Botulinum toxin for chronic anal fissure after biliopancreatic diversion for morbid obesity

Serafino Vanella, Giuseppe Brisinda, Gaia Marniga, Anna Crocco, Giuseppe Bianco, Giorgio Maria

AIM: To study the effect of botulinum toxin in patients with chronic anal fissure after biliopancreatic diversion (BPD) for severe obesity.

METHODS: Fifty-nine symptomatic adults with chronic anal fissure developed after BPD were enrolled in an open label study. The outcome was evaluated clinically and by comparing the pressure of the anal sphincters before and after treatment. All data were analyzed in univariate and multivariate analysis.

RESULTS: Two months after treatment, 65.4% of the patients had a healing scar. Only one patient had mild incontinence to flatus that lasted 3 wk after treatment, but this disappeared spontaneously. In the multivariate analysis of the data, two registered months after the treatment, sex ($P = 0.01$), baseline resting anal pressure ($P = 0.02$) and resting anal pressure 2 mo after treatment ($P < 0.0001$) were significantly related to healing rate.

CONCLUSION: Botulinum toxin, despite worse results than in non-obese individuals, appears the best alternative to surgery for this group of patients with a high risk of incontinence.

Key words: Botulinum toxin; Anal diseases; Anal fissure; Severe obesity; Bariatric surgery; Biliopancreatic diversion

INTRODUCTION

Approximately two-thirds of individuals living in the United States are overweight, and of those, almost half are obese$^{[1,2]}$. The incremented prevalence of obesity is associated with an increase in the frequency of obesity comorbidity$^{[3,4]}$, which is responsible for $> 2.5$ million deaths per year worldwide$^{[5-9]}$.

In 1991, the US National Institutes of Health established guidelines for the surgical therapy of morbid obesity (body mass index $\geq 40$ or $\geq 35$ in the presence of significant comorbidity), now referred to as bariatric surgery$^{[10]}$. The literature on postoperative weight loss and the problems associated with various bariatric surgical procedures is extensive and has been summarized elsewhere.

Bariatric surgery in morbidly obese individuals re-
verses, eliminates, or significantly ameliorates diabetes, hyperlipidemia, hypertension, and obstructive sleep apnea. These benefits occur in the majority of patients who undergo surgery. Biliopancreatic diversion (BPD) is a malabsorptive bariatric technique that is successful in achieving long-lasting weight lost in super-obese patients. In fact, the diarrhea (steatorrhea) that is expected after any malabsorptive technique can sometimes cause significant nutritional changes and anal disease. These patients are frequently referred to a coloproctology clinic due to hemorrhoids, fissures, and anal sepsis and fistula due to changes in quality and quantity of their feces. The aim of this technique in maintaining long-term weight loss is to produce malabsorption for fats and starch, which will lead to an important qualitative change in the patient’s feces. In a recent long-term study of outcome after BPD, Marinari et al found that 85.5% of patients scored good or better; however, little is known about the direct consequences of changes in defecation and flatulence after BPD. In addition, a change in the equilibrium of intestinal flora and bacterial overgrowth syndrome may also increase malodorous gas and discomfort. Changes in bowel habit may have a direct influence on quality of life reported by patients after BPD. Although many of the possible metabolic side effects can be controlled with vitamin complexes, micronutrients and protein prophylactics, chronic diarrhea can be the cause of different proctological disorders. These lead to an increase in the assistance required by these patients, who therefore need intervention and re-intervention, with a consequential increase in health expenditure.

The most commonly recorded anal disorders seen in this type of patient are hemorrhoids, fissures, abscesses, and anal fistulae mainly due to repeated, continuous diarrhea and changes in qualitative composition that are typical of steatorrhea in malabsorptive processes. The aim of this study was to analyze the results of botulinum toxin treatment in chronic anal fissure after BPD.

MATERIALS AND METHODS

This was an open label study. We present the results of the treatment with type A botulinum toxin in patients affected by chronic anal fissure developed after BPD for morbid obesity, observed at the Department of Surgery of the University Hospital “A. Gemelli” in Rome. Patients were sent to our Proctological Center after 1 year of the University Hospital “A. Gemelli” in Rome. All patients gave written informed consent to participate in the study.

Treatment target

In every treated patient, the primary target was the complete healing of the fissure. The healing was documented by inspection, physical examination and reported symptoms. Treatment was considered satisfactory only when a complete cicatrization was obtained.

