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Review Article

Recent Developments in Chronic Prostatitis and Pelvic Pain Syndrome

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Abstract

Chronic prostatitis or chronic pelvic pain syndrome (CP/CPPS) is a versatile, clinical condition, which poses challenges for both patients and clinicians. The aim of this review is to survey the literature for recent developments in the etiology, diagnosis and treatment of CP/CPPS.

There are many theories regarding the development of CP/CPPS but they require further investigation. To check for chronic bacterial prostatitis, bacteria are cultured from expressed prostatic secretions whereas CP/CPPS is a bacterial condition. CP/CPPS commonly presents with voiding dysfunction, sexual dysfunction and pain in the pelvic region. Diagnosis is mainly based on clinical presentation. The use of questionnaires enhances conclusions and the assessment of quality of life.

Treatment is often challenging and demands a multidisciplinary approach as the etiology may involve both biological and psychological factors. Medical therapy usually involves 4 to 6 weeks of antibiotics. Non-steroidal anti-inflammatory drugs (NSAIDS) and alpha-blockers are often being used in combination with innovative minimally invasive manipulations, physical therapy and psychological therapy. There is no known cure for CP/CPPS. Management of the symptoms is often a frustrating struggle for both patients and clinicians, requiring a great deal of patience.

INTRODUCTION

Chronic prostatitis remains a mysterious phenomenon for many clinicians and practitioners. Prostatitis is a common condition worldwide, affecting approximately 2-10% of adult men; the majority are diagnosed with chronic prostatitis or chronic pelvic pain (CP/CPPS) rather than acute or chronic bacterial prostatitis [1,2].

The term 'prostatitis' is often confusing because in 1994 the National Institute of Health (NIH) used it to refer to four clinically distinct and unrelated conditions. This was based upon clinical syndromes which replaced the old diagnostic schemes that included terms such as 'non-bacterial prostatitis' and 'prostatodynia' [3].

In brief, chronic bacterial prostatitis (Class II) is associated with bacteria, which are identified after prostate massage. They frequently appear following a recurrent urinary tract infection (UTI) in men between and/or after partially treated acute prostatitis (Class I). Acute bacterial prostatitis (Class I) is beyond the scope of this review. A characteristic of CP/CPPS (Class III) is pelvic pain with no clear identifiable pathology that is often associated with various painful symptoms–including pain and discomfort in the lower abdomen or suprapubic area, genitalia, lower back and thigh, perineum penis and testicles. Lower urinary tract symptoms (LUTS), ejaculatory pain and erectile dysfunction are also often associated with CPPS. The presence or absence of white blood cells (WBC) in urinary samples differentiate class IIIa and class IIIb respectively. Class IV refers to the asymptomatic inflammatory prostatitis as defined by histopathologic examination [3].

Due to the frustration that CP/CPPS creates for both clinicians and patients, this review focuses on current assessment and management strategies regarding CP/CPPS and discussion of the etiologic factors of this challenging syndrome.

MATERIALS AND METHODS

The Cochrane Library, Pubmed, and EBSCO databases were surveyed find English literature from January 1990 to May 2017 on the etiology, assessment and management of CP/CPPS. The following terms were used with or without the combination of

the previous topics: *prostatitis*, *chronic nonbacterial prostatitis*, *chronic abacterial prostatitis*, chronic *pelvic pain syndrome*. We limited the search to full text articles, which included reviews, clinical trials, and observational studies.

The pathophysiology of CP/CPPS

Various theories have been proposed to explain CP/CPPS. It is likely that this syndrome is characterized by multifactorial, potential etiologies from a spectrum of causative mechanisms or a cascade of events triggered by one or more initiating factors as described in the following sections.

Infectious

The exact cause of CP/CPPS remains undefined and various theories have been developed ~~by~~ using animal models and epidemiological research [4].

