DIAGNOSIS AND MANAGEMENT OF PROSTATITIS

ANTHONY J. SCHAEFFER

Department of Urology, Northwestern University Medical School, Chicago, Illinois, USA

ABSTRACT

Prostatitis is a common syndrome characterized by infection, pain and/or inflammation of the prostate or surrounding tissues. The diagnosis requires a careful history, physical exam, and localization of inflammation and/or infection to the prostate. Bacterial prostatitis is uncommon and characterized by acute or recurrent infection of the bladder, which usually responds to antimicrobial therapy. Chronic pelvic pain syndrome is common and characterized by pelvic pain, no infection and varying degrees of prostate inflammation. Because the etiology is unknown, empiric therapy is utilized and only moderately successful. Asymptomatic prostatitis is detected in the course of evaluation for BPH, prostate cancer or infertility and should only be treated if the underlying condition warrants therapy.

Key words: prostate, prostatitis, inflammation, infection, prostatodynia

INTRODUCTION

Prostatitis syndrome is one of the most common entities encountered in clinical practice. It is estimated that up to 50% of adult men experience complaints of symptoms of prostatitis at some time in their lives. The symptoms of prostatitis may mimic the symptoms of bladder outlet obstruction from prostatic hyperplasia, which may further confuse the clinician. The differentiation of infectious from non-infectious prostatitis is essential and requires careful attention to details of specimen collection. New drugs have improved therapy and hold promise for better results in the future.

EPIDEMIOLOGY/CLASSIFICATION

The diagnosis of prostatitis is made at an estimated two million outpatient visits each year in the United States. Prostatitis is the most common urologic diagnosis for men under the age of fifty, and the third most common urologic diagnosis for those over fifty (1). The epidemiological literature quotes anywhere from 9-50% of all men will be diagnosed with prostatitis at some time in their life (2,3). Traditionally, prostatitis has been classified as acute or chronic bacterial, chronic nonbacterial, or prostatodynia with the latter two categories comprising roughly 90% of all prostatitis cases (Table 1). Recently, the National Institutes of Health Consensus Conference on Prostatitis developed a more specific classification scheme for prostatitis. Categories I and II (i.e. acute or chronic bacterial prostatitis) are based on symptoms and identification of bacteria in the urine or expressed prostate secretions (EPS) respectively. Chronic Pelvic Pain Syndrome (CPPS), Category III, is based on symptoms of chronic pelvic pain and Category IV refers to asymptomatic patients with coincidental finding of prostate inflammation in patients undergoing evaluation for benign prostatic hyperplasia, prostate cancer, or infertility.

DIAGNOSIS

The cornerstone of diagnosis and treatment for prostatitis remains appropriate localization of inflammation and infection to the prostate as described
## Table 1 - Traditional and NIH classification of prostatitis syndromes (modified from Stamey TA: Pathogenesis and Treatment of Urinary Tract Infections. Baltimore, Williams & Wilkins, p.344, 1980)

<table>
<thead>
<tr>
<th>Traditional Prostatitis</th>
<th>NIH</th>
<th>Pain in the Prostate</th>
<th>Evidence of Inflammation (EPS) (1)</th>
<th>Culture Positive (EPS)</th>
<th>Culture Positive (Bladder)</th>
<th>Common Etiologic Bacteria</th>
<th>Rectal Examination (Prostate)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Bacterial Prostatitis (2)</td>
<td>I</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+^3</td>
<td>Enterobacteriaceae</td>
<td>Abnormal</td>
</tr>
<tr>
<td>Chronic Bacterial Prostatitis (3)</td>
<td>II</td>
<td>±</td>
<td>+</td>
<td>+</td>
<td>+^3</td>
<td>Enterobacteriaceae</td>
<td>Normal</td>
</tr>
<tr>
<td>&quot;Nonbacterial&quot; Prostatitis (4)</td>
<td>IIIA</td>
<td>±</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>?</td>
<td>Variable</td>
</tr>
<tr>
<td>Pelviperineal Pain Prostatodynia</td>
<td>IIIB</td>
<td>±</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>Variable</td>
<td></td>
</tr>
<tr>
<td>Asymptomatic Prostatitis</td>
<td>IV</td>
<td>-</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>Variable</td>
<td></td>
</tr>
</tbody>
</table>

