A Comprehensive Review of the Diagnosis, Treatment, and Management of Urologic Chronic Pelvic Pain Syndrome

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Abstract

Purpose of Review Urologic chronic pelvic pain syndrome (UCPPS) is a chronic, noncyclic pain condition which can lead to significant patient morbidity and disability. It is defined by pain in the pelvic region, lasting for greater than 3 to 6 months, with no readily identifiable disease process. The aim of this review is to provide a comprehensive update of diagnosis and treatment of UCPPS.
Recent Findings UCPPS encompasses chronic pelvic pain syndrome or chronic prostatitis (CP/CPPS) in men and interstitial cystitis or painful bladder syndrome (IC/PBS) in women. Underlying inflammatory, immunologic, and neuropathic components have been implicated in the pathogenesis of UCPPS.
Summary For optimal patient management, an individualized and multimodal approach is recommended. Medical management and physical therapy are the mainstays of treatment. Injection therapy may offer additional relief in medically refractory patients. Further minimally invasive management may include spinal cord and peripheral nerve stimulation, though evidence supporting efficacy is limited.

Keywords Urologic chronic pelvic pain syndrome · Chronic prostatitis · Interstitial cystitis · Painful bladder syndrome

Introduction

Urologic chronic pelvic pain syndrome (UCPPS) is a chronic, noncyclic pain condition that can cause significant discomfort and disability [1]. It is defined by pain in the pelvic region that lasts for greater than 3 to 6 months and has no identifiable disease process [2]. Often a diagnosis of exclusion, this common but complex condition affects both men and women [3]. The term "UCPPS" includes chronic pelvic pain syndrome or chronic prostatitis (CP/CPPS) in men and interstitial cystitis or painful bladder syndrome (IC/PBS) in women [4]. Nearly 25% of adult women experience pelvic pain, most of whom are unable to find an identifiable cause, despite extensive workup by multiple practitioners [5]. Among men who are seen for prostatitis in the outpatient setting, over 90% are diagnosed with CP/CPPS, indicating its high incidence among urologic disorders [6]. Still, the incidence of UCPPS is likely highly underestimated due to patient hesitancy in reporting urogenital problems [7].

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Patient risk factors can include various triggers such as previous infection, surgery, chemical irritation, or trauma [8]. In addition, psychosocial factors such as depressive symptoms, physical disability, sexual functioning, home environment, occupation, socioeconomic status, and individual response to pain can further worsen symptoms [9]. Prior colonoscopy and CP/CPPS have also been correlated [10]. Among male patients, low total testosterone levels may be associated with the presence of CP/CPPS, although findings are less robust [11].

Patients diagnosed with UCPPS all present with chronic pelvic pain; however, a wide range of symptom types and severity is often reported [7, 12]. For some, this pain is debilitating, and significantly lowers quality of life. In addition to pelvic pain, patients can have a range of organ involvements, including urogenital, neurologic, endocrine, gastrointestinal, or musculoskeletal systems [13]. Often a psychological component such as a comorbid mood disorder or individual inability to cope may contribute to the exacerbation of symptoms [14]. Patients with a surgical history often have associated fibrotic changes, further complicating pelvic nerve anatomy [7].

As such, diagnosis and management of UCPPS can be challenging. Patients may further delay medical attention due to the sensitive nature and perceived stigma of urogenital medical care [9]. Often patients will see multiple providers and experience frustration with their care and with the chronicity of their condition [15]. In addition to the impact on quality of life, UCPPS contributes to a high economic burden through large direct and indirect financial costs [8, 16]. Due to the complexity of UCPPS, a wide range of treatment options is currently available with variable success. This review, therefore, aims to summarize the current understanding of UCPPS, with a focus on treatment options.

Pathophysiology

The etiology of UCPPS is historically difficult to determine, as the pathophysiology is complex and can vary by patient population and/or disease subtype [13]. Several theories and associated findings are currently under investigation. One common cause of pelvic pain involves nerve entrapment or injury. In a recent study of 26 patients with UCPPS, Quaghebeur et al. found significant pudendal nerve mechanosensitivity in 85% of affected patients, suggesting a neuropathic or injurious component to the pain mechanism [17]. Pudendal nerve entrapment can occur at multiple pelvic anatomic landmarks, including the sacrotuberous ligament, ischiorectal space, and pubic symphysis area [18]. Rarely, persistent damage of the cauda equina nerve roots can present as chronic pelvic pain [19].