CP/CPPS may stem from a bacterial origin; however, the majority of studies have not been able to identify a single bacterium responsible for it. Most experts believe that inflammatory and non-inflammatory CP/CPPS are both noninfectious disorders. Many attempts to find an "uncultivable bacteria" by 16S DNA technology have failed to identify a 'hidden' pathogen in urine or expressed prostatic secretions (EPS) [5–8].

A recent study by Nickel, et al., analyzed the urine of 110 CP/CPPS patients and 115 controls using the Meares-Stamey four-glass lower urinary tract localization tract (Figure 1). They found a higher incidence of *Burkholdriacenocepacia* in the VB1 when compared to controls. However, its etiologic significance remains unclear [9].

Noninfectious

Neurological factors: Growing evidence supports a significant neuropathic component to CP/CPPS as the prostate is abundant with autonomic innervation from the sympathetic and parasympathetic nervous system. It is only recently that researchers have begun to understand the complexity of the overlapping neuropathways of pelvic organs (10). The nociceptive and inflammatory sensation of pain results from the activation of C and A lambda neural fibers respectively. The nociceptive type of pain is a response to chemical or thermal stimuli which indicates danger or damage, whereas the inflammatory response results from the interaction between pro-inflammatory cytokines and the nervous system which causes a sensitization phenomenon and may lead to pain that has no clear stimulus. Sensitization of pain can be mediated via the peripheral nervous system or the central nervous system (dorsal horn of the spinal cord).

The peripheral sensitization theory postulates an inflammation trigger, which initiates an activation cascade of nociceptive neurons by reducing the activation threshold thereby causing pain. This condition creates recurrent, persistent and expansion of the of pain even in the absence of a specific stimulus [11].

Central sensitization is considered to play an important role in chronic pain. It is thought that the chronic signals from damaged peripheral nerves result~~s~~ in abnormal regeneration of the neural pathway within the dorsal horn of the spinal cord and thereby causes sensitization in the central nervous system.

The pain that is associated with CP/CPPS is similar in many respects to neuropathic pain, which is defined as pain of a neural origin. In CP/CPPS the trigger of this type of pain is thought to be a result of injury to the peripheral nervous system, which is partially attributed to an excitotoxic state (dysfunctional pain) [11].

Endocrine factors: Another theory is that sex hormones may play an important role in the development of CPPS. Experiments on laboratory rats show that estrogen induces autoimmune inflammation. The exact mechanism is still unknown; however the increase in the transcription of pro-inflammatory genes was observed in the presence of estrogen [12,13].

Immunological factors: Animal models as well as human studies suggest that noninfectious inflammation might be secondary to immunologically mediated inflammatory response caused by some unknown antigens or even an autoimmune process.

The autoimmune theory is a prominent one behind the etiology and pathogenesis of CP/CPPS. An in-vitro observation made by Casas-Ingaramo et al., suggests that T-cells sensitized by the male accessory gland have been shown to expand and differentiate by themselves in response to prostate antigens only [14]. In humans, some observations an autoimmune process as well. For example, markers for cytotoxic T-cells were found in the expressed prostatic excretion (EPS) of men with CP/CPPS. This cell type is not normally present in the immune process involved in antimicrobial "defense" but is more consistent with autoimmune inflammation or secondary remodeling injury tissue [15]. Another study which examined the presence of regulatory T-cells marker (FOXP3) in CP/CPPS individuals as compared to controls, found a reduced FOXP3 expression in the CP/CPPS group; this might be associated with human immune disorders [16].

Finally, a 9% incidence of intraprostatic spermatozoa was noted at autopsy [17]. This observation suggests that a non-prostatic substance such as spermatozoa may be present in the prostate, explaining an eventual (not of prostatic origin) target antigen causing the autoimmune response.

Psychological factors: A psychological etiology of CP/CPPS should be seriously considered. Psychological factors include personality variables such as clinical depression, panic disorders, anxiety, poor social interaction, and poor coping skills that manifest as a magnification of any situation that may worsen pelvic pain [18]. Psychological stress, which is believed to be frequently associated with CPPS, creates a vicious cycle which further increases the perception of pain [19]. In a large case control study, the most common psychologic finding among men with CP/CPPS as compared to controls, was clinical depression and panic disorders. This finding highlights their importance [20]; therefore, clinicians and other therapists involved in the treatment of patients with CP/CPPS should not ignore that many of these patients have a stressful personality with a tendency to catastrophizing their situations. It is also possible that suffering from such a challenging disease may be the cause rather than the result of these personality changes.

