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Guideline No. 445: Management of Chronic Pelvic Pain

(En français : Gestion de la douleur pelvienne chronique)

The English document is the original version; translation may introduce small differences in the French version.

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This clinical practice guideline was prepared by the authors and overseen by the SOGC Clinical Practice Gynaecology committee. It was reviewed by the SOGC's Urogynaecology committee (now part of the Clinical Gynaecology Committee) and approved by the SOGC Guideline Management and Oversight Committee.

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This document reflects emerging clinical and scientific advances as of the publication date and is subject to change. The information is not meant to dictate an exclusive course of treatment or procedure. Institutions are free to amend the recommendations. The SOGC suggests, however, that they adequately document any such amendments.

Informed consent: Patients have the right and responsibility to make informed decisions about their care, in partnership with their health care provider. In order to facilitate informed choice, patients should be provided with information and support that is evidence-based, culturally appropriate, and personalized. The values, beliefs, and individual needs of each patient in the context of their personal circumstances should be considered and the final decision about care and treatment options chosen by the patient should be respected.

Language and inclusivity: The SOGC recognizes the importance to be fully inclusive and when context is appropriate, gender-neutral language will be used. In other circumstances, we continue to use gendered language because of our mission to advance women's health. The SOGC recognizes and respects the rights of all people for whom the information in this document may apply, including but not limited to transgender, nonbinary, and intersex people. The SOGC encourages health care providers to engage in respectful conversation with their patients about their gender identity and preferred gender pronouns and to apply these guidelines in a way that is sensitive to each person's needs.

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KEY MESSAGES

1. Health care providers should have a systematic approach to identify and address possible factors initiating and perpetuating pain such as gynaecologic, urologic, gastrointestinal, myofascial/musculoskeletal, and psychosocial contributors, as well as clinical findings suggestive of central sensitization/nociplastic pain.
2. Interventions that are supported by moderate to strong evidence include pain neuroscience education, physiotherapy, psychological treatment, tricyclic antidepressants, and menstrual suppression.
3. Interdisciplinary management based on a biopsychosocial model that includes psychological treatment, physiotherapy, and medical treatment, offers more comprehensive and effective care than stand-alone treatments for individuals with chronic pelvic pain.

RECOMMENDED CHANGES IN PRACTICE

1. Pain neuroscience education should be included as an important component of chronic pelvic pain management.
2. Chronic overlapping pain conditions should be identified and treated.
3. Management interventions deemed effective for generalized chronic pain are an option for chronic pelvic pain when nociplastic pain is likely.
4. Opioid medications are not recommended for long-term management of chronic pelvic pain.

ABSTRACT

Objective: To provide evidence-based recommendations for the management of chronic pelvic pain in females.

Target Population: This guideline is specific to pelvic pain in adolescent and adult females and excluded literature that looked at pelvic pain in males. It also did not address genital pain.

Benefits, Harms, and Costs: The intent is to benefit patients with chronic pelvic pain by providing an evidence-based approach to management. Access to certain interventions such as physiotherapy and psychological treatments, and to interdisciplinary care overall, may be limited by costs and service availability.

Evidence: Medline and the Cochrane Database from 1990 to 2020 were searched for articles in English on subjects related to chronic pelvic pain, including diagnosis, overlapping pain conditions, central sensitization, management, medications, surgery, physiotherapy, psychological therapies, alternative and complementary therapies, and multidisciplinary and interdisciplinary care. The committee reviewed the literature and available data and used a consensus approach to develop recommendations. Only articles in English and pertaining to female subjects were included.

Validation Methods: The authors rated the quality of evidence and strength of recommendations using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach. See online Appendix A (Tables A1 for definitions and A2 for interpretations of strong and conditional [weak] recommendations).

Intended Audience: Family physicians, gynaecologists, urologists, pain specialists, physiotherapists, and mental health professionals.

Tweetable Abstract: Management of chronic pelvic pain should consider multifactorial contributors, including underlying central sensitization/nociplastic pain, and employ an interdisciplinary biopsychosocