Research article

Urinary incontinence during pregnancy – of predictive value?

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Abstract

Objective: Primary objective was to find the prevalence of urinary incontinence during pregnancy by its type and see how predicative it was of urinary incontinence in the long term. Associated risk factors were identified as secondary objectives.

Method: Antenatal clinics provide opportunistic screening for diseases of the breast, the heart or even the mind. Preventive gynaecological issues could also be addressed likewise. It was felt worth investigating the prevalence of urinary incontinence during pregnancy without undue harassment to the patients by way of a few routinely directed questions about urinary incontinence.This information is rarely volunteered due to embarrassment.

867 women attending antenatal clinics led by the author at teaching hospital Peradeniya, in 2007 and 2008 were interviewed during pregnancy and those incontinent were followed up post-partum at 6 weeks,one and two years till 2010. Patients were inquired regarding the predominant type of incontinence, when the symptoms were mixed(stress and urge both).

Results: Of the 867 women analysed, 209 (24%) had some form of urinary incontinence. 110 (52%) of those affected had pure or predominant stress, and 15 (7%) pure or predominantly urge incontinence. The rest 41% had a mixed picture.

154 were still incontinent at 6 weeks post-partum. The proportion of the type of incontinence had changed. 97 (63%) had pure or predominantly stress incontinence, 6 (3.9%) had pure or predominantly urgency and the 33.1% had a mixed picture.

47 of them still complained of some degree of incontinence at the end of one year despite interventions of pelvic floor exercise (PFE),for urodynamic stress incontinence or mixed symptoms.

The patterns were even more stress predominant. 33(70%). 2 patients (4.2%) had pure urgency and the rest 12 (25.5%) had a mixed pattern.

End of 2 years only 43 patients of those incontinent at 1year were traceable at least over the telephone. Only 23 complained of persistence of the problems.

Of the 23 with symptoms at 2 years, 2 were pregnant again. 18 (78%) had pure SI and the rest 5 (22%) had a mixed picture.

This indicated that pure stress incontinence tended to persist more than urge incontinence probably indicating some form of permanent damage to the pelvic floor. Older and women of higher parity were found to be more affected again indicating possibility of such injury.

Conclusions: The finding that of the initial 209 women who leaked urine during pregnancy 23 remained incontinent at the end of 2 years (11%), is evidence that incontinence during pregnancy is of some predictive value in finding those likely to have long term incontinence.The symptoms of stress incontinence persisted despite PFE. This places doubts on benign physiological and hormonal change theories which is discussed alongside these findings. Incontinence during pregnancy maybe a useful indicator of injury sustained to the pelvic floor providing early warning for trouble ahead with the sphincter mechanism. Questions about urinary incontinence as a routine during antenatal consultations may be a worthwhile exercise. This would take only a very short time and should be encouraged as a higher percentage would have remained incontinent had PFE not been initiated as an ethical intervention.

Key words: pregnancy, urinary incontinence

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Introduction and objectives

Primary objective of this study was to find the prevalence of urinary incontinence during pregnancy by its type and see how predicative it was of long term urinary incontinence. Associated risk factors were identified as secondary objectives.

Antenatal clinics provide opportunistic screening for diseases in the Breast and the Heart. Preventive gynaecology could also commence likewise. It was felt worth investigating the prevalence of urinary incontinence during pregnancy without undue harassment by way of a few routinely directed questions about urinary incontinence. This information is rarely volunteered due to embarrassment.

Sri Lankan women will rarely volunteer symptoms of urinary incontinence unless it is severe enough to interfere with daily routine, often through embarrassment. Incontinence could be urge or stress or a combination of both and could be due to benign reasons like the pressure on the bladder exerted by the gravid uterus. But most commonly cited cause of pregnancy-related urinary incontinence or urgency is weak pelvic floor muscles. This might be temporary as the pelvic floor muscles relax to prepare for labour. It was felt interesting to see whether this was essentially the case specially when there is predominant or pure stress incontinence, more indicative of sphincter dysfunction. It is possible that those with abnormally weak pelvic floors get unmasked by the challenge of pregnancy-allowing early warning for continued surveillance and interventions.

