Int Urogynecol J (2015) 26:1115–1121
DOI 10.1007/s00192-015-2663-3

ORIGINAL ARTICLE

Clustering of pelvic floor disorders 20 years after one vaginal or one cesarean birth

Maria Gyhagen · Sigvard Åkervall · Ian Milsom

Received: 10 December 2014 / Accepted: 18 January 2015 / Published online: 24 February 2015

Abstract
Introduction and hypothesis The objective was to assess the prevalence and risk factors for co-occurring pelvic floor disorders (PFDs): urinary incontinence (UI), symptomatic pelvic organ prolapse (sPOP), and fecal incontinence (FI), 20 years after one vaginal (VD) or one cesarean (CS) delivery.
Methods We carried out a registry-based national cohort study of primiparae who delivered during the period 1985–1988 and had no further deliveries. Medical Birth Registry data were linked to data from postal questionnaires distributed 20 years post-partum (response rate 65.2 %, n=5,236). Main outcome measures were prevalence and risk factors for combined and isolated PFDs.
Results The prevalence of any PFD was 46.5; 31.7 % had one symptom and 14.8 % had two or more. Co-occurring symptoms doubled after VD (17.1 %) compared with CS (8.4 %) (adjOR 2.26; 95 % CI 1.84–2.79). The strongest association was observed between VD and having all three symptoms (adjOR 5.20; 95 % CI 2.73–9.91), followed by the combination of sPOP and UI (adjOR 3.38; 95 % CI 2.24–5.10). The degree of frustration perceived by the women because of pelvic floor dysfunction increased with each additional co-occurring PFD (p<0.001). The strongest risk factors for clustering of PFDs were: VD (OR 2.19; 95 % CI 1.75–2.73), family history (OR 2.03; 95 % CI 1.73–2.34), and ≥2 degree tear (OR 1.78; 95 % CI 1.24–2.55). Vacuum extraction and episiotomy were not risk factors.
Conclusions The prevalence of co-occurring PFDs was high and was doubled in women after VD compared with CS. Women with UI most likely had it as an isolated symptom, whereas FI and sPOP more often occurred in combination.

Keywords Epidemiology · Cesarean section · Combinations · Long-term · Pelvic floor dysfunction · Vaginal delivery

Introduction

Pelvic floor disorders (PFDs) such as urinary incontinence (UI), pelvic organ prolapse (POP), and fecal incontinence (FI) constitute a huge health problem, globally affecting hundreds of millions of women [1]. Each year millions of women throughout the world undergo corrective surgery at considerable expense and personal suffering for all three conditions [1].

There are at present a large number of reports on single PFDs in women [1], but only limited information is available about co-occurring PFDs. A few studies have presented data on "double incontinence" (DI=concomitant urinary [UI] and fecal [FI] incontinence) [2, 3] and more recently on combinations of several PFDs [4, 5].

Numerous risk factors for the development of UI, POP, and FI have been identified and several of these risk factors are shared by one or more types of PFD [1]. Vaginal delivery has been implicated as an important risk factor for all three types of PFD [1]. However, confining the evaluation to single

Electronic supplementary material The online version of this article (doi:10.1007/s00192-015-2663-3) contains supplementary material, which is available to authorized users

M. Gyhagen · I. Milsom
Department of Obstetrics and Gynecology, Sahlgrenska Academy at Gothenburg University, Gothenburg, Sweden

M. Gyhagen (✉)
Department of Obstetrics and Gynecology, Södra Älvsborgs Hospital, 501 82 Borås, Sweden
e-mail: maria.gyhagen@vgregion.se

S. Åkervall
Department of Surgery, Södra Älvsborgs Hospital, 501 82 Borås, Sweden

symptoms of PFD produces several problems. Simultaneously occurring symptoms may exaggerate the subjective impact of the reported disorder and at the same time conceal the effect of co-occurring conditions. A higher prevalence of co-occurring symptoms after vaginal delivery (VD) compared with cesarean section (CS) may also be a possible indication of more extensive and/or more severe pelvic floor damage. Hence, clustering of PFDs could potentially be used to evaluate the long-term effects of obstetric risk factors and interventions.