(1) Expressed prostatic secretion.
(2) Acute bacterial prostatitis is nearly always accompanied by bladder infection.
(3) Characterized by recurrent bacteriuria, at varying intervals up to several months, after stopping antimicrobial therapy.
(4) Chronic Pelvic Pain Syndrome

### Figure 1 - Segmented culture technique for localizing urinary infections in the male to the urethra or the prostate (ref. 4).

1st. voided 10 ml. → Mid-stream culture → Prostatic massage → 200 ml. later → E.P.S. Prostatic secrections → Voided 1st voided 10 ml. after massage.
Figure 1 - Describes the four-glass test of sequential voided urine collected both before and after prostatic massage. The urine and expressed prostatic secretion (EPS) are examined microscopically to identify inflammation and are cultured to determine the type, number and antimicrobial sensitivity of the pathogens.

To define prostatitis, the degree of inflammatory changes that can be found in normal prostatic fluid must first be determined. Available data suggest that white blood cells are rarely present in normal prostatic fluid. Schaeffer et al. (5) studied 119 consecutive patients with no history, symptoms, or physical findings (excluding prostatic fluid evaluation) of urinary tract inflammation, normal prostate gland by digital examination, and fewer than 10 white blood cells per high-power field in the first 10 ml of voided urine and no, or insignificant, growth on urine culture (Figure-2). Of these patients, 31 were judged to have no urologic disease and they had prostatic fluid containing 0.7 ± 0.41 white blood cells per high-power field (mean ± standard error of mean), and 88, with a variety of noninflammatory urologic diseases, had 3.8 ± 0.83 white blood cells per high-power field in the prostatic fluid. There were two white blood cells or less per high-power field observed in 97 percent of the patients with no urologic disease and in 75 percent with noninflammatory urologic disease and normal prostate glands by digital examination. Only 13 of the 119 patients in these two groups had ten white blood cells or more per high-power field. Blacklock (6) and Anderson & Weller (7) have reported similar results. It appears therefore that clinically significant inflammation is present when prostatic fluid contains ten or more white blood cells per high-power field. The white blood cell count in prostatic secretions also rises significantly in healthy men for several hours after sexual intercourse and ejaculation.

Acute bacterial prostatitis is diagnosed by identification of bacteria in the urine of a patient with an acute, septic urinary tract infection. Chronic bacterial prostatitis is diagnosed by culture of any uropathogen in the EPS in the absence of urethral pathogens (VB1 urine culture), or a cultured EPS or VB3 bacterial count at least ten times greater than cultured VB1 or VB2 urine.

Chronic pelvic pain syndrome is diagnosed by the complaint of pelvic pain for a total of 3 months within the last 6 months. Patients are excluded if they have any of the conditions listed on Table-2.

Patients with asymptomatic prostatitis are identified by elevated levels of white blood cells in the EPS (for example to determine if prostatic inflammation is associated with an elevated prostate specific antigen level) or ejaculate (in a patient being evaluated for infertility).

PATHOGENESIS

The pathogenesis of bacterial prostatitis is unknown. Presumably ascending urethral infection after a vaginal or rectal inoculation of the urinary meatus during sexual intercourse plays an important role. As such, the prostatitis may represent an extension or a complication of urethritis. Evidence of reflux of urine into the prostatic ducts has come from crystallographic analysis of prostatic calculi where constituents found in urine, but foreign to prostatic secretions, were discovered. Direct bacterial inoculation from the rectum or hematogenous or lymphatic spread to the prostate may also occur.
Prostatitis

Bacterial prostatitis is caused by the usual uropathogens. Escherichia coli and other members of the Enterobacteriaceae family, such as Klebsiella and Proteus species, predominate. Pseudomonas and Enterococcus faecalis are less common. Mixed infections involving two or more strains or classes of microorganisms are not uncommon (8).

