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The Role of Obesity on Urinary and Anal Incontinence in women: a review

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1. Introduction

Up to 39% of the global population are classified as overweight or obese(1) and no country to date has been able to reverse the obesity epidemic(2). Overweight and obesity are defined as abnormal or excessive fat accumulation that presents a risk to health. A body mass index (BMI) over 25 is considered overweight, and over 30 is obese(1). The prevalence of overweight and obesity has been steadily increasing since 1999 and is becoming a significant health and financial burden worldwide(2).

Figure 1 Actual and predicted prevalence of obesity through 2030

Reprinted from Obesity and Severe Obesity Forecasts Through 2030, by Finkelstein EA, Khavjou OA, Thompson H, Trogon JG, Pan L, Sherry B, et al. 2012, American journal of preventive medicine 42(6):563–70. (3) Copyright 2020 Elsevier

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In 2015 excess weight contributed to 4.0 million (2.7-5.3 million) deaths and 120 million (84-158 million) disability-adjusted life years (DALYs) (4.9% [3.5-6.4%] of all-cause DALYs) among adults globally(4).

Obesity has been linked to the pathophysiology of several health conditions, including urinary and anal incontinence.

The EPICONT study (Epidemiology of Incontinence in the County of Nord-Trøndelag) analysed data from 34,755 women and demonstrated that obesity had a significant impact on stress, urgency and mixed urinary incontinence(5).

Table 1 Multiple regression analysis of the association between BMI and UI. Value expressed as OR (95%CI)

Reprinted from 'Are smoking and other lifestyle factors associated with female urinary incontinence? The Norwegian EPINCONT Study', by Hannestad YS, Rortveit G, Daltveit AK, Hunskar S. 2003, BJOG: An International Journal of Obstetrics and Gynaecology 110(3):247–54. Copyright 2020 John Wiley and Sons (6)

Previous evidence suggests that the odds ratio for the presence of UI is 1.6 per 5 unit increase in body mass index (BMI) and the prevalence of UI in women in the 'morbidly obese' category seeking weight loss surgery is as high as 67%(7).

Urinary incontinence (UI) is a common condition with a prevalence of 25% in the general population(5,8). It has a significant impact on individuals and wider society (9). Several different aetiological factors have been identified in epidemiological studies, such as previous hysterectomy, pregnancy and operative vaginal birth(10). In younger women the prevalence is lowest, increasing towards menopause and then rising more steadily from the age of 60(9).

There is a significant economic burden associated with UI. The direct costs of UI care in the US has been estimated to be more than 16 billion dollars per year(11). A systematic review by Milsom et al in 2014 demonstrated that there is an increasing burden in the US as well as

several other countries(12). Annual cost of illness estimates for UUI, including direct and indirect costs, have been reported as up to 7 billion Euros for six western countries including Canada and the UK(13).

Studies on quality of life (QoL) have highlighted a potentially detrimental impact regardless of age(14,15), with anxiety, depression and sexual dysfunction commonly reported (16–18). Pace et al found an inverse correlation of the total female sexual function index in the domains arousal, orgasm, lubrication and satisfaction with increased BMI in postmenopausal women(19).

The joint IUGA/ICS report on the terminology for female pelvic organ prolapse has defined anal incontinence as involuntary loss of flatus or faeces(20). The symptom of faecal incontinence has been defined as the complaint of involuntary loss of solid and/or liquid faeces, and flatal incontinence is the involuntary loss of flatus (gas)(21,22)

There is patient reluctance to seek medical help for anal incontinence, potentially due to embarrassment, but compounded by infrequent screening by healthcare providers. Subsequently there are limited epidemiological data for AI and the magnitude of the condition remains largely unknown(23), especially in comparison to UI. In a recent study of 457 women presenting for benign gynaecological care, only 17% of women with FI were questioned about these symptoms by their clinician. Johansen et al reported that less than a third of patients with FI had disclosed this to a healthcare provider(23).

Response rates to questionnaires highlight this difference in willingness to disclose symptoms of AI compared to UI, with only 60-70% of participants responding to AI-related questionnaires, whereas a review of epidemiological studies with 230,000 respondents reported median response rates for UI questionnaires of 80%(10,24).

Barriers in disclosing incontinence have been reported including social expectations, lack of 'trusted space' for disclosure, confusion surrounding the medical terminology and meaning, and emotional, social and psychological consequences(25).