The aim of this study was to investigate the late prevalence, risk factors, and subjective impact of combinations of the three most common PFDs in women 20 years after one VD or one CS.

Materials and methods

Participants in the SWEPOP (Swedish Pregnancy, Obesity and Pelvic floor) study were obtained from the Medical Birth Registry of the National Board of Health and Welfare, Sweden. Inclusion criteria for participation were primiparity with one single birth 1985–1988 and no further births. Exclusion criteria were multiparity and multifetal or ongoing pregnancy. Obstetric data were combined with information from a 2008 postal questionnaire, 20 years after the birth. A description of the study population based on a flow chart and cohort characteristics, including an analysis of the non-responders, has been described in detail previously [6].

The questionnaire included 31 questions about current height and weight, urinary or bowel incontinence and genital prolapse, menstrual status, hysterectomy, the menopause and hormone treatment and has been described previously [6] (Appendix 1). UI was defined according to the International Continence Society [7]. Symptomatic pelvic organ prolapse (sPOP) was diagnosed according to a validated five-item questionnaire [8] and was defined by the key symptom 'feeling of a vaginal bulge' (often/sometimes/infrequently). FI was defined as the involuntary loss of feces, solid or liquid. Double incontinence (DI) was defined as the combination UI and FI. An isolated symptom was defined as one single symptom of UI, sPOP, or FI. Combined or co-occurring symptoms were present if two or three of these were affirmed.

The feeling of frustration in relation to each PFD was assessed by the Incontinence Impact Questionnaire—Short form (IIQ-7): "Has (the symptom) affected you?—Do you feel frustrated because of the (symptom)?". Responses were on a four-step ordered category scale from "not at all" to "greatly" (1–4p) for each symptom. Family history for each PFD was assessed by the question: "Has your mother been troubled by (the symptom)?"

Statistical analysis was performed using SAS 9.1 (SAS Institute, Cary, NC, USA). Prevalence and 95 % confidence intervals (CI) were calculated. Chi-squared test was used to compare categorical variables and Student's *t* test to compare continuous variables. Logistic regression analysis was used to demonstrate independent risk factors for combined PFDs, while controlling for some obstetric and non-obstetric potential risk factors and confounders. Potential risk factors were mode of delivery, maternal age at delivery, current BMI, infant birth weight, episiotomy, vacuum extraction (VE) and ≥2nd degree perineal tear. Adjusted prevalence was calculated using analysis of variance, taking the variables maternal age, infant birth weight, and current BMI into account. Odds ratio (OR) and its 95 % CI were calculated from the logistic regression model. A p value of <0.05 was considered statistically significant. Subgroup analysis of the VD cohort was performed to address disproportion, which in this context was defined as a mother of short stature (≤160 cm) and a child ≥4,000 g. The number needed to avoid combined symptoms was calculated as the inverse of the absolute risk reduction, where risk reduction was the difference between the adjusted prevalence of VD compared with CS.

Ethical approval for the SWEPOP study was obtained from the regional (reference no. 381–07, 13 August 2007) and National (34–9148/2007, 26 October 2007) ethical review boards. All women received written information and gave written consent before participation in the study.

Results

The questionnaire was returned by 65.2 % of the available women. The basic characteristics of the women included have been described in detail previously [6]. The prevalence of any PFD in the total cohort was 46.5 %. Figure 1 includes a Venn diagram illustrating the overlapping prevalence of UI, sPOP, and FI. The overall prevalence of isolated symptoms of UI, sPOP, and FI was 31.8 % and the prevalence of those with co-occurring symptoms was 14.8 %. Approximately one third of the women with a PFD had co-occurring symptoms. The most common combination was UI+FI in 41.7 %, followed by UI+sPOP in 33.4 %. The overall prevalence of DI was 9.1 % (Fig. 1). All three PFDs (UI+sPOP+FI) occurred in 2.9 %. Women with UI were most likely to have it as an isolated symptom (Fig. 1).