The role of gram-positive bacteria, other than enterococci, as pathogens in prostatitis is controversial. Although uncommon, chronic bacterial prostatitis caused by Staphylococcus aureus has been documented, usually as a consequence of a hospital-acquired catheter-associated infection. The etiologic role of other gram-positive bacteria (e.g. Staphylococcus epidermidis and saprophyticus, micrococci, non-group D streptococci, and diphtheroids) is doubtful. These organisms are considered skin inhabitants, existing as urethral commensals rather than true pathogens. Furthermore, they do not cause relapsing recurrent urinary tract infections in untreated patients.

Transrectal ultrasonography detects prostatic calculi in 75 percent of middle-aged men and nearly 100 percent of elderly men (8). Moreover, transrectal ultrasonography is capable of demonstrating stones in as many as 70 percent of men who have no other radiologic signs of prostatic stones (9). Prostatic stones usually are tiny and occur in small clusters; at times; however, the stones may be large and extensively involve the prostate. Bacteria do not usually colonize these calculi and cause no harm provided they remain confined to the prostate. In certain men with prostate calculi and recurrent urinary tract infections, however, the stones have been shown to become colonized and to be the source of bacterial persistence (10,11). Similar to colonized renal calculi, prostatic stones can be permeated with bacteria that are protected from the action of antimicrobial agents. Because prostatic calculi are exceedingly common in adult men, and because these stones can become colonized during or after formation, it is plausible that unrecognized colonized stones are often responsible for the failure of antimicrobial therapy to cure chronic bacterial prostatitis.

The pathophysiology of inflammatory CPPS may involve the intraprostatic reflux of urine inciting a chemical inflammatory response (12); however, other factors such as an autoimmune mediated process may also be responsible (13). Past studies implicated Ureaplasma, Mycobacterium, or, most commonly, Chlamydia as pathogens responsible for the development of inflammatory CPPS, yet recent well designed studies have found no evidence that these pathogens are involved in the etiology of inflammatory CPPS (14,15). The common urodynamic findings in patients with inflammatory CPPS include synergistic voiding (normal relaxation of the pelvic floor musculature during the act of voiding) with incomplete relaxation of the bladder neck and prostatic urethra. The urinary peak flow is usually decreased with the urethral pressure profile demonstrating an increased maximal urethral closing pressure at rest (16). It

Table 2 - Exclusion criteria for chronic pelvic pain syndrome

- Prostate, Bladder or Urethral CA
- Unilateral Orchalgia (testicular pain), without pelvic symptoms
- Active Urethral Stricture
- Any Neurological disease/disorder affecting the bladder
- Inflammatory Bowel Disease
- Intravesical BCG
- TURP (transurethral resection of the prostate)
- TUIP (transurethral incision of the prostate)
- TUIBN (transurethral incision of the bladder neck)
- TUMT (transurethral microwave therapy)
- TUNA (transurethral needle ablation)
- Balloon Dilation
- Any other prostate surgery
- Cryotherapy
- Thermal Therapy
is felt that the high pressure generated from smooth muscle spasm of the bladder neck and prostatic urethra during the act of voiding leads to intraprostatic reflux of urine.

The pathophysiology of non-inflammatory CPPS involves a poorly understood chronic myofascial pain complex that may be related to a tension myalgia of the pelvic floor musculature. Pathologically there are no specific findings and no evidence of inflammation in the prostatic gland. Accordingly, urodynamic findings are nondistinguishable from those of patients with inflammatory CPPS.

The pathophysiology of asymptomatic prostatitis is unknown and probably multifactorial. Factors that may cause symptoms of prostatitis (IIIA), e.g., infectious agents that are difficult to culture or an autoimmune response, may play a role in the pathogenesis of asymptomatic prostatitis.

