EDITORIAL

Incontinence: An Underappreciated Problem in Obesity and Bariatric Surgery

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Driven by the obesity epidemic, the number of bariatric operations performed in the United States has increased markedly, by one estimate from ∼20,000 in 1998 to 140,000 in 2004, and plateaued thereafter [1]. While diarrhea is a recognized consequence of intestinal bypass procedures, Roberson and colleagues in this issue of the journal highlight, for the first time, that fecal incontinence (FI) may begin or worsen after bariatric surgery [2]. Of note, 48% of women and 42% of men reported FI for liquid stools while 21% of women and 30% of men reported solid FI after surgery. Moreover, 55% of women and 31% of men reported their symptoms were worse after surgery. While these figures may be affected by response rates (48%) or, since surveys were only conducted after surgery, by recall bias, they draw attention for the first time to an underappreciated and significant problem since FI can be a devastating symptom which can substantially impair quality of life [3, 4].

Consistent with studies in the general population, diarrhea, cholecystectomy, stress, and mixed urinary incontinence were risk factors for FI by univariate analysis [5, 6]. However, only diarrhea remained a significant risk factor in multivariate analysis. The high prevalence of FI, not only in women but also in men, who are less prone to pelvic floor injury, also underscores the contribution of diarrhea. Stool consistency is a critical factor affecting fecal continence. Indeed, even healthy subjects find it harder to retain continence for low-viscosity (i.e., 100 cPois) than high-viscosity material (10,000 cPois) in the rectum, particularly when anal sphincter endurance is reduced [7]. In addition to fat malabsorption resulting from biliopancreatic diversion, an alteration in intestinal and colonic bacterial flora after bariatric surgery may also contribute to diarrhea [8].

Obesity is a risk factor for urinary incontinence [9]. Moreover, in a large trial of 338 obese women with urinary incontinence, a 6-month structured weight loss program was more effective than education alone for weight loss (i.e., average weight loss of 8% body weight for treatment versus 1.6% for control group) and reducing the frequency of urinary incontinence (47.4 vs. 28.1%) [10]. This benefit was more pronounced for stress than urge urinary incontinence and supports the concept that obesity predisposes one to urinary incontinence. In the Roberson study, urinary incontinence improved in one-third of patients after bariatric surgery; whether this improvement was related to actual weight loss is unclear. In contrast to urinary incontinence, the relationship between obesity per se (i.e., before bariatric surgery) and FI is less clearly defined. In one study of 256 morbidly obese (BMI ≥ 35 kg/m^2) women, the prevalence of anal incontinence (i.e., gas and stool) was 67%, which seems very high [11]. The BMI was not a risk factor for anal incontinence in that study, perhaps because all women were morbidly obese. However, it is difficult to compare this figure to population-based estimates, which generally focus on fecal leakage alone. Another case-control study observed that the risk of incontinence for flatus (but not feces) was higher in morbidly obese women [12]. In the National Health and Nutrition Examination Survey, obesity was not a significant risk factor for FI by multivariate analysis [6]. In contrast, a recent population-based case-control study from Olmsted County suggests that increased BMI is associated with a higher risk (OR 1.1 per unit, 95% CI, 1.004, 1.1) of FI [13].

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The mechanisms by which obesity predisposes one to urinary and FI are partly understood. Obesity is a risk factor for diarrhea [14] and accelerated colonic transit [15]. Besides predisposing to acute leakage by virtue of higher viscus (bladder or rectal) pressure, it is suggested that a higher intra-abdominal pressure may damage the pelvic floor in obese people [9]. Indeed, a systematic review of seven studies observed that intra-abdominal pressures were higher, averaging 9–14 mmHg, in morbidly obese patients than in non-obese subjects (i.e., average of 5–7 mmHg) [16]. While the evidence for increased intra-abdominal pressures in overweight subjects is mixed, two studies suggest that obesity is also associated with a higher intra-abdominal pressure. Moreover, relative to the supine position, the increase in intra-abdominal pressure in the upright position is more pronounced in morbid obesity, suggesting that the abdominal cavity may function as a hydraulic system in which intra-abdominal contents may exert pressure on pelvic viscera. Also, a longitudinal study found that a higher BMI was prospectively associated with stress but not urge incontinence in midlife, which reinforces the potential long-term impact of obesity on the pelvic floor [17]. Moreover, among women aged ∼50 years, the risk of stress urinary incontinence was higher among those who were obese for a longer (30 years or longer) than a shorter (5–10 years) duration [17].

There are several important messages from the Roberson study. First, they reinforce the need to manage obese patients by an integrated team, including gastroenterologists. Second, patients undergoing bariatric surgery should be asked if they have FI before and after surgery since a majority of patients with FI will not disclose the symptom to their physician unless asked [18, 19]. Anorectal function should be evaluated with a careful digital rectal examination, supplemented by anal manometry, particularly for patients who have incontinence for solid stool, which reflects more severe anal weakness than incontinence for liquid stool [20, 21]. Third, loperamide should be considered for managing diarrhea; loperamide improved fecal continence in obese subjects treated with orlistat [22]. While bacterial overgrowth may also cause diarrhea after gastric bypass surgery [23, 24], the diagnosis of overgrowth in this setting is challenging since normal values for small-bowel bacterial flora composition after Roux-en-Y bypass surgery are unknown. Moreover, it is difficult to access the more distal Roux limb to collect a sample for bacterial overgrowth with a standard endoscope and breath tests may be false-positive because of rapid small-intestinal transit. Pelvic floor retraining by biofeedback therapy should be considered in patients with FI who do not respond to conservative measures alone [25]. Fourth, a structured weight-loss program, and if surgery is necessary, a sleeve gastrectomy, should be considered in obese patients who have diarrhea or

FI before gastric bypass surgery. Long-term weight loss after sleeve gastrectomy is comparable to gastric bypass and greater than after banding procedures; a sleeve gastrectomy may cause mild dumping but does not cause malabsorption [26]. In the Roberson study, diarrhea was not significantly more common after gastric bypass than after banding procedures, perhaps because only a small proportion (13%) of patients had a gastric bypass in this study.

Bariatric surgery is more effective than non-surgical options for weight loss and ameliorating metabolic syndrome and inducing remission in diabetes mellitus [26]. So far, safety outcomes have focused on major adverse events (e.g., anastomotic leakage, pneumonia) and mortality, which is <0.5% after laparoscopic gastric banding surgery [27, 28]. Future studies should focus on morbidity and quality of life, including diarrhea and FI, after bariatric surgery. This is particularly important since bariatric surgery is increasingly performed for adolescents who may sustain anorectal injury after bariatric surgery (e.g., during childbirth), compounding the threat to fecal continence posed by diarrhea. Further studies are also necessary to evaluate the contribution of impaired anorectal continence mechanisms to FI after bariatric surgery.

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