In comparison with CS, VD had a stronger association with both isolated (OR 1.26; 95 % CI 1.10–1.45) and co-occurring symptoms (OR 2.26; 95 % CI 1.84–2.79; Table 1). The strongest association was between VD and the combination of sPOP+UI (OR 2.68; 95 % CI 1.86–3.87) and the triple combination sPOP+UI+FI (OR 5.20; 95 % CI 2.73–9.91; Table 1). Of 148 women with the triple combination 90.5 % (134 out of 148; 95 % CI 84.8–94.3) had a VD. Figure 2 illustrates a Venn diagram of the overlapping prevalence of UI, sPOP, and FI in para 1 women 20 years after one VD or one CS. Based on these results the number needed to treat

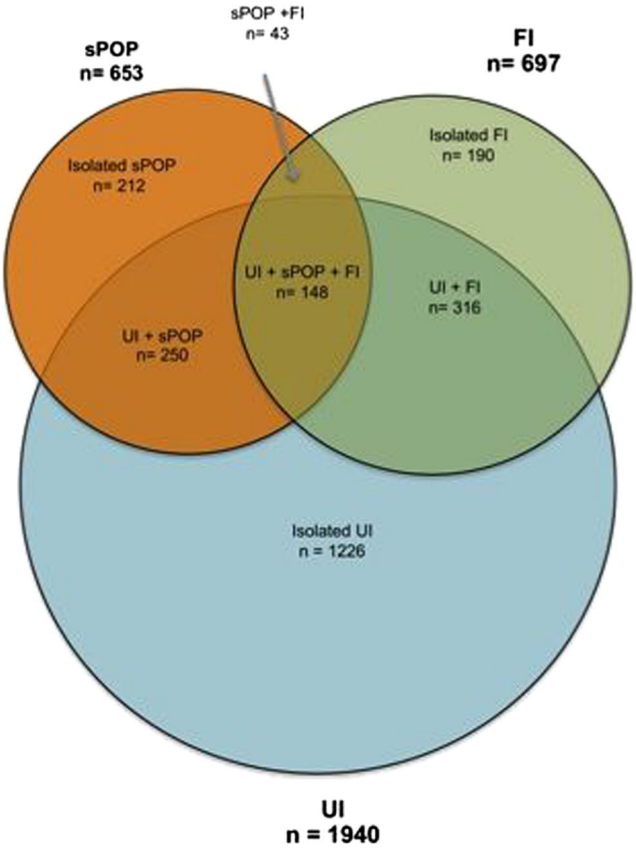

Fig. 1 Venn diagram illustrating the overlapping prevalence of urinary incontinence (*UI*), symptomatic pelvic organ prolapse (*sPOP*), and fecal incontinence (*FI*) in para 1 women 20 years after childbirth

(NNT) by CS to avoid one case with isolated symptoms was 20 and 11–12 to avoid one case with co-occurring symptoms (Table 1).

The logistic regression analysis of risk factors for the combined symptoms of PFDs showed that VD, current BMI, maternal age, ≥2-degree tears, and family history were risk factors (Table 2). In the total cohort the odds risk for combined PFDs increased by 2 % yearly for maternal age (OR 1.02; 95 % CI 1.01–1.04) and by 6 % for each unit increase in current BMI (OR 1.06; 95 % CI 1.05–1.08). Episiotomy and vacuum extraction were not risk factors (Table 2).

The degree of frustration experienced by the women as a result of pelvic floor dysfunction increased with each additional co-occurring PFD ($p<0.001$; Table 3). UI daily or weekly was more common in women with UI combined with another PFD than with UI alone (OR 1.42; 95 % CI 1.17–1.71). UI in combination with other PFDs was associated with an increased risk of bothersome UI (OR 2.21; 95 % CI 1.74–2.82) compared with women with UI alone.

The prevalence of co-occurring PFDs was 28.1 % in women with a VD who had disproportion (i.e. height ≤160 cm with an infant birth weight ≥4,000 g) compared with 15.8 % in women ≤160 cm with an infant with a birth weight <4,000 g (OR 2.07; 95 % CI 1.18–3.64) and 7.9 % in short mothers after CS, irrespective of infant birth weight (OR 4.54; 95 % CI 2.42–8.49; Table 4). The NNT by CS to avoid one case with two or more PFDs in women with disproportion was 5.