MANAGEMENT

I - Acute bacterial prostatitis

Acute bacterial prostatitis usually presents in a relatively young man with dramatic onset of fever, malaise, low back or perineal pain, and myalgia for several days prior to onset of symptoms of urinary frequency, dysuria, urgency, and varying degrees of bladder outlet obstruction. Palpation usually but not always reveals a tender, hard, irregular prostate that is warm to touch. Prostatic massage should be avoided because of the risk of bacteremia, but gentle pressure on the prostate may induce copious amounts of purulent prostatic secretions. Since cystitis usually accompanies acute bacterial prostatitis, the responsible bacterial pathogen can be isolated from bladder urine. Escherichia coli and other members of the Enterobacteriaceae family account for 95% of cases; Pseudomonas and enterococcus (Streptococcus faecalis) microorganisms are less common. Serum chemistry reveals an elevated white blood cell count.

General Therapy

Most patients are quite toxic and may require admission to the hospital. Supportive measures such as analgesics, antipyretics, hydration, bed rest, and stool softeners should be instituted. If the patient cannot urinate, urethral catheterization may further exacerbate the prostatic inflammation and lead to complications such as acute epididymitis. Therefore use of a small urethral catheter or a suprapubic tube are advised.

Antimicrobial Therapy

Patients with acute bacterial prostatitis usually respond dramatically to antimicrobial drugs that normally do not achieve therapeutic levels in prostatic fluid. This is because the inflamed prostate permits diffusion of drugs from the bloodstream to the prostate. If the patient is septic, we obtain blood cultures and administer gentamicin sulfate or tobramycin sulfate, 3 to 5 mg per kg of body weight per day, divided into three intramuscular or intravenous doses, plus ampicillin, 1 gram administered intravenously every 6 hours or an intravenous fluoroquinolone such as ciprofloxacin (Cipro) 200-400 mg every 12 hours or ofloxacin (Floxicin) 200-400 mg every 12 hours. If the patient can take oral antimicrobial agents, a fluoroquinolone such as ciprofloxacin (Cipro) 500 mg every 12 hours, ofloxacin (Floxicin) 300 mg every 12 hours, norfloxacin (Noroxin) 400 mg every 12 hours, lomefloxacin (Maxaquin) 400 mg every day or enoxacin (Penetrex) 400 mg every 12 hours can be utilized. Although two weeks of therapy is probably adequate, four weeks is preferred to ensure that all bacteria are eliminated from the prostate.

Operative Therapy

Surgical intervention is generally not indicated for acute bacterial prostatitis. Prostatic abscess is a rare complication that should be suspected in patients whose symptoms and clinical courses do not respond to appropriate antimicrobial therapy. If a large, localized, tender, fluctuant mass is palpated within the prostate, ultrasound or computed tomography and perineal or transurethral drainage should be performed. If the prostate gland is hard, several months may be required before it returns to normal consistency. Granulomatous prostatitis in the absence of tuberculosis and rare mycotic infection of the prostate is a histologic stage of resolving acute bacterial prostatitis, which is usually detected as a local area of prostatic induration suspicious of carcinoma. Ex-
except for exclusion of carcinoma, no special therapy is warranted.

II - Chronic Bacterial Prostatitis

Chronic bacterial prostatitis is characterized by relatively asymptomatic periods in between episodes of recurrent bacteriuria. The infection is caused by small numbers of bacteria in the prostatic fluid and is very difficult to eradicate with most antimicrobial therapy. The common pathogens are the Enterobacteriaceae and species of Pseudomonas. Enterococcus (Streptococcus faecalis) is also a definite cause of chronic bacterial prostatitis. Other gram-positive organisms have been implicated much less frequently and rarely cause recurrent bacteriuria. Mixed infections involving two or more strains or classes of microorganisms are uncommon.

Hematospermia and painful ejaculation occur infrequently. Prostate examination is nondiagnostic.

General Therapy

Appropriate oral antimicrobial therapy usually controls the acute episode of cystitis. Hot sitz baths and antipyretics are also helpful. Septic episodes requiring hospitalization and parenteral therapy occur rarely.