Baseline assessment and operative technique

All patients underwent pretreatment evaluation, including clinical inspection and anorectal manometry. In all the patients, age and sex were registered. We divided the patients into four age groups to make the examined population more homogeneous (<30, 31-40, 41-50 and > 50 years). Specific data relative to the anal diseases, association with other anal or systemic pathologies, allergies, and previous surgery have been registered in every patient. Defecation pattern, fecal consistency, evacuation frequency, and eventual utilization of laxatives or enemas have been particularly emphasized. As regards duration of symptoms, two groups have been identified (<7 mo or >7 mo). Finally, we divided our population into two groups according to whether they had <7 or >7 fecal evacuations/wk.

Anorectal manometry was performed at rest and after maximum voluntary contraction, and was compared with the normal range for our laboratory. The resting anal tone and maximal squeeze pressure (i.e., the maximal voluntary increase above the resting tone) were measured according to a stationary pull-through technique. One and two months after treatment, patients underwent the same examination as performed at baseline.

Type A botulinum toxin was diluted with saline solution; after individuating the internal anal sphincter, the toxin was injected with a 27 gauge needle; during the procedure the patient was laying on the left side. The injection was given in the anterior or posterior midline. During the procedure, no local or systemic sedative was administered. The phials were containing 100 IU of botulinum toxin A (Botox, Allergan, Irvine, CA, United States) were stored at a temperature of -20°C and diluted in saline solution at the moment of its utilization. As regards the treatment with botulinum toxin, there were three groups according to the number of units injected: low dose (20 IU injected for single treatment); middle dose (30 IU);
and high dose (50 IU). The patients received different doses of botulinum toxin to test which dose may have a lower complication rate and higher healing rate.

**Statistical analysis**

Data have been analyzed with statistical standard methods. At the outset, we performed a univariate analysis with all the factors potentially influencing the course of disease using the $\chi^2$ test or Fisher’s exact test for categorical data and the ANOVA test for continuous data divided in more than two groups. Subsequently, we executed a logistic multivariate regression, constructing models with the factors potentially influencing the course of disease, which by univariate analysis had a $P$ value $< 0.25$. Additionally, we inserted age and sex into our multivariate analysis. Data were processed using GraphPad® Prism Software (GraphPad, San Diego, CA, United States). $P < 0.05$ was considered statistically significant, regardless of the test used.

**RESULTS**

Demographic data and parameters registered at the first observation are reported in Table 1. Specifically, we observed 21 (35.6%) male patients and 38 (64.4%) females, aged between 21 years and 61 years (average: 40.49 ± 10.63 years). We did not observe prevalence of the considered disease in patients of a particular age: we observed 12 (20.3%) patients aged $\leq$ 30 years, 19 (32.2%) aged 31-40 years, 15 (25.4%) aged 41-50 years, and 13 (22.0%) aged $> 50$ years.

The anal fissure was localized posteriorly in 91.5% and anteriorly in 5.1% of the patients. In 31 (52.5%) patients, symptoms started in the 7 mo before the clinical observation and treatment with botulinum toxin. 98.3% of cases (58 patients) were referred with post-defecatory pain, which tended to persist independently from the evacuation in 22 patients (37.3%) and during the night in 17 (28.8%). Bleeding, even mild, was reported in 44 patients (74.6%), and 22.0% complained of mucorrhea. The relaxation of puborectal muscle was documentable at the physical examination in almost all patients (98.3%). Associated anal or systemic pathology due to obesity was observed in only 11.9% and 39.0% of patients, respectively.

As regards defecation characteristics, 50 patients (84.7%) were referred for diarrhea and evacuation of feces with decreased consistency. In 89.8% of cases, the number of weekly evacuations was $> 7$. Only three patients (5.1%) were referred for forced evacuations, a sense of incomplete evacuation, and continuous utilization of laxatives and evacuative enemas.

In almost all patients, botulinum toxin was injected into the internal anal sphincter, at the anterior midline (56 cases). No patients received local anesthesia and/or systemic sedation, and in all patients, the internal sphincter was easily identified with digital palpation alone (Table 2).