The prevalence of AI is estimated to range between 7 and 15% in community-dwelling adults, however the prevalence in care home residents is reported to be at least three times higher(26,27).

-This difference in prevalence is primarily due to the increased prevalence of dementia in care home populations, a disorder with direct negative impact on anal sphincter control. Other contributors such as altered stool consistency and immobility are also likely to play a role. There is evidence to suggest that care home residents with FI often have coexisting UI, known as double incontinence(27). In population based studies UI has been found to coexist in 50% of patients with FI(28).

Risk factors for AI include acquired structural abnormalities for example secondary to childbirth and obstetric anal sphincter injuries, functional disorders such as Irritable bowel syndrome and Inflammatory bowel disease, and neurological disorders such as multiple sclerosis and dementia (30). The impact of AI can be variable and detrimental in some patients with feelings of dignity loss and social isolation(29).

The impact of obesity on AI has not been as widely researched. Women with AI have higher BMIs compared to the general population(30). In a study by Varma et al, obesity was independently associated with a 20% higher prevalence of FI per 5-unit increase in BMI(31).

The World Health Organisation has recognised the increasing prevalence of obesity as a global epidemic, with 2.8 million people dying each year as a result of being overweight or obese. Given the associations between obesity and pelvic floor disorders and the socioeconomic burden of UI and AI, the aim of this review was to evaluate the current evidence on the effects of obesity on the pelvic floor. An overview of the relationship between obesity and urinary and anal incontinence, as well as outcomes of continence surgery treatments and weight loss, is warranted to better inform clinical practice.

2. Pathophysiology of Incontinence

2.1 Pathophysiology of urinary incontinence in obese populations

Several studies have investigated the association between obesity and urinary incontinence. A systematic review by Hunskaar et al investigated the factors that predispose overweight and obese women to develop UI(32). The study suggested that intra-abdominal pressure increases with obesity, weakening the pelvic muscles and pelvic innervation. The prolonged effect on the pelvic musculature, nerve supply and supporting structures due to chronic strain, may cause pelvic floor muscle weakness and have a negative impact of pelvic organ function(33).

This increase in pressure can be demonstrated in urodynamic results which show an increased maximal intravesical peak pressure during cough, however it does not seem to alter urethral sphincter function as obese patients have a similar Valsalva leak point pressure and comparable maximal urethral closure pressure to non-obese controls(34–36). Coexisting excess weight and morbid obesity causes a rise in intra-abdominal pressure which seems to reach a value of 12cmH₂O. This has been demonstrated to be significantly different to healthy, non-obese controls with BMI<30kg/m² and mean intra-abdominal pressure of 0+-1.2cmH₂O (p<0.0001), contributing to the development of UI symptoms(37).

In addition, the associated oxidative stress from adipose tissue has been postulated to increase prevalence and severity of incontinence through alterations in collagen metabolism. Visceral adipose tissue is considered an endocrine organ in itself, and in the overweight and obese population the secretion of inflammatory cytokines and factors is dysregulated, for example tumour necrosis factor alpha (TNF alpha) and interleukin 6 (IL-6) (38). Leptin activates nicotinamide adenine dinucleotide phosphate (NADPH) oxidases stimulating production of reactive oxygen species such as hydrogen peroxide, H₂O₂, also contributing to increased oxidative stress in obesity(39).

A study by Liu et al showed that exogenous H₂O₂ had two-way regulatory effects on collagen metabolism(40). After incubation for 24 hours in vitro with human uterosacral ligament fibroblasts, lower concentrations of H₂O₂ stimulated the anabolism of COL1A1

(Collagen type 1 alpha 1), whereas a higher concentration promoted catabolism. They also noted upregulation of transforming growth factor beta 1 (TGF- β 1) and proteolytic enzymes such as matrix metalloproteinase-2 (MMP-2), which promote collagen catabolism, with increasing oxidative stress. It was concluded that oxidative stress contributed to collagen metabolic disorder in the human pelvic fibroblasts.

It has been therefore demonstrated that both physical and biochemical stresses of obesity on pelvic floor neuromusculature seem to predispose to the development of urinary incontinence.

