

# Urinary incontinence after obstetric anal sphincter injuries (OASIS)—is there a relationship?

Inka Scheer · Vasanth Andrews · Ranee Thakar ·  
Abdul H. Sultan

Received: 23 January 2007 / Accepted: 5 July 2007  
© International Urogynecology Journal 2007

**Abstract** This study aimed to compare urinary symptoms and its impact on women's quality of life after obstetric anal sphincter injuries (OASIS) with a matched control group in the short term. The study group consisted of 100 primiparous women with OASIS and 104 controls who sustained a second-degree tear or had a mediolateral episiotomy performed. All women completed a validated International Consultation on Incontinence Questionnaire-Short Form (ICIQ-SF) questionnaire 10 weeks after delivery. Compared to controls, significantly more women with OASIS reported overall urinary incontinence (21.2 vs 38%,  $p=0.005$ ) and had significantly worse quality of life score (incontinence score: 2.42 vs 1.2;  $p=0.008$ ). Significantly more women with OASIS suffered from stress urinary incontinence (33 vs 14%;  $p=0.002$ ; OR 3.06; CI=1.54–6.07) than controls. Logistic regression analysis revealed that OASIS and a prolonged ( $>50$  min) second stage of labour were independent risk factors for the development of stress urinary incontinence. This study highlights the importance of inquiring about urinary incontinence in women with OASIS.

**Keywords** Urinary incontinence · Vaginal delivery · Anal sphincter tear

Presented to the German Forum Urodynamikum 2006 by IS (11.03.2006) and received the Eugen-Rehfisch Award of the German Forum Urodynamikum.

I. Scheer · V. Andrews · R. Thakar (✉)  
Urogynaecology Unit, Mayday University Hospital,  
530, London Road,  
Croydon CR7 7YE, UK  
e-mail: ranee.thakar@mayday.nhs.uk

## Introduction

Urinary incontinence (UI) occurs in 53% of women over 20 years of age [1]. The prevalence of urinary incontinence during pregnancy is reported to be as high as 44% [2] but appears to vary between 7 [3] and 34% [4] after delivery. The aetiology is unknown, but intrapartum pudendal neuropathy [5–9] and muscle trauma altering the urethral support [10, 11] have been incriminated.

Obstetric anal sphincter injuries (OASIS) occur in up to 19% of vaginal deliveries [12] and are an established risk factor for the development of anal incontinence [13, 14]. Urinary incontinence after OASIS has been reported in 13–46% [15–18]. However, long term follow-up of women with OASIS reveals conflicting results. Fornell et al. [18] demonstrated a strong correlation between OASIS and faecal and urinary incontinence. Women with OASIS had a higher risk of urinary incontinence compared to women without a tear or a small perineal tear [18]. However, no explanation was given as to why women with OASIS were at a higher risk of developing urinary incontinence. By contrast, Otero et al. [19] found no significant increase in urinary incontinence 18 years after OASIS when compared to parity-matched controls [19]. Others have identified a range of risk factors for the development of urinary incontinence after childbirth, namely, vaginal delivery when compared to caesarean section [20–22], vacuum extraction, [17] forceps delivery [22], pre-existing urinary incontinence, [17] older maternal age [20], and prolonged second stage of labour [7, 9].

Very little data is available on postpartum urinary incontinence and quality of life. Glazener et al. [20] reported recently that around 50% of incontinent women were affected by their urinary incontinence symptoms

postpartum in terms of hygiene, home work, and social life, but they used a non-validated questionnaire.

The aims of our study were to establish the prevalence of urinary incontinence after OASIS in primiparous women on short-term follow-up to identify risk factors and evaluate its impact on quality of life.

## Materials and methods

Primiparous women with OASIS were followed up at 3 months postpartum in a dedicated one-stop perineal clinic, where they completed the validated International Consultation on Incontinence Questionnaire-Short Form (ICIQ-SF) questionnaire [23, 24]. The control group consisted of primiparous women who sustained a spontaneous second degree tear or had a mediolateral episiotomy. The control group were recruited between February 2003 and January 2004 and also participated in another trial that has previously been published [25]. All women in this group had a second perineal examination in the immediate postpartum period by an experienced research fellow as well as an endoanal scan (repeated at 3 months). Therefore, none of the women had “occult” or unrecognized injuries.