Discussion

Overall, 47 % of the women in this study had one or more PFD 20 years after one birth. Approximately one third with any PFD symptom had two or more PFDs, and the prevalence of co-occurring PFDs was doubled after VD compared with CS, affecting every sixth woman. The degree of frustration experienced due to PFD increased with each additional co-occurring PFD. Women with UI most likely had it as an isolated symptom whereas FI and sPOP occurred more often in combination. However, UI co-occurring with other PFDs was more bothersome and frequent. The strongest risk factors for the clustering of PFDs were: VD, family history, and ≥2-degree tears.

According to the present study and earlier reports, the prevalence of co-occurring PFDs is high and increases with age [9, 10], parity, and BMI [4, 9, 10]. In a cross-sectional population study by Nygaard et al. [10] 27 % of women in the age stratum 40–59 years had one or more PFDs. The prevalence of one or more PFDs was 34 % in a study by Rortveit et al. on 2,106 women who were 40–69 years and members of a Medical Health Program [5]. In the present study, which included para 1 women with a mean age of 51 years, the prevalence of one or more PFD was substantially higher (47 %), which to some extent can be explained by the inclusion of nulliparous women and a more restrictive definition of UI in the above-cited studies.

The present study has identified VD, current BMI, maternal age, ≥2-degree tear, and family history as independent risk factors for co-occurring PFDs. Wu et al. [9] studied the prevalence of PFDs in women in the USA. The prevalence of one or more PFDs was higher after VD (30.4 %) than after CS (18.4 %) and that reported in non-pregnant women (11.5 %). After adjusting for confounders, higher BMI, greater parity, and hysterectomy were associated with higher odds of one or more pelvic floor disorders. However, mode of delivery was non-significant in their multivariate analysis [9].

In the study by Rortveit et al. 82 % of women with any UI experienced it as an isolated symptom [5]. The proportion in our study was 63 %. The higher proportion with isolated UI in the study of Rortveit et al. may be explained by the fact that 18 % of the women in their study were nulliparous and were probably also a somewhat healthier being based on a Medical Health Program. In the same study, 60 % with any FI experienced it in combination with at least one additional PFD and 49 % with sPOP experienced it in combination with at least one additional PFD. In the current study the corresponding combinations were 73 % for FI and 68 % for sPOP. DI

Table 1 Crude and adjusted (Adj) prevalence and odds ratio of isolated and combinations of pelvic floor disorders (PFDS) in relation to mode of delivery

	VD *N*=3,740, *n* (%)	CS *N*=1,387, *n* (%)	Crude OR (95%CI)	VD (*N*=3,740) %	CS (*N*=1,387) %	AdjOR (95%CI)
1 PFD only						
sPOP only (*n*=212)	171 (4.6)	41 (3.0)	1.57 (1.11–2.22)	4.4	3.2	1.42 (1.01–1.99)
UI only (*n*=1,226)	932 (24.9)	294 (21.2)	1.23 (1.06–1.43)	25.2	20.5	1.31 (1.13–1.52)
FI only (*n*=190)	124 (3.3)	66 (4.8)	0.69 (0.51–0.93)	3.5	4.5	0.77 (0.57–1.05)
Women with 1 PFD only (either UI, sPOP or FI; *n*=1,628)	1,227 (32.8)	401 (28.9)	1.20 (1.05–1.37)	33.1	28.1	1.26 (1.10–1.45)
≥2 PFDs						
sPOP+UI only (*n*=250)	225 (6.0)	25 (1.8)	2.80 (1.92–4.06)	6.0	1.9	2.68 (1.86–3.87)
sPOP+FI only (*n*=43)	36 (1.0)	7 (0.6)	1.92 (0.85–4.32)	1.0	0.5	1.97 (0.88–4.43)
UI+FI only[a] (*n*=316)	237 (4.3)	79 (5.7)	1.12 (0.86–1.46)	6.52	5.2	1.27 (0.97–1.67)
Any combination of≥2 PFDs (*n*=757)	632 (16.9)	125 (9.0)	2.05 (1.68–2.51)	17.1	8.4	2.26 (1.84–2.79)
sPOP+UI+FI[a] (*n*=148)	134 (3.6)	14 (1.0)	3.64 (2.09–6.34)	3.6	0.7	5.20 (2.73–9.91)
DI[a] (*n*=464)	371 (9.9)	93 (6.7)	1.53 (1.21–1.94)	10.2	6.0	1.78 (1.39–2.27)