Anatomical disruptions are also associated with neuropathic origins of pain. Post-surgical changes within the lower spine or pelvis from procedures such as pelvic ring fixation or vaginal mesh placement can lead to the development of adhesions, with subsequent nerve entrapment [20, 21]. UCPPS can also present as a post-operative complication as can be observed following rectal cancer resection and hysterectomy [22, 23]. Anatomic abnormalities such as a duplicate cervix and vagina or bicornis bicollis uterus can disrupt regular female anatomy, further contributing to pain [13, 24]. Men with CPPS had significantly lower pelvic floor muscle mobility with a full bladder when compared with controls without CPPS, indicating that pelvic floor dysfunction contributes to prostate-related pain by altering tension in the puboprostatic ligaments [25].

In addition to neuropathic changes, UCPPS has underlying inflammatory and immunologic components [26]. Chronic inflammation stimulates catecholamine overactivity and smooth muscle proliferation, causing muscle spasms and pelvic pain [27–29]. Pelvic cytokines can cross the blood-brain barrier, contributing to mental health symptoms. In addition, prostatic interstitial cells of Cajal (ICCs) increase catecholamine synthesis, further stimulating excitatory neurotransmission [30–32]. While the NIH defines UCPPS by a lack of identifiable bacterial source on traditional culture, Nickel et al. found that urinary concentrations of *Burkholderia cenocepacia* were significantly increased in UCPPS patients [33, 34]. While no significant correlation was found, the door remains open for an infectious-related pathophysiology [28].

UCPPS is also associated with an imbalance of neurotransmitters [35]. CP/CPPS patients have lower GABA levels and high choline levels, which may be associated with a decrease in inhibitory neurotransmission and negative mood symptoms, respectively [29, 36]. Chronic pain is also triggered by chronic peripheral or central inflammation and/or nerve injury, through mast cell activity and formation of reactive oxidative species [26, 28]. Such inflammation can damage sperm protein, DNA, and membrane integrity, causing infertility [37].

In addition to these findings, many clinical studies have identified coexisting emotional and behavioral symptoms with UCPPS [38]. Osório et al. found an association with mood disorders in women with chronic pelvic pain (CPP), suggesting that increased pain sensitivity may create and perpetuate a cycle of pelvic irritation, pain, depression, and anxiety [39]. While the existence of triggers remains controversial, Sutcliffe et al. reported the association of recent urinary tract infection (UTI) symptoms and sexual activity with flare onset [40]. Triggers may also include food sensitivities unique to each patient and commonly include spicy foods, coffee, or alcoholic beverages [8]. Finally, it is important to rule out any malignancy that may present as UCPPS, such as an adenomyoma, a tumor compressing the pudendal nerve, or intravascular papillary endothelial hyperplasia [41, 42]. Proposed mechanisms of UCPPS are summarized in Table 1.

Table 1 A summary of proposed mechanisms causing and contributing to chronic pelvic pain

Mechanistic theory	Description
Neurotransmitters	• An imbalance of neurotransmitters, particularly decreased GABA and increased choline, alter pain processing and contribute to both mood and psychiatric symptoms of chronic pelvic pain [29, 36].
Pelvic floor dysfunction	• Patients with chronic pelvic pain have decreased pelvic floor muscle mobility, which alters tension in supporting ligaments resulting in pelvic pain [25].
Anatomy	• Anatomical anomalies can contribute to chronic pelvic pain syndrome or chronic prostatitis (CP/CPPS) through entrapment and injury of the pudendal nerve and lumbosacral plexus [18].
Malignancy	• Malignancy must be ruled out as a cause of CP/CPPS such as those causing compression of the pudendal nerve [41].
Post-surgical changes	• Pelvic surgeries may result in adhesions or anatomical changes, causing chronic pelvic pain [20, 22, 23].
Referred pain and psychiatric	• Some CPPS patients have hypersensitivity to pain, and the damage to nociceptive fibers from this hypersensitivity leads to a susceptibility to referred pain in deep organs [43]. Increased hypersensitivity to pain in referred areas is associated with both psychological distress and disease severity [43].
Behavioral triggers	• While previously thought to play a role in inciting episodes of chronic pelvic pain, only recent UTI symptoms and recent sexual activity are associated with flare onset [40].
Inflammation and autoimmunity	• Chronic inflammation leads to smooth muscle proliferation and muscle spasms which cause pelvic pain [27, 32]. Cytokines produced in the pelvis may cross the blood-brain barrier and increase mental health symptoms [44]. Changes in the expression of interstitial cells of Cajal leading to increased catecholamine release play a role in chronic pelvic pain [30, 32, 45, 46]. Mast cells are also critically involved in its pathophysiology [47, 48].
Infection	• In the past, infection was thought to be a cause of chronic pelvic pain, but the NIH now classifies CP/CPPS by its lack of identifiable bacterial source, though patients are often misdiagnosed at first as having a urinary tract infection [33].
Gut microbiome	• Some differences in gut microbiome have been found in men with CP/CPPS versus those without it, possibly identifying it as a potential area of treatment [33, 49].