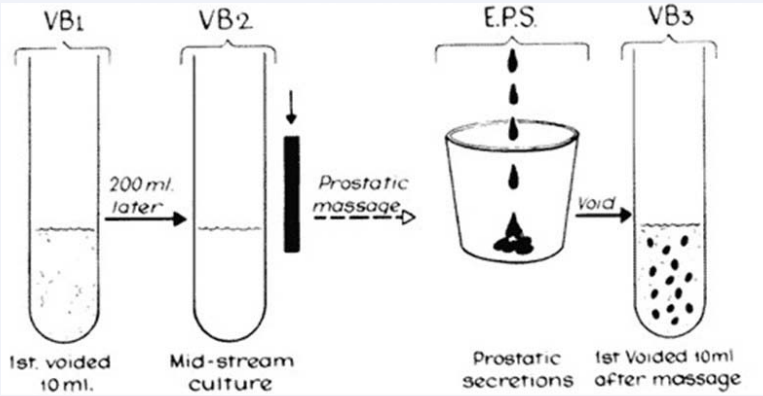

Figure 1 Meares-Stamey four-glass lower urinary tract localization test for chronic prostatitis and chronic pelvic pain syndrome. The voided bladder (VB1) include the first 10mL of urine and represents the urethral specimen. The voided bladder-2 (VB2) specimen is similar to mid-stream urine collection and represents the bladder urine. Expressed prostatic secretion (EPS) should be collected directly into a sterile container during prostatic massage. The voided bladder-3 (VB3) specimen, the first 10mL of urine after prostatic massage, includes any EPS trapped in the prostatic urethra. Recently a shortened 2 glass test has been adopted (pre and post prostatic massage urine sample)

Pelvic floor muscle factors: Several research groups have suggested that a myofascial pain syndrome with abnormal pelvic muscle spasm is the primary source of CP/CPPS [21]. A review of the National Institute of Health (NIH) study which examined 384 men with CP/CPPS and 121 asymptomatic controls who had complete physical examinations, shows that more than half of the CP/CPPS patients had abdominal or pelvic pain compared to only 7% of the controls [22]. Additional research suggests that the pain originates specifically at the muscle attachments of the pelvic girdle such as the sacrum, coccyx, ischial tuberosity, pubic rami, and the endopelvic fascia [22,23]. It is hypothesized that the formation of myofascial trigger points in these areas is the result of mechanical abnormalities of the hip and lower extremities, chronic holding patterns such as those that occur during toilet training, constipation, sexual abuse, sports that create chronic pelvic stimulation, trauma, recurrent infections, and surgery [21]. According to a recent study by Antolak, et al., pudendal nerve entrapment can also explain some of the pain experienced by CP/CPPS patients (as it leads to subsequent neuropathic pain) [24].

DIAGNOSTIC EVALUATION

CP/CPPS is characterized by chronic pelvic pain, which is often accompanied by LUTS; the clinical course of the disease is not predictable. The patient often experiences a relapsing-remitting pattern with unstable severity and frequency [25]. Patients often complain of pain or discomfort that may be located in the perineum, anus, lower back, inguinal area and genitals. Post-ejaculatory pain is common and often may indicate a pelvic floor muscle pathology [26]. Basic diagnostic steps include urine analysis and urine culture before and after prostatic massage, and once bacterial growth is detected, chronic bacterial prostatitis is diagnosed (or classified as "chronic prostatitis" type IIIA). The absence of bacterial growth in urine culture before and after prostatic massage and the persistence of symptoms most likely suggests that the patient is suffering from CP/CPPS (or classified as "chronic prostatitis" type IIIB).

