Management of chronic primary pelvic pain syndromes

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Abstract

Management of chronic pelvic pain remains a huge challenge for care providers and a major burden for health care systems. Treating chronic pain that has no obvious cause warrants an understanding of the difficulties in managing these conditions. Chronic pain has been recently accepted as a disease in its own right by the World Health Organisation (WHO), with chronic pain without obvious cause being classified as chronic primary pain. Despite innumerable treatments that have been proposed and tried so far for chronic pelvic pain, unimodal therapeutic options are mostly unsuccessful, especially in non-selected individuals. In contrast, individualised multimodal management of chronic pelvic pain seems ~~to be~~ most promising ~~approach~~ and may lead to an acceptable situation for a large proportion of patients. In this review, the interdisciplinary and interprofessional European Association of Urology (EAU) Chronic Pelvic Pain Guideline Group gives a contemporary overview on the most important concepts to successfully diagnose and treat this challenging disease ~~entity.~~

Introduction

Chronic pain syndromes are highly prevalent with significant negative impact on the quality of life (QoL) of affected individuals [1]. This article provides an overview of the aetiology, classification, diagnosis and management of patients with chronic pelvic pain syndromes for urologists caring for such patients. The evidence underpinning this review has been gathered through systematic literature searches performed by the European Association of Urology (EAU) Chronic Pelvic Pain Guideline Group with regular updates to incorporate the latest available evidence into clinical practice. [2]. For more detailed information, the 2021 EAU Guidelines on Chronic Pelvic Pain is available in print and online [2].

Definitions and terminology

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage (International Association for the Study of Pain Taxonomy). Chronic pain refers to pain lasting more than three months. Now included in the 11th revision of the World Health Organisation International Classification of Diseases (ICD-11)~~.~~ ~~In ICD-11~~, the term chronic primary pain refers to pain that has no clear underlying cause and chronic secondary pain is used for pain associated with another diagnosis or recognised pathology. Chronic pelvic pain (CPP) refers to persistent continuous or recurrent pain perceived in structures related to male or female pelvis for at least three months, but a longer period of more than 6 months may be appropriate for cyclical pain.

Aetiology and pathophysiology

Peripheral and central mechanisms

Animal and clinical research have indicated that many underlying chronic pain mechanisms are centrally mediated with central sensitisation and neural pathway modulation maintaining pain ~~perception~~ in the absence of an on-going peripheral trigger or pathology [3]. Perception of a painful stimulus (nociception) requires transmission of information to higher centres and activated pain pathways are modulated at the spinal cord level by a number of pathways ascending and decending. Peripherally, acute pain mechanisms can lead to sensitisation of nociceptive transducers and activation of silent afferents that increase afferent

signalling and maintain pain. Central sensitisation [4] is responsible for a decrease in response threshold and an increase in the magnitude and duration of dorsal horn neuron response. Increased signalling to the central nervous system (CNS) amplifies what is perceived from a peripheral stimulus (hyperaesthesia) so that non-painful stimuli are perceived as painful (allodynia) and noxious stimuli are magnified with ~~an~~ increased ~~level of~~ pain (hyperalgesia). In visceral hyperalgesia, sub-threshold non-perceptible visceral stimuli are perceived resulting in painful sensations such as a feeling of fullness or a need to void/defecate. Convergence of afferents from visceral and somatic sites onto the same second order projection neurons may result in pain being perceived at a different location to its site of origin (referred pain) as higher centres fail to distinguish the source of the nociceptive signal.

Sensitisation of autonomic nervous system afferent fibres can cause a sensitivity to sympathetic stimulation and modification of the efferent output may produce end-organ dysfunction and functional abnormalities. Sensitisation of somatic efferent pathways may explain trophic changes found in somatic tissues. Central mechanisms are also important in the pathogenesis of muscle hyperalgesia with muscle tenderness and trigger points implicated as a source of pain.

Psychological mechanisms

Pain processing is complex as nociceptive pathway activation is associated with emotional, cognitive, behavioural and sexual responses that involve neural networks rather than distinct centres. Psychological processes affect supratentorial processing of pain, producing inhibition and facilitation of nociceptive signals, influencing their appraisal and interpretation and modulating the response and experience of pain. Functional MRI has indicated that psychological modulation of visceral pain probably involves multiple pathways that result from a persistent strengthening of synapses (long-term potentiation) in response to patterns of activity [5]. Psychological factors are relevant to the maintenance of pelvic pain as beliefs about pain contribute to its experience and symptom-related anxiety and central pain amplification may be measurably linked [6]. The various mechanisms of CNS facilitation, amplification and failure of inhibition, mean that there is no simple relationship between physical findings, pain experienced and resulting distress and restriction of activities.