Demographic data and labour details were collected prospectively.

Statistical analyses were performed using the Statistical Package for Social Sciences (SPSS, Chicago, IL, version 12.0). Differences in proportions of categorical variables were calculated using the chi-square test. Differences between the groups were assessed using Student's *t* tests and Mann–Whitney *U* tests for parametric and nonparametric data, respectively. Univariate analysis have been performed to determine risk factors given in odds ratio (OR) and 95% confidence intervals (CI). In a logistic regression analysis, the following variables have been entered in the model to determine independent risk factors for overall and stress urinary incontinence: OASIS, second

**Table 1** Characteristics of women after OASIS and controls

	OASIS, <i>n</i> =100	Controls, <i>n</i> =104	<i>P</i> value
Age, mean (SD)	28.9 (5.1)	28.0 (5.5)	0.23 <sup>a</sup>
BMI (SD)	25.04 (5.1)	24.59 (4.5)	0.58 <sup>a</sup>
Instrumental delivery, <i>n</i> (%)	39 (39)	29 (28)	0.07 <sup>b</sup>
Second stage, minutes (SD)	66.42 (55)	55.10 (40)	0.11 <sup>a</sup>
Epidural, <i>n</i> (%)	28 (28)	28 (27)	0.83 <sup>b</sup>
Birth weight, g, mean (SD)	3,457 (535)	3,231 (423)	0.001 <sup>a</sup>
Head circumference, mm, mean (SD)	343 (16)	336 (15)	0.005 <sup>a</sup>
Mediolateral episiotomy (%)	60 (60)	41 (39)	0.002 <sup>b</sup>

<sup>a</sup> Student's *t* test

<sup>b</sup> Chi-square test

**Table 2** Frequency of leakage

	OASIS, <i>n</i> =100	Controls, <i>n</i> =104
Never	62	82
Once a week or less	19	11
2–3/week	7	5
Once/day	6	2
Several times/day	6	3
All the time	—	1

stage of labour >50 min, and head circumference > median of 339.6 mm.

Ethical approval for the control group was approved by the Croydon Ethics and Research Committee. Data of women having OASIS was collected from the perineal clinic. This questionnaire forms part of our normal evaluation of patients with OASIS. Patients gave written consent to use the data from questionnaires for scientific publications, and this was sanctioned by the Croydon Research and Development Committee.

## Results

Between July 2002 and January 2005, of the 102 primiparous with a diagnosis of an OASIS in our unit, 100 women returned for the postnatal visit and were included in this study (return rate 98%). One hundred and four primiparous controls were seen postpartum. In the control group, 95% of the invited women participated in this study and 86% attended the follow up. The postpartum visit took place between 5 and 15 (mean, 10±5) weeks after delivery. In the control group, OASIS were classified and repaired according to the Royal College of Obstetricians and Gynaecologists (RCOG) guidelines [26]. Forty-eight women sustained a 3a tear [less than 50% of the external anal sphincter (EAS) involved], 42 women a 3b tear ( $\geq$ 50% of the EAS involved), 5 women a 3c tear [EAS and internal anal sphincter (IAS) torn], and 5 women sustained a fourth degree tear (EAS, IAS, and anal epithelium torn).

Demographic data and labour details for women with OASIS and controls are given in Table 1.

Age, length of second stage of labour, and instrumental delivery did not differ significantly between the study group (OASIS) and controls (Table 1).

The OASIS group reported significantly more symptoms of urinary incontinence (leakage never: OASIS 62%, controls 78.8%) and a larger volume of urinary leakage (moderate amount: OASIS 32%, controls 0%,  $p=0.017$ , Tables 2 and 3).