2.2 Pathophysiology of anal incontinence in obese populations

There is limited evidence on AI and obesity but some studies indicate a higher prevalence of AI in the obese, with one study of 256 morbidly obese women reporting a prevalence of AI in 67%(41). The aetiology of this is likely multifactorial. Altered stool consistency is one of the proposed contributors to AI as obesity has been associated with increased intestinal motility and diarrhoea(42). In an observational study by Pares et al of obese patients undergoing evaluation for weight loss, symptoms of AI were found in 32.7% and those with incontinence reported significantly higher percentages of altered bowel habits with non-formed stools (35.2%, $p=0.004$)(45). A prospective case-matched study by Brochard et al reported similar findings. They compared obese patients to age- and sex-matched non-obese patients with AI(43). The authors of this study suggested that diarrhoea was significantly associated with obesity in FI patients (OR 2.94, [1.22-7.19], $p=0.0158$) and recommended a management focused on stool consistency when managing AI in these patients .

When reviewing evidence from anorectal manometry investigations, obese patients have higher upper- and lower-part resting pressures, higher intra-abdominal pressure during effort and increased maximum tolerable volume(43).

A study by Ellington et al, also showed that baseline resting and squeeze pressures in multivariable analyses of anal manometry were increased in obese women with FI compared to normal and overweight women (44).

Table 2 Anal manometry parameters

Reprinted from, The effect of obesity on fecal incontinence symptom distress, quality of life, and diagnostic testing measures in women by Ellington DR, Polin MR, Szychowski JM, Deng L, Richter HE. 2013 Int Urogynecol J. 2013;24(10):1733-1738 (44)

A recent study on asymptomatic women showed that age, BMI and parity influences anorectal motion. Using magnetic resonance (MR) defaecating proctogram the authors showed that in younger women, increasing BMI was associated with a more obtuse median anorectal resting angle, 107 degrees in those with BMI \geq 35 and 97 degrees in women with BMI less than 25(45). This is perhaps because of the increased intraabdominal pressure associated with increased BMI that may lead to greater perineal descent at rest. Perineal descent has long since been associated with chronic straining and constipation, but is also found in those with anal incontinence and pudendal nerve neuropathy which may be caused by chronic stretch and pressure forces on the pelvic floor(46).

3. Risk factors for incontinence and obesity

3.1 Age, obesity and Incontinence

Data is limited regarding the cumulative effect of age on symptom severity in the obese.

Given that most data are derived from community-dwelling study participants, generalisation of findings is not possible. Obesity in childhood is associated with functional constipation and functional non-retentive faecal incontinence(47); however, its effect specifically on anal incontinence is not as clear. A recent large-scale population study of 6,803 children and adolescents reported on the prevalence of faecal incontinence, daytime urinary incontinence, nocturnal enuresis and nocturia(48). Faecal incontinence was reported in 11.2%, within the school entry group (mean age 6.45years), and in 2.1% of adolescents

(mean age 13.9years). Obesity was found to be associated with faecal incontinence in first grade boys (OR 1.86, CI 1.10-3.15)

compared with normal weight, however no association was found with increasing BMI in adolescents. A possible reason for this may be that obesity-associated constipation, and subsequent incontinence, has spontaneously resolved by adolescence or been successfully treated. 21.8% of children and 4.5% of adolescents reported daytime urinary incontinence (DUI), and no significant association was found with obesity. It may be that the impact of weight on pelvic floor function becomes evident after a longer period of obesity.

Varma et al reported a prevalence of FI of 24.2% (511/2106) in community dwelling women older than 40 years (49). A positive correlation of age with faecal incontinence rates was observed after stratification by age. The multivariable analysis revealed a not statistically significant (OR 1.1, 95% CI 1, 1.2, $p=.15$) trend whereby increments of age (per 5 years) increased the odds of developing FI by approximately 10%. It remains unclear however whether there is interplay between obesity and age on risk of FI as obesity was not controlled for in this cross sectional study. The study authors did recognise the limitations of a cross sectional study in determining casual associations.

3.2 Parity, obesity and incontinence

Childbirth is well known to be associated with pelvic floor dysfunction; incontinence both urinary and anal, and pelvic organ prolapse(50). Effects are most pronounced with increasing parity and vaginal birth and are thought to be due to neuromuscular effects antepartum and intrapartum, and perineal trauma at the time of delivery(51–53).

Obesity has a positive association with gestational diabetes mellitus (GDM) and cephalopelvic disproportion (CPD) therefore instrumental delivery rates, incidence of prolonged second stage and rates of obstetric anal sphincter injury (OASIS) could be higher, with increased rates of UI and AI as associated sequelae in obese women.

