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Chronic prostatitis presenting with dysfunctional voiding and effects of pelvic floor biofeedback treatment

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Study Type – Therapy (case series)
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OBJECTIVE

To investigate the features of chronic prostatitis presenting with dysfunctional voiding (DV) and the effects of pelvic floor biofeedback (PFB).

PATIENTS AND METHODS

The study included 21 patients, diagnosed by having symptoms for ≥3 months, including urinary frequency and urgency, voiding difficulty, upper abdominal or perineal discomfort, and with a score of ≥1 on the first and second part of the National Institutes of Health Chronic Prostatitis Symptom Index (NIH-CPSI). Patients with bacterial prostatitis, urethritis, interstitial cystitis, urethral stricture and neurogenic bladder were excluded. All patients had a urodynamic examination, to assess the uroflow curve, maximum urinary flow rate (Q_{max}), maximum detrusor pressure during the storage phase ($P_{det.max}$), maximum urethral pressure (MUP) and the maximum urethral closure pressure (MUCP) were recorded. PFB was carried out in patients with non-neurogenic detrusor sphincter dyssynergia, and the effects evaluated after 10 weeks.

RESULTS

Before and after PFB treatment the mean (SD) Q_{max}, $P_{det.max}$, MUP, MUCP were 8.2 (4.1) vs 15.1 (7.3) mL/s, 125.1 (75.3) vs 86.3 (54.2) cmH_2O, 124.3 (23.3) vs 65.4 (23.0) cmH_2O and 101.5 (43.6) vs 43.5 (16.7) cmH_2O, all significantly different ($P < 0.05$). The respective differences in the pain, urination and life impact subdomain scores, and total scores, of the NIH-CPSI were 4.0 (2.0) vs 2.2 (1.7), 7.9 (2.1) vs 2.2 (1.9), 9.6 (2.7) vs 2.9 (2.6) and 21.7 (4.8) vs 8.4 (4.6), and all differences were significant ($P < 0.05$).

CONCLUSIONS

There might be DV in patients with chronic prostatitis and lower urinary tract symptoms. Urodynamics showed a low Q_{max} and increasing intravesical pressure and, in some patients, increasing urethral pressure. Urodynamics could be used to help in the diagnosis, and to select the most appropriate treatment. PFB had satisfactory short-term effects on these patients.

KEYWORDS

chronic prostatitis, dysfunctional voiding, urodynamics, biofeedback

INTRODUCTION

Chronic prostatitis (CP) is a common diagnosis in men and is mainly treated using antimicrobial and anti-inflammatory drugs, with a relatively low cure rate and high recurrence rate. This shows that there might be different mechanisms for these patients' symptoms, most of whom are assumed to have nonbacterial CP and are given empirical treatments. Urodynamic examinations have been used in patients with recurrent nonbacterial CP who present with chronic LUTS, and some appeared to have dysfunctional voiding (DV). In the present study, we investigated the features of CP presenting with DV and the effects of pelvic floor biofeedback (PFB), in 21 patients with non-neurogenic detrusor sphincter dyssynergia (DSD).

PATIENTS AND METHODS

Between July 2007 and January 2009 we analysed retrospectively 21 men diagnosed as having nonbacterial CP with DV. The diagnosis of CP conformed to the recommendations of the National Institutes of Health (NIH) criteria: symptoms for >3 months, including urinary frequency and urgency, voiding difficulty, upper abdominal or perineal discomfort, etc.; scores of ≥1 for the first and second part of NIH CP Symptom Index (CPSI); treated once by antibiotics and α-blockers for >6 months, with poor effects [1]. Bacterial prostatitis, urethritis, interstitial cystitis, urethral stricture and neurogenic bladder were excluded from the analysis through detailed medical history-taking, systematic physical examination, urine culture, analysis of expressed prostatic secretion, cysto-urethrography and cystoscopy.

All patients had a urodynamic examination, including uroflowmetry, a uroflow curve, maximum flow rate (Q_{max}), a cystometrogram in the filling phase and pressure-flow electromyography (EMG) by synchronous detection in the voiding phase (the EMG was recorded by surface electrodes), and measurements of bladder compliance, maximum detrusor pressure during the storage phase (Pdet.max), static urethral

pressure profile, maximum urethra closure pressure (MUCP) and the maximum urethral pressure (MUP). Urodynamics was conducted according to the recommendations of the ICS after discontinuation of all drugs for 3–5 days.