**Healing after treatment with botulinum toxin**

We observed 45 and 26 patients at 1 mo and 2 mo, respectively. One month after treatment with botulinum toxin, healing was observed in 68.9% of patients. Only one patient developed mild incontinence to flatus that
rested 3 wk after treatment but disappeared spontaneously. Two months after treatment, no patient had incontinence. At the same time, the complete cicatrization of the fissure, with no residual specific symptoms, was evident in 65.4% of patients. Healing persisted for a period of 32.2 ± 33.9 mo (range: 0-141 mo).

**Manometric results**
At the first observation, before treatment, resting anal pressure was 107.1 ± 20.0 mmHg and maximal voluntary squeezing was 78.2 ± 17.0 mmHg. One month after treatment with botulinum toxin, the mean resting pressure and maximum voluntary squeeze pressure were 21.2% (84.4 ± 23.6 mmHg, \( P < 0.0001 \)) and 9.3% (70.9 ± 18.3 mmHg, \( P = 0.03 \)) lower, respectively, than the respective baseline values. Two months after treatment, the mean resting anal pressure was similar to the 1-mo value (\( P = 0.7 \)) and was 23.2% lower than the baseline value (82.3 ± 22.7 mmHg, \( P < 0.0001 \)). The maximum voluntary squeeze pressure did not differ significantly from the 1-mo value (\( P = 0.9 \)) and was 10.4% lower than the baseline value (70.1 ± 19.2 mmHg, \( P = 0.05 \)) (Table 3).

**Univariate analysis**
The considered parameters were compared with the healing rate at 1 mo and 2 mo after treatment with botulinum toxin. No demographic parameter had a direct influence on healing rate (Table 4). Both age and sex did not show a statistically significant difference (\( P = 0.42 \) and \( P = 0.11 \), respectively). None of the clinical parameters had an influence on the results. Dose and site of injection did not have a significant effect on healing rate.

**Multivariate analysis**
One month after treatment (Table 5), no significant relationship was revealed with any parameter. Two months after treatment, sex (\( P = 0.01 \)), baseline resting anal pressure (\( P = 0.02 \)) and resting anal pressure 2 mo after treatment (\( P < 0.0001 \)) were significantly related to healing rate (Table 6).

**DISCUSSION**
It has been shown that the more aggressive a bariatric technique is, the better are the results in terms of fat loss and maintenance of that fat loss over time. and although there is an improvement in comorbidity, the price paid by the patient, in relation to undesirable and adverse side effects and associated illnesses derived from these aggres-

---

**Table 2  Results of treatment with botulinum toxin A**

| Botox dose | Frequency | Percent (%) |
|------------|-----------|-------------|
| 20 IU      | 3         | 5.1         |
| 30 IU      | 11        | 18.6        |
| 50 IU      | 45        | 76.3        |

**Table 3  Manometric data before, and 1 and 2 mo after botulinum toxin treatment (mean ± SD)**

| Time         | Resting anal pressure (mmHg) | Maximum voluntary anal squeeze pressure (mmHg) |
|--------------|------------------------------|-----------------------------------------------|
| Baseline     | 107.1 ± 20.0                 | 78.2 ± 17.0                                   |
| 1 mo         | 84.4 ± 23.6^a                | 70.9 ± 18.3^a                                 |
| 2 mo         | 82.3 ± 22.7^b                | 70.1 ± 19.2                                   |

All patients were included in all evaluations. ^\( P = 0.036, ^{^a}P < 0.001 \) vs baseline.

**Table 4  Univariate analysis of parameters registered 1 and 2 mo after treatment**

| Risk factor                      | \( P \) value^a | \( P \) value^b |
|----------------------------------|-----------------|-----------------|
| Age                              | 0.4275          | 0.4034          |
| Sex                              | 0.1147          | 0.4128          |
| Duration of symptoms             | 0.4566          | 0.4018          |
| Post-defecatory anal pain         | 0.6889          | NA              |
| Nocturnal anal pain               | 0.1008          | 0.1039          |
| Anal pain unrelated to defecation | 0.1507          | 0.1039          |
| Bleeding                         | 0.4669          | 0.1585          |
| Mucorrhea                        | 0.5826          | 0.2081          |
| Other anal pathologies            | 0.1656          | 0.1591          |
| Extra-anal associated pathologies| 0.3223          | 0.133           |
| Defecation pattern                | 0.3825          | 0.3972          |
| Stool consistency                 | 0.3825          | 0.3291          |
| Number of evacuations/wk          | 0.1656          | 0.4197          |
| Straining                        | 0.2244          | 0.2615          |
| Sensation of incomplete evacuation| 0.2244          | 0.7323          |
| Laxative use                     | 0.2244          | 0.2615          |
| Enemas/ suppositories use         | 0.2244          | 0.7323          |
| Botulinum toxin dose              | 0.8151          | 0.1635          |
| Resting anal tone                 | 0.6889          | NA              |
| Maximal voluntary anal squeeze    | 0.2094          | 0.6179          |
| Puborectal muscle relaxation      | 0.3111          | NA              |
| MVAS 0 (mmHg)                     | 0.2457          | 0.7281          |
| MVAS 1 (mmHg)                     | 0.5292          | 0.4133          |
| MVAS 2                            | NA              | 0.8272          |
| RAP 0 (mmHg)                      | 0.284           | 0.1322          |
| RAP 1 (mmHg)                      | 0.3513          | 0.0985          |
| RAP 2                             | NA              | 0.172           |
| Site of injection                 | 0.0818          | 0.5815          |