The ICIQ urinary incontinence score (Table 4) was significantly higher in women with OASIS (2.42±3.7).

**Table 3** Amount of leakage

	OASIS, n=100	Controls, n=104
None	62	82
Small amount	3	21
Moderate amount	32	—
Large amount	3	1

compared to controls ( $1.20 \pm 2.7$ ,  $p=0.008$ ). However, the grade of OASIS did not affect the ICIQ urinary incontinence score ( $p=0.507$ ).

Quality of life (QoL), as assessed by the ICIQ-SF, was significantly worse after OASIS (0.88 vs 0.31,  $p=0.01$ ).

The different types of urinary incontinence are shown in Table 5. Significantly more women after OASIS reported urinary leakage while coughing and sneezing (OASIS 30%, controls 11.5%,  $p=0.005$ ) or while they were physically active (OASIS 12%, controls 3.8%,  $p=0.03$ ). No significant differences were found regarding urinary leakage during any of the following: sleep, before reaching the toilet, or after urinating (Table 5).

Risk factors (univariate analysis) for postpartum urinary incontinence are shown in Tables 6 and 7. The median duration of the active second stage of labour in our cohort was 50 min, and the median head circumference was 339.6 mm. OASIS, a second stage of labour longer than 50 min (median), and a head circumference larger than 339.6 mm (median) were significant risk factors for overall urinary incontinence and stress urinary incontinence (Tables 6 and 7). In a logistic regression analysis, OASIS and a second stage of labour >50 min were independent risk factors for stress urinary incontinence (Table 8).

## Discussion

Previously published studies on OASIS have focused mainly on anal symptoms [9, 12, 13], and only few studies [12, 15–17, 19, 27] have investigated urinary incontinence. There have been three studies with short-term follow up of

**Table 5** Type of urinary incontinence according to symptoms

When does urine leak?	OASIS, n=100	Controls, n=104	P value
Never, n (%)	62 (62)	82 (78.8)	0.005 <sup>a</sup>
When you cough, sneeze, and physically active, n (%)	33 (33)	15 (14.4)	0.002 <sup>a</sup>
When you are asleep, n (%)	3 (3)	0	0.116 <sup>b</sup>
Before you can get to the toilet (%)	18 (18)	10 (9.6)	0.104 <sup>b</sup>
When you have finished urinating (%)	2 (2)	2 (1.9)	0.678 <sup>b</sup>
For no obvious reason (%)	6 (6)	2 (1.9)	0.272 <sup>b</sup>
All the time (%)	0	1 (1)	1.0 <sup>b</sup>

<sup>a</sup>Chi-square test

<sup>b</sup>Fisher's exact test

urinary symptoms after OASIS [12, 16, 27]. Urinary symptoms were reported in up to 31% [16] with a high incidence of urge urinary incontinence [12], but a validated questionnaire was not used [12, 16] nor did they compare their findings to women without OASIS [16]. Longer term urinary symptoms after OASIS have been evaluated in three studies [15, 17, 19]. In a retrospective study using a non-validated questionnaire, Wagenius and Laurin [15] reported no difference in urinary symptoms 4 years after OASIS compared to a control group. The prevalence of urinary incontinence was 13% for OASIS and 9% for controls. In a prospective study ( $n=72$ ), Tetschner et al [17] reported urinary symptoms in 32% of women and demonstrated a relationship between the degree of anal sphincter rupture (1, less than one third of anal sphincter muscle affected; 2, more than one third affected; 3, complete rupture of the anal sphincter without damage to the anal mucosa) and urinary incontinence at 2 to 4 years after OASIS. Women with a total rupture of the anal sphincter had a greater risk of developing urinary incontinence compared to women with a partial rupture [17]. However, symptoms were not evaluated using a validated questionnaire nor was there a control group. Otero et al [19] used a validated questionnaire and found no difference in almost all urinary symptoms 18 years after OASIS compared to parity-matched controls. Women with OASIS reported