Risk factors

Many factors can increase an individual's susceptibility to developing chronic pain. Genetics play a role as family clusters of pain conditions have been reported with subtle changes in receptors and their transmitters ~~described~~. Developmental, environmental and social factors are important as twin studies have shown that the impact of genetics on the variation in individual susceptibility for some pain syndromes is low [7]. The endocrine system is important in visceral function and stress related to significant life events may alter development of the hypothalamic-pituitary-adrenal axis and the chemicals released [8]. Upregulation of corticotrophin-releasing hormone has been implicated in several pain states and may exert its effect by acting on mast cells. Stress can also influence pain levels through dysregulation of serotonergic pathways and evidence suggests that sex hormones may modulate nociception and pain perception [9].

Classification of chronic pelvic pain syndromes

CPP conditions can be subdivided into pain syndromes (chronic primary pain) that have no obvious causative pathology and non-pain syndromes (chronic secondary pain) that have classical well-defined aetiology (e.g. infection, neoplasia). Pain syndromes are conditions in which pain is the main symptom and pain as a disease process is considered the cause.

Chronic primary pelvic pain syndromes (CPPPS) are a diagnosis of exclusion and refer to the occurrence of CPP when there is no proven infection or obvious local pathology accounting for the pain. The term syndrome encompasses the negative emotional, cognitive, behavioural, sexual and functional consequences of chronic pain and encourages a holistic approach to management with multidisciplinary input. In the absence of well-

defined aetiological mechanisms, CPPPS are classified by describing them in terms of their symptoms, signs and, where possible, investigations. This phenotyping has clinical and research validity and should include disturbances of organ or system function caused by changes in their control mechanisms. Spurious terminology must be avoided, especially if it implies an unproven causality. Terms that end in "itis" should only be used if infection and/or inflammation has been proven to be the cause of the pain.

Pain perception in CPPPS may focus on a particular pelvic organ/structure, may affect more than one pelvic organ and can be associated with systemic disorders such as chronic fatigue syndrome and fibromyalgia. When pain is localised to a single organ, some specialists use specific end-organ terms (e.g. primary bladder pain syndrome). For non-specific, poorly localised pelvic pain affecting more than one organ site, the generic term CPPPS should be applied. Despite a general tendency to move away from end-organ nomenclature, a diagnosis or name ascribed to a set of symptoms can provide patients with a sense of being understood, may help with acceptance of the problem as chronic, resolve unfounded fears about its implications and encourage engagement with therapeutic endeavours and self-management.

Prevalence

Pelvic pain syndromes increase with age but information on their true prevalence is limited by variations in diagnostic criteria, evaluation tools and symptom overlap with other conditions. Using a vague definition of continuous or episodic pain situated below the umbilicus over six months, one study reported that CPP was one of the commonest diagnoses in primary care ~~units~~ in Great Britain with monthly and annual prevalence rates of 21.5/1,000 and 38.3/1,000 respectively [10].

Functional disturbances

Sexual dysfunction
Studies of men with pelvic pain have reported higher chances of suffering from erectile and ejaculatory dysfunction [11]. Women with CPP have more sexual problems than patients with any other type of chronic pain disorder with sexual avoidance, dyspareunia and "vaginismus" most commonly reported [12]. Psychological factors (low self-esteem, depression, anxiety), physiological factors (such as fatigue, nausea and pain) and pain medications (opioids, selective serotonin re-uptake inhibitors) can contribute to loss of libido, delay ejaculation and affect sexual function.

Pelvic floor muscle dysfunction
An association between pelvic pain and muscular dysfunction (especially overactivity) is now recognised and has been reported in patients with CPP [13, 14]. Repeated or chronic muscular overload can activate trigger points within the pelvic floor and adjacent (abdominal, gluteal and iliopsoas) muscles. Trigger points are hyper-irritable spots within taut muscle bands that prevent full muscle lengthening and restrict range of movement. Pain is aggravated by trigger point pressure or sustained/repeated pelvic floor muscle contraction such as pain related to voiding or defecation.

