	0.079
Wexner's score	11.4±4.5	12.0±3.8	0.343
Incontinence episodes/ month	8.8± 16.4	12.7±26.7	0.331

Table 1: Type and severity of fecal incontinence (FI) symptoms.

The resting anal pressure and rectal sensation were lower in group 2. The structural defects of AS were significantly more frequent in group 1 (Table 2) . No significant differences were found in frequency of electrophysiological test abnormalities, between both groups.

	Group 1. n=49	Group 2. n=199	P
Resting anal pressure	51.2±22.3	41.0±20.9	0.003
Pressure with voluntary squeeze	91.5±33.9	90.9±36.5	0.921
Sensory threshold	17.4±6.6	21.1±13.2	0.007
Ultrasound abnormality	31 (63.3)	62 (31.2)	<0.001
- Internal AS abnormality	3 (6.1)	10 (5.0)	
- External AS abnormality	22 (44.9)	41 (20.6)	
- IAS + EAS abnormality	6 (12.2)	11 (5.5)	
AS-EMG abnormality	36 (73.5)	126 (63.3)	0.120
- AS-EMG unilateral abnor.	19 (38.8)	36 (18.1)	0.008
- AS EMG bilateral abnor.	17 (34.7)	90 (45.2)	
PNTML	26 (53.1)	75 (37.7)	0.020
- PNTML unilateral abnor.	14 (28.6)	39 (19.6)	0.082
- PNTML bilateral abnor.	12 (24.5)	36 (18.1)	

Table 2: Results of anorectal manometry (AM), endoanal ultrasound (EUS), anal sphincter (AS) electromyography (EMG), pudendal nerve terminal motor latency (PNTML) in both groups.

In the group 1, 24 of the 49 patients who initiate their symptoms immediate postpartum, had abnormalities in both, ultrasound and EMG. In group 2, 90 of the 199 who consider their symptoms not related with any vaginal delivery or medical problem, had only EMG abnormalities, with normal anal sphincter structure in ultrasound (Table 3).

	Group 1 n=49	Group 2. n=199	<i>P</i>
<i>Normal ultrasound and EMG</i>	6 (12,2)	47 (23.6)	<0.001
Only ultrasound anormal	7 (14.3)	26 (13.1)	
Only EMG anormal	12 (24.5)	90 (45.2)	
Both ultrasound and EMG anormal	24 (49)	36 (18.1)	

Table 3: Differences in ultrasound and neurological abnormalities of anal sphincter, isolated and in combination.

In a logistic regression model, variables independently associated with combined abnormalities of EMG and ultrasound of anal sphincter, were : onset of symptoms in the immediate postpartum, history of anal sphincter tears and lower resting anal pressure ($R^2= 0.176$)

Interpretation of results: Half of the women with FI symptoms, initiated after delivery, had a combination of abnormal ultrasound and EMG. A small proportion had anal sphincter disruption isolated (ultrasound abnormality). Isolated abnormalities in neurophysiological tests were significantly more common, in women with FI in whom the onset of their symptoms was late in their life and in whom the onset of the symptoms, was not related to any medical condition.

Concluding message: When we evaluate women with FI symptoms after vaginal delivery, structural (ultrasound) and neurological evaluation (EMG), both may be necessary. If neurological abnormality is observed, these patients could be considered at high risk for FI in future years, even if a normal anal sphincter with ultrasound was found. Prospective studies with a cohort of patients with and without symptoms after delivery are necessary.

<i>Specify source of funding or grant</i>	None
<i>Is this a clinical trial?</i>	No
<i>What were the subjects in the study?</i>	HUMAN
<i>Was this study approved by an ethics committee?</i>	No
<i>This study did not require eithics committee approval because</i>	This study was proceeeded within conventional evaluation in our clinical practice
<i>Was the Declaration of Helsinki followed?</i>	Yes
<i>Was informed consent obtained from the patients?</i>	Yes