**Table 4** Urinary incontinence and score in women with OASIS and controls

	OASIS, n=100	Controls, n=104	P value
Overall urinary incontinence, n (%)	38 (38)	22 (21.2)	0.008 <sup>a</sup>
Quality of life score Mean (SD)	0.88 (1.79)	0.31 (0.91)	0.01 <sup>b</sup>
Incontinence score, mean (SD)	2.42 (3.7)	1.20 (2.7)	0.008 <sup>b</sup>

<sup>a</sup>Chi-square test

<sup>b</sup>Mann–Whitney U test

**Table 6** Risk factors for overall urinary incontinence (univariate analysis)

	OR (CI)
OASIS	2.28 (1.23–4.25)
Second stage of labour > median 50 min	2.28 (1.16–4.45)
Birth weight > median of 3,380 g	0.96 (0.62–1.48)
Head circumference > median of 339.6 mm	1.84 (1.10–3.06)
Mediolateral episiotomy	1.36 (0.88–2.09)
Epidural	1.19 (0.76–1.86)
Instrumental delivery	1.47 (0.99–2.15)

**Table 7** Risk factors for stress urinary incontinence (univariate analysis)

	OR (CI)
OASIS	3.06 (1.54–6.07)
Second stage of labour > median 50 min	2.63 (1.26–5.50)
Birth weight > median of 3380 g	0.96 (0.62–1.48)
Head circumference > median of 339.6 mm	1.84 (1.10–3.06)
Mediolateral episiotomy	1.36 (0.88–2.09)
Epidural	1.19 (0.76–1.86)
Instrumental delivery	1.39 (0.93–2.08)

significantly more frequent leakage of small amounts of urine when compared to controls ( $p=0.04$ ). However, the proportion of women who had sought treatment for urinary incontinence was similar in both groups. More recently, Borello-France et al. [27] performed a large prospective study investigating the relationship between anal sphincter tears and postpartum faecal and urinary incontinence. At 6 weeks and 6 months, they found no significant difference in the prevalence of urinary incontinence between the OASIS and control group. However, there were considerable differences between the groups in terms of episiotomy rates, instrumental deliveries, birth weight, and length of labour.

Our study was conducted prospectively with a representative control group in which all women had either a spontaneous second-degree tear or a mediolateral episiotomy. All women in the control group had a repeat perineal examination and an endoanal scan immediately after delivery and at 3 months postpartum. Therefore, none of the control group had “occult” or unrecognised anal sphincter injuries [13, 25]. This was to ensure that in terms of the perineal trauma, the only real difference between the study and the control groups were that the study group sustained additional anal sphincter injury. In our study, postnatal stress urinary incontinence at 10 weeks after OASIS was reported in 33% compared to 14% of the control group ( $p=0.002$ ), and women with OASIS reported a worse impact on their quality of life compared to controls (QoL score, OASIS 0.88; controls, 0.31;  $p=0.01$ ).

The limitation of this study is that we did not perform urodynamic studies. However, this is in keeping with our

clinical practice, whereby we always initiate pelvic floor exercises and only perform urodynamics in those women who seek surgical intervention. Nevertheless, our data shows that at 3 months postpartum, urinary symptoms occurred significantly more frequently after OASIS.

There are two possible hypotheses for the association between OASIS and urinary incontinence. Firstly, it may be associated with the extended mechanical trauma that resulted in the anal sphincter injury. Recent evidence suggests that the urethra has no ligaments attaching it anteriorly to the pubic bone [28]. Ventro-laterally, the urethra is enclosed by the levator ani, its fascia and a ventral urethral connective tissue bridge connecting both sides. Dorsally, the urethra is closely connected to the anterior vaginal wall [28]. In a prospective study, Dietz and Lanzarone [29] demonstrated that vaginal delivery is associated with avulsion of the levator ani from the pelvic sidewall in one third of women. Apart from soft tissue trauma, they reported a weak but significant association between avulsion injury and worsening or de novo stress urinary incontinence [29]. It remains to be determined whether trauma to the posterior vaginal wall and perineum that occurs with OASIS also traumatises the urethral support mechanisms and consequently leads to the development of stress urinary incontinence.