Clinical assessment

History
CPPPSs are symptomatic diagnoses so history is key in evaluating patients ~~with CPP.~~ Specific disease-associated pelvic pain must be ruled out and red flag symptoms investigated by the relevant end-organ

specialist. Some patients can relate pain onset to an acute event such as surgery, sepsis or trauma, but for most it will be idiopathic. Burning is the commonest descriptor for neuropathic-type pain, but crushing and electric are also used. Patients may report the feeling of a swelling or foreign body, such as a golf or tennis ball, in the rectum or perineum.

Enquiring about pelvic organ function is important for phenotyping a patient's condition: lower urinary tract function and the influence of micturition on pain, anorectal function and the relationship between bowel habit and pain, sexual function and gynaecological symptoms. In women the temporal relation of pain to the menstrual cycle may help define the aetiology. A sexual history, including previous sexually transmitted infections, urethral/vaginal discharge, previous sexual trauma and a woman's cervical smear history is mandatory. A full urogynaecological history is important in individuals who have had continence or prolapse surgery using non-absorbable mesh. Dysfunction affecting two or more pelvic organs~~,~~ should raise suspicion of pelvic floor muscle dysfunction or central sensitisation

Determining disease severity, its progression and treatment response requires validated symptom-scoring instruments. They are recommended for basic evaluation and therapeutic monitoring of patients with CPPPS. Where the primary treatment outcome is pain relief, it is useful to agree a clinically meaningful level of relief before starting treatment. The most reliable methods for assessing pain are a five point verbal scale (none, mild, moderate, severe, very severe pain), a visual analogue special scale or a 0-10-point numerical scale. Generic QoL measures are important and the Brief Pain Inventory provides a broad assessment of the impact of pain on various aspects of life [15]. In males, sexual dysfunction can be evaluated using the International Index of Erectile Function and Premature Ejaculation Diagnostic Tool. The Female Sexual Function Index (FSFI) is a brief, multi-dimensional self-report instrument developed for assessing key dimensions of sexual function in women including desire, subjective arousal, lubrication, orgasm, satisfaction, and pain.

Direct questioning about the patient's view of what is wrong and their concerns can be more helpful than anxiety questionnaires. Anxiety about pain often refers to fears about missed pathology (particularly cancer) [6] or uncertainties about treatment and prognosis. Depression or depressed mood are common in chronic pain [16], often due to losses (work, leisure activities, social relationships, etc) related to chronic pain.

Physical evaluation

Clinical examination (with a chaperone present) helps to confirm or refute initial impressions gained from the history. Appropriate consent must be obtained including the risk of exacerbating pain during examination. Abdominal and pelvic examination including the external genitalia aims to exclude gross pelvic pathology and demonstrate sites of tenderness. Neurological examination is considered an integral part of the assessment and undertaken if appropriate. Many authors recommend assessing for cutaneous allodynia along the dermatomes of the abdomen (T11-L1) and the perineum (S3) and recording the degree of tenderness. The bulbocavernosus reflex in men provides information about the intactness of the pudendal nerves. A general musculoskeletal (tender point) evaluation, including muscles outside the pelvis, may help diagnose myofascial aspects of pelvic pain [17].

When assessing pelvic floor muscle function, a vaginal or rectal examination should be performed according to the International Continence Society report [18]. An internal examination is important for diagnosing pelvic organ prolapse and cervical abnormalities in women. Perianal dermatitis can be a sign of faecal incontinence or diarrhoea and anal fissures may be overlooked. Digital rectal examination is used to assess anal sphincter tone, the rectum, muscle tenderness and trigger points (including puborectalis), and prostate abnormalities including pain on palpation.

Investigations

There is no specific diagnostic test for CPPPS. Investigations are used to identify and exclude specific diseases associated with pelvic pain and for phenotypic description of pain syndromes. Investigations should be performed according to appropriate guidelines to exclude diseases with known aetiologies that present with symptoms identical to those of CPPPS.

Phenotyping
Given the polysymptomatic nature of CPPPS, clinical phenotyping systems can aid and standardise assessment of affected individuals by setting out series of domains that should be considered. Clinical phenotyping systems promote holistic patient care and potentially simplify treatment by promoting goal-directed multimodal therapy. UPOINTS is a promising system despite possibly under-assessing relevant psychological variables [19]. Having clear records for each system affected will significantly help support treatment.