MVAS 0: Maximal voluntary anal squeeze (pretreatment); MVAS 1: Maximal voluntary anal squeeze (1 mo after the treatment); MVAS 2: Maximal voluntary anal squeeze (2 mo after the treatment); RAP 0: Resting anal pressure (pretreatment); RAP 1: Resting anal pressure (1 mo after treatment); RAP 2: Resting anal pressure (2 mo after treatment). NA: Not applicable. 1: 1 mo after treatment; 2: 2 mo after treatment.
Table 5  Multivariate analysis of parameters registered 1 mo after treatment

| Risk factor                        | P value | T Ratio | 95% CI           |
|-----------------------------------|---------|---------|------------------|
| Age                               | 0.8286  | 0.2183  | -0.02207 to 0.0178 |
| Sex                               | 0.4231  | 0.8115  | -0.2156 to 0.5011 |
| MVAS 0                            | 0.9669  | 0.04185 | -0.01058 to 0.01102 |
| Site of injection                 | 0.7336  | 0.3433  | -0.2783 to 0.3911 |
| Maximal voluntary anal squeeze    | 0.3667  | 0.9273  | -0.6433 to 0.2409 |
| Enemas/suppositories use          | 0.5132  | 0.6612  | -0.5860 to 1.149  |
| Sensation of incomplete evacuations| 0.7892  | 0.2696  | -0.8144 to 1.063  |
| Straining                         | 0.8183  | 0.2317  | -0.9509 to 1.195  |
| Number of evacuations/wk          | 0.4590  | 0.7496  | -0.03187 to 0.01473 |
| Anal pain unrelated to evacuation | 0.6136  | 0.5099  | -1.341 to 0.8041  |
| Nocturnal anal pain               | 0.5195  | 0.6513  | -0.7694 to 1.492  |
| Other anal pathologies            | 0.4454  | 0.7727  | -0.3460 to 0.7966  |

MVAS 0: Maximal voluntary anal squeeze (pretreatment).

Table 6  Multivariate analysis of parameters registered 2 mo after treatment

| Risk factor                        | P value | T Ratio | 95% CI           |
|-----------------------------------|---------|---------|------------------|
| Age                               | 0.3991  | 1.059   | -0.01680 to 0.00750 |
| Sex                               | 0.0111  | 2.957   | 0.1110 to 0.7129 |
| Nocturnal pain                    | 0.8100  | 0.2454  | -0.3677 to 0.4619 |
| Bleeding                          | 0.2130  | 1.310   | -0.4982 to 0.1221 |
| Mucorrhea                         | 0.3122  | 1.052   | -0.6795 to 0.2345 |
| Other anal pathologies            | 0.0712  | 1.965   | -0.8284 to 0.03926 |
| Extra-anal pathologies            | 0.2659  | 1.163   | -0.4816 to 0.1446 |
| Dose of botulinum toxin           | 0.0771  | 1.920   | -0.02639 to 0.001535 |
| RAP 0                             | 0.0225  | 2.588   | 0.001689 to 0.01872 |
| RAP 1                             | 0.8975  | 0.1314  | -0.08417 to 0.007451 |
| RAP 2                             | < 0.0001| 5.953   | -0.03038 to -0.01420 |

RAP 0: Resting anal pressure (pretreatment); RAP 1: Resting anal pressure (1 mo after treatment); RAP 2: Resting anal pressure (2 mo after treatment).

