The Association Between Late-Onset Fecal Incontinence and Obstetric Anal Sphincter Defects

Michael Oberwalder, MD; Adam Dinnewitzer, MD; M. Khurrun Baig, MD; Klaus Thaler, MD; Kathy Cotman, BS; Juan J. Nogueras, MD; Eric G. Weiss, MD; Jonathan Efron, MD; Anthony M. Vernava III, MD; Steven D. Wexner, MD

Hypothesis: Endoanal ultrasonographic results have demonstrated that clinically occult anal sphincter damage during vaginal delivery is common. This may or may not be associated with postpartum fecal incontinence (FI). Bayesian meta-analysis of the literature revealed that at least two thirds of obstetric sphincter disruptions are asymptomatic in the postpartum period. Women with postpartum asymptomatic sphincter damage may be at increased risk for FI with aging compared with those without sphincter injury.

Design: Case series.

Setting: Tertiary referral center.

Patients: After excluding patients with other possible causes of FI, the histories of 124 consecutive women with late-onset FI after vaginal delivery were analyzed.

Main Outcome Measures: Endoanal ultrasonographic findings, pudendal nerve terminal motor latency assessment, and anal manometric results.

Results: Eighty-eight women (71%) with a median of 3 vaginal deliveries had sphincter defects on endoanal ultrasonographic results. The mean incontinence score, squeeze and resting pressures, median age at last delivery, and median duration of FI were not significantly different between patients with and without sphincter defects. Pudendal neuropathy was more frequent in patients without sphincter defects (10 [30.3%], left side; 12 [36.4%], right side) than in patients with sphincter defects (12 [14.3%] and 16 [19.3%], respectively), with the difference nearly reaching statistical significance ($P = .054$ and $P = .059$, respectively). The median age at onset of FI in patients with a sphincter defect was 61.5 years vs 68.0 years in those without a sphincter defect, which was not statistically significant ($P = .08$).

Conclusion: Analysis of the current patient population revealed that 88 women (71%) with late-onset FI after vaginal delivery had an anatomical sphincter defect. Thus, FI related to anal sphincter defects is likely to occur even in an elderly population who had experienced vaginal deliveries earlier in life.

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From the Department of Colorectal Surgery, Cleveland Clinic Florida, Weston (Drs Oberwalder, Dinnewitzer, Baig, Thaler, Nogueras, Weiss, and Wexner) and Naples (Drs Efron and Vernava); and the Department of Biostatistics and Epidemiology, Cleveland Clinic Foundation, Cleveland, Ohio (Ms Cotman).

VAGINAL DELIVERY WITH RESULTANT occult anal sphincter damage is believed to be a principal cause of fecal incontinence (FI) in women. Its exact incidence was unknown until a series published by Sultan et al\textsuperscript{4} in 1993 reviewed endoanal ultrasound (EAUS) findings before and after vaginal delivery and found sphincter defects in 35% of primiparous and 44% of multiparous women. In these women, only 20% reported symptoms of FI.

There is wide variation\textsuperscript{4-8} in the incidence of clinically occult anal sphincter injuries diagnosed by EAUS (11%-35%) after the first vaginal delivery. Other ranges include the incidence of de novo injuries (3%-12%) after subsequent deliveries and disruptions associated with subsequent FI (0%-68%). A Bayesian meta-analysis\textsuperscript{9} of the literature was used to produce 1 inference from 5 studies while accounting for the potential heterogeneity among the study populations. The meta-analysis included 717 vaginal deliveries\textsuperscript{4-8} and revealed a 27% incidence (95% confidence interval [CI], 22%-31%) of sphincter defects in primiparous women, an 8% incidence (95% CI, 5%-13%) of new sphincter defects in multiparous women, and a 30% incidence (95% CI, 23%-36%) of symptomatic defects (M.O. and S.D.W., unpublished data, August 2002).

To our knowledge, there are no published data regarding women living with occult postpartum asymptomatic sphincter disruptions. These women may be at an increased risk of developing FI with increasing age compared with women without sphincter injury. This study sought to assess an association between late-onset FI and sphincter defects in women.
EAUS has been described in detail elsewhere.11 Two investigators (J.J.N. and E.G.W.) performed EAUS using a scanner with a rotating endoprobe (Bruel & Kjaer, Naerum, Denmark) and a 7- or 10-MHz transducer. The technique of EAUS had a sphincter defect and became the defect group; 59 had a defect in the external anal sphincter, 6 had a defect in the internal anal sphincter, and 23 had defects in both sphincters. The no defect group consisted of 36 patients (29%) without any defect. Eighty-eight (71%) of the 124 patients who underwent EAUS had a sphincter defect and became the defect group; 59 had a defect in the external anal sphincter, 6 had a defect in the internal anal sphincter, and 23 had defects in both sphincters. The no defect group consisted of 36 patients (29%) without any defect.