Patient Assessment: Diagnosis and Clinical Presentation

Though UCPPS carries significant disease burden, a disproportionately small number of patients voice their complaints. Patients often refrain from discussing symptoms with their physician for a number of psychological and social reasons. Wood et al. found that patients felt ashamed, which hindered their willingness to disclose symptoms to physicians [9]. The inherently erratic nature of UCPPS and unpredictability of symptoms further prevents many patients from describing their ailments with clarity [9]. Patients who were aware of their diagnosis and were interested in receiving treatment were also discouraged by the lack of effective therapy. To counteract these hesitancies, healthcare providers are encouraged to show empathy and support for patients potentially affected by UCPPS and to participate in activism related to treatment development [9].

Proper evaluation of UCPPS requires a general gynecologic and urologic workup with an organized approach. Upon symptom inquiry, clinicians should be suspicious of chronic pain in the perineum, testicles, tip of penis, or pubic areas that has lasted for at least 3 months. Palpation of the pelvic floor and prostate, urine culture, pre- and post-prostate massage urine test, cystoscopy, Pap smear, urodynamics, and ultrasound can be used to exclude other diagnoses. Some women can present with concurrent abdominal wall pain [50]. Patients with UCPPS can also exhibit tenderness in the pelvic floor, suprapubic area, pubic symphysis, and posterior superior iliac spine [6]. Finally, symptomatically similar diseases such as UTI, bacterial prostatitis, benign prostate hypertrophy (BPH), overactive bladder, pelvic floor dysfunction, malignancy, calculi, interstitial cystitis, and irritable bowel syndrome must be ruled out before diagnosis of UCPPS [51].

Peripheral nerve mechanosensitivity may play a role in the pathophysiology of UCPPS. Quaghebeur et al. recently found that the musculoskeletal pain in UCPPS is correlated with minor nerve injuries [17]. Others have considered a solely neurogenic basis. Pudendal neuropathy can be determined using a pinprick sensory evaluation; Antolak et al. found that such neuropathy was present in every patient exhibiting symptoms of UCPPS [52]. Furthermore, Passavanti et al. pointed to the utility of brain neuroimaging for diagnostic assessment, although a detailed understanding of structural anomalies must be further investigated [7].

Many psychiatric comorbidities are also present in patients with UCPPS [53]. Psychosocial factors often amplify symptoms, and while psychosocial self-management has been effective in many other pain conditions, its impact on UCPPS is unclear [54]. Klotz et al. found a correlation between psychosomatic and myofascial symptoms, noting that pressure on muscular trigger points can cause pain in seemingly unrelated parts of the body, such as the pelvic floor [55]. Other clinicians have suspected a relationship between mental health and the development of UCPPS, with an estimated 91% prevalence of somatoform disorders and an estimated 50% prevalence of mood disorders [14]. In keeping patient evaluation systematic and thorough, a 6-point symptom classification was developed to include urinary, psychosocial, organ-specific, infectious, neurologic/systemic, and tenderness of skeletal muscle (UPOINT) domains [56].

Conservative Management

For optimal patient management, an individualized and multimodal approach is recommended [57]. Successful management of any chronic condition begins with a discussion of realistic goals of care. This is essential when addressing the symptoms of UCPPS. Since chronic pelvic pain is often associated with dysfunctional pelvic floor muscles, many treatment plans begin with physical therapy [58]. Methods include manual therapy to facilitate muscle relaxation, therapeutic exercises to improve the range of motion and flexibility, and biofeedback for strength training [59]. Biofeedback is achieved by monitoring pelvic floor muscle contraction through perineal electrodes [59]. This positive feedback is used to monitor strength training and muscle relaxation. If physical therapy is unable to relieve a patient of pain at trigger points, injection with a nerve-blocking agent such as lidocaine and bupivacaine can provide symptomatic improvement [60]. For example, a patient with residual tenderness can receive a pudendal nerve block at trigger points [60]. Although providing focused physical therapy for pelvic floor muscle conditioning is challenging, its use within a multidisciplinary treatment approach is promising [61, 62].

Pharmaceutical management is another essential component of UCPPS treatment; alpha-blockers, antibiotics, acetaminophen, nonsteroidal anti-inflammatory drugs, and gabapentenoids have been demonstrated to offer symptomatic relief [63••]. Alpha-adrenergic blockers such as doxazosin are commonly used to control pain in UCPPS. NSAIDs and acetaminophen are used frequently as first-line therapy but should be initiated on a 4- to 6-week trial and only continued if effective [64]. Recent evidence has shown that anti-inflammatory drugs may be ineffective in alleviating pain caused by UCPPS [64]. Gabapentenoids are also effective, with gabapentin yielding more promising symptomatic relief than pregabalin [65]. Magri et al. found that patients who incorporated *Serenoa repens* extracts, lycopene supplements, selenium supplements, and antibacterial agents experienced greater relief, particularly in treating the urinary and infectious domains of UCPPS [56]. Patients who do not respond to these first line agents may be treated with duloxetine and other selective 5-serotonin and norepinephrine reuptake inhibitors, which provide safe and effective pain management when paired with doxazosin [66]. Muscle relaxants, 5-phosphodiesterase inhibitors, and anxiolytics have also all been used second-line with promising reports, but further studies are necessary to assess long-term efficacy [55, 56, 67].

While relieving functional impairment in UCPPS is often the primary treatment goal, psychological burden must also be considered. The prevalence of somatoform, mood, and anxiety disorders is significant, and effective treatment involves addressing all biopsychosocial factors that contribute to chronic pelvic pain [14]. Conventional use of biofeedback and cognitive behavioral therapy (CBT) can be beneficial in addressing comorbid depression or anxiety, as depression is often correlated with symptom severity and can have a large impact on quality of life [14]. Although CBT monotherapy often fails to completely relieve psychological symptoms, the addition of CBT or psychodynamic therapy to a pharmacological regimen can improve psychological status and decrease pain severity [15, 68, 69]. This multimodal management is also associated with improved sexual function [70]. Furthermore, new evidence links the myofascial element of UCPPS to psychosomatic pathologies, emphasizing the need for psychological intervention [55].

The effect of nonpharmacologic therapies on UCPPS has also been studied. Salama and Abouelnaga found that medication-refractory patients treated with radial extracorporeal shock wave therapy experienced a significant decrease in pain domain, urine score, and improvement in quality of life [71]. Guu et al. also noted a positive response in 81.8% of refractory patients who received low-intensity extracorporeal shock wave therapy, with waist circumference as a significant predictor of a positive treatment response [72••]. This improvement was quantified by the National Institutes of Health Chronic Prostatitis Symptom Index (NIH-CPSI), a scoring system used to quantify UCPPS symptoms [51]. A meta-analysis by Qin et al. found that acupuncture and electro-acupuncture decreased the severity of UCPPS symptoms based on the NIH-CPSI score when used with alpha-blockers and antibiotics [63, 73]. Acupuncture techniques were often found to be more effective than alpha-blockers; however. long-term improvement of continued acupuncture therapy is still under review [63, 74].

Other therapeutic trials aimed at relieving UCPPS symptoms have examined curcumin and calendula extract suppositories. A phase II, randomized, single-blinded placebo-controlled clinical trial found significant improvement in the NIH-CPSI score in patients treated with such suppositories [70].

Practicing yoga is also therapeutic for UCPPS, with patients reporting a significant decrease in pain and an improvement in quality of life on the visual analog scale (VAS) score [75]. A preliminary study by Ying et al. found that topical essential oil significantly reduced perineal discomfort in patients with UCPPS after 4 weeks of use [76]. Chronic pelvic pain may also be reduced by direct inhibition of preganglionic afferent neurons. Transcutaneous electrical nerve stimulation significantly improved painful stimuli by providing simultaneous inputs in larger myelinated nerve fibers, thus "closing" the hypothetical gate in the dorsal horn of the spinal cord [77]. Finally, the gut microbiome may also impact UCPPS, as over half of men taking the bioflavonoid quercetin had improvements in pain and quality of life [49].

Minimally Invasive Interventional Therapy

Trigger Point Injections

Similar to its role in persistent myofascial pain, trigger point injection (TPI) therapy may offer some benefit to patients with UCPPS. Methods vary, but typically, a local anesthetic, botulinum toxin, or a dry needle is used to target the piriformis, iliococcygeus, pubococcygeus, levator ani, coccygeus, obturator internus, or superficial and deep transverse perinei [78]. It is thought that needling itself may reduce symptoms in the absence of medication administration [79]. Adverse effects include soreness, hematoma, hemorrhage, and syncope [80]. While the mechanism of action by which TPIs may improve chronic pelvic pain is unclear, theories include mechanical disruption of muscle fibers, interruption of a positive feedback loop, dilution of nociceptive substances, vasodilation removing excess metabolites, and a release of nociceptive/inflammatory biochemicals [81, 82]. Recently, Tadros et al. found that half of participants with CPPS had symptom improvement following a combination of trigger point injections and pelvic floor physical therapy, further supporting a multimodal approach [60].

Botulinum toxin (BTX) has shown promising results in patients with chronic pelvic pain [83, 84]. Women with MFFP who were treated with BTX and physical therapy demonstrated improvement in pelvic pain symptoms and a decreased number of pelvic pain trigger points [83]. BTX can also help relieve lower urinary tract symptoms [85]. In addition, transurethral injection of BTX led to a significant decrease in NIH-CPSI scores within 1 month of injection [86]. These patients experienced improved quality of life with no major side effects, while patients receiving placebo injections had worsening symptoms [86]. Although initially effective, the duration of symptomatic improvement following BTX injections can vary among patients. Abdel-Meguid et al. found that while intraprostatic injections of BTX significantly reduced the NIH-CPSI score in nonbacterial CPPS,

improvements declined after 9–12 months [84]. While the exact mechanism of BTX treatment is uncertain, Zhang and Smith suggest that denervation of cholinergic motor neurons and subsequent reduction in sphincter tone may inhibit sensory and motor nerve stimulation, thus reducing pain [87].

While TPIs are typically limited to dry needling, local anesthetic, or botulinum toxin, the addition of a steroid component can also enhance pain relief for some patients. Although rarely used as monotherapy, steroid injections are often combined with local anesthetics in a single injection. Langford et al. found that using a combination of bupivacaine and corticosteroids in pudendal nerve injections provided immediate and continuous pain relief for 3–5 weeks in patients with chronic pelvic pain [88]. Additionally, Koo and O'Brien reported a case of chronic prostatitis which had maintained complete relief at 21 months follow-up after treatment with a prostatic injection of methylprednisolone and levobupivacaine [89]. Among female patients, post-surgical and post-partum chronic pelvic pain often impacts sexual function and reduces the quality of life. In a study of 53 women, perineal injection of a bupivacaine, hyaluronidase, and hydrocortisone mixture provided significant symptomatic relief for 89% of sexually active women and 69% of sexually inactive women [90]. Additionally, Shim et al. reported that a woman who developed strictures following a vaginal septum revision had a substantial decrease in pain after triamcinolone injections at the transverse vaginal septum, most likely due to the softening of collagen by the steroids [91]. While steroid use can be effective, its use is not recommended for patients with immunosuppression, those with diabetes mellitus, or those taking systemic steroids [92].

