Urol Res (2000) 28:316–318

ORIGINAL PAPER

A. Mehik · P. Hellström · O. Lukkarinen
A. Sarpola · O. Alfthan

Prostatic tissue pressure measurement as a possible diagnostic procedure in patients with chronic nonbacterial prostatitis/chronic pelvic pain syndrome

Received: 27 October 1999 / Accepted: 24 March 2000

Abstract Forty-two patients with chronic nonbacterial prostatitis (CNP) and twelve men without any urological complaints or history underwent intraprostatic tissue pressure measurement with a Stryker intracompartmental pressure monitor device. The pressures were measured under spinal anesthesia in connection with various surgical procedures. Tissue pressure was monitored at 10, 60 and120 s after an injection of 1 ml saline. Significantly ($P < 0.001$) higher intraprostatic pressure values were registered at all the three time points in the patients with CNP compared to the controls. Our study shows that patients with CNP have elevated intraprostatic tissue pressures, probably reflecting increased tissue resistance and a poor tissue microcirculation status. It seems that this method can be used as a diagnostic tool to differentiate between various causes of chronic pelvic pain in the male. The aim is to develop further this method so that it is also suitable for outpatient use.

Key words Prostatic tissue pressure · Tissue pressure measurement · Chronic prostatitis/chronic pelvic pain syndrome

Introduction

Chronic prostatitis (CP) is a common syndrome, and half of all men are said to suffer from typical symptoms during their life [1, 8, 9, 19, 20, 22, 23].

A. Mehik (✉) · P. Hellström · O. Lukkarinen
Department of Surgery, Division of Urology,
University of Oulu, 90220 Oulu, Finland
e-mail: amehik@cc.oulu.fi
Fax: +358-8-3155318

A. Sarpola
Department of Public Health Science and General Practice,
Oulu University, Finland

O. Alfthan
Professor Emeritus, Helsinki, Finland

The cause of the most prevalent, nonbacterial form of chronic prostatitis, or chronic pelvic pain syndrome (inflammatory), the name proposed by the National Institutes of Health for future use in clinical practice and research, still remains unknown [13, 24].

There are no specific symptoms or signs, other than persistent pain [12, 16, 26, 28]. Apart from pain, a large variety of functional and somatic urological problems, such as frequency, urgency, dysuria and decreased urinary flow rate, have been reported widely [1, 9, 16, 17, 20, 26, 27, 28].

We earlier described a method to measure intraprostatic tissue pressure and showed that the pressure was elevated in patients with chronic prostatitis symptoms as compared to patients with symptomatic benign prostatic hyperplasia (BPH) or controls [21].

The purpose of the present study was to further develop the method, and hence it included only patients with definite CP symptoms.

Patients and methods

Two groups of patients were included in this study. Group A had 42 patients with painful chronic nonbacterial prostatitis and no obstructive symptoms. In all, 53 measurements were performed: in nine cases the measurement was performed twice and in one case three times. This was because the recurrence of the disease necessitated a second and third treatment under spinal anaesthesia.

To exclude most of the patients with prostatic hyperplasia and obstruction, we included only prostatitis patients with prostates smaller than 40 ml that had a maximum flow of urine over 10 ml/s and a residual volume of less than 50 ml after voiding. We were well aware that this group was not homogeneous, because we did not perform any pressure-flow studies and some symptoms of obstruction could be still possible. The mean age of the patients was 52 years, range 33–79 years. The diagnosis of chronic nonbacterial prostatitis was made primarily on the basis of a typical disease history, a physical examination, laboratory tests, such as prostatic-specific antigen (PSA), urine culture, expressed prostatic secretion by Nickel's pre- and postmassage test (PPMT) [22] and transrectal ultrasonography (TRUS).The urine cultures of all patients were negative and PPMT showed more than ten leukocytes and no bacteria per high power field. Patients with fewer leukocytes than ten per power field or with bacteria were excluded.

The prostate volumes were measured by TRUS (Medical Ultrasound Scanner, Type 2001- Leopold- class I, Type B, multiplane probe Type 8551/7 MHz/, Bruell and Kjaer Medical, Denmark), and the mean volume was 23 ml, range 12–37 ml.