If a relationship of incontinence during pregnancy were to persist beyond puerperium, symptoms during pregnancy could serve as a predictor of troubles ahead. It may become a worthwhile exercise to make routine inquiry with a view of at least advocating benign measures like PFE to minimize progression.

Method

867 women attending antenatal clinics supervised by the author for booking, in 2007 and 2008 were followed up after delivery at 6 weeks, and at end of one and two years, only if they had complained of incontinence during pregnancy. They were re-interviewed again in the last visit to see whether incontinence cropped up later than the first antenatal visit.

Patients with mixed symptoms of stress and urgency were inquired regarding the predominant pattern (which symptom bothered them most). Data concerning the prevalence of urinary incontinence was collected by a structured questionnaire and clinical assessment performed only where there was leakage 6 weeks post-partum. Pelvic floor muscle strength was not measured and pad testing was not resorted to. Inspection was done to see overt injury and demonstrable leak. Only the persistence of symptoms was recorded for analytic purposes. Such incontinent patients were requested to attend my gynaecology clinic with a special emphasis on uro-gynaecology. Faecal or incontinence of flatus indicative of pelvic floor dysfunction was inquired upon but not included in the study as no one strangely admitted those symptoms perhaps through embarrassment. Symptoms were categorized as stress or predominantly stress as stress incontinence, predominantly or pure urgency as urgency and when it was not pure or predominantly of any of them as mixed.

Parity, (but not gravidity) and mode of any prior deliveries were noted as they could indicate injury to the pelvic floor. Patients on medications that are known to affect bladder functions were excluded. Details of the socio economic class was not inquired into as vast majority of the patients attending the state run clinic were from the lower middle or lower socioeconomic strata.

Patients with evidence of overt urinary tract infection were excluded by performing routine UFR and culture where indicated. The short term urgency or other forms of incontinence due to bladder infections and irritation was not the sought out issue but the incidence of unrecognised pelvic floor dysfunction. Those who were identified less likely to present for follow up at Peradeniya hospital (visiting only for confinement) were not included. Patients with any significant history of psychiatric disorder were excluded for being unreliable witness of bladder symptoms, being less likely to present for follow up. Any medications like imipramine could also mask symptoms of urgency.

Results

The mean age of the 867 women recruited was 27.4 yrs ranging from 16 to 43 yrs.

The age distribution was as; under 16-25 (430); 26-35 (308) and those over 36 (129).

The total number of incontinent anytime during the antenatal period was 209. (overall 24%)

Among each group the incidence of incontinence during the pregnancy by age group was;

16-26 age group -59 of 430 patients (13.7%); 26-35 age group - 90 out of 308 patients (29%); and in the over 36 age group -60 out of 129 patients (46.5%) indicating an increase with age.

The parity was analysed in relation to incidence of incontinence. 444 were in their first pregnancy, 301 had delivered one child (183 normal vaginal delivery; 106 by LSCS, 12 by assisted vaginal delivery); and the rest (122) had delivered more than one child (56 had one or more LSCS). The 209 total number of incontinent patients were distributed by parity as 54 of the 444 (12%) for those in first pregnancy ; 101 of 301(33.6%) who had delivered one child by any means, and 54 among of the 122 (44.3%) who had delivered more than 2 children.

As the study was not intended to establish the impact of vaginal birth over caesarean and assisted deliveries. Detailed complex analysis by type of delivery was not done. However of the 301 who had one delivery with 101 incontinent amongst them; the 106 women who had delivered by LSCS had lower percentage of incontinence. Only 20 (18.8%) of the patients within that group of 106, having previously delivered one child by LSCS leaked urine. Those 183 delivering vaginally unassisted 75 leaked urine (41%). Of the 12 who had undergone assisted vaginal deliveries 6 (50%) leaked urine.

The primary objective of the investigation was to find the persistence of the incontinence by type of symptom, and persistence over time.