Adjusted for current BMI, infant birth weight, and maternal age

UI urinary incontinence, *sPOP* symptomatic pelvic organ prolapse, *FI* fecal incontinence, *DI* double incontinence (UI and FI), *VD* vaginal delivery, *CS* cesarean section

[a] In this cohort 316 women had UI+FI only and 148 women had UI and FI in combination with sPOP. DI was reported by 464 women

occurred in 10 %, of the women who were aged 45–55 years in the study reported by Slieker-ten Hove et al. [2] compared with 9 % in our study, which was considerably higher than that reported by Rortveit et al. (3.0 %) [5].

Several studies have focused on DI [2, 3, 11, 12], presumably because it is the most severe and debilitating manifestation of PFD [2]. DI afflicted approximately one in ten in both our study and the Slieker-ten Hove study. In our study, one third of women with DI also had sPOP, which was part of the triple combination UI+FI+sPOP. Interestingly, DI without sPOP (i.e. UI+FI only) was not significantly associated with VD, in contrast to the co-occurrence of DI with sPOP, where the effect of VD was marked. There was also a stronger association between VD and isolated UI than that between VD and DI, which has been described by others [13]. UI was more often an isolated symptom compared with sPOP and FI. This may indicate that there is a difference in pathogenesis between the different PFDs and the occurrence of DI with sPOP and DI without sPOP.

Earlier results from the SWEPOP study described the importance of disproportion for the development of PFD [14]. The prevalence of co-occurring PFDs was almost doubled in women with a VD who had disproportion compared with women ≤160 cm tall with an infant birth weight <4,000 g and was almost four times higher than in women who had undergone a CS, irrespective of infant birth weight. It has been postulated that women with disproportion are subjected to greater pelvic floor trauma during VD [15].

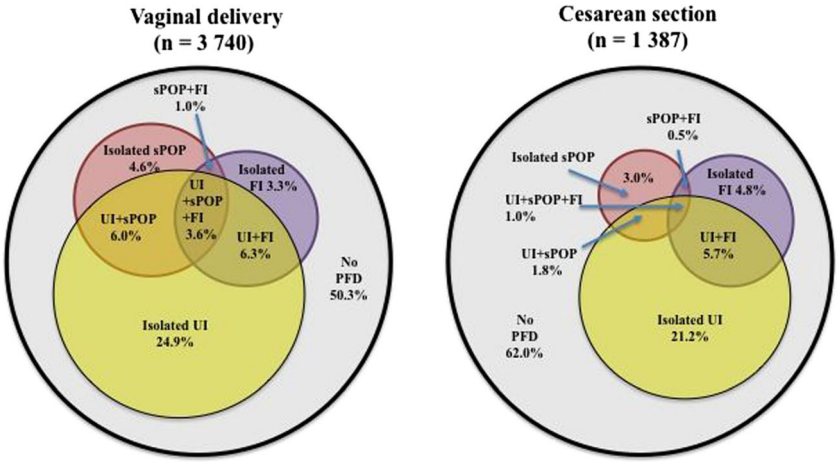

Fig. 2 Venn diagram illustrating the overlapping prevalence of urinary incontinence (*UI*), symptomatic pelvic organ prolapse (*sPOP*), and fecal incontinence (*FI*) in para 1 women 20 years after one vaginal delivery or one cesarean section

Table 2 Logistic regression analysis of risk factors for co-occurring (≥2) pelvic floor symptoms

	Total cohort (*N*=5,093) OR (95 % CI)
VD vs CS	2.19 (1.75–2.73)
BMI current	1.06 (1.05–1.08)
Birth weight	1.01 (0.99–1.02)
Maternal age	1.02 (1.01–1.04)
Tear ≥2 degree	1.78 (1.24–2.55)
Vacuum extraction	1.03 (0.83–1.29)
Episiotomy	0.92 (0.71–1.20)
Family history	2.03 (1.73–2.34)

VD vaginal delivery, *CS* cesarean section, *OR* odds ratio

It has been reported that the major component of PFD may be attributed to VD-related injuries [16]. Trauma to the levator ani muscle due to avulsions, over-distension, reduced muscle strength, and increased hiatal area are all interrelated [17] and have been shown to be strongly and primarily associated with POP [18]. This is in accordance with the finding of the present study, that isolated sPOP was more strongly associated with VD in comparison with UI and FI and that the three outcomes with the strongest association with VD all included sPOP.