The PSA values in group A were: mean 2.1, range 0.5–7.0 (by using a monoclonal fluoroimmunoassay, Delphia – Dual, Wallac Oy, Turku, Finland). All the patients with a PSA value over 3.0 underwent sextant random biopsies for detecting possible cancer of prostate, despite their negative digital rectal examination (DRE) and TRUS findings.

Group B had 12 patients with with no prostatic disease and without any urological history. Fifteen measurements were taken: in three cases the measurement was performed from the both sides of the prostate gland) The mean age was 45 years, range 35–55 years. The mean volume of the prostate was 25 ml, range 17–33 ml. The mean PSA value was 2.2; range 0.3–3.2. All these patients had been admitted for hemorrhoid surgery.

Prostatic pressure was measured with a Stryker intracompartmental pressure monitor system (295–1 pressure monitor, 295–2 Quick Pressure Monitor Set: Bio Tec Instruments, Model DPM-1, Winooski, Vt, USA). This tissue pressure monitor is widely used in orthopedics to measure compartmental pressure in patients with tibial bone fracture [30,31]. The side-holed needle for the injection of sterile physiological solution was 18 G in size and 2.5 inches (6 cm) long. The monitor was calibrated before the pressure measurements, as instructed by the manufacturer.

The following method of measuring prostatic tissue pressure was used as described in our first study [21], and we now added to this two extra readings (at 60 s and 120 s). All the patients were in the lithotomy position. Spinal anesthesia was used in all cases. Some of the patients with CNP underwent transurethral needle ablation (TUNA) to control intractable pain [4]. The perineal area was washed. The correct position of the needle was confirmed by TRUS. One milliliter of sterile physiological saline solution was injected into a randomly chosen apex of the prostate. The pressure readings were recorded after 10, 60 and 120 s. All patients received prophylactic antibiotic medication 1 h before the procedure (ciprofloxacine 500 mg) [1]. There were no cases of urinary tract infection, urosepsis or any other complications after the tissue pressure measurement and all patients were discharged after the procedure in a good condition.

Statistics

Statistical analyses included a variance test and Student's *t*-test with Bonferroni's corrections.

Results

The intraprostatic tissue pressure readings in each study group are presented in Table 1.

A statistically highly significant difference was found between the patients with CNP and the controls at 10, 60 and 120 s ($P < 0.001$).

Discussion

We showed in our previous study that intraprostatic tissue pressure can be measured and we found a high pressure level in the patients suffering from chronic nonbacterial prostatitis [21]. In the initial study, we had a slightly confusing situation because the patients with CNP also showed signs of obstruction caused by BPH and all underwent TURP. In the present study, therefore, we tried to obtain maximum homogeneity of the case group by excluding all patients with obstructive symptoms. Consequently, we had a relatively homogeneous group of patients suffering from the "classical" symptoms of chronic nonbacterial prostatitis, and most significantly of all, the primary symptom of all these patients was a strong sensation of pelvic pain. We also increased the number of patients in the control group. The mean age of the patients in the control group was lower than in the case group, but their prostate volumes and PSA levels were comparable to those in the case group.

The results of the present study clearly confirmed the earlier findings of significantly elevated tissue pressure in patients with CNP. In the previous study, tissue pressure was measured only after 10 s [21]. As far as we can see, the readings taken at 10 s illustrate the elasticity and resistance of prostatic tissue. The decreasing values of tissue pressure at 60 and 120 s probably give an estimate of the prostate tissue capillary status and/or microcirculation capability and/or oncotic/hydrostatic pressure and are possibly related to Starling's forces [18, 25]. The increased tissue pressure may eventually cause pain, like any local tissue edema with the well-known pathophysiological signs and symptoms (edema, pain, cyanosis, blood circulation disturbances, hypoxia, acidosis, lesions of organ function, etc.) which are in accordance with the etiology of many other types of chronic pain in the human being [10, 11, 14, 15, 30, 31].