Of 867 women analysed 209 (24%) had some form of incontinence by the end of antenatal period. 110 (52%) of those affected had pure or predominant stress, and 15 (7%) pure or predominant urge incontinence. The rest 41% had a mixed picture where clear predominance of one symptom over the other could not be established.

During the first visit in all 867 presenting in first visit in first trimester only 12 had incontinence with just 2 stating it preceded the current pregnancy. These 2 patients were multiparous and had predominantly stress incontinence. The number rose steeply to 209 by the end of the antenatal period by around 36-40 week interview, indicating the problem likely arose in the mid trimester.

(6 of the patients had urgency and 4 had a mixed pattern and 2 had stress in the first trimester interview).

Only 204 0f those with incontinence during pregnancy (209), presented for follow up at 6 weeks. (5 lost for follow up). Of these 204 who had incontinence during pregnancy and presenting for follow up 154 were still incontinent! The proportion of the type of incontinence had changed. 97 (63%) had stress incontinence, 6 (3.9%) had urgency and 51 (33.1%) had a mixed picture indicating that pure or predominantly stress incontinence tended to linger on more than urge incontinence.

Of the 663 who originally did not complain of incontinence during pregnancy 22 complained of incontinence following the pregnancy (new case load). 14 were following caesarean deliveries involving catheterizations of bladder. These data were not included in the analysis as the objective was to see the frequency of persistence or regression of the symptoms as found during the antenatal period and not caused by the mode of delivery.

Of the 154 who were incontinent at 6 weeks 133 patients were traced at around approximately one year since delivery at the gynaecology outpatient clinic.

Of these 133 traceable incontinent patients 104 had followed the advice given on PFE, and 4 used solifenacin for ethical reasons as incontinence was severely disturbing them.

47 of them still had some degree of incontinence at one year despite interventions at gynae clinic.

The patterns were again even more stress predominant. 33 (70%). 2 (4.2%) had pure urgency and the rest 12 (25.5%) had a mixed pattern.

At the end of 2 years only 43 patients of the incontinent at 1 year clients (n=47) were traceable at least over the phone and only 23 complained of persistence of the symptoms. 20 reported being cured spontaneously or due to PFE, the exact contribution indeterminable.

Of the 23 with symptoms at 2 years; 2 were pregnant again. 18 (78%) had pure SI and the other 5 (22%) had a mixed picture.

Of the initial 209 women who were incontinent during their antenatal period initially, 23 remained incontinent even at the end of 2 years which places doubts on simple physiological change but more on the possibility of some permanent injury taking place.

Conclusions

Urinary incontinence has a frequent association with pregnancy in nearly a quarter of women. Only 2 of our study had any such symptoms before the pregnancy.

Parity and age had a clear relationship with the incidence of incontinence; increasing with age and number of deliveries. History of vaginal delivery had a higher association with incontinence during subsequent pregnancy.

Assisted vaginal deliveries, admittedly too small in number for drawing statistically significant conclusions, seemed to fare worst with a 50% incidence. Whether it was due to an event like prolonged labour indicating the assistance or the procedure itself could not be ascertained as it was beyond the scope of the study.

The pattern was mostly of pure or predominantly stress over urgency regardless of the timing.

Stress incontinence tended to persist with time than urgency which mostly disappeared with time.

Effects of PFE /solifenacin may have made the result less marked as it was felt unethical to withhold treatment for the sake of the study.

Of the initial 867 women studied 23 (2.65%) remained incontinent even at the end of 2 years with stress predominance, raising the possibility of structural injury than mere physiological changes to account for symptoms. (This is a raw figure with the dropouts included in the denominator).

Discussion

Urinary incontinence in pregnancy is encountered infrequently by obstetricians probably because it is simply not inquired about during the antenatal visits. This is despite reported prevalence rates before, during and after pregnancy of 3.6-11%, 42-62%, and 5-38%, respectively from numerous studies done world over[12,13,14,15,16]. The wide variation in incidence amongst studies quoted may be due to different study designs and different methods of assessing incontinence.