However, a large retrospective study of 45,557 births showed that perineal trauma rates were reduced among obese women and that OASIS was not significantly associated with BMI(54).

Table 3 Association between BMI and third and fourth degree perineal tears

Reprinted from 'Effect of body mass index on the incidence of perineal trauma' by Durnea CM, Jaffery AE, Gauthaman N, Doumouchsis SK. 2018, *Int J Gynaecol Obstet.* 141(2):166-170. Copyright 2020 John Wiley and Sons (54)

This is consistent with the findings from Lindholm and Altman who studied all singleton vaginal deliveries in Sweden 2003-2008 (n=210,678). The rate of OASIS was 4.25% and they concluded that increasing BMI showed a significant near-dose-response type of protective effect against third and fourth degree lacerations(55).

4. Outcomes of continence interventions

4.1 Outcomes of urinary incontinence interventions in obese women

The majority of existing research into outcomes of continence surgery in the obese population is from studies evaluating mid urethral slings. In contrast research concerning efficacy of bulking agents is more limited.

A meta-analysis by Xia et al demonstrated that objective success rates after mid urethral sling were lower in women with BMI>25kg/m² compared to normal BMI, although no significant difference was found between overweight (BMI 25-<30kg/m²) and obese (BMI>30kg/m²)(56). There was no significant difference in subjective cure between BMI groups.

Studies, often with short to mid-term follow up have shown a trend towards favourable results and increased cure rates in patients with a lower BMI, although statistical significance was not reached(57,58).

Secondary analysis of a randomised controlled trial evaluating mid urethral slings reported that at 5 years, non-obese women reported higher rates of objective and subjective cure,

with 76.7% subjective cure rate, compared to 53.6% in obese women ($P = 0.002$, RD 23.2%, 95% CI 8.0-38.3%)(59).

Operative outcomes are also negatively influenced by severity of obesity with morbidly obese patients twice as likely to report failure following mid urethral sling(60). The incidence of urinary urgency incontinence was comparable in both groups; however, bothersome symptoms were more likely to persist in obese women (58.9% vs 42.1%).

Since the 2018 mesh pause, or high vigilance restriction period however, the use of mesh for SUI has been effectively halted, except in cases where there is no alternative and delay is unacceptable (61), replaced by alternative SUI surgical treatments such as urethral bulking and colposuspension.

Bladder neck suspension procedures have evidence of long-term efficacy with success rates comparable to mid urethral slings(62), and most recent studies have reported on the efficacy and safety of laparoscopic colposuspension(63). As synthetic mesh mid urethral slings continue to face scrutiny, the Burch colposuspension, first described in 1961(64), is becoming increasingly popular as a primary procedure for stress incontinence or in those where urethral bulking has failed. However, data concerning the efficacy of Burch colposuspension in the overweight and obese women, and the impact of BMI on treatment efficacy and longevity, is limited.

Onabotulinum toxin A is the mainstay of treatment for refractory detrusor overactivity (DO), OAB and urgency incontinence, with reported success compared to placebo in several randomised controlled trials(65–67). The most recent randomised trial reported on the higher risk of treatment failure and association with non-response (defined as 'no change' or 'worse') on the Patient Global Impression of Improvement (PGI-I) scale, OR 1.07, 95%CI 1.0, 1.16, $P = 0.065$, with increased BMI(68).

4.2 Outcomes of anal incontinence interventions in obese women

Behavioural treatment that aims to reduce stool inconsistency is the primary treatment for anal incontinence. Various treatment alternatives have been proposed, including bowel training, biofeedback, anti-diarrhoeal drugs and bulk laxatives (in cases of chronic constipation). None of these methods have been evaluated in obese populations. Sphincteroplasty remains the cornerstone of treatment in cases of damaged anal sphincter. A study on 15 obese and 64 non-obese women, followed-up for a median period of 64 months(68) showed that although the risk of complications was comparable between the two groups, improvement was less evident in obese patients. Perianal bulking has shown promising results in obese patients undergoing gastric bypass surgery who are often affected by incontinence post operatively(69). However, robust data to support efficacy in the obese symptomatic populations are lacking, and considering the limited efficacy of bulking agents in the treatment of UI they cannot be yet recommended for the treatment of FI.

5. Impact of weight loss on incontinence

5.1 Urinary incontinence following weight loss

It has been noted that weight loss leads to statistically significant reductions in intravesical pressure, bladder-to-urethra pressure transmission during cough and urethral axial mobility(70). Bump et al demonstrated significant reduction in incontinence episodes and need to use absorptive pads at 1 year after surgically induced weight loss(70).