Chronic prostatitis is a diffuse and obscure term, but very commonly and widely used by general practitioners and naturally also by urologists. Pain in the lower abdomen and the genital region is the primary symptom in these patients, but a variety of micturitional symptoms accompany the pelvic pain. The etiology of the chronic inflammatory syndrome is still unknown, and several causes have been proposed, such as possible urine reflux into the prostatic ducts with chemical irritation of the tissue, ascending infection from the urethra or descending infection from the bladder, hematogenic or lymphogenic spreading of infective organisms, autoimmune syndrome and a false reaction of the host to microbes and/ or spermatozoa or to cellular remnants after heavy antibiotic treatment [1, 4, 8, 9, 12, 16, 20, 22, 23, 26, 28]. It also seems that sympathetic activity, causing spasms in the prostatic smooth muscles or the bladder neck, may affect microcirculation in tissue with reduced oxygenation and altered tissue metabolites [2, 6, 7, 14, 15, 16, 17, 21]. Urodynamic changes have also been shown in patients with CNP and prostatodynia [13, 17, 27].

In conclusion, in the light of our two studies, we are convinced that when a real inflammatory process with tissue edema is present long enough in the prostatic tissue (glandular ducts or stroma). This causes an elevated tissue pressure resulting in all pathophysiologically known entities (functional and/or organic disturbances) at the level of capillary microcirculation and in the nerve fibers up to the most severe physiological endpoint, i.e., possible tissue scarring with the known clinical signs (rigidity through fibrosis, occlusion of ducts with stagnation of fluids, nerve conducting

Table 1 Pressure readings (mmHg) of intraprostatic tissue with the Stryker intracompartmental device at 10 s (I), 60 s (II) and 120 s (III). Case group A, control group B. (*N* number of patients, *n* number of pressure measurements)

Group	*N*	*n*	Mean	SD	95%CI–Mean	Min	Max
I							
A	42	53	89.60*	20.7	83.9–95.3	20	149
B	12	15	44.53	17.7	34.7–54.4	13	78
II							
A	42	53	47.15*	23.2	40.8–53.5	12	103
B	12	15	24.33	9.5	14.4–34.3	12	38
III							
A	42	53	33.85*	20.5	28.8–39.5	4	95
B	12	15	13.67	5.6	7.8–19.5	7	23

*$P < 0.001$ between A and B groups

disturbances after demyelinisation of nerve fibres, etc.) [2, 4, 10, 11, 14, 16, 17].

This pressure can be measured by using our method, and a differential diagnosis between the various causes of chronic pelvic pain can be obtained. The only drawback in our pressure measurement is that the available standard equipment from Stryker, requires some form of anesthesia that may or may not influence the results of the measurements. The use of spinal anesthesia is more convenient for the patient in the measurement of prostatic tissue pressure. It also prevents this method from being used widely in the screening of outpatients.

We will continue our research in this field to make this measurement as handy and easy as transrectal biopsy of the prostate gland. We will also try to find out the possible connections between increased intraprostatic tissue pressure, age, PSA, prostate volume, duration of prostatitis symptoms and the validated prostatic symptoms scores (NIH-CPSI) [24].