Vanella S et al. Botulinum toxin after bariatric surgery

sive techniques, is high[13,24]. The etiology of anal pathology in this group of patients is multifactorial. One trigger-factor present after the intervention (as well as poor dietary habits), is diarrhea (steatorrhea), due to chemical irritation of the liquid feces. BPD produces an increase in the number of bowel movements and a decrease in their consistency due to malabsorption of fat[25]. Diarrhea generally appears during the first months after intervention and then it stabilizes between the 18th and 24th months when in general, fat loss has also stabilized.

Fich et al[25] showed a significant increase in the frequency of phase 3 of the migrating motor complex in patients after Billroth II and Roux-en-Y operations. Change in consistency of bowel motions after BPD was even greater than after Roux-en-Y gastric bypass, with nearly 80% of patients reporting loose stools or diarrhea, even 6 years after the intervention.

A high prevalence of fecal incontinence in both women and men after bariatric surgery has been described[26-28]. Women with diarrhea were four times as likely to have fecal incontinence and over half perceived their fecal incontinence to be worse after surgery[29]. Furthermore, women who perceived their diarrhea to be worse after surgery were significantly more likely to have fecal incontinence. This suggests that fecal incontinence in this population may be due to an underlying weakness in continence mechanisms that is clinically expressed after surgery because of altered stool consistency and delivery or changes in diet. Roberson and colleagues highlighted, for the first time, that fecal incontinence may begin or worsen after bariatric surgery[14].

In the general population, diarrhea, cholecystectomy, stress, and mixed urinary incontinence were risk factors for fecal incontinence in univariate analysis. However, only diarrhea remained a significant risk factor in multivariate analysis. Stool consistency is a critical factor affecting fecal continence. In addition to fat malabsorption resulting from BPD, an alteration in intestinal and colonic bacterial flora after bariatric surgery may also contribute to diarrhea[27].

Bacterial overgrowth is one of the complications related to diarrhea caused by the excluding loop in patients who undergo bariatric surgery. It occurs when a lack of equilibrium between the colonic flora and pathogenic bacteria is present. When untreated, it can evolve into malnutrition, anemia, poor absorption syndrome and can worsen diarrhea[28,29].

Anal fissure is a linear ulceration of the distal part of the anal canal, extending below the dentate line to the anal verge[30]. It is a very common pathology affecting the quality of life of subjects who, in most cases, do not have any other significant comorbidity. Most of the fissures are localized in the posterior midline; in 10% of females and 1% of males, they are localized anteriorly[31,32].

The etiology of anal fissure is multifactorial and not completely understood. There is a general agreement that trauma of the anal canal represents the initial insult. Hard bowel movements are the most common antecedent, but diarrhea may also be associated with the onset of fissure symptoms[31]. Anal hypertonicity and subsequent decreased blood flow to the anoderm are now recognized as pivotal factors in the pathogenesis of anal fissures. The increased internal sphincter pressure in patients with fissures reduces the blood flow to this area even further. Sphincterotomy reduces the anal canal pressure and improves anodermal blood flow at the posterior midline, resulting in fissure healing, which provides further evidence that abnormal activity in the sphincter contributes to the development of a fissure.

Although anal fissures are generally associated with constipation, in 7%, diarrhea could be the trigger as passing irritant liquid feces is a factor in the deterioration of the situation more often than with the patients who are constipated. This appeared together with the increase in the number of bowel movements following dietetic transgressions. Diarrhea/steatorrhea, due to malabsorption of fat and bacterial flora after bariatric surgery may also contribute to diarrhea[27].
in anal fissure is high anal pressure; therefore, different therapeutics strategies are needed to resolve the internal sphincter spasm, temporarily or permanently. The most used surgical procedure is partial lateral internal sphincterotomy[39]. The procedure has been considered the most effective treatment for anal fissure, although up to 10% of patients have recurrence, and > 35% of patients may experience incontinence. In fact, continence mechanisms can be acutely unbalanced by the execution of sphincterotomy: To avoid the permanent sequences of sphincterotomy, especially in multiparous women with a high risk of incontinence, alternative treatments can be adopted, such as botulinum toxin injection or topical application of nitrates[31,34]. The advantage in using these therapeutic approaches is that they cause a significant decrease in anal resting tone, inducing fissure healing, without permanent damages to anal sphincters[39].

