Botulinum toxin for spastic GI disorders

To the Editor:

We read with interest the review on botulinum toxin for spastic GI disorders by Drs. Zhao and Pasricha.[1] This excellent review confirms that the management of spastic GI disorders has undergone extensive re-evaluation and renewed emphasis during the past few years. This is attributable to the development of neurochemical treatment.

Since the discovery in the late 1970s that botulinum neurotoxin inhibits neuromuscular transmission, this powerful poison has been studied extensively. One of the most recent applications is the treatment of various GI disorders.[2,3] Beginning with the pioneering work of Dr. Pasricha in the treatment of esophageal achalasia, clinical application has expanded to encompass a range of GI conditions and continues to increase. Botulinum neurotoxin is not only potent in blocking skeletal neuromuscular transmission, but also cholinergic nerve endings in the autonomic nervous system. The capability to inhibit contraction of the GI smooth muscle was first suggested by in vitro observations and later demonstrated in vivo; it has also been shown that botulinum neurotoxin does not block nonadrenergic, noncholinergic responses mediated by nitric oxide.

In the section of their review devoted to anismus and related conditions, Zhao and Pasricha state that relief of symptoms is reported to be temporary, usually lasting less than 5 months in most patients. We have treated 14 women with anterior rectocele by injection of botulinum toxin into the puborectalis.[4] After 2 months, symptom as well as defecographic improvement was noted in 9 patients (64%). All patients were followed for a mean of 18 ±4 months during which there was no relapse of symptoms in any case. No complications or side effects were observed during follow-up, with the exception of one patient who developed mild incontinence for flatus 15 days after treatment with 100 units of botulinum toxin: incontinence persisted for 1 week and spontaneously disappeared. At the 1-year evaluation, incomplete or digitally assisted rectal evacuation had not been reported by any patient. Rectocele was not found at physical examination. The resting anal pressure (60 ±19 mm Hg; $p = 0.7$) and the maximum voluntary pressure (45 ±22 mm Hg; $p = 0.9$) were not changed compared with baseline values. The pressure during straining (49 ±12 mm Hg) was reduced as compared with baseline values ($p = 0.01$), and was not significantly lower than resting pressure ($p = 0.08$). Defecography revealed recurrent rectoceles in 4 patients, but all of them were asymptomatic. Rectocele depth was 1.9 ±1 cm ($p = 0.00001$ versus baseline value) and rectocele area 3.1 ±0.9 cm^2 ($p = 0.00001$ versus baseline value). Anorectal angle was 119 ±26 degrees ($p = 0.01$ versus baseline value).

Rectocele, herniation of the anterior rectal wall into the lumen of the vagina, is a frequent finding in women patients and its clinical relevance is questionable: from 20% to 81% of both asymptomatic women and patients with constipation may have rectoceles. If less than 2 cm in diameter, rectoceles are usually accepted as a normal finding, both in constipated patients and in healthy subjects, whereas those with a diameter of more than 2 cm can cause outlet obstruction and rectal emptying difficulties. Although it has been suggested that some rectoceles may be caused by failure of relaxation or paradoxical contraction of the puborectalis muscle during attempted evacuation, the exact pathogenesis is unclear. However, it is important to identify a rectocele when it is the primary cause of intractable obstructed evacuation. Rectoceles may cause mild to severe anorectal symptoms that are usually associated with chronic constipation. It is probable that straining aggravates the rectocele, enlarges it, and makes evacuation even more difficult. In patients with rectocele and paradoxical sphincter reaction, defecation has to occur through the unrelaxed pelvic floor. Although simple to diagnose, rectocele has proved to be difficult to treat. A wide variety of surgical approaches have been proposed with the aim of assuring rectal emptying by reducing the dimension of the rectocele. However, the results of operation are often disappointing with regard to emptying difficulties. Surgical repair—either vaginal, transperineal or transanal—does not always alleviate symptoms and in some patients impairs fecal continence. Transanal repair, furthermore, may compromise anal sphincter pressures. An alternative approach should be considered when the anal sphincter is lax.

We believe that botulinum toxin injection is a safe treatment for patients with pelvic floor disorders. Our observations indicate that outlet obstruction is the main cause for anterior rectocele in certain patients and provide evidence that botulinum toxin may be a remedy in these patients. The duration of efficacy of the injections remains to be measured, and repeated treatments are probably necessary. The optimal dose of botulinum toxin also remains to be determined; a placebo controlled study with long-term follow-up is warranted.

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