It appears that a mutual relationship exists between CP/CPPS and other systemic syndromes and the attention for review of body systems is mandatory. During patient evaluation, one may often encounter other, non-related chronic pain, bowel dysfunction, depression, stress and feeling of hopelessness or helplessness [27]. A recent publication examined the prevalence, type, location, and timing of pain in over 1500 men with CP/CPPS. Perineal pain or discomfort was the most common complaint (63%), followed by testicular pain (58%), pain in the pubic or suprapubic region (42%), and penile pain (32%). Painful ejaculation and voiding was reported in 45% and 43% of the patients respectively (28). Inguinal, lower back, and thigh pain as well as psoas muscle related pain also been described [23]. Due to these various clinical presentations, a thorough history taking at presentation is required.

The most scientific way to qualify and quantify this bizarre clinical scenario involves the use of questionnaires. The National Institute of Health Chronic Prostatitis Symptoms Index (NIH-CPSI; Table 1.) which has been validated as a nine-question survey that covers the three domains of pain (location, frequency and severity), urinary and quality of life symptoms arranged into a score, is most commonly used. A six point improvement in the total score is considered a clinically significant improvement [29].

In order to bridge the gap between a symptom based diagnosis and future mechanistic approach for the diagnosis of CP/CPPS, the UPOINT questionnaire (urinary, psychological, organ specific, infection, neurologic/system, and tenderness of skeletal muscle; Table 2) was suggested by Nickel, et al. [30]. This questionnaire evaluates the symptoms of CP/CPPS according to the various organ systems potentially involved in the pathophysiology of CPPS. Recent studies favor the UPOINT questionnaire [31,32].

One must note, that there is a strong correlation between pain, psychological factors and quality of life (QoL) [27,33]. Medical journals dealing with pain have also noted that pain combined with psychological stress tends to be stable over time, with negative effects of comorbid pain and cognitive factors on functioning and wellbeing [34,35]. In addition, daily physical

NIH –Chronic Prostatitis Symptom Index (NIH-CPSI)

Pain or Discomfort

1. In the last week, have you experienced any pain or discomfort in the following areas?

	Yes	No
a. Area between rectum and testicles (perineum)	☐1	☐0
b. Testicles	☐1	☐0
c. Tip of the penis (not related to urination)	☐1	☐0
d. Below your waist, in your pubic or bladder area	☐1	☐0

2. In the last week, have you experienced:

	Yes	No
a. Pain or burning during urination?	☐1	☐0
b. Pain or discomfort during or after sexual climax (ejaculation)?	☐1	☐0

3. How often have you had pain or discomfort in any of these areas over the last week?

☐ 0 Never
☐ 1 Rarely
☐ 2 Sometimes
☐ 3 Often
☐ 4 Usually
☐ 5 Always

4. Which number best describes your AVERAGE pain or discomfort on the days that you had it, over the last week?

☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐
0 1 2 3 4 5 6 7 8 9 10
NO PAIN — PAIN AS BAD AS YOU CAN IMAGINE

Urination

5. How often have you had a sensation of not emptying your bladder completely after you finished urinating, over the last week?

☐ 0 Not at all
☐ 1 Less than 1 time in 5
☐ 2 Less than half the time
☐ 3 About half the time
☐ 4 More than half the time
☐ 5 Almost always

6. How often have you had to urinate again less than two hours after you finished urinating, over the last week?

☐ 0 Not at all
☐ 1 Less than 1 time in 5
☐ 2 Less than half the time
☐ 3 About half the time
☐ 4 More than half the time
☐ 5 Almost always

Impact of Symptoms

7. How much have your symptoms kept you from doing the kinds of things you would usually do, over the last week?

☐ 0 None
☐ 1 Only a little
☐ 2 Some
☐ 3 A lot

8. How much did you think about your symptoms, over the last week?

☐ 0 None
☐ 1 Only a little
☐ 2 Some
☐ 3 A lot

Quality of Life

9. If you were to spend the rest of your life with your symptoms just the way they have been during the last week, how would you feel about that?