The evidence for shared risk factors between sPOP and UI (partly biological and partly environmental) is also supported by findings from family history studies [19, 20]. Family history is of particular interest, because it may be closely linked to the overall precondition for the development of all PFDs. The structure of the female pelvis and pelvic floor is a compromise between the requirements for the controlled evacuation of urine and feces and the ability to allow the passage of a fetus through the pelvic hiatus at delivery. The fact that PFDs are more common among nulliparous women or women who have undergone a CS [1, 6, 14, 21] as opposed to men of the same age, indicates that the female pelvic floor already from the outset is inherently weaker because of its wider pelvis, the width of its hiatus, and the arrangement, size, and strengths of the pelvic floor muscles, especially around the three outlets.

Table 3 Degree of frustration (as measured by IIQ-7) due to pelvic floor dysfunction in women with one, two or three PFDs in the total cohort

Number of PFDs	Number of women with PFD (*N*=2,051)	Degree of frustration, mean (SD)
1	1,302	1.50 (0.71)
2	601	2.25 (1.30)
3	148	5.03 (2.26)

Adjusted for current BMI, infant birth weight, and maternal age

Significance of difference: 1 PFD vs 2 PFDs, $p<0.001$; 2 PFDs vs 3 PFDs, $p<0.001$

PFD pelvic floor dysfunction, *SD* standard deviation, *IIQ-7* Incontinence Impact Questionnaire—Short Form

Table 4 Prevalence and odds ratio of co-occurring pelvic floor disorders in women grouped according to maternal height and fetal weight and mode of delivery

	All combinations of co-occurring pelvic floor disorders	
	%	[a]AdjOR (CI 95 %)
VD Maternal height ≤160 cm and IBW ≥4 kg (*n*=75)	28.1	
vs	vs	2.07 (1.18–3.64)
VD Maternal height ≤160 cm and IBW <4 kg (*n*=456)	15.8	
VD Maternal height ≤160 cm and IBW ≥4 kg (*n*=75)	28.1	
vs	vs	4.54 (2.42–8.49)
CS Maternal height ≤160 cm (all fetal weights) (*n*=380)	7.9	

IBW infant birth weight, *VD* vaginal delivery, *CS* cesarean section

*Chi-squared test was used for statistical comparison between groups

[a] Prevalence and odds ratios (OR) were adjusted for current weight and maternal age

These observations are supported by family studies that demonstrate an earlier onset and a more severe form of POP and UI in subjects with genetic predisposition [20, 22]. A possible genetic predisposition may also partly explain the occurrence of PFDs in nulliparous women and in those who have undergone CS delivery.

The main strengths of this study are the high response rate, the large study population recruited according to strict inclusion criteria from a national cohort of singleton primiparae and the long-follow-up time. There are also advantages of studying singleton parae 1, as the first delivery is considered to exert the greatest risk increase for PFDs [1, 23]. The inclusion of women regardless of maternal health status, and maternal and fetal complications, is considered a strength, as it allows for a greater generalization of results and therefore gives a better basis for consultation about mode of delivery. Some limitations of this study must, however, be considered. First, analyses of the non-responder group suggest a small selection bias on our results; however, acting in both directions (more younger women in the non-responders leading to overestimation of results; more overweight/obesity and heavier babies in non-responders leading to underestimation). Second, predisposition to participate in studies has been shown to be higher in symptomatic women, whereby the prevalence of PFDs might be overestimated [24]. Third, symptoms of PFD were self-reported. However, several studies have shown that self-reporting is consistent [25] and valid when symptoms exist at the time of the report [26, 27]. This study lacks information on whether PFDs were present before or during pregnancy, or started after delivery. The occurrence of PFDs in primiparous

women before and after a single delivery has recently been reported by other authors [28]. However, there is little evidence to suggest any difference in the prevalence of PFDs before or during pregnancy in women grouped according to mode of delivery. Nor was it possible to assess the effects of the length of the second stage of labor, as this is not documented in the MBR. The validity of family history in determining the heritability of PFDs has been questioned because of insufficient control of shared environmental factors within families and a lack of adjustment for mother's age. Results may also be overestimated because daughters with PFDs more likely discussed the problem with their mother. Together with classical twin studies, it has however been estimated that heritability might contribute to about half of the phenotypic variation for PFD [29].

