Management of Prostatitis

Dr. Richard LO
MD(UCLA), MCPS(Manitoba), FCSHK, American Board of Urology, FHKAM(Surgery)
Consultant Surgeon, Pedder Clinic

The term “Prostatitis” has been used to describe a whole constellation of genitourinary symptoms in men. Men with pain or discomfort from the subumbilical area to the groin, with or without voiding symptoms, are all labelled as having Prostatitis. This is a result of poor understanding of the aetiology of these painful syndromes, and confusion over bacterial infection as a real aetiology that caused these symptoms.

Prostatitis is a rather common ailment amongst men, with the prevalence estimated to be 9 to 16% of the male population, or 3.8/1000 person-years. At any one time, 2 to 10% of men will experience prostatitis-like symptoms. Of those afflicted, 1 in 4 will have more than one episode per year, with 16% of the patients having persistence of their symptoms. It is estimated that in some countries, prostatitis comprises 3 to 12% of a urologist’s outpatient workload.

The National Institute of Health classified prostatitis into four main categories:

- Category I – Acute Bacterial Prostatitis
- Category II – Chronic Bacterial Prostatitis
- Category III – Chronic Pelvic Pain Syndrome (CPPS)
- Category IIIA – Inflammatory CPPS
- Category IIIB – Non-inflammatory CPPS
- Category IV – Asymptomatic Inflammatory Prostatitis

Categories I and II are associated with bacterial infections of the urine, while the aetiology of the latter two are obscure. Microbiological, immunological, neurological, inflammatory and psychological causes, singly or in combination, have been implicated but never conclusively proven in the CPPS group. Statistically, Categories I and II account for no more than 10-15% of the group.

The *sine qua non* in the diagnosis of bacterial prostatitis is a documented bacteriuria. Acute bacterial prostatitis is heralded by high fevers, chills, perineal pain, severe dysuria and other lower urinary tract symptoms (LUTS). Digital rectal examination will show an enlarged and exquisitely tender prostate. The patient is acutely ill and appears toxic. Urine microscopy will show pyuria and the culture will reveal significant bacteriuria, usually a coliform of intestinal origin. Management of acute bacterial prostatitis consists of empirical intravenous antibiotics, usually an aminoglycoside with a third-generation cephalosporin or carbopenam if the local sensitivity profile so dictates. With deferescence and clinical improvement after IV antibiotics, the patient can be switched to oral antibiotics according to the sensitivity profile of the particular organism, for another three to four weeks. This is the only opportunity to avert an acute bacterial prostatitis progressing to the chronic version.

In the normal, non-inflamed state, the blood-prostate barrier blocks diffusion of most serum contents from entering the prostate. In acute bacterial prostatitis, however, this barrier to antibiotics diffusion is broken down because of the intense inflammation, and provides the only scenario in which bacteria in the prostate could be successfully eradicated.

The single most important feature in the diagnosis of chronic bacterial prostatitis is the recognition of a chronic, relapsing bacterial cystitis, with the same organism and identical sensitivity pattern. As the bacteria are now resident in the prostate and are ‘protected’ by this impregnable blood-prostate barrier layer, they will not be exposed to any antibiotic, and therefore there will be no selection of resistant organisms. The patient may have recollection of an episode resembling acute bacterial prostatitis, but the recurrent pattern is a helpful hint to the astute physician. The symptoms, however, are much less pronounced, and usually consist of dysuria, frequency and urgency only.

Further documentation of the bacteria originating from the prostate is needed to cinch the diagnosis. The 4-glass segmental urine culture described by Meares and Stamey, (Diagram 1) compares the bacterial colony counts in the midstream urine versus those in the prostatic fluid obtained by prostate massage (EPS), and the washout portion from the urethra after massage (VB3). If there is a two-log increase (EPS/VB3 >> VB2), and the organism is identical to the one recovered from a midstream culture whilst infected, the diagnosis of chronic bacterial prostatitis is confirmed. Treatment of chronic bacterial prostatitis, once proven, is actually
the simplest: as the organism and sensitivity profile are known. A midstream urine is sent for culture if practical. The patient is empirically prescribed a 3 to 4-day course of a sensitive antibiotic, which should be sufficient. In the event the patient is still symptomatic after the antibiotic treatment, the possibility exists that a different offending organism had actually caused a de novo cystitis and it was not a recurrence of the original chronic bacterial prostatitis. (Remember there are over 150 serotypes of E. coli!)

The main reason why patients do not improve after antibiotics is that it was not a bacterial infection after all. Those who fall into Category III or Chronic Pelvic Pain Syndrome (CPPS) have similar but yet dissimilar symptoms from their bacteria-infected cohorts. Groin, perineal, suprapubic and low back pain are common complaints. Lower urinary tract symptoms are sometimes associated features. The predominant symptom in all these patients, however, is pain. A single course of antibiotics is widely prescribed on first presentation of men with LUTS/CP/CPPS, and is acceptable practice in a primary care situation. If symptoms persist, it is mandatory to reassess the urine culture for bacteriuria, or at least microscopically for pyuria. (Clinical ‘improvement’ after antibiotics, unfortunately, does not necessarily establish the diagnosis of bacterial infection.) In the tertiary referral setting, it is not unusual to see patients who were prescribed a protracted course of multiple antibiotics, urinary analgesics and anxiolytics.

When a patient with suspected CPPS is referred, proper bacteriological studies with segmental urine cultures (after an appropriate washout period), and microscopic examination of the expressed prostatic secretion should be performed, to definitively rule out any bacterial origin. If the cultures are negative, these patients are treated symptomatically and empirically. Further use of antibiotics is futile.

α-adrenergic blockers have been shown, in randomised placebo-controlled studies, to have a modest benefit in selected patients. The subset who improves is usually those with recent onset of moderate to severe symptoms, and the treatment duration should be six weeks or more. A similar line of reasoning is used to recommend the use of skeletal muscle relaxants, but the response is variable and not supported by Level I evidence. Anti-inflammatory agents have been used, with limited success, but the analgesic effect may provide symptomatic relief to some. The use of hormonal treatment is based on the conviction that the prostate is the culprit. Hormones should not be used in those without voiding type of LUTS.

Physical therapy is used to supplement the shortcomings of medical treatment in CP/CPPS. These consist of prostate massage (long since abandoned by mainstream Urology), myofascial trigger point release, acupuncture and biofeedback. None of these have any proven efficacy or long-term benefits in large-scale, well-designed studies. Similarly, it is extremely hazardous to recommend surgery or even minimally-invasive therapies like microwave thermotherapy, as the efficacy of these modalities remains unproven, and should only be used as a last ditch effort. As the chief complaint and presenting symptoms are pain, pain amelioration is the main treatment goal in this group of patients.