Antibacterial Therapy

Cure of bacterial prostatitis appears to correlate best with the level of antimicrobial drug in the prostatic fluid rather than its level in serum or prostatic tissue. Trimethoprim and the fluoroquinolones do diffuse into prostatic fluid and have the best documented success in curing chronic bacterial prostatitis due to susceptible pathogens. Long-term therapy (8-12 weeks) appears to be more effective than short-term therapy (2 weeks) in achieving bacteriologic cures. The following recommendations are made for treatment of nonazotemic men with documented culture susceptible pathogens infecting the prostate:

1)- TMP-SMX (Septra or Bactrim), one double-strength tablet (160 mg of TMP and 800 mg of SMX) orally twice daily for 12 weeks; or
2)- Trimethoprim (Proloprim or Trimpex), two tablets (100 mg each) orally twice daily for 12 weeks; or
3)- Ciprofloxacin (Cipro), 250 mg every 12 hours for 4 weeks; or
4)- Enoxacin (Penetrex), 400 mg every 12 hours for 4 weeks; or
5)- Lomefloxacin (Maxaquin), 400 mg every day for 4 weeks; or
6)- Norfloxacin (Noroxin), 400 mg every 12 hours for 4 weeks; or
7)- Ofloxacin (Floxin), 200 mg every 12 hours for 4 weeks.

Specific therapy must always be tailored to meet the individual patient’s needs and drug tolerance (see also the manufacturer’s official directive in the use of these agents.) Patients not cured by antimicrobial therapy can be kept comfortable and abacteriuric by use of continuous low-dose suppressive daily therapy with an appropriate oral antimicrobial agent such as nitrofurantoin (50 mg capsule) or trimethoprim-sulfamethoxazole (TMP-SMX; a single, regular strength tablet each day). Bacteriuria will usually recur following cessation of therapy.

Operative Therapy

Transurethral resection of the prostate is the only alternative, short of radical prostatectomy, for surgical management of bacterial prostatitis. However, transurethral prostatectomy can be curative only if all foci of infected tissue and calculi are removed. Since most inflammation in chronic prostatitis occurs in the periphery of the gland and all the ducts from the peripheral zone empty into the urethra distal to the verumontanum, radical transurethral resection with removal of all foci of infected stones and tissues is difficult to achieve and carries a high risk of urinary incontinence. Approximately one-third of patients with well-documented bacterial prostatitis have been cured by this technique.

III - Chronic Pelvic Pain Syndrome (CPPS)

Diagnosis and treatment of this syndrome is controversial. CPPS (in the clinical setting, “prostatitis”) is most frequently synonymous with pain in the pelvic or perineum frequently associated with urinary urgency frequency dysuria or poor urine flow. The syndrome has been called prostatodynia because the symptoms have been judged to be of prostatic origin.
However, many patients are probably unable to differentiate prostatic pain from pelvic or perineal symptoms. CPPS therefore is a more appropriate term to describe this condition. Evidence of inflammation in the EPS is variable suggesting that different factors (e.g., infections, inflammation, musculoskeletal) may contribute to the etiology and pathogenesis of the syndrome. It is not surprising therefore that a wide variety of agents have been used to treat patients with CPPS and that the outcomes are so varied.

III.A - Inflammatory CPPS (Nonbacterial Prostatitis)

Nonbacterial prostatitis is about eight times more common than bacterial prostatitis. The clinical significance of evidence for prostate inflammation, particularly in asymptomatic patients, has been questioned. However, recognition that identifiable groups of patients (such as those with infertility, i.e., category IV) have significant increased leukocyte counts indicates that nonbacterial prostatitis may be indicative of an underlying disease.