Peripheral Nerve Block

Peripheral nerve blocks have also been used for the management of CPPS. Pudendal nerve blockade or ablation may relieve chronic perineal pain via interruption of pudendal innervation within the penis, clitoris, bulbospongiosus muscle, ischiocavernosus muscles, perineum, and anus [93–95]. Additionally, the superior and inferior hypogastric plexuses, ilioinguinal nerve, iliohypogastric nerve, and genitofemoral nerve have been targets of nerve blocks for chronic pelvic pain [95]. Providers may block the superior hypogastric for sympathetically mediated pain pathways associated with cancer-related pain rather than CP/CPPS [96]. Inferior hypogastric plexus blocks can be performed for chronic pain conditions of the lower pelvic viscera, particularly in females, as well as pelvic cancer pain [97, 98]. Ganglion impar blocks may also be utilized; patients with chronic pelvic and perineal pain who were given ropivacaine ganglion impar blocks had a short-term reduction in pain intensity as well as some intermediate-term effects in up to 50% of patients [99]. A summary of injection therapy used for the treatment of CPP is presented in Table 2.

Table 2   A summary of injection therapies used for the management of chronic pelvic pain

Therapy	Description and current use	Advantages	Disadvantages
Injection therapies			
Trigger point injections	• Local anesthetic, botulinum toxin, or dry needling of the pelvic floor may reduce symptoms by mechanically disrupting muscle fibers, interrupting a positive feedback loop, diluting nociceptive substances, and vasodilation [79, 81, 82] • Used in patients with persistent myofascial pain if more conservative treatments have failed	• Proven symptomatic improvement for up to 6 months and can be done by the individual as needed [60, 100, 101]	• May cause soreness, hematoma, hemorrhage, and/or syncope [80]
Botox injections	• Botulinum toxin can cause symptomatic improvement by denervation of cholinergic motor neuron inhibition [83, 84] • Used in addition to other forms of therapy or for patients who have failed conservative methods [83]	• Symptomatic improvement of pelvic pain and a decrease in the number of pelvic pain trigger points [83] • Significantly decrease NIH-CPSI scores and improve quality of life [86] • They may also relieve lower urinary tract (LUT) symptoms [85]	• The length of symptomatic improvement is uncertain and may decline between 9 and 12 months [84]
Blocks and neurolysis			
Steroid	• Muscles injections are primarily used for targeted symptom relief • Steroid injections are not typically used, but oral and rectal steroid suppositories treat LUT inflammation, improve storage symptoms, relieve perineal pain, and improve the quality of life in men with chronic pelvic pain syndrome or chronic prostatitis (CP/CPPS) [102, 103]	• Obturator externus injections with local anesthetics reduce pain scores and decrease hip pain [92]	• Few serious adverse events [102, 103]
Pudendal nerve block	• Pudendal nerve blocks or ablation can relieve chronic perineal pain as it innervates the penis, clitoris, bulbospongiosus muscle, ischiocavernosus muscles, perineum, and anus [93–95] • Used in patients experiencing pudendal neuropathy such as having pain when sitting on a toilet [104]	• Both diagnostic and therapeutic of pudendal neuralgia • Relieves chronic pelvic pain symptoms [93, 105]	• Particle embolization and infarction of pudendal artery [95] • Proximity to other nerves within the tight pudendal canal can cause unintentional nerve blocks [105]
Superior hypogastric, ganglion impar, intrathecal and epidural block; sympathetic block	• Blocking the superior and inferior hypogastric plexuses, along with the ganglion impar, can improve symptoms [97] • Superior hypogastric blocks are used more for cancer-related pain and less for CP/CPPS [96] • Inferior hypogastric plexus blocks are done more for cancer and chronic pain [97, 98]	• Short-term reduction in pain intensity as well as some intermediate-term effects in up to 50% of patients [99]	• Transient paresthesia is the most common side effect, but other possibilities include rectal puncture, hematoma formation, vasculature rupture, and infection [97]

Neuromodulation

Although some patients with UCPPS have successful pain management with injection therapy, many experience minimal relief. Sacral nerve modulation has been effective in treating patients with urinary urge incontinence, urgency, frequency, and fecal incontinence [106, 107]. Roy et al. examined its use at the S3 nerve root for patients with chronic pelvic pain, particularly if physical exam findings were associated with the S3 sacral nerve origin [108]. Although earlier studies discouraged its use, sacral neuromodulation techniques have gained popularity, particularly for treating women with IC/PBS [106]. Recently, Cappellano et al. reported improved pain control in 18 of 19 patients who received a permanent sacral nerve

electrode implantation at a mean follow-up of 21.3 months [109]. Although promising, neuromodulation procedures carry the risk of cerebrospinal fluid leak, lead instability, and lead migration [109]. Additional studies with larger sample sizes are needed to evaluate its efficacy in both genders.