Conclusion

In this large national cohort study of para 1 women we found that the prevalence of co-occurring PFDs 20 years after birth was high. Approximately one third of the women with a PFD had two or more PFDs. The prevalence of co-occurring PFDs was doubled in women after VD compared with CS. Risk factors for the clustering of PFDs were: VD, family history, ≥2-degree tears, maternal age, and current BMI. The effect of the resultant damage to the afflicted individual, as measured by the degree of frustration experienced because of pelvic floor dysfunction, increased with each additional co-occurring PFD. This study provides preliminary evidence that the clustering of PFDs may be used as an indicator of the extent of vaginal trauma following delivery.

Funding The study was supported by a National LUA/ALF grant no. 11315 and the Region of Västra Götaland, grants from The Göteborg Medical Society and Hjalmar Svenssons Fund. The funding source played no role in the study design, data analysis, data interpretation or writing of the report. MG and IM had full access to all study data and had final responsibility for the decision to submit for publication.

Authors' contributions M. Gyhagen, S. Åkervall, I. Milsom all contributed to protocol/project development, data collection, data analysis, manuscript writing, and editing.

Conflict of interest None.

References

1. Milsom I, Altman D, Cartwright R, Lapitan MC, Nelson R, Sillén U, Tikkanen K (2013) Epidemiology of urinary incontinence (UI) and other lower urinary tract symptoms (LUTS), pelvic organ prolapse (POP) and anal (AI) incontinence. In: Abrams P, Cardozo L, Khoury S, Wein A (eds) Incontinence. Health Publications, Paris, pp 15–107
2. Slieker-ten Hove MC, Pool-Goudzwaard AL, Eijkemans MJ, Steegers-Theunissen RP, Burger CW, Vierhout ME (2010) Prevalence of double incontinence, risks and influence on quality of life in a general female population. Neurourol Urodyn 29:545–550
3. Botlero R, Bell RJ, Urquhart DM, Davis SR (2011) Prevalence of faecal incontinence and its relationship with urinary incontinence in women living in the community. Menopause 18:685–689
4. Lawrence JM, Lukacz ES, Nager CW, Hsu J-WY, Luber KM (2008) Prevalence and co-occurrence of pelvic floor disorders in community-dwelling women. Obstet Gynecol 111:678–685
5. Rortveit G, Subak LL, Thom DH et al (2010) Urinary incontinence, faecal incontinence and pelvic organ prolapse in a population-based, racially diverse cohort: prevalence and risk factors. Female Pelvic Med Reconstr Surg 16:278–283
6. Gyhagen M, Bullarbo M, Nielsen TF, Milsom I (2013) The prevalence of urinary incontinence 20 years after childbirth: a national cohort study in singleton primiparae after vaginal or caesarean delivery. Br J Obstet Gynecol 120:144–151
7. Abrams P, Cardozo L, Fall M et al (2003) The standardisation of terminology in lower urinary tract function: report from the standardisation sub-committee of the International Continence Society. Urology 61:37–49
8. Tegerstedt G, Miedel A, Maehle-Schmidt M, Nyren O, Hammarstrom M (2005) A short-form questionnaire identified genital organ prolapse. J Clin Epidemiol 58:41–46
9. Wu MJ, Vaughan CP, Goode PS et al (2014) Prevalence and trends of symptomatic pelvic floor disorders in U.S. women. Obstet Gynecol 123:141–148
10. Nygaard I, Barber MD, Burgio KL, Kenton K, Meikle S, Schaffer J et al (2008) Prevalence of symptomatic pelvic floor disorders in US women. JAMA 300:1311–1316
11. Roberts RO, Jacobsen SJ, Reilly WT, Pemberton JH, Lieber MM, Talley NJ (1999) Prevalence of combined faecal and urinary incontinence: a community-based study. J Am Geriatr Soc 47:837–841
12. Uustal Fornell E, Wingren G, Kjølhede P (2003) Prevalence of urinary and faecal incontinence and symptoms of genital prolapse in women. Acta Obstet Gynecol Scand 82:280–286
13. Manning J, Eyers AA, Korda A, Benness C, Solomon MJ (2001) Is there association between faecal incontinence and lower urinary dysfunction? Dis Colon Rectum 44:790–798
14. Gyhagen M, Bullarbo M, Nielsen TF, Milsom I (2013) The prevalence and risk factors for pelvic organ prolapse 20 years after childbirth: a national cohort study in singleton primiparae after vaginal or caesarean delivery. Br J Obstet Gynecol 120:152–160
15. Gudmundsson S, Henningsson A-C, Lindqvist P (2005) Correlation of birth injury with maternal height and birthweight. Br J Obstet Gynecol 112:764–767
16. Patel DA, Xu X, Thomason AD, Ransom SB, Ivy JS, DeLancey JOL (2006) Childbirth and pelvic floor dysfunction: An epidemiologic approach to the assessment of prevention opportunities at delivery. Am J Obstet Gynecol 195:23–28
17. Van Delft K, Sultan AH, Thakar R, Schwertner-Tiepelmann N, Kluivers K (2014) The relationship between postpartum levator ani avulsion and signs and symptoms of pelvic floor dysfunction. Br J Obstet Gynecol 121:1164–1172
18. Dietz HP, Franco AVM, Sheck KL, Kirby A (2012) Avulsion injury and levator hiatal ballooning: two independent risk factors for prolapse? An observational study. Acta Obstet Gynecol Scand 91:211–214
19. Jack GS, Nikolova G, Vilain E, Raz S, Rodríguez LV (2006) Familial transmission of genitovaginal prolapse. Int Urogynecol J Pelvic Floor Dysfunct 17:498–501
20. McLennan MT, Harris JK, Kariuki B, Meyer S (2008) Family history as a risk factor for pelvic organ prolapse. Int Urogynecol J 19:1063–1069