Secondly, as a prolonged second stage was also an independent risk factor, stress urinary incontinence may be attributed to associated neuropathic damage. A prolonged second stage of labour is believed to exacerbate pudendal nerve damage [5, 6, 9] and can alter electromyographic findings [7]. However, in a prospective study, Sultan et al. [9] found no significant difference in pudendal nerve terminal motor latencies in continent and incontinent women post partum. Allen et al. [7] reported that only a few women, who sustained damage to the pelvic floor, complained of urinary incontinence post partum. There is also evidence to suggest that some degree of recovery of pudendal nerve function occurs with time [5, 9]. Dolan et al. [30] performed a prospective longitudinal study in which pelvic floor neurophysiology was performed antenatally in a cohort of 96 primigravid, and some of these women were followed up for 15 years. Their findings were contradictory, as they found that while the motor unit potential duration increased at 7 years, the vaginal squeeze pressure also

**Table 8** Logistic regression analysis of independent risk factors for overall urinary incontinence stress urinary incontinence

	Overall urinary incontinence		Stress urinary incontinence	
	OR (CI)	P value	OR (CI)	P value
OASIS	0.55 (0.28–1.11)	0.095	2.65 (1.22–5.74)	0.013
Second stage of labour > median 50 min	0.50 (0.25–1.0)	0.005	2.32 (1.07–5.06)	0.034
Head circumference > median of 339.6 mm	0.54 (0.26–1.11)	0.093	1.68 (0.76–3.73)	0.201

increased during the same period. They concluded that the aetiology of stress incontinence was complex, and in the absence of a definitive neurophysiological test to quantify neuropathy, the role of denervation/reinnervation in the pathophysiology of urinary incontinence remains unclear.

## Conclusion

Women who sustain OASIS are more likely to develop stress urinary incontinence. Furthermore, these women suffer a significant deterioration in quality of life. As incontinence is an embarrassing symptom and a social stigma, this study highlights the need for clinicians to directly inquire about both urinary and anal incontinence in women who sustain OASIS. Prevention of this silent affliction could only be considered until further research enables a better understanding of the pathophysiology of urinary incontinence in women who sustain OASIS.

**Acknowledgement** The authors are grateful for the statistical advice kindly given by Sally Kerry from the Community Health Science Department at St. George's University of London. Funding for this study was provided by the Mayday Childbirth Charity Fund, Croydon, UK.