Our findings indicated while urgency incontinence seemed self-limiting, stress predominant incontinence remained unresolved over time in a significant proportion of patients. The attention of discussion thus focuses more on stress urinary incontinence (SUI) with likely involvement of the pelvic floor and the urethral sphincter. Stress Urinary incontinence could also be due to detrusor hyperreflexia /over active bladder states but the absence of associated symptom of urgency points more towards genuine/ urodynamic stress incontinence. It was impractical and felt invasive to perform urodynamic assessments to make a precise diagnosis of the aetiology of the SUI though the resources were available at the urology laboratory.

A significantly higher incidence of SUI is reported in pregnant women than in non-pregnant women[10]. Prior Research on pregnant women with SUI has found significantly decreased PFM strength in incontinent pregnant women compared with continent pregnant women[8]. Pregnancy due to hormonal effects may result in reduced PFM strength, which may manifest as SUI. However, the pathophysiology of pregnancy-related SUI remains uncertain and multifactorial.

The association between obesity in pregnancy and SUI is reported by many. Glazener[11] reports that women with UI in antenatal period had a BMI > 25 kg/m^2 [OR 1.68, 95% confidence interval (CI) 1.12-2.43]. Zhu et al[24] reported that the risk of SUI increased with increasing prenatal BMI [odds ratio (OR = 1.037)]. Liang et al[5] reported that women with a prepregnancy BMI >30 kg/m^2 were at elevated risk of developing SUI during pregnancy.

Physiological weight gain in pregnancy and the weight of the developing foetus and the resultant increased pressure on the PFM and bladder, may cause a degree of urethral hypermobility[25,26]. Furthermore, excess maternal weight gain not subjected to analysis in this study, is reported to impair blood flow and innervations to the bladder and urethra[26].

Multigravidity is reported to decrease pelvic floor strength at a rate of 22-35% beginning at a gestational age of 20 weeks and lasting until 6 weeks postpartum[7]. We analysed parity and found a similar pattern. Hilde et al[30], in a study of 300 nulliparous women at 18-22 weeks' gestational age showed that continent against incontinent pregnant women had significantly higher PFM strength.

Increase of progesterone and relaxin during pregnancy may have a bearing in the development of SUI[30]. Relaxin is said to stimulate tissue growth in the lower urinary tract and increase urethral pressure. Bladder-

neck hypermobility from early pregnancy has been reported 36 more so in incontinent over continent women.

A substantial decrease in the total collagen content in women with SUI is reported by many[25,27]. They also report a both quantitative and qualitative reduction in collagen.

Trauma at childbirth is well known to cause damage to the pudendal nerve, levatorani muscle, fascial pelvic supports, and the anal sphincters[27]. This damage reduces PFM strength results in increased bladder-neck and urethral mobility[28]. 3D scans of the pelvic floor demonstrates avulsion of pubo-coccygeus muscles in a significant proportion of women following partus. The result would be a descent of the vesico - urethral junction below pelvic floor[1].

Abnormal urethral pressure profile parameters were measured in almost all pregnant women who experienced SUI during pregnancy suggesting an inherent weakness of the urethral sphincter mechanism in the pathogenesis of SUI[31]. Therefore the damage precedes possible trauma at child birth.

Enlargement of the uterus with the progressive increase in foetal weight, especially at the third trimester appears to make matters worse. These put extra pressure direct on the bladder, which may lead to descent of the bladder-neck and a reduction of the bladder capacity explaining events on the basis of simple mechanics.

Increased urinary frequency and urgency are early symptom of pregnancy often attributed to hormonal changes again. The urges tend to reduce in the second trimester but it did seem severe enough to cause incontinence troubling some patients attending our clinics.

PFE is always the first-line intervention to treat SUI as most cases resolved with reversal of pregnancy changes.