A randomised controlled trial by Subak et al demonstrated similar findings(71). Following a weight reduction programme, women were more likely to experience statistically significant improvements in continence and quality of life at 6 months follow up. A reduction in as little as 5-10% baseline weight could confer 50% reduction in UI episodes, with benefits in both urgency and stress incontinence. Urodynamics studies also showed a reduction in bladder pressure with weight loss indicating higher bladder pressure in overweight and obese women is a contributor to UI.

A Cochrane review in 2015 aimed to determine the effectiveness of specific lifestyle interventions including weight loss on adult urinary incontinence(72). Four trials with a total of 4,701 women reported on the effect of weight loss using low calorie diet and an exercise programme compared to no treatment. All four trials reported that women allocated to the intervention group had a statistically significant reduction in weight from baseline compared to the control groups. There was evidence, albeit 'low' quality, that weight loss programmes were associated with higher improvement rates based on women's self-report, and also higher cure and improvement rates based on quantifiable symptoms. The outcome measures of these studies were variable, including number of weekly incontinence episodes and only one small trial assessed effect using disease-specific quality of life measures (Incontinence Impact Questionnaire, IIQ and the Urogenital Distress Inventory, UDI). This trial showed that at three month follow up, women in the intervention group reported that urinary incontinence had less adverse impact (median IIQ scores, 40 women in analysis, 37 versus 89, P value 0.01) and was less distressing (median UDI scores, 40 women in analysis, 104 versus 195, P value < 0.0001) compared with the control group(71).

The positive effect of weight loss was consistently reported despite this variation in outcome measures, which strongly suggests that weight loss has a beneficial effect in treating urinary incontinence.

Taking into account the modest success rates of behavioural interventions for weight loss, one may expect that the impact of surgical procedures would be more clear. A recent systematic review evaluated the impact of bariatric surgery on obese women with UI. Data from 33 cohort studies including 2910 women were included with median follow up of 12 months. Improvement or resolution of any type of UI was reported in 56% (95% CI 48-63%), with larger reductions seen in UUI than SUI after bariatric surgery. A significant reduction ($p < 0.001$) in symptom questionnaire scores was also reported, with Urogenital Distress Inventory (UDI) scores reduced by 13.4 points (95% CI 7.2-19.6)(73). However, quality of

evidence was graded as very low, and 3% of patients reported worsening or development of new onset UI following bariatric surgery.

Another systematic review and meta-analysis measured effect on incontinence specific quality of life and incontinence cure rate following bariatric surgery(74). Analysis of 3 studies included 3225 women showed that incontinence specific quality of life scores were improved by 14% (weighted mean difference = -14.79; CI = -18.47 to -11.11; I² = 87.1%), while the cure rates for any type of UI reached 59% (95% CI = 51 to 66%). Short term follow up and study heterogeneity were noted to be limitations to these data.

5.2 Anal incontinence following weight loss

By comparison, the impact of bariatric surgery on AI is less encouraging. A systematic review by Montenegro et al reported the findings from 20 studies (3684 patients) which showed a modest relative risk reduction in FI episodes after bariatric surgery, although the finding was not significant (OR, 0.80; 95% CI, 0.53-1.21; p = 0.29) (78)

The relationship between weight loss and reduction in FI episodes is based on the assumption that stool inconsistency associated with obesity is the aetiological basis of increased FI prevalence.

A study which investigated pelvic floor dysfunction in 46 obese women reported that there was no significant difference in internal or external anal sphincter size or mean anorectal angle during squeeze and during defecation between BMI groups, <35, 35-40, >40kg/m² (75). However, FI decreased significantly after bariatric surgery from 23% preoperatively, to 9.2% at 6 months and 5.7% at 12 months follow up (p=0.001)(75).

Similarly, a systematic review by Poylin et al also demonstrated a reduction in faecal incontinence after Roux-en-Y procedure although the link between bariatric surgery and diarrhoea were unclear (80)

6. Conclusion

Obesity is linked to the development and severity of both urinary and anal incontinence. Increased intra-abdominal pressure and chronic strain on the pelvic neuromusculature contributes to UI, and oxidative stress from visceral adipose tissue is likely to have a negative effect on the collagen and supportive structures of the pelvic floor, which confer continence. With regards to AI in obese individuals, stool consistency seems to be the main contributing factor, more so than intra-abdominal pressure.