We reported the results of observation and treatment with type A botulinum toxin of patients affected by chronic anal fissure who had undergone Scopinaro’s BPD. We observed a prevalence of anal fissure in the female patients, but we did not observe any difference between the age groups. One month after treatment with botulinum toxin, healing was observed in 68.9% of patients. Two months after treatment, complete healing of the fissure without any specific symptoms was present in 65.4% of cases. Healing persisted for a mean 32.2 ± 33.9 mo. The number of evacuations per week did not significantly relate to persistence of fissure at 1 mo and 2 mo follow-up. We think that not only the frequency of bowel movements but also the altered composition of feces can play a role in the development of anal disorders in these patients. Mild incontinence to flatus with botulinum treatment was probably related to the diffusion of botulinum toxin to the external anal sphincter. The multivariate analysis of the parameters registered at the first control revealed no statistically significant values, instead, at the second control, sex, baseline resting anal pressure and resting anal pressure 2 mo after treatment had a significant influence on healing, by multivariate analysis. In particular, male sex had a direct influence on healing and high resting anal pressure pre- and post-treatment was associated with persistent anal fissure.

Despite the high dose of botulinum toxin, the healing rate in this study, and the reduction of resting anal canal pressure and maximum anal canal squeeze pressure were lower than in our previous trials[18,19,30,34,35]. The results of the present study were different from our previous observation of 100 patients: 50 treated with 50 IU Botox (group I) and 50 with 150 IU Dysport (group II)[35]. We considered the difference in group I. One month after treatment, fissure healing was present in 82% of the patients in our previous study and in 68.9% of the patients in the present trial (P = 0.15). In patients in previous trials, a 38.7% decrease of resting anal pressure, compared to pretreatment values, was registered; in the present study, we observed a lower decrease of resting anal pressure (21.2%). The reduction of maximal anal squeezing was 22.4% and 9.3% in the previous and present study, respectively. Two months after treatment, 92% and 65.4% of the patients in the previous and present study, respectively, (P = 0.007) had a healing scar. The reduction of resting anal canal pressure was lower than in our previous trial (23.2% vs 41.8%). The decrease of maximum anal canal squeeze pressure was lower than in our previous study (10.4% vs 20.2%)[35].

We think that the increase of baseline resting anal pressure in bariatric patients is secondary to repeated, continuous diarrhea and changes in qualitative composition that are typical of steatorrhea in malabsorptive processes. The reduction of resting anal pressure and maximal anal squeezing after treatment, lower than in the general population, and the association between persistent high levels of resting anal pressure and a low healing rate can result from chronic irritation of the anal canal by liquid stools. The reduction of resting anal pressure favors healing but the results are worse than in the general population because the anal hypertonicity is not the principal pathogenetic factor of chronic anal fissure in bariatric patients.

In summary, we believe that, in patients who undergo BPD, chronic steatorrhea can be the cause of different anal disorders. It is of utmost importance to be conservative in the treatment of anal disorders for bariatric patients. It is also important to remember that the anatomy of the anal canal should be preserved as much as possible so as to avoid incontinence, because in these patients, at least one of the continence mechanisms is already altered. Botulinum toxin, despite worse results than in non-obese population, appears the best alternative to surgery for this group of patients with high risk of incontinence. The effectiveness of other treatments such as nitrates depends on patient compliance, which may be poor in bariatric patients. Moreover, repeated evacuations and altered fecal composition can decrease the pharmacological effect of nitrates. Botulinum toxin injection has the advantage of a good healing rate, can be repeated if necessary, and the possible incontinence is temporary.

**COMMENTS**

**Background**

Anal fissure is a split in the distal anal canal. It is a common problem that causes substantial morbidity in people who are otherwise healthy. There is accumulating agreement that bariatric surgery is currently the most efficacious and enduring treatment for clinically severe obesity, and as a result, the number of bariatric surgery procedures performed has risen dramatically in recent years. Biliopancreatic diversion (BPD) by Scopinaro is a malabsorptive technique that is successful in achieving long-lasting weight loss in super-obese patients. The diarrhea that is expected after the procedure can sometimes cause significant nutritional changes and anal disease.