Management

The management of CPPPS is based on a bio-psychosocial model with active patient involvement. Ensuring appropriate patient information and understanding improves adherence to treatment and underpins self-management. Single interventions rarely work in isolation and multimodal interventions addressing affected domains need to be considered within a personalised management strategy. A general overview of available treatment options is outlined below, with CPPPS-specific management detailed in subsequent sections. Where no evidence based treatments exist, CPPPS management should be underpinned by the principles that apply to other chronic pain disorders.

Physical therapy
Patients with CPP often have poor to absent pelvic floor muscle function [20] and stretching affected muscle groups helps regain length and function. Pelvic floor relaxation techniques taught by specialised physiotherapists can reduce pelvic floor over-activity and help interrupt the pain-spasm cycle. Myofascial trigger points can be treated by manual therapy and dry or wet needling, but strong evidence for effectiveness of these techniques is lacking [21, 22]. Encouraging chronic primary pain sufferers to remain physically active has general health benefits, but exercise has been shown to reduce pain and improve QoL especially for professionally-led supervised group exercise [23].

Psychological therapy
Early identification and management of psychological symptoms such depression and anxiety may ameliorate pain and reduce distress. Psychological interventions can be directed at the pain itself to reduce its impact on life or at adjustment to pain to improve mood, function and reduce healthcare use, with or without pain reduction [24]. A systematic review of the few heterogeneous trials of psychologically based treatment for pelvic pain found some short-term benefits for pain comparable to that achieved by pharmacotherapy, but this was not sustained at follow-up.

Pharmacological treatment
Few studies have investigated medications used for CPPPS [25], so the evidence for pharmacotherapy is derived from findings for general chronic pain. If drug benefit is limited by side-effects, then dose titration is used to determine the lowest effective dose.

Simple analgesia
Paracetamol is an antipyretic analgesic with a central mechanism of action that is well tolerated with few side effects [26]. Non-steroidal anti-inflammatory agents (NSAIDs) are anti-inflammatory, antipyretic analgesics that act peripherally by inhibiting the enzyme cyclooxygenase. NSAIDs have a higher incidence of side effects and evidence is lacking for their use in CPP.

Neuromodulators
Neuromodulators are used to modulate neuropathic or centrally mediated pain. The evidence for treatment of CPP is lacking but is present for other painful conditions. Several classes are available but all have side-effects that ~~may~~ limit use. The UK National Institute for Health and Clinical Excellence has reviewed the pharmacological management of neuropathic pain with recent guidance on neuromodulator use in chronic pain [23, 27].

Despite being an off-label indication, several antidepressants have been recommended for treating chronic primary pain. Tricyclic antidepressants have anxiolytic effects with multiple mechanisms of action including acetylcholine receptor blockade, inhibition of serotonin and noradrenaline re-uptake, and blockade of H1 histamine receptors. Amitriptyline is most commonly used with doses ranging from 10 to 150 mg/day but nortriptyline and imipramine are alternatives. Duloxetine is the only licensed serotonin-noradrenaline re-uptake inhibitor antidepressant with evidence for use in neuropathic pain. There is moderately strong evidence of benefit in diabetic neuropathy and fibromyalgia at a dose of 60 mg/day but side-effects often limit use [28].

The anticonvulsant carbamazepine has evidence of moderate benefit for neuropathic pain, but is no longer a first choice agent because of potentially serious side effects [29]. Other anticonvulsants include gabapentin and pregabalin for neuropathic pain, but the evidence for primary pelvic pains is limited with at least one publication suggesting gabapentin does not help. As a consequence, anticonvulsants are best administered by pain specialists familiar with their use.

Opioids
Although opioids may be beneficial in chronic non-cancer pain in small numbers of patients at low doses in a managed setting, there is mounting evidence of a limited role in this population. Opioids can have harmful effects on the endocrine and immune systems with a growing understanding of opioid-induced hyperalgesia, in which patients taking opioids paradoxically~~,~~ become more sensitive to painful stimuli [30]. Side-effects are common, including constipation, nausea, opioid tolerance and psychological changes, with the risk of harm increasing substantially at doses above 120mg/day morphine equivalence. Opioids should be administered by clinicians experienced in their use with arrangements made for formal monitoring, follow-up and review. Opioids Aware is an excellent web-based resource for patients and health care professionals (https://fpm.ac.uk/opioids-aware) [30].