General Therapy

Since the etiology is unknown, treatment is empiric and often unrewarding. Two important conditions should be considered in a differential diagnosis, interstitial cystitis and carcinoma in-situ of the bladder. Thus for selected patients it may be reasonable to obtain urine specimens for cytology and perform endoscopic evaluation under anesthesia (17). Some experts advocate that an initial trial of antimicrobials is warranted for inflammatory CPPS. The antimicrobial regimen consists of an initial 28-day course of a fluoroquinolone or a six-week trial of TMP/SMX as upwards of 40% of these patients will demonstrate clinical improvement (18). This initial trial of antimicrobials is supported by the fact that men with inflammatory CPPS demonstrate a higher incidence of significant bacterial infection on prostate biopsy culture than noninflammatory CPPS patients (19). If Chlamydia or Ureaplasma species are likely causes of urethritis associated with prostatitis, we recommend a clinical trial with minocycline (Minocin), 100 mg orally twice daily for 10 days. Unless the response is favorable, further treatment is probably not indicated. Continued empiric administration of other antimicrobial drugs is almost invariably ineffective and engenders considerable expense, anxiety, and dissatisfaction. Instead, efforts should be made to educate the patient with a frank discussion about the unknown and probably noninfectious etiology of the condition and efforts to relieve symptoms. Videourodynamics may be obtained in order to exclude other diagnoses such as primary bladder neck obstruction and pseudodyssynergia of the external urethral sphincter, both of which are common voiding dysfunction conditions in men less than fifty years of age misdiagnosed with CPPS (20). Several treatment options may be employed for inflammatory CPPS including pharmacological alpha blockade of the bladder neck, nonsteroidal anti-inflammatory agents, warm sitz baths, prostatic massage, biofeedback and transurethral microwave thermotherapy of the prostate. We generally recommend hot sitz baths for symptomatic flare-ups. Many patients obtain symptomatic relief after short courses of anti-inflammatory agents such as ibuprofen (Motrin), 400 to 600 mg orally three or four times daily. Patients with obstructive voiding symptoms may benefit from therapy with an alpha-blocker such as terazosin (Hytrin) 5-mg orally once daily, doxazosin (Cardura) 4-mg orally once daily, or tamsulosin hydrochloride (Flomax) 0.4 mg once daily. This treatment has been reported to achieve a clinical success rate of approximately 58% (21), yet better prospective data and the use of standardized diagnostic criteria need to be employed in future studies. All patients should undergo urodynamics prior to the initiation of treatment in order to confirm the diagnosis of CPPS. Concomitant use with beta-blockers or verapamil will increase the sensitivity of alpha 1 induced postural hypotension. The antihypertensive effect of clonidine is decreased with the use of an alpha 1 blocker. The cardiovascular adverse effects of hypotension and palpitations occur in approximately 4% of all patients. Headache and dizziness may occur in 5-9% of patients on long term therapy. Irritative voiding symptoms may respond to therapy with anticholinergics, such as propantheline (Pro-Banthine) 15 mg orally, four times daily or oxybutynin chloride (Ditropan), 5
mg orally, two or three times daily. Oxybutynin chloride ( Ditropan) is an anticholinergic agent that inhibits the muscarinic action of acetylcholine on smooth muscle. Clinically, oxybutynin exhibits an antispasmodic effect on detrusor smooth muscle thus diminishing involuntary bladder contractions associated with the CPPS in some patients. Normal starting dosage is 5 mg every six hours for symptomatic relief with titration to a dose of 10 mg. Contraindications include hypersensitivity to the drug, acute closure glaucoma, urinary retention, and intestinal atony. The most common side effects are dry mouth, flushing, and headache.

Tolterodine tartrate (Detrol) is a novel muscarinic receptor antagonist that inhibits detrusor smooth muscle in similar fashion as oxybutynin. The apparent advantage of this medication over oxybutynin is twofold: 1) early studies reveal it is as efficacious as oxybutynin, yet better tolerated with less side effects because of its higher selectivity for bladder detrusor smooth muscle (22), and 2) it is dosed just two times a day as compared to oxybutynin which is dosed four times a day. Recommended starting dose is 1 mg with titration to 2 mg as needed. Contraindications to the drug are the same as those for oxybutynin. The cost of tolterodine may be considered a disadvantage, as it is not generic with retail cost approaching $1 per tablet.

Occasionally, therapeutic prostatic massage and dietary restrictions regarding the use of alcoholic beverages, coffee, and spicy foods are beneficial.