We are inclined to address only obstetric issues in the obstetrics clinics and gynaecology in the gynae clinics not realizing that these arbitrary boundaries inconvenience the patients being shuffled between multiple clinics conducted on different dates due to lack of multidisciplinary approach. Understanding these issues can be useful for health-care professionals including field midwives when counselling and following up pregnant women. It would help detect audit and refer all forms of urinary incontinence during pregnancy and the puerperium. Even PFE is best achieved when motivated by a field worker. Obesity and high BMI before pregnancy are potentially modifiable risk factors for SUI. Weight reduction may be an effective alongside to PFE in minimising the problem.

However a reasonable number of women remained incontinent at the end of 2 years follow up despite PFE allows us to diagnose those who may have had substantial pelvic floor injury. The avulsion of the muscle is palpable and demonstrable on 3D pelvic floor scanning as the routine in some hospitals and specialized centres like the pelvic floor institute in Boston USA. Merely delivering a baby without brain damage and avoiding maternal mortality is superseded by higher expectations of quality which includes avoidance of pelvic floor injury and resultant mental and sexual health issues. For a better quality of life during longer post-menopausal years, the practice of good obstetrics and preventive gynaecology earlier on during the reproductive life is paramount.

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References

1. King JK, Freeman RM. Is antenatal bladder neck mobility a risk factor for postpartum stress urinary incontinence? Br J Obstet Gynaecol 1998; 105: 1300-7. [PubMed] [Google Scholar]

2. Handa VL, Harvey L, Fox HE, Kjerulff KH. Parity and route of delivery: does cesarean delivery reduce bladder symptoms later in life? Am J Obstet Gynecol 2004; 191(2): 463-9. doi: 10.1016/j.ajog.2004.03.031. PMID: 15343222.

3. Sharma JB, Aggarwal S, Singhal S, Kumar S, Roy KK. Prevalence of urinary incontinence and other urological problems during pregnancy: a questionnaire based study. Arch Gynecol Obstet 2008; 279(6): 845-51.

4. van de Pol GG, Van Brummen HJ, Bruinse HW, Heintz AP, van der Vaart CH. Is there an association between depressive and urinary symptoms during and after pregnancy? Int Urogynecol J Pelvic Floor Dysfunct 2007; 18: 1409-15.

5. Liang CC, Chang SD, Lin SJ, Lin YJ. Lower urinary tract symptoms in primiparous women before and during pregnancy. Arch Gynecol Obstet 2012; 285(5): 1205-10.

6. van Brummen HJ, Bruinse HW, van de Pol G, Heintz AP, van der Vaart CH. Bothersome lower urinary tract symptoms 1 year after first delivery: prevalence and the effect of childbirth. BJU Int 2006; 98(1): 89-95.

7. FitzGerald MP, Graziano S. Anatomic and functional changes of the lower urinary tract during pregnancy. Urol Clin N Am 2007; 34(1): 7-12.

8. Morkved S, Salvesen KA, Bo K, Eik-Nes S. Pelvic floor muscle strength and thickness in continent and incontinent nulliparous pregnant women. Int Urogynecol J Pelvic Floor Dysfunct 2004; 15: 384-90.

9. Hojberg KE, Salvig JD, Winslow NA, Lose G, Secher NJ. Urinary incontinence: prevalence and risk factors at 16 weeks of gestation. Br J Obstet Gynecol 1999; 106: 842-50.

10. MacLennan AH, Taylor AW, Wilson DH, Wilson D. The prevalence of pelvic floor disorder and their relationship to gender, age, parity and mode of delivery. Br J Obstet Gynecol 2000;107:1460-1470.

11. Glazener CM, Herbison GP, MacArthur C, et al. New postnatal urinary incontinence: obstetric and other risk factors in primiparae. Br J Obstet Gynaecol 2006; 113(2): 208-17.

12. Chaliha C, Kalia V, Stanton SL, Monga A, Sultan AH. Antenatal prediction of postpartum urinary and fecal incontinence. Obstet Gynecol 1999; 94: 689-94.

13. Marshall K, McConnell V, Whelan M, Totterdal D, Walsh DM. Urinary incontinence and constipation during pregnancy and after childbirth. Physiotherapy 1996; 82: 98-103.