The pudendal nerve is a potential target for generalized pelvic pain, as its origin from S2–S4 may stimulate a greater number of nerves to reach a larger area of pain [110]. Although several techniques are used in practice, the STAR and Bock techniques have shown promising results through a shorter operatory time and fewer skin punctures [111]. Symptom improvement through pudendal stimulation, however, is no greater than relief through sacral neuromodulation [112]. In addition to these nerve targets, individual cases have shown success through conus medullaris stimulation, although the high electrode energy requirement for adequate pain management may suggest an alternative use as an ablative target [113]. Dorsal column spinal cord stimulation (SCS) has also been explored, although the complex nature of pelvic innervation has led to inconsistent results with lead placement [114].

Surgical Procedures

If patients do not experience adequate pain relief from noninvasive, minimally invasive, or neuromodulating interventions, surgery may be cautiously considered. Such procedures are typically reserved for patients who present with an identifiable lesion or a specific condition that can be targeted by invasive intervention. Any surgical intervention should be considered in conjunction with a multidisciplinary and multimodal approach, with attention given to other organ systems including musculoskeletal, urological, gastroenterological, and psychological [115].

Among female patients presenting with UCPPS, surgical intervention is beneficial when identifying adhesions or endometriosis [116•]. Several studies have reported the use of hysterectomy for pain management, although its use remains controversial. Yamamoto et al. found that among 106 patients, 67% of women experienced pain improvement 46 months post-hysterectomy, while 8% experienced worse pain [117]. In contrast, Brichant et al. found that among 48 patients undergoing exploratory laparoscopy, 98% of cases demonstrated a previously undiagnosed pelvic anomaly [111]. Of these women, 59% experienced symptomatic improvement after undergoing surgical treatment of their lesions. Therefore, patient selection is critical before performing a hysterectomy due to high morbidity and limited benefit. Hysterectomy remains a viable option for refractory pain if a thorough evaluation has excluded the involvement of other organ systems [112]. To date, few studies have examined the efficacy of laparoscopic uterosacral nerve ablation, with earlier studies showing no difference in patients who received an ablation versus those who did not [118, 119].

Among male patients, there is currently insufficient evidence to recommend surgical intervention for CP/CPPS. The possibility of bacterial origin remains uncertain, further pointing away from the benefit of a surgical intervention [28]. Some studies do report that prostatectomy is effective within a carefully selected patient population; Schoeb et al. found a significant decrease in pelvic pain in four patients with refractory CP/CPPS after robotic radical prostatectomy (RRP), with a mean follow-up of 34 months [120]. Still, RRP carries the risk of loss of sexual function, and patients must understand the risks and benefits [121]. Larger-scale studies are needed to further investigate the efficacy of RRP.

Conclusion

UCPPS is a common yet debilitating chronic condition that affects both men and women and contributes significant economic burden and healthcare costs. Although it remains difficult to diagnose and is incurable, a multidisciplinary approach is the cornerstone of any treatment plan, and several tools exist to aid providers in making clinical decisions.

Current recommendations place strong emphasis on a thorough history and physical assessment [122]. Multiple factors contribute to the complex presentation of pelvic pain; addressing these will promote better outcomes for patients. Patients should also be routinely evaluated for mood or physical comorbidities and screened for any psychosocial factors. Central to the framework of treatment is establishing a collaborative and trusting relationship between the patient and provider, which will further aid in educating and empowering patients, as well as managing expectations [123]. This will facilitate open communication and help providers determine optimal treatment plans for short- and long-term care while helping reduce the stigma that can surround pelvic pain [62].

Of equal importance is using the biopsychosocial model to create a multimodal treatment plan to target physical pain, mood disorders, and any existing comorbidities. Given the variability of pain presentation and pathophysiology, successful management and higher patient satisfaction is often correlated with individualized therapy. Although current recommendations support the initial use of multimodal and minimally invasive treatment, patients with refractory pain have shown improvement with more invasive therapies. Looking forward, further large-volume and long-term studies are needed to determine optimal pain management for patients with UCPPS.

Compliance with Ethical Standards

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Papers of particular interest, published recently, have been highlighted as:
• Of importance
•• Of major importance

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