The diagnostic criteria of DV included: nothing abnormal detected in the history, and no symptoms on an examination for neurological diseases; transient and intermittent closure the of external sphincter during voiding detected by EMG and fluoroscopic cysto-urethrography; a higher external sphincter EMG activity with no abdominal pressure increase in the voiding phase. Uroflowmetry was assessed individually to show any discontinuity in a diagram of urinary flow, in conditions with as little external interference as possible.

PFB was used by patients with DV after the urodynamic tests. An anal electrode was used to record the EMG. The abdominal pressure was measured through a 6 F balloon catheter in the rectum. Patients were maintained in the optimum position for pelvic floor relaxation and were taught to void while sitting, with the thighs spread to relax the perineum, the back straight and tilted slightly forward. They were taught to perceive differences in relaxation and contraction of the anal sphincter, and instructed to constrain it while simultaneously keeping the abdominal pressure curve stable. Each session lasted ≈30 min, was administered twice or three times a week, and could be done intermittently for several weeks. After nearly 10 weeks, the NIH-CPSI and uroflowmetry with EMG were repeated to evaluate the results. Throughout the sessions, all the antibiotics, α-blockers and anticholinergic agents were suspended.

Continuous variables are expressed as the mean (SD) and assessed statistically using a *t*-test; categorical variables were assessed statistically by the chi-square test. The NIH-CPSI scores in the CP group were evaluated using the Wilcoxon matched-pairs signed-ranks test; in all tests, $P < 0.05$ was considered to indicate statistically significant differences.

RESULTS

The comparison of the urodynamic results in the 21 patients before and after PFB is shown in Table 1; all variables were significantly different ($P < 0.05$) with a clear increase in Q_{max} and clear decrease in Pdet.max, MUP and MUCP, indicating a relief of LUTS. The NIH-CPSI scores before and after biofeedback are also shown in Table 1; the scores for pain, urination, life impact and total score decreased significantly ($P < 0.05$), indicating an effect of the relief of LUTS by PFB in patients with CP and DV.

Mean (SD) variable	Before	After
Urodynamics		
Q_{max}, mL/s	8.2 (4.1)	15.1 (7.3)
Pdet.max, cmH_2O	125.1 (75.3)	86.3 (54.2
MUP, cmH_2O	124.3 (23.3)	65.4 (23.0
MUCP, cmH_2O	101.5 (43.6)	43.5 (16.7
NIH-CPSI scores		
Discomfort	4.0 (2.0)	2.2 (1.7)
Urination	7.9 (2.1)	2.2 (1.9)
Life impact	9.6 (2.7)	2.9 (2.6)
Total	21.7 (4.8)	8.4 (4.6)

TABLE 1 The urodynamic variables and NIH-CPSI scores before and after PFB; all differences were significant ($P < 0.05$)

DISCUSSION

DV is secondary to voluntary contraction of the external sphincter during micturition, which can be voluntarily suppressed by the patient in the absence of known neurological disease, differentiated from external DSD, which is a serious urological condition with involuntary contraction of or inability to relax the external sphincter, frequently seen in patients with spinal cord injury and multiple sclerosis. The aetiology of DV is the emotional and psychological aspects or the reaction to adverse conditions (e.g. inflammation, infection, pelvic disease, etc.) [2]. Kaplan *et al.* [1] thought that the psychological stress was related to voiding disorders in men with nonbacterial CP, and some of those who in fact had a contraction of the external urinary sphincter during voiding had been misdiagnosed and empirically treated for nonbacterial CP. Our previous research [3] also analysed the possible mechanism that increased the mental burden caused by pelvic floor dysfunction, and then increased the prostatic urethral pressure, which caused LUTS in some patients with CP, manifested as voiding symptoms and mostly classified as IIIB (leukocytosis with no proof of bacterial infection); Hinman and Baumann [4] had a similar view.

We surmised that the pathogenic process in patients with CP and DV was a type of acquired abnormality, caused by psychological factors. The relationship between DV and CP might be a chronic mental burden caused by a disorder of the pelvic floor muscles, LUTS and then CP; or inflammation and infection due to CP induced with DV. Although the causes of CP/chronic pelvic pain syndrome are still unclear, there are three important factors: (i) there is a mutually promoting 'vicious circle' between CP and DV; (ii) CP presenting with DV is often misdiagnosed as only CP and urodynamics are ignored; (iii) patients with CP and DV are often treated as having only CP, with unsatisfactory cure rates and further development of disease.