14. Thorp JM, Norton PA, Wall LL, Kuller JA, Eucker B, Wells E. Urinary incontinence in pregnancy and the puerperium: a prospective study. Am J Obstet Gynecol 1999; 181: 266-73.

15. Spellacy E. Urinary incontinence in pregnancy and the puerperium. J Obstet Gynecol Neonatal Nurs 2001; 30: 634-41.

16. Morkved S, Bo K. Prevalence of urinary incontinence during pregnancy and postpartum. Int Urogynecol J Pelvic Floor Dysfunct 1999; 10: 394-8.

17. Schytt E, Lindmark G, Waldenstrom U. Symptoms of stress incontinence 1 year after childbirth: prevalence and predictors in a national Swedish sample. Acta Obstet Gynecol Scand 2004; 83: 928-36.

18. Burgio KL, Zyczynski H, Locher JL, Richter HE, Redden DT, Wright KC. Urinary incontinence in the 12-month postpartum period. Obstet Gynecol 2003; 102: 1291-8.

19. Hvidman L, Foldspang A, Mommsen S, Nielsen JB. Postpartum urinary incontinence. Acta Obstet Gynecol Scand 2003; 82: 556-63.

20. Liang CC, Wu MP, Lin SJ, Lin YJ, Chang SD, Wang HH. Clinical impact of and contributing factors to urinary incontinence in women 5 years after first delivery. Int Urogynecol J 2013; 24(1): 99-104.

21. Whitford HM, Alder B, Jones M. A cross-sectional study of knowledge and practice of pelvic floor exercises during pregnancy and associated symptoms of stress urinary incontinence in North-East Scotland. Midwifery 2007; 23(2): 204-17.

22. Huebner M, Antolic A, Tunn R. The impact of pregnancy and vaginal delivery on urinary incontinence. Int J Gynecol Obstet 2010; 110(3): 249-51. [PubMed] [Google Scholar]

23. Viktrup L, Lose G, Rolff M, Barford K. The symptom of stress incontinence caused by pregnancy or delivery in primiparas. Obstet Gynecol 1992; 79: 945-9.

24. Zhu L, Li L, Lang JH, Xu T. Prevalence and risk factors for peri-and postpartum urinary incontinence in primiparous women in China: a

prospective longitudinal study. Int Urogynecol J 2012; 23(5): 563-72.

25. Pregazzi R, Sartore A, Troiano L, et al. Postpartum Urinary symptoms: prevalence and risk factors. Obstet Gynecol 2002; 103(2): 179-82.

26. Greer WJ, Richter HE, Bartolucci AA, Burgio KL. Obesity and pelvic floor disorders: a systematic review. Obstet Gynecol 2008; 112(2 Pt 1): 341-9.

27. Dietz HP, Schierlitz L. Pelvic floor trauma in childbirth-myth or reality? Aust N Z J Obstet Gynaecol 2005; 45(1): 3-11.

28. Keane DP, Sims TJ, Abrams P, Bailey AJ. Analysis of collagen status in premenopausal nulliparous women with genuine stress incontinence. Br J Obstet Gynaecol 1997; 104(9): 994-8.

29. Wijma J, Weis Potters AE, Tinga DJ, Aarnoudse JG. The diagnostic strength of the 24-h pad test for self-reported symptoms of urinary incontinence in pregnancy and after childbirth. Int Urogynecol J Pelvic Floor Dysfunct 2008; 19(4): 525-30.

30. Hilde G, Staær-Jensen J, Ellström Engh M, Braekken IH, Bo K. Continence and pelvic floor status in nulliparous women at midterm pregnancy. Int Urogynecol J 2012; 23(9): 1257-63.

31. van Geelen JM, Lemmens WA, Eskes TK, Martin CB., Jr. The urethral pressure profile in pregnancy and after delivery in healthy nulliparous women. Am J Obstet Gynecol 1982; 144(6): 636-49.