☐ 0 Delighted
☐ 1 Pleased
☐ 2 Mostly satisfied
☐ 3 Mixed (about equally satisfied and dissatisfied)
☐ 4 Mostly dissatisfied
☐ 5 Unhappy
☐ 6 Terrible

Scoring the NIH-Chronic Prostatitis Symptom Index Domains

Pain: Total of items 1a, 1b, 1c, 1d, 2a, 2b, 3, and 4 = ____

Urinary Symptoms: Total of items 5 and 6 = ____

Quality of Life Impact: Total of items 7, 8, and 9 = ____

Table 1 Phenotypic approach to the management of the chronic prostatitis/chronic pelvic pain syndrome. The National Institute of Health Chronic Prostatitis Symptom Index (NIH-CPSI).

Urinary	Associated lower urinary tract symptoms
Psychological	Clinical depression with catastrophic attitude about CP/CPPS symptoms
Organ specific	Pain associated with the voiding cycle or prostate specific tenderness
Infection	Positive culture of either urine or EPS
Neurologic/systemic	Pain outside the pelvis or other pain disorders
Tenderness	Pain or tenderness in the lower abdominal or pelvic musculature as palpated on physical exam.

Table 2 Domains of the UPOINT classification system.
Abbreviations: CP/CPPS, chronic prostatitis/ chronic pelvic pain syndrome; EPS, expressed prostatic secretions;

activities are strongly dependent upon symptoms of chronic pain and depression alone or in combination [36]. A recent study shows that among forty-four CP/CPPS patients and spouses who have been followed for over 2 years showed that both patients and their spouses have experienced a decrease in mental QoL, depression and anxiety [37].

Treatment of CP/CPPS

The treatment of this complex syndrome is neither simple nor uniform and usually involves a wide range of drugs and manipulations, which have been widely studied over the years. Due to its diverse symptomatology from potential multiple etiologies, it requires a great deal of patience, sympathy, and understanding on the part of the physician regarding the physical and mental difficulties experienced by the patient.

The most widely utilized categories of treatments include antibiotics, anti-inflammatory medications, neuromodulators, alpha-blockers, pelvic floor physical therapy (PFPT) and cognitive behavioral therapy (CBT).

Antibiotics: For most clinicians, the use of antibiotics as a first line treatment is almost intuitive because it is believed that an unknown causative "organism" probably plays some role in the etiology of their patient's misery. Often patients who have been treated with multiple courses of antibiotics do not improve.

There is little debate regarding the use of antibiotics, specifically quinolones and macrolides, when bacteria is cultured from the prostatic fluid and the diagnosis of chronic bacterial prostatitis is determined. The effectiveness of these antibiotics in clearing up bacterial infections in chronic bacterial prostatitis may reach a rate of over 70% and improve pain and sexual related symptoms [38,39]. When CP/CPPS is involved, repeated courses of antibiotics are not helpful, as there is no evidence of a cultured pathogen [40]. Some patients, temporarily report symptom improvement when they are treated with antibiotics even when cultures are negative. This phenomenon may be partially explained because of the anti-inflammatory effects of these drugs in addition to their anti-microbial properties [41,42]. Therefore, the justifications for antibiotic treatment for CP/CPPS patients are strong placebo effect, the eradication or suppression of non-cultured microorganism and the independent anti-inflammatory effect of some antibiotics. It is advised to consider empiric antibiotic therapy for category IIIA chronic prostatitis with re-evaluation of its effects after 2-4 weeks of therapy. Antibiotics might be continued for 4-6 weeks if the patient reports positive effects from treatment.

Anti-inflammatory medications: Steroids and non-steroidal anti-inflammatory drugs (NSAIDS) may provide temporary relief [43]. A randomized controlled study by Nickel, et al., of 161 men who were treated with either rofecoxib, a COX-2 inhibitor, or a placebo, showed a modest improvement in NIH-CPSI scores of the refocoxib group compared to the placebo group, with a significant improvement in QoL [44].