ENDOANAL ULTRASOUND

One hundred seventeen patients had PNTML measurements on the left side, and 116 had PNTML measurements on the right side. The number of pathologic PNTML measurements on the left vs the right side between the defect and no defect groups nearly reached statistical significance ($P = .054$ and $P = .059$, respectively) (Table 1). The mean CCFIS score was 13.7 in the defect group compared with 12 in the no defect group ($P = .13$), and the mean squeeze ($P = .22$) and resting ($P = .31$) pressures were lower than normal limits in both groups, to a similar degree (Table 2).

PATIENT HISTORY

Both groups of patients had the last vaginal delivery at approximately the same median age ($P = .55$) and had FI for approximately the same median time ($P = .24$) before initial examination. The median age at onset of FI in patients with a defect was 61.5 years vs 68.0 years in those without a defect; this was not statistically significant ($P = .08$). The median number of prior vaginal deliveries was 3 in the defect group and 2 in the no defect group, which was not statistically significant ($P = .08$) (Table 2).

COMMENT

Endoanal ultrasound results have demonstrated that clinically occult anal sphincter disruption during vaginal delivery is common and may or may not be associated with postpartum FI.14 Bayesian analysis of the literature estimated that in 26.9% (95% CI, 22.5%-31.4%) of women, a disruption of the anal sphincter occurs with the first vaginal delivery. The risk of acquiring a sphincter de-
fect with subsequent vaginal deliveries was estimated to be 8.5% (95% CI, 4.9%-12.7%). A 29.7% incidence of symptomatic defects and a 3.4% incidence of postpartum FI without a sphincter defect were calculated. Combining all of this information, the probability of postpartum FI due to a sphincter defect is 77% to 83% (M.O. and S.D.W., unpublished data, August 2002).

A specific subset of women with postpartum symptomatic defects may gain back anal continence across time. However, it is unknown what subset of women with initially asymptomatic defects develop FI across time.

In the current study group of women who experienced late-onset FI, 71% of the patients had a defect on EAUS results. This finding correlated closely with the 77% to 83% probability of defects in cases of postpartum FI revealed by a meta-analysis of the literature. Moreover, the onset of FI in patients with a defect was at a median age of 61.5 years vs 68.0 years in patients without a defect, although this difference did not reach statistical significance. The mean CCFIS score in the current study was 13, implying severe impairment of continence that may have a deleterious effect on quality of life.

There is some controversy regarding the effect of pudendal nerve injury during vaginal delivery on the development of FI. Sultan et al.11 found prolonged PNTML 6 weeks after vaginal delivery in 16% of primiparous and 15% of multiparous women; however, in their study, there was no association between the change of PNTML and the development of symptoms. Moreover, abnormal latencies were significantly associated with a sphincter defect. Additionally, only 3% of multiparous women had abnormal values prior to delivery. In a subsequent study, Sultan et al.13 reported that only one third of pathologic PNTML remained prolonged after 6 months.

Conversely, a recent study14 of 80 primiparous and multiparous reported that pathologic postpartum PNTML measurements recover to the predelivery level within 2 months. Furthermore, prior to delivery, PNTML measurements showed no significant difference between primigravidae and multipligrae.

Another study15 of 168 primiparous women who had vaginal delivery reported that 6 weeks after delivery, 10% of patients had pathologic PNTML measurements and intact anal sphincters on EAUS results, while 9% had both pathologic PNTML measurements and anal sphincter defects. Among the 31% of women who had pudendal latencies of more than 2.4 milliseconds, prolongation persisted until 6 months post partum.

In the current study, pathologic PNTML measurements on both the left and right sides were more frequent in patients with no defect compared with those with a defect. This finding was marginally statistically significant (P = .054 and P = .059, respectively). The origin of the nerve damage in the current study population is unclear. It may have been acquired during vaginal delivery because patients with pathologic PNTML measurements represent a subset of women in whom the pudendal nerves did not fully recover after vaginal delivery. Alternatively, pudendal neuropathy may have been acquired later in life. Finally, the higher median age of the current study population with no defects may partially explain the higher incidence of pathologic PNTML measurements in that group because it has been demonstrated that age is correlated with pudendal neuropathy.16

The current study cannot determine the potential risk for an asymptomatic woman with a sphincter defect to develop FI later in life because no control group of equal parity and age is available. However, the high incidence of defects in patients with late-onset FI in the current study as well as in women with postpartum FI, as demonstrated by the meta-analysis, is remarkable. Thus, after ruling out or treating other possible causes of FI, the anal sphincter should be evaluated with EAUS. Defects can be surgically repaired with functional success rates of up to 60%.17

Analysis of the current patient population revealed that 71% of women with late-onset FI after vaginal delivery had an anatomical sphincter defect. This finding correlated closely with the 77% to 83% probability of defects in cases of postpartum FI in a recent meta-analysis of the literature. Thus, FI related with anal sphincter defects is likely to occur even in an elderly population who had experienced vaginal deliveries earlier in life.