21. Gyhagen M, Bullarbo M, Nielsen TF, Milsom I (2014) Faecal incontinence 20 years after one birth - A comparison between vaginal delivery and caesarean section. Int Urogynecol J 25:1411–1418
22. Slieker-ten Hove MC, Pool-Goudzwaard AL, Eijkemans MJ, Steegers-Theunissen RP, Burger CW, Vierhout ME (2009) Symptomatic pelvic organ prolapse and possible risk factors in a general population. Am J Obstet Gynecol 200:184.e1−184.e7
23. Milsom I, Ekelund P, Molander O, Arvidsson L, Areskoug B (1993) The influence of age, parity, oral contraception, hysterectomy and menopaus on the prevalence of urinary incontinence in women. J Urol 149:1459–1462
24. Thom DH, van den Eeden SK, Ragins AI, Wassel-Fyr C, Vittinghof E, Subak LL et al (2006) Difference in prevalence of urinary incontinence by race/ethnicity. J Urol 175:259–264
25. Grodstein F, Frett R, Lifford K, Resnick N, Curhan G (2003) Association of age, race, and obstetric history with urinary symptoms among women in the Nurses' Health Study. Am J Obstet Gynecol 189:428–434
26. Diokno AC, Brown MB, Brock BM, Herzog AR, Normolle DP (1988) Clinical and cystometric characteristics of continent and incontinent noinstitutionalized elderly. J Urol 140:567–571
27. Herzog AR, Fultz NH (1990) Prevalence and incidence of urinary incontinence in community dwelling populations. J Am Geriatr Soc 38:273–281
28. Durnea CM, Khashan AS, Kenny LC, Tabirca SS, O'Reilly BA (2014) The role of prepregnancy pelvic floor dysfunction in postnatal pelvic morbidity in primiparous women. Int Urogynecol J 25(10): 1363–1374
29. Wennberg A-L, Altman D, Lundholm C, Klint A, Iliadou A, Peeker R et al (2011) Genetic influences are important for most but not all lower urinary tract symptoms: a population-based survey in a cohort of adult Swedish twins. Eur Urol 59:1032–1038