References

1. Aagard J, Madsen PO (1992) Diagnostic and therapeutic problems in prostatitis. Therapeutic position of ofloxacin. Drugs Aging 2: 196
2. Ashton H (1975) The effect of increased tissue pressure on blood flow. Clin Orthop 113: 15
3. Barbalias GA, Meares EM Jr, Sant GR (1983) Prostatodynia: clinical and urodynamic characteristics. J Urol 130: 514
4. Blacklock NJ (1991) The anatomy of the prostate: relationship with prostatic infection. Infection 19[Suppl 3]: 111
5. Chiang PH, Tsai EM, Chiang CP (1997) Pilot study of transurethral needle ablation (TUNA) in treatment of nonbacterial prostatitis. J Endourol 11: 367
6. Coffey DS (1992) The molecular biology, endocrinology and physiology of the prostate and seminal vesicles In: Walsh PC, et al (eds) Campbell's urology, 6th edn. Saunders, Philadelphia, p 225
7. Crowe R, Chapple CR, Burnstock G (1991) The human prostate gland: a histochemical and immunohistochemical study of neuropeptides, serotonin, dopamine b-hydroxylase and acetyl-cholinesterase in autonomic nerves and ganglia. Br J Urol 68: 53
8. Domingue GJ Sr (1998) Cryptic bacterial infection in chronic prostatitis: diagnostic and therapeutic implications. Curr Opin Urol 8: 45
9. Domingue GJ Sr, Hellstrom WJG (1998) Prostatitis. Clin Microbiol Rev 10: 604
10. Ebbehøj N, Svedsen LB, Madsen P (1984) Pancreatic tissue pressure: techniques and pathophysiological aspects. Scand J Gastroenterol 19: 1066
11. Ebbehøj N, Borly L, Madsen P, Svedsen LB (1986) Pancreatic tissue pressure and pain in chronic pancreatitis. Pancreas 1: 556
12. Egan KJ, Krieger JL (1997) Chronic abacterial prostatitis – a urological chronic pain syndrome. Pain 69: 213
13. Executive summary: chronic prostatitis workshop, December 1995, Bethesda, MD: National Institutes of Health- National Institutes of Diabetes and Digestive and Kidney diseases, 1995: Appendix 1: 1–15
14. Gelberman RH, Szabo RM, Williamson RV, Hargens AR, Yaru NC, Minteer-Convery MA (1983) Tissue pressure threshold for peripheral nerve viability. Clin Orthop 178: 285
15. Guyton AC, Granger HJ, Taylor AE (1971) Interstitial fluid pressure. Physiol Rev 51(3): 527
16. Krieger JN (1984) Prostatitis syndromes: pathophysiology, differential diagnosis, and treatment. Sex Transm Dis 11(2): 100
17. Hellstrom WJG, Schmidt RA, Lue TF, Tanagho EA (1987) Neuromuscular dysfunction in nonbacterial prostatitis. Urology 30: 183
18. Hinghofer-Szalkay H (1978) Gewebedruck und Gewebecompliance und ihre praktische Bedeutung. Wien Med Wochenschr 7: 213
19. McNaughton MC, Barry MJ (1998) Epidemiology of chronic prostatitis. Curr Opin Urol 8: 33
20. Meares EM Jr (1992) Prostatitis and related disorders. In: Walsh PC, et al (eds) Campbell's urology, 6th edn. Saunders, Philadelphia, p 810
21. Mehik A, Hellstrom P, Lukkarinen O, Sarpola A, Alfthan O (1999) Increased intraprostatic pressure in patients with chronic prostatitis. Urol Res 27: 277
22. Nickel JC (1997) The pre and post massage test (PPMT): a simple screen for prostatitis. Tech Urol 3: 38
23. Nickel JC (1998) Prostatitis: myth and realities. Urology 53: 362
24. Nickel JC, Nyberg LM, Hennefent M, for the International Prostatitis Collaborative Network (1999) Research guidelines for chronic prostatitis: consensus report from the first National Institutes of Health International Prostatitis Collaborative Network. Urology 54: 229
25. Peters RM, Hargens AR (1981) Protein vs electrolytes and all of the Starling Forces. Arch Surg 116: 1293
26. Roberts RO, Lieber MM, Bostwick DG, Jacobsen SJ (1997) A review of clinical and pathological prostatitis syndromes. Urology 49: 809
27. de la Rosette JJMCH, Karthaus HFM, de Boo Th, Debruyne FMJ (1992) Research on "prostatitis syndromes": the use of alfuzosin (a new alpha-1 receptor blocking agent) in patients mainly presenting with micturition complaints of an irritative nature and confirmed urodynamic abnormalities. Eur Urol 22: 222
28. Stewart C (1988) Prostatitis. Emerg Med Clin North Am 6: 391
29. Tanagho EA (1992) Anatomy of the lower urinary tract. In: Walsh PC, et al (eds) Campbell's urology, 6th edn. Saunders, Philadelphia, p 52
30. Whitesides TE, Haney ThC, Morimoto K, Harada H (1975) Tissue pressure measurements as a determinant for the need of fasciotomy. Clin Orthop 113: 43
31. Witschger PM, Wegmuller M (1994) Apparative Muskeldruckmessung beim akuten und chronischen Compartmentsyndrom. Z Unfallchirurg versicherungsmed 87: 45