**Research fronts**

Treatment of chronic anal fissure has undergone a transformation in recent years from surgical to medical, both approaches sharing the common goal of reducing spasm of the internal anal sphincter. Surgical sphincterotomy results in a healing rate up to 95%. Despite this, it carries a significant risk of persistent disturbance in anal continence. This is believed to be the first study of the use of botulinum toxin injection for treatment of chronic anal fissure in patients undergoing BPD. This article, unlike others that focus only on the positive effects...
of bariatric surgery on improvement of comorbidity in obese patients, highlights the side effects that may affect the quality of life and draws new boundaries in the treatment of chronic anal fissure after BPD.

**Innovations and breakthroughs**

Different types of bariatric operations might alter bowel habits as a consequence of the surgical technique used. Disordered bowel habits and anal diseases might influence quality of life after bariatric surgery. There are few papers in the literature devoted to the study of the anal disorders after bariatric surgery. Prospective randomized trials are needed to estimate the true incidence and to establish the etiology of proctological disorders in these patients.

**Applications**

The treatment of chronic anal fissure in these high-risk patients should have the minimum risk of fecal incontinence. Botulinum toxin injection has the advantages of an excellent healing rate and can be repeated if necessary. Any incontinence, as a complication, however, is transient.

**Peer review**

This is an interesting paper about the usefulness of botulinum toxin A in healing chronic anal fissure after BPD for morbid obesity. The authors report complete healing in 65.4% of patients.

