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Relief by botulinum toxin of voiding dysfunction due to prostatitis

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Obstructive urinary symptoms due to chronic non-bacterial prostatitis are due to incomplete relaxation of the bladder neck or inappropriate contraction of the external urethral sphincter during voiding.¹ Patients may respond to α -adrenergic blocking drugs; if effective, treatment may need to be continued indefinitely. Botulinum toxin has been used to weaken striated^{2,3} and smooth¹ muscles. Toxin injections into the external urethral sphincter to relieve urethral obstruction were described by Dykstra and Sidi.³ Apart from treatment of detrusor-sphincter dys-synergia, we have found no reports in the literature on the treatment of voiding dysfunction in non-bacterial prostatitis.

Four consecutive men (mean age 30.7 [SD 5.7] years) with chronic non-bacterial prostatitis and poor bladder emptying because of spastic external urethral sphincter (mean duration of symptoms 18 [3] months), who failed to respond to tamsulosin 0.4 mg once daily for more than 4 months were enrolled. All the patients were examined, had uroflowmetric studies to assess times of urinary flow (TQ) and maximum urinary flow (TQmax), maximum flow (Qmax), average flow (Qave), and total urinary volume (Vcomp), and had anorectal manometry at rest (RT) and after maximum contraction (MC). An increased value of TQ and TQmax with a normal value of Qmax was taken to be indicative of incomplete relaxation of bladder neck. 1, 4, and 8 weeks after treatment, patients underwent the same assessments.

With the patient lying on his left side, a 26-gauge monopolar needle electrode was inserted in the perineum in the anterior midline, about 1.5-2.0 cm from the anus and directed toward the prostatic apex, without sedation or local anaesthesia. 30 U of type A botulinum toxin were injected. No local complications or systemic side-effects were seen. Within 1 week of injection all patients had a striking improvement in their voiding; none complained of urinary incontinence. At 4 weeks, three patients showed a continuing improvement. At 8 weeks, the same three patients were satisfied with the therapy and none of them complained of urinary incontinence. The patients were followed up for a mean of 12 months. No relapse occurred in the three patients who improved. 6 weeks after treatment,

the fourth patient reported worsening voiding. He was also depressed and required fluoxetine. He was re-treated with 50 U of botulinum toxin and the urinary symptoms improved. Uroflowmetric study showed a decrease in TQ and TQmax values at 1, 4, and 8 weeks compared with baseline values (table). Other variables were not changed.

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Enterohaemorrhagic Escherichia coli in Ngoila (Cameroon) during an outbreak of bloody diarrhoea

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Ngoila (2°37' N, 14°1' E, population 400) is in a rain-forest region of Cameroon. In November, 1997, there was an outbreak of red diarrhoea. After investigations by the District Health Authorities, metronidazole and cotrimoxazole were suggested to control the epidemic. By March, 1998, the Ministry of Health had reports of 237 cases and 44 deaths. The illness evolved slowly and deaths were not reported before day 7. Viral haemorrhagic fever was suspected and epidemiological investigations were started. We identified 22 patients, between March 25 and March 28 who presented with bloody diarrhoea or dysentery, duration of illness 3-58 days (median 7). Cramping abdominal pain, fever, and dehydration were noted in 96%, 43%, and 37%. Available antiparasitic and antibacterial agents were not effective against the illness.

Stools and blood samples were collected from 22 patients either with dysentery or bloody diarrhoea, aged 2-60 years. Stool samples and serum samples were frozen in liquid nitrogen. Stool samples stored in merthiolate-iodine-formaldehyde solution were screened for parasites. Stool cultures set up in the field were screened 2 days later for enteropathogenic bacteria. Serum samples were investigated at the Institut Pasteur de Bangui for Ebola Virus (sequence detection by RT PCR, ELISA IgM capture assay, ELISA antigen detection with IgM capture), Marburg virus (IgG and IgM), Rift Valley fever virus (ELISA antigen detection, FI ISA-IgM capture assay, viral isolation on Vero E6 cells), yellow fever virus (IgM), Rift Valley fever virus (IgG), CCHF virus (IgG) and hanta viruses (Hantaan, Seoul, Pumala). All were negative. Serum samples were negative for Ebola virus in the Institut Pasteur, Paris.

Non-polio enterovirus was detected in 12 (54.5%) patients; parasites, including flagellates, amoebae, and helminths in 17 (77.3%); *Entamoeba histolytica histolytica* in seven. Stool cultures identified 11 (50%) patients infected with *Escherichia coli* 0157:H7; nine (41%) with *Shigella dysenteriae* type 1; and two (9%) with *S boydii*. Two patients were infected with both *S dysenteriae* type 1 and *E coli*

	Baseline	1 week	4 weeks	8 weeks
TQ (s)	49.3 (8.7)	23.2 (15.3)*	22.2 (17.5)*	26.5 (8.5)*
TQmax (s)	20.7 (7.5)	8.0 (1.8)*	9.7 (5.9)*	13.2 (4.4)*
Qmax (mL/s)	16.7 (6.4)	15.2 (6.8)†	15.0 (3.4)†	21.6 (10.2)†
Qave (mL/s)	11.0 (5.1)	9.9 (4.5)†	8.7 (1.5)†	12.3 (5.4)†
Vcomp (mL)	353.5 (220.1)	200.7 (86.4)†	215.0 (97.4)†	405.2 (66.7)†
RT (mm Hg)	70.0 (8.2)	65.0 (10.0)†	59.2 (8.3)†	60.7 (16.2)†
MC (mm Hg)	90.0 (29.4)	66.0 (11.4)†	56.2 (15.9)†	72.7 (37.5)†

† test for paired samples. *p<0.05 vs baseline value. †p>0.05 vs baseline value.

Uroflowmetric and anorectal manometric values in four patients