Cannabinoids
The evidence base for cannabinoid use in pain is weak and well conducted trials are necessary [Moore et al., 2021].

Nerve blocks
Nerve blocks may have a diagnostic and therapeutic role for pain management, but the evidence base for these interventions for chronic non-malignant pain is weak [31]. Injection of local anaesthetic and steroid at a nerve injury site may produce therapeutic actions by blocking sodium channels and reducing inflammation and swelling.

Urological pain syndromes

Primary prostate pain syndrome
Primary prostate pain syndrome (PPPS) refers to persistent or recurrent episodic pain (reproduced by prostate palpation) for a minimum of 3 months with no proven infection or obvious local pathology. The terms chronic prostatitis and prostadynia should be avoided. No single aetiological explanation has been identified and PPPS probably develops in susceptible men exposed to unidentified initiating factors. Infection should be excluded

with microscopy and culture of voided urine pre- and post-prostate massage (two-glass test) being a useful bacterial localisation screening procedure [32]. In high risk men, PSA testing and MRI scanning may be considered after appropriate counselling. The National Institute of Health consensus classification of prostatitis includes infection (types I and II), which are best considered as specific disease-associated pelvic pain [33]. The National Institute of Health Chronic Prostatitis Symptom Index is a validated symptom-scoring tool.

Primary bladder pain syndrome

Primary bladder pain syndrome (PBPS) refers to persistent or recurrent pain perceived suprapubically in the bladder area, accompanied by at least one other symptom, such as worsening pain with filling, transient relief with voiding and daytime and/or night-time urinary frequency. Terms such as interstitial cystitis (IC) and painful bladder syndrome are no longer recommended.

The cause is thought to be an initial unidentified bladder insult leading to urothelial damage, neurogenic inflammation and pain. An infective cause has not been confirmed. Defects in the urothelial glycosaminoglycan layer have been implicated with a role for mast cell histamine release proposed. PBPS prevalence ranges from 0.06% to 30% with a female predominance (about 10:1) [34, 35] but no clear racial difference [36]. There is increasing evidence that children can be affected [37].

Urinalysis and urine culture (including culture for TB if sterile pyuria) should be checked with urine cytology also recommended in high risk groups. Pain in PBPS does not correlate with cystoscopic or histologic findings, but these are important for diagnosis, ruling out confusable conditions and defining phenotypes. The European Society for the Study of Interstitial Cystitis has suggested a standardised scheme of sub-classifications [38] to acknowledge differences and make it easier to compare various studies. The O'Leary-Sant Symptom Index (Interstitial Cystitis Symptom Index) is a symptom-scoring instrument validated in a large study [39]. Intravesical therapies and surgical intervention should be considered when conservative approach fails. Botulinum toxin application, neuromodulation and transurethral resection have been shown effective in subsets of patients.

Primary scrotal pain syndrome

Primary scrotal pain syndrome refers to persistent or recurrent episodic pain perceived within the contents of the scrotum. No specific pathology is identifiable, but an injury or intervention along the course of the ilioinguinal, genitofemoral and pudendal nerves that innervate the scrotum can cause pain perceived in that area. Urinary tract and sexually transmitted infections need to be excluded. Scrotal ultrasound does not help in diagnosis or treatment of scrotal pain, but excludes confusable conditions. Microsurgical denervation of the spermatic cord can be considered in patients who have failed conservative and pharmacological treatment, as it can provide good long-term symptomatic relief in patients with testicular pain responding to spermatic cord nerve block [40].

Primary urethral pain syndrome

Primary urethral pain syndrome can affect men and women and refers to chronic or recurrent episodic pain perceived in the urethra. As with PBPS, epithelial damage and neuropathic hypersensitivity following urinary tract infection are thought to be important. There is no specific treatment, but laser therapy of the trigone has been reported with good results [41].

Non-urological pain syndromes

Primary vulvar pain syndrome

Primary vulvar pain syndrome (PVPS) refers to pain in the vagina or female external genital organs that persists for more than 3 months and can be generalised or focal. It is poorly understood and usually precedes

dyspareunia. The terms vulvodynia and chronic vaginal pain are no longer recommended. In generalised PVPS, pain occurs in different areas of the vulva at different times and may be constant or intermittent. Touch or pressure does not initiate it but can exacerbate it. In focal PVPS, the pain is at the vaginal introitus and described as a burning sensation that only develops after touch or pressure, such as during penetration.