**REFERENCES**

1. Bennett JM, Mehta S, Rhodes M. Surgery for morbid obesity. *Postgrad Med* 2007; 83: 8-15
2. Fobi MA. Surgical treatment of obesity: a review. *J Natl Med Assoc* 2004; 96: 61-75
3. Dixon JB. Obesity and diabetes: the impact of bariatric surgery on type-2 diabetes. *World J Surg* 2009; 33: 2014-2021
4. Simard B, Turcotte H, Marceau P, Biron S, Houël FS, Lebel S, Marceau S, Boulet LP. Asthma and sleep apnea in patients with morbid obesity: outcome after bariatric surgery. *Obes Surg* 2004; 14: 1381-1388
5. Zingmond DS, McCorgy ML, Ko CY. Hospitalization before and after gastric bypass surgery. *JAMA* 2005; 294: 1918-1924
6. Buchwald H, Avidor Y, Braunwald E, Jensen MD, Pories W, Fahrbach K, Schoelles K. Bariatric surgery: a systematic review and meta-analysis. *JAMA* 2004; 292: 1724-1737
7. Livingston EH. Obesity, mortality, and bariatric surgery death rates. *JAMA* 2007; 298: 2406-2408
8. Maggard MA, Shugarman LR, Sitttorp M, Maglione M, Segerman HJ, Livingston EH, Nguyen NT, Li Z, Mojica WA, Hilton L, Rhodes S, Morton SC, Shekelle PG. Meta-analysis: surgical treatment of obesity. *Ann Intern Med* 2005; 142: 547-559
9. Steinbrook R. Surgery for severe obesity. *N Engl J Med* 2004; 350: 1075-1079
10. NIH conference. Gastrointestinal surgery for severe obesity. Consensus Development Conference Panel. *Ann Intern Med* 1991; 115: 956-961
11. Van Hee RH. Bilipancreatico gastro intestinal diversion in the surgical treatment of morbid obesity. *World J Surg* 2004; 28: 435-444
12. Scopinaro N, Adami GF, Marini GM, Gianetta E, Traverso E, Friedman D, Camerini G, Baschieri G, Simonelli A. Bilipancreatic diversion. *World J Surg* 1998; 22: 936-946
13. Scopinaro N, Marini G, Camerini G, Papadia F. Bilipancreatic diversion for obesity: state of the art. *Surg Obes Relat Dis* 2005; 1: 317-328
14. Roberson EN, Gould JC, Wald A. Urinary and fecal incontinence after bariatric surgery. *Dig Dis Sci* 2010; 55: 2606-2613
15. Potoczna N, Hartmann SE, Steffen R, Briggs R, Bier N, Horber FF. Bowel habits after bariatric surgery. *Obes Surg* 2008; 18: 1287-1296
16. Wasserberg N, Hamoui N, Petrone P, Crookes PF, Kaufman HS. Bowel habits after gastric bypass versus the duodenal switch operation. *Obes Surg* 2008; 18: 1563-1566
17. Marini GM, Papadia FS, Briatore L, Adami G, Scopinaro N. Type 2 diabetes and weight loss following biliopancreatic diversion for obesity. *Obes Surg* 2006; 16: 1440-1444
18. Brisinda G, Maria G, Bentivoglio AR, Cassetta E, Gui D, Albannese A. A comparison of injections of botulinum toxin and topical nitroglycerin ointment for the treatment of chronic anal fissure. *N Engl J Med* 1999; 341: 65-69
19. Maria G, Cassetta E, Gui D, Brisinda G, Bentivoglio AR, Albannese A. A comparison of botulinum toxin and saline for the treatment of chronic anal fissure. *N Engl J Med* 1998; 338: 217-220
20. DeMaria EJ. Bariatric surgery for morbid obesity. *N Engl J Med* 2007; 356: 2176-2183
21. Adams TD, Gress RE, Smith SC, Halverson RC, Simper SC, Rosamond WD, Lamotte MJ, Stroup AM, Hunt SC. Long-term mortality after gastric bypass surgery. *N Engl J Med* 2007; 357: 753-761
22. Fich A, Neri M, Camilleri M, Kelly KA, Phillips SF. Stasis syndromes following gastric surgery: clinical and motility features of 60 symptomatic patients. *J Clin Gastroenterol* 1990; 12: 505-512
23. Chen CC, Gatmaipan K, Koepp S, Barber MD, Chand B, Schauer PR, Brehbauer SA. Obesity is associated with increased prevalence and severity of pelvic floor disorders in women considering bariatric surgery. *Surg Obes Relat Dis* 2009; 5: 411-415
24. Lawrence JM, Lukacz ES, Liu IL, Nager CW, Luber KM. Pelvic floor disorders, diabetes, and obesity in women: findings from the Kaiser Permanente Continence Associated Risk Epidemiology Study. *Diabetes Care* 2007; 30: 2536-2541
25. Lawrence JM, Lukacz ES, Nager CW, Hsu JW, Luber KM. Prevalence and co-occurrence of pelvic floor disorders in community-dwelling women. *Obstet Gynecol* 2008; 111: 678-685
26. Shamliyan T, Wyman J, Bliss DZ, Kane RL, Wilt TJ. Prevention of urinary and fecal incontinence in adults. *Evid Rep Technol Assess (Full Rep)* 2007; 1-379
27. Wasserberg N, Haney M, Petrone P, Ritter M, Emami C, Rosca J, Siegmund K, Kaufman HS. Morbid obesity adversely impacts pelvic floor function in females seeking attention for weight loss surgery. *Dis Colon Rectum* 2007; 50: 2096-2103
28. Crea N, Papa G, Di Betta E, Greco F, Casella C, Vilardi A, Mettmergerger F. Long-term results of biliopancreatic diversion with or without gastric preservation for morbid obesity. *Obes Surg* 2011; 21: 139-145
29. Crookes PF. Surgical treatment of morbid obesity. *Ann Rev Med* 2006; 57: 243-264
30. Z’graggen K, Guweidhi A, Steffen R, Potoczna N, Biral R, Walther F, Komminoth P, Horber F. Severe recurrent hyperglycemia after gastric bypass surgery. *Obes Surg* 2008; 18: 981-988
31. Lindsey I, Jones OM, Cunningham C, Mortensen NJ. Chronic anal fissure. *Br J Surg* 2004; 91: 270-279
32. Elia Guedea M, Gracia Solanas JA, Royo Dachary P, Ramirez Rodriguez JM, Aguililla Diago V, Martinez Diez M. [Prevalence of anal diseases after Scopinaro’s biliopancreatic bypass for super-obese patients]. *Cir Esp* 2008; 84: 132-137
33. Brisinda G, Vanella S. Chronic anal fissure: Surgical or reversible neurochemical sphincterotomy? *Nat Rev Gastroenterol Hepatol* 2009; 6: 694-695
34. Brisinda G, Cadeddu F, Brandara F, Marniga G, Maria G. Randomized clinical trial comparing botulinum toxin injections with 0.2 per cent nitroglycerin ointment for chronic anal fissure. *Br J Surg* 2007; 94: 162-167
35. Brisinda G, Albanese A, Cadeddu F, Bentivoglio AR, Mabiosomi A, Marniga G, Maria G. Botulinum neurotoxin to treat chronic anal fissure: results of a randomized “Botox vs. Dysport” controlled trial. *Aliment Pharmacol Ther* 2004; 19: 695-701

S-Editor Shi ZF  L-Editor Kerr C  E-Editor Zheng XM