## References

- Harrison GL, Memel DS (1994) Urinary incontinence in women: its prevalence and its management in a health promotion clinic. *Br J Gen Pract* 44(381):149–152
- Chaliha C, Kalia V, Stanton SL et al (1999) Antenatal Prediction of postpartum urinary and fecal incontinence. *Obstet Gynecol* 94 (5 Pt 1):689–693
- Viktrup L, Lose G, Rolff M et al (1992) The symptom of stress incontinence caused by pregnancy or delivery in primiparas. *Obstet Gynecol* 79:945–949
- Wilson PD, Herbison RM, Herbison GP (1996) Obstetric practice and the prevalence of urinary incontinence three months after delivery. *BJOG* 103:154–161
- Snooks SJ, Swash M, Setchell M et al (1984) Injury to the innervation of pelvic floor sphincter musculature in childbirth. *Lancet* 2:546–550
- Snooks SJ, Badenoch DF, Tiptaft RC et al (1985) Perineal nerve damage in genuine stress incontinence. An electrophysiological study. *Br J Urol* 57:422–426
- Allen RE, Hosker GL, Smith ARB et al (1990) Pelvic floor damage and childbirth: a neurophysiological study. *Br J Obstet Gynaecol* 97:770–779
- Sampselle CM (1990) Changes in pelvic floor strength associated with childbirth. *J Obstet Gynecol Neonatal Nurs* 19:371–377
- Sultan AH, Kamm MA, Hudson CN (1994) Pudendal nerve damage during labour: prospective study before and after childbirth. *BJOG* 101:22–28
- Peschers UM, Schaer G, Anthuber C et al (1996) Changes in vesical neck mobility following vaginal delivery. *Obstet Gynecol* 88:1001–1006
- King JK, Freeman RM (1998) Is antenatal bladder neck mobility a risk factor for postpartum stress incontinence. *BJOG* 105:1300–1307
- Fenner D, Genberg B, Brahma P et al (2003) Fecal and urinary incontinence after vaginal delivery with anal sphincter disruption in an obstetrics unit in the United States. *Am J Obstet Gynecol* 189(6):1543–1549
- Sultan AH, Kamm MA, Hudson CN et al (1993) Anal sphincter disruption during vaginal delivery. *N Engl J Med* 329:1905–1911
- Zetterstrom JP, López A, Anzén B et al (1999) Anal incontinence after vaginal delivery: a prospective study in primiparous women. *BJOG* 106:324–330
- Wagenius J, Laurin J (2003) Clinical symptoms after anal sphincter rupture: a retrospective study. *Acta Obstet Gynecol Scand* 82:246–250
- Nazir M, Stien R, Carlsen E et al (2003) Early evaluation of bowel symptoms after primary repair of obstetric perineal rupture misleading. *Dis Colon Rectum* 46(9):1245–1250
- Tetzschner T, Sørensen M, Lose G et al (1996) Anal and urinary incontinence in women with obstetric anal sphincter rupture. *BJOG* 103:1034–1040
- Ustal Fornell E, Wingeren G, Kjølhede P (2004) Factors associated with pelvic floor dysfunction with emphasis on urinary and fecal incontinence and genital prolapse: an epidemiological study. *Acta Obstet Gynecol Scand* 83:383–389
- Otero M, Boulvain M, Bianchi-Demicheli F et al (2006) Women's health 18 years after rupture of the anal sphincter during childbirth. II Urinary incontinence, sexual function, and physical and mental health. *Am J Obstet Gynecol* 194(5):1260–1265
- Glazener CMA, Herbison GP, MacArthur C et al (2006) New postnatal urinary incontinence: obstetric and other risk factors in primiparae. *BJOG* 113:208–217
- Foldspang A, Hvidman L, Mommsen S et al (2004) Risk of postpartum urinary incontinence associated with pregnancy and mode of delivery. *Acta Obstet Scand* 83:923–927
- Farrell S, Allen VM, Baskett TF (2001) Parturition and urinary incontinence in primiparas. *Obstet Gynecol* 97(3):350–356
- Avery K, Donovan JL, Abrams P (2001) Validation of a new questionnaire for incontinence: the international consultation on incontinence questionnaire (ICIQ). *Neurourol Urodyn* 20:86
- Donovan JL, Badia X, Corcos J (2002) Symptoms and quality of life assessment. In: Abrams P, Cardozo L, Khoury S, Wein A (eds) *Incontinence*, 2nd edn. Health Publication Bristol, pp 269–316
- Andrews V, Sultan AH, Thakar R et al (2006) Occult anal sphincter injuries—myth or reality. *BJOG* 113(2):195–200
- Adams EJ, Fernando RJ (2001) Management of third and fourth degree tears following vaginal deliveries. Royal College of Obstetricians and Gynaecologists 2001; Guideline No. 29
- Borello-France D, Burgio KL, Richter HE et al (2006) Fecal and urinary incontinence in primiparous women. *Obstet Gynecol* 108 (4):863–872
- Fritsch H, Mitterberger M, Barbel G et al (2006) What are the supportive structures of the female urethra. *Neurourol Urodyn* 25 (2):128–134
- Dietz HP, Lanzarone V (2005) Levator trauma after vaginal delivery. *Obstet Gynecol* 106(4):707–712
- Dolan LM, Hosker GL, Mallett VT et al (2003) Stress incontinence and pelvic floor neurophysiology 15 years after first delivery. *BJOG* 110:1107–1114