Transurethral microwave thermotherapy is reserved for treatment of refractory inflammatory CPPS patients. Limited success in symptom relief has been reported with this modality. Standard Procedure: transurethral placement of a microwave applicator into the prostatic urethra is performed under direct vision, and the periurethral prostatic tissue is heated to a temperature of 45-60°C for approximately one hour. The tissue hyperthermia induced by the microwaves (frequency of 915-2450 MHz) theoretically ablates sensory neural components of the prostate thus explaining symptom relief in some patients (23). Further work is needed to elucidate the effects of thermotherapy on prostatic tissue.

III.B - Noninflammatory CPPS (Prostatodynia)

The term prostatodynia has been suggested for men with symptoms that mimic prostatitis, especially “prostatic pain,” but who have negative cultures and no evidence of inflammation in the expressed prostatic secretions. Although some of these symptoms may be of prostatic origin, the term is misleading if the patient’s assessment of the etiology of his discomfort is inaccurate. Musculoskeletal abnormalities are probably responsible for much of the symptomatology. Some patients with this syndrome have apparent functional obstruction in the bladder neck and prostatic urethra. These patients may respond favorably to therapy with an alpha-blocking agent such as those listed above, once daily at bedtime. Other patients with apparent tension myalgia of the pelvic floor respond best to treatment with warm sitz baths diathermy, muscle relaxants, and physiotherapy, with or without the use of diazepam (Valium), 5 mg orally three times daily. Some patients have emotional disturbances that benefit from psychiatric consultation.

There is no evidence to support alcohol intake, caffeine, or tobacco use as risk factors for prostatitis. However, animal studies suggest that a daily intake of dietary soy protein may play a protective role against the development of prostatitis (24).

Prostatic massage may be utilized for the CPPS patient with a congested prostate from sexual inactivity. Firm digital pressure applied to the prostate two to three times each week has been anecdotally proven to relieve symptoms in some patients. Biofeedback has been used for various dysfunctional voiding disorders with success rates reported as high as 70% (25). Biofeedback involves training the patient to selectively contract and relax the muscles of the pelvic floor on a voluntary basis in order to eventually use this technique for the interruption of pelvic myofascial pain attacks associated with CPPS. In addition, the patient undergoes a bladder-training program that involves a progressive increase in the interval between voids to no less than four hours. Biofeedback involves patient recognition and eventual correction of the symptoms associated with CPPS. This appears to be a promising treatment protocol for patients with CPPS, yet
prospective studies involving pre- and post treatment urodynamics, post void residuals, and voiding diaries are needed.

The sequelae of CPPS include the potential for male factor infertility, and an association with psychiatric illness that should not be overlooked by the physician. Treatment refractory CPPS has a proven sickness impact on quality of life similar to that of patients with a history of myocardial infarction, angina, and Crohn’s disease (26). Berghuis et al. (27) suggest that as many as 43% of CPPS patients suffer from some form of significant psychological distress from their condition.

IV - Asymptomatic Prostatitis

Therapy for asymptomatic prostatitis is only recommended if the cofactor e.g. infertility, elevated prostate specific antigen is judged to be potentially caused by inflammation. Empiric antimicrobial therapy or anti-inflammatory drugs listed above may reduce inflammation and resolve the clinical problem.

CONCLUSION

Prostatitis is a common problem with multiple etiologies. Less than 10% of men have bacterial prostatitis. Acute bacterial prostatitis presents as an acute, serious event and usually responds to prompt antimicrobial therapy. Chronic bacterial prostatitis is always associated with recurrent urinary tract infections. Bacterial localization studies and long term antimicrobial therapy can cure two-thirds of these patients. Chronic Pelvic Pain Syndrome is the most common type of prostatitis and most likely is caused by a variety of autoimmune, inflammatory and muscular disorders of the prostate and other pelvic organs. Most current management is empiric and of limited benefit but research suggests that exciting progress and therapies are forthcoming.

REFERENCES


Correspondence address:
Anthony J. Schaeffer, M.D.
Dept. of Urology, Univ. Northwestern
Tarry Building 11-715
303 East Chicago Avenue
Chicago, Illinois, USA, 60611-3008
Fax: ++ (1)(312) 908-7275
E-mail: ajschaeffer@nwu.edu

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