Pentosanpolysulfate is a semisynthetic mucopolysaccharide that is similar to naturally occurring glycosaminoglycans that form a protective barrier in the epithelium of the urinary system. It is approved for the treatment of interstitial cystitis and has shown some limited response in treating CP/CPPS [45].

Finasteride is an inhibitor of the enzyme 5-alpha reductase, which prevents the transformation of testosterone to its active form dihydrotestosterone (DHT). Therefore, reducing prostatic volume and halting prostate growth could suggest an optional disease regression. However, finasteride failed to be effective as demonstrated by Nickel, et al, [46].

Herbal anti-inflammation substances have shown some efficacy in treating CP/CPPS. Bioflavonoids may reduce prostatic inflammation in CP/CPPS. Although their mechanism of action in the process of inflammation is not yet clarified, they may be offered to patients with focal prostatic tenderness without infection. Quercetin, a bioflavinoid, has been tested and found to reduce inflammation associated with lipopolysaccharids and obesity induced inflammation in rat models [47,48]. Quercetin has also been associated with improved CPSI scores in 30 men who were randomized to quercetin treatment versus placebo [49].

Neuromodulators in the management of pain : Neuropathic pain is believed to be the dominant type of pain in CP/CPPS. Neuromodulating medications such as amytriptiline, gabapentin and pregabalin have become popular in the treatment of neuropathic pain and studies are beginning to define their role in the treatment of CP/CPPS.

Pregabalin has been tested for its effectiveness in CP/CPPS in a double–blind trial of 324 men, 218 of whom were randomized to pregabalin and placebo. It was found that treatment was associated with improvement in global pain score and total improvement in CPSI-NIH questionnaires [50].

Amytriptiline is a tricyclic antidepressant (TCA) sometimes used to improve both voiding symptoms and pain in CP/CPPS patients. One early study of 22 patients that included 12 men found resolution of symptoms in 11 patients and significant improvement in 6 others [51]. A subsequent placebo controlled study of 50 men suffering from interstitial cystitis showed an improvement in pain and urinary symptoms as measured by the O'Leary-Sant score [52].

Gabapentin is also known for its efficacy in treating chronic pain conditions and was originally reserved for chronic neuropathic pain. This medication has not been studied in depth regarding the treatment of CP/CPPS but the few studies that were done showed some effectiveness in refractory genitourinary pain mainly when combined with amitriptyline [53,54].

Alpha-Blockers: Alpha-adrenergic receptor blockers (α-blockers) are known for their role in improving lower urinary tract symptoms (LUTS) in men suffering from benign enlargement of the prostate. The urine flow disorders of some of the CP/CPPS patients become prominent. This may explain the utility of these medications in CP/CPPS as part of the notion that urine flow improves when using alpha blockers. Recently, alpha blockers have been shown to have some role in the inhibition of prostate cell proliferation and the induction of cell apoptosis [55,56].

In 2008, a multicenter, randomized, double blind, placebo control study, examined the efficacy of alfuzosin, a commonly used α blocker, in reducing symptoms in 272 previously untreated men suffering from CPPS. The study failed to show a reduction in the symptoms rate (as measured by NIH-CPSI) in the treated group compared to the placebo group. One reason may have been that patients were included whether or not they had LUTS, often making the treatment inappropriate [55].

A meta-analysis of 734 patients in nine studies found an improvement in pain scores (the majority based on NIH CPSI questionnaires) following treatment with alpha-blockers for more than 3 months when compared to the placebo group. This improvement was more significant when urinary complaints were involved rather than pain. This finding suggests that the use of alpha-blockers should be considered in patients who complain of voiding issues and is more effective when combined with other therapies [57].

Pelvic Floor Physical Therapy (PFPT): Pelvic floor physical therapy (PFPT) with a skilled therapist has been found to be extremely useful in the treatment of CP/CPPS, especially in patients who failed previous treatments [21].