The diagnosis of DV mainly depends on pressure-flow and synchronous EMG detection, with voiding cysto-urethrography. Urodynamic studies show a continuous detrusor contraction accompanied by higher perianal EMG activity in the voiding phase. Urethrography shows complete or partial closure of the membranous urethra. Kaplan *et al.* [1] reported a decrease in Q_{max} and increase in Pdet.max in DV. Moreover, Liao *et al.* [5] confirmed an increase in MUP and MUCP in patients with CP. These results were consistent with those in the present study. In summary, the urodynamic features of CP with DV were a decrease in Q_{max} and increase in Pdet.max, MUP and MUCP.

We suggest for that for patients with CP and voiding symptoms, especially those with recurrent CP, it is necessary to undertake urodynamic testing and a detailed examination of the nervous system to investigate possible DV, which is important for selecting the appropriate therapy. During the urodynamic study, the influence of the surrounding environment while voiding should be minimized.

Since 1970, when Hinman and Bauman [4,6] originally coined the term, the non-neurogenic neurogenic bladder has been difficult to treat. The first patient described by Hinman and Bauman [4] was treated successfully with hypnosis. Some researchers [7] reported that diazepam or α-blockers could be used to alleviate the tension of the external and internal urethral sphincter, but the effect is not ideal in clinical practice. Otherwise, patients with CP appearing as DV have had prolonged treatment with α-blockers and antibiotics before further treatment.

Using specific devices, PFB can provide signals reflecting pelvic muscle movement, although the signals must be converted to those sensed by the patients, e.g. visible and acoustic signals, to teach patients to constrain and relax the pelvic muscles selectively, while keeping other muscles relaxed. Porena *et al.* [8] used PFB to treat 43 children with DSD; voiding disorders in all the patients improved dramatically. Achieving pelvic floor muscle and sphincter coordination again was thought to have a positive influence on the neuromuscular mechanism controlling all bladder and sphincter activities. Cornel *et al.* [9] treated 31 patients with CP using PFB, and the effect was significant.

Based on these results, physical biofeedback therapy might be a useful means to correct pelvic floor dysfunction, which is closely related to CP with DV. After a 10-week course of therapy, all subdomains of the NIH-CPSI decreased markedly. Voiding symptoms were alleviated, with urodynamic values improved simultaneously. These satisfying effects further support that PFB has satisfying short-term effects for patients with CP and DV, but further studies are needed to assess the mid- and long-term curative effects and side-effects. Another shortcoming of the present study was that there was no PFB 'placebo' group as a control. These patients with CP and DV had prolonged treatment with α-blockers and antibiotics before further treatment, but the effect was not ideal.

From our experience, PFB is a course of active learning, which has a close relationship with the patients' comprehension and educational levels, and the capability of the medical staff to demonstrate and explain the method. Three points are important: (i) the patient must respond positively to the detailed instructions to correct erroneous movements; (ii) the duration and intensity of each treatment should be suitable, so that patients do not feel fatigue in the pelvic floor; (iii) patients need encouragement to complete the prolonged course of therapy, and the economic burden of hospitalization during the first 1 or 2 weeks.

In conclusion, patients with CP presenting as DV have pelvic floor dysfunction and many abnormal urodynamic features, i.e. decreased Q_{max} and increased Pdetmax and MUCP. Many symptoms arise from pelvic floor dysfunction rather than other causes. The short-term effect of PFB is satisfactory, but the long-term effects need a further follow-up study.

CONFLICT OF INTEREST

None declared.

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Abbreviations: **CP**, chronic prostatitis; **DV**, dysfunctional voiding; **PFB**, pelvic floor biofeedback; **DSD**, detrusor sphincter dyssynergia; **NIH**, National Institutes of Health; **CPSI**, CP Symptom Index; **Q_{max}**, maximum urinary flow rate; **EMG**, electromyography; **Pdet.max**, maximum detrusor pressure during the storage phase; **MUCP**, maximum urethral closure pressure; **MUP**, maximum urethral pressure.