As a consequence, weight loss through lifestyle changes or bariatric surgery can confer significant improvements in UI, but the effect on AI is less pronounced.

Evidence concerning the surgical treatment of AI in the obese population is scarce and inadequate to inform clinical practice. Studies on urinary continence procedures in the obese have not been extensive enough to evaluate different surgical interventions in obese women, and evidence exists on selected procedures only. Although the transobturator tape has superior efficacy as compared to single incision tapes, obesity is associated with poorer long term subjective and objective surgical outcomes. There is limited evidence regarding outcomes of urethral bulking and colposuspension in the obese, and further research is needed into surgical treatments for AI.

Nevertheless, and in addition to its other numerous beneficial effects promoting overall wellbeing and longevity, weight loss should have a prominent place in treatment pathways for the management of UI and AI.

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SXD: Conception of idea, analysis of included articles, review and editing of manuscript.

JL: Review of articles for inclusion, drafting and editing manuscript

VP: Involved in conception of idea. Reviewed included articles and contributed to initial manuscript draft. All authors accept responsibility for the paper as published.

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Figure Legend

Figure 1: Actual and predicted prevalence of obesity through 2030

Table Legend

Table 1: Multiple regression analysis of the association between BMI and UI.

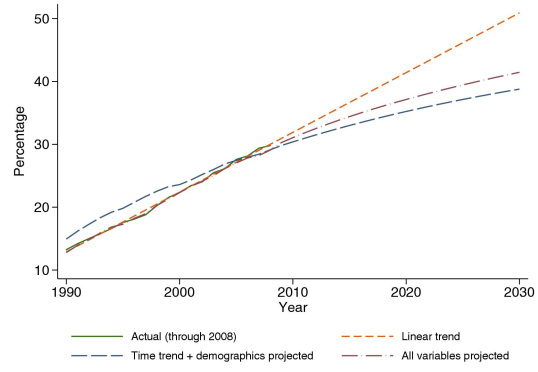
	Incontinence		Stress type incontinence		Mixed type incontinence		Urge type incontinence	
	All	Severe	All	Severe	All	Severe	All	Severe
Body mass index								
<25	1	1	1	1	1	1	1	1
25–29	1.4 (1.3–1.5)	2.0 (1.7–2.3)	1.4 (1.2–1.5)	1.9 (1.5–2.4)	1.7 (1.5–1.9)	2.3 (1.9–2.8)	1.1 (0.9–1.3)	1.6 (1.1–2.4)
30–34	1.9 (1.7–2.1)	3.1 (2.6–3.7)	1.7 (1.6–2.0)	2.8 (2.1–3.6)	2.3 (2.0–2.7)	3.5 (2.8–4.3)	1.5 (1.2–1.9)	3.0 (1.9–4.6)
35–39	2.4 (2.1–2.8)	4.2 (3.3–5.3)	2.0 (1.7–2.5)	3.2 (2.1–4.8)	3.5 (2.9–4.3)	5.5 (4.1–7.4)	1.4 (0.9–2.1)	2.4 (1.2–4.9)
40+	2.7 (2.1–3.5)	5.0 (3.4–7.3)	2.4 (1.7–3.3)	4.2 (2.2–7.9)	3.7 (2.7–5.2)	6.0 (3.7–9.6)	1.8 (0.9–3.5)	3.8 (1.3–11.1)

Table 2: Anal manometry parameters

Characteristic	Overall (n = 407)	Normal (n = 124)	Overweight (n = 123)	Obese (n = 160)	p
Resting Pressure mmHg	36.2 ± 19.6	31.2 ± 18.1	32.5 ± 17.1	43.0 ± 20.6	<0.0001
Squeeze Pressure mmHg	74.0 ± 36.5	68.0 ± 32.3	67.6 ± 31.5	83.6 ± 41.0	<0.0001
Capacity Cc	114.9 ± 55.0	104.3 ± 46.9	117.5 ± 59.5	121.1 ± 56.4	0.031

Table 3: Association between BMI and third and fourth degree perineal tears

Factor	Odds ratio (95% confidence interval)	P value
BMI		
<25	1	-
25 to ≤30	1.14 (0.99-1.30)	0.058
30 to <35	0.89 (0.71-1.10)	0.275
≥35	0.88 (0.63-1.23)	0.446



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