Pelvic floor muscle dysfunction may be present in many patients with CP/CPPS in a RCT including 384 men with CP/CPPS and 121 controls, pelvic floor muscle tenderness was noted in 51% of the patients and only in 7% of the controls [22]. This finding may help therapists identify this dysfunction and treat it successfully. PFPT include paradoxical relaxation, myofascial release and stretching exercises [58]. A study of 113 patients who received trigger-point release had a decrease in visual analog pain scores at 6 months. Another study has suggested daily therapy with an improvement in CPSI scores after 6 days in 60% of patients who were examined [59].

In addition to pain, PFPT has also been shown to be effective in the treatment of sexual dysfunction such as painful ejaculation and erectile dysfunction [60].

Cognitive behavioral therapy: As mentioned earlier, individual response to pain and the various attempts to manage it, can be affected by impaired coping mechanisms in patients with CP/CPPS. Therefore, it is essential to recognize it and address it properly as it can dramatically improve patient's QoL [61].

Cognitive behavioral therapy may help guide patients to healthy coping strategies. Because it is known that poor coping mechanisms contribute to the disease, different programs have been developed for intensive cognitive behavioral therapy in an attempt to assist the management of the pain. However, no large studies; have been conducted to validate its effectiveness [62]. The modest effect of cognitive behavioral therapy on chronic neck pain has been demonstrated by a recent review by Monticone M., et al., however this area of psychological management should continue to be investigated [63]. Due to the high rates of psychological co-morbidity with CP/CPPS, offering psychological counseling should be a part of every practitioner's treatment strategy.

Other Minimally invasive manipulations for refractory patients (lower recommendation level):

Prostate heat therapy: The use of transurethral microwave thermotherapy (TUMT) falls into the 300-3000MHz range of electromagnetic spectrum and is used for the generation of heat energy, which causes irreversible damage to prostate cells without markedly affecting the urethra. It is used as part of the treatment modality for BPH. Urinary irritation and obstruction are major side-effects of the procedure and require a few days of bladder drainage post-treatment.

The effectiveness of TUMT has been demonstrated by Choi, et al., who reported normalization of symptoms in almost half of the 78 patients who suffer from treatment resistant CP/CPPS [64]. Nickel and Sorenson observed a short term response to TUMT in a smaller group of CP/CPPS patients [65]. We have also found ~~a~~ limited complete responses and temporary relief in 12 patients treated with TUMT [66].

Intraprostatic injections: The penetration of antibiotics into the prostate tissue is limited by the blood-prostate barrier [67]. Due to this limitation, a sophisticated method of antimicrobial delivery into the prostate gland was developed. Antibiotics are often combined with lidocaine and steroids. Researchers have attempted to reach the prostate using transurethral, transperineal, and transrectal approaches. The success of injections in the various protocols and schedules has been reported to be less than 50% [68].

Additional treatments such as *injections of Botulinum A* and *shock waves* to the perineum have also been suggested. Zimmerman, et al., looked into a group of 60 patients who were treated with a reflector that produces shock waves directed at different points of the perineum. The control group received a mock treatment. The treatment protocol involved 3000 weekly shock waves (maximum total energy flow density 0.25 mJ/mm^2; frequency: 3Hz) for 4 weeks. The results indicated that there was some pain relief as measured by the NIH-CPSI [69].

CONCLUSION

The various treatments and manipulations suggest that we are faced with a complex, undetermined condition that may be affected by different etiologic factors. The course of this syndrome is quite unpredictable and can be characterized by "attacks" that last for days or even weeks followed by "remissions" lasting sometimes for years. Many health practitioners feel helpless

when confronted by these suffering individuals. Standard treatment is based on antibiotics, frequently combined with alpha-blockers. Other minimally invasive procedures such as heat therapies, intraprostatic injections combined with physiotherapy and psychological consultations have been suggested. As no "one bullet treatment" presently exists, treatments follow some guideline protocol. The problem however becomes frustrating when attacks recur despite the protocol treatment. That is the point where less evidence-based treatments are sometimes offered, just to not disappoint the patients.

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