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Corresponding author and reprints: Steven Wexner, MD, Department of Colorectal Surgery, Cleveland Clinic Florida, 2950 Cleveland Clinic Blvd, Weston, FL 33331 (e-mail: mcderme@ccf.org).

Table 2. Results of Fecal Incontinence (FI) Parameters in Patients With and Without Sphincter Defects

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sphincter Defect</th>
<th>No. of Patients</th>
<th>Mean Value</th>
<th>SD</th>
<th>P Value*</th>
</tr>
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<tbody>
<tr>
<td>Incontinence score</td>
<td>No</td>
<td>29</td>
<td>12.07</td>
<td>5.13</td>
<td>.13</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>81</td>
<td>13.72</td>
<td>4.45</td>
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<tr>
<td>Resting pressure</td>
<td>No</td>
<td>28</td>
<td>28.32</td>
<td>11.43</td>
<td>.31</td>
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<tr>
<td></td>
<td>Yes</td>
<td>78</td>
<td>25.58</td>
<td>14.27</td>
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<tr>
<td>Squeeze pressure</td>
<td>No</td>
<td>28</td>
<td>36.54</td>
<td>23.98</td>
<td>.22</td>
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<td></td>
<td>Yes</td>
<td>78</td>
<td>30.18</td>
<td>19.71</td>
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<table>
<thead>
<tr>
<th>Variable</th>
<th>Sphincter Defect</th>
<th>No. of Patients</th>
<th>Median</th>
<th>Min/Max</th>
<th>P Value†</th>
</tr>
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<tbody>
<tr>
<td>Age at last delivery, y</td>
<td>No</td>
<td>36</td>
<td>30.50</td>
<td>16/44</td>
<td>.55</td>
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<td></td>
<td>Yes</td>
<td>88</td>
<td>29.00</td>
<td>19/47</td>
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<tr>
<td>Duration of FI, mo</td>
<td>No</td>
<td>36</td>
<td>2.00</td>
<td>0.30/26.00</td>
<td>.24</td>
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<tr>
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<td>2.50</td>
<td>0.40/29.00</td>
<td></td>
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<tr>
<td>Age at onset of FI, y</td>
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<td>68.00</td>
<td>44.00/83.00</td>
<td>.08</td>
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<tr>
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<td>Yes</td>
<td>88</td>
<td>61.50</td>
<td>28.00/80.00</td>
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<tr>
<td>No. of vaginal deliveries</td>
<td>No</td>
<td>36</td>
<td>2.00</td>
<td>1/5</td>
<td>.08</td>
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<td></td>
<td>Yes</td>
<td>88</td>
<td>3.00</td>
<td>1/11</td>
<td></td>
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Abbreviations: Max, maximum; Min, minimum.
*Derived using the t test.
†Derived using the Wilcoxon rank sum test.
REFERENCES


IN OTHER AMA JOURNALS

ARCHIVES OF INTERNAL MEDICINE
Venous Thromboembolism in Patients Undergoing Laparoscopic and Arthroscopic Surgery and in Leg Casts
David Bergqvist, MD, PhD; Gordon Lowe, MD, PhD
The risk of venous thrombosis and need for prophylaxis in patients having undergone minimally invasive procedures and in patients immobilized in a leg plaster are poorly defined. We performed a literature search to evaluate the risk of developing venous thromboembolism after 2 minimally invasive procedures, laparoscopic surgery and arthroscopy, and in patients with lower limb plaster casts. Despite problems of “contamination” because some surgeons use prophylaxis in some of these patients, we were able to determine that (1) laparoscopic cholecystectomy can be considered a low-risk procedure and therefore routine use of prophylaxis is probably not justified; (2) patients undergoing arthroscopic knee surgery are at low to moderate risk and thus prophylaxis is optional; and (3) patients with plaster cast immobilization because of trauma have a moderate risk of thrombosis and should receive prophylaxis. (2002;162:2173-2176)

Corresponding author and reprints: David Bergqvist, MD, PhD, Department of Surgical Sciences, University Hospital, SE-751 85 Uppsala, Sweden (e-mail: david.bergqvist@kirurgi.uu.se).