Primary anorectal pain syndrome

Primary anorectal pain syndrome (PAPS) refers to continuous, recurrent or episodic pain perceived in the anal canal and/or rectum in the absence of proven infection or local pathology. The Rome III criteria for functional anorectal pain disorders should be fulfilled for 3 months with symptom onset at least six months before diagnosis [42]. Pain may be continuous (chronic proctalgia) or intermittent with episodic cramping, aching or stabbing pain lasting several seconds to 30 minutes with no pain between episodes (proctalgia fugax). Most patients with intermittent PAPS do not report it to their physicians with pain attacks occurring less than five times a year in over half of patients. Bowel dysfunction is common with excessive straining, anal digitation in dyssynergic (paradoxical) defecation and a sensation of anal blockage reported by some affected individuals. During examination, exquisite tenderness during posterior traction on the puborectalis muscle ("levator ani syndrome") is thought to be due to pelvic floor muscle over-activity

Chronic post-surgical pain

Chronic post-surgical pain (CPSP) is defined as pain that develops or increases in intensity after a surgical procedure and persists beyond the healing process (more than 3 months). It has now been classified by ICD-11 as a chronic pain condition. Abdominopelvic operations with higher risk of CPSP include bariatric surgery, inguinal hernia repair, vasectomy, hysterectomy and caesarean section.

Post-vasectomy scrotal pain syndrome occurs in 2-20% of men who have undergone a vasectomy [43]. Underlying mechanisms are poorly understood, but the risk is significantly lower with no-scalpel technique [44]. Reversal of vasectomy can cure symptoms especially if patency is achieved. An RCT reported high rates of symptomatic improvement (80%) with pulsed radio-frequency to the ilioinguinal and genitofemoral nerves but follow-up was limited to 3 months. The evidence for epididymectomy is poor and is less likely to provide benefit if the epididymis has a normal sonographic appearance.

Post-inguinal hernia repair pain develops in up to 10% of patients at 6 months and may present with groin or scrotal pain. The risk is higher following laparoscopic rather than open surgery [45]. Limited evidence from case series has shown that neurectomy of damaged nerves can lead to symptomatic improvement.

The incidence of CPSP following hysterectomy is difficult to determine as pain is a common indication for the operation. Rates approximate 28% so careful case selection and management of patient expectation is important [46]. There is also a significant incidence of CPSP at 12 months following caesarean section so careful counselling is needed in non-emergency cases .

Flexible polypropylene plastic mesh implants developed and inserted to treat urinary stress incontinence and uterovaginal prolapse now carry a significant 'health and safety warning 'with complication rates close to 10% that include chronic pelvic pain, chronic infections, erosion into surrounding structures (vagina, bladder and urethra) and nerve and musculoskeletal damage [47, 48]. Mesh-related complications have a significant impact on patients 'QoL so early recognition is important. Mesh removal may be necessary in difficult-to-treat pain and should be provided within multi-disciplinary tertiary settings [49]. This is complex surgery requiring removal of dense scar tissue and reconstruction of the vagina, urethra and bladder, but can have beneficial and durable effects on chronic pain [50].

Conclusions

CPPPSs are symptomatic diagnoses that have significant negative impact on affected individuals. Specific disease-associated causes of pelvic pain need to be excluded. Phenotyping is important for diagnosis and provides an approach to management of affected patients by promoting goal-directed treatment of the functional, emotional, behavioural, sexual and physical consequences. Evidence based treatments for CPPPSs are limited so management follows the principles used for treating other chronic pain disorders.

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Abbreviations

CNS	Central nervous system
CPP	Chronic Pelvic Pain
CPPPS	Chronic primary pelvic pain syndrome
EAU	European Association of Urology
FSFI	Female Sexual Function Index
ICD-11	World Health Organisation International Classification of Diseases 11th Revision
PPPS	Primary prostate pain syndrome
PBPS	Primary bladder pain syndrome
PVPS	Primary vulval pain syndrome
PAPS	Primary anorectal pain syndrome
QoL	Quality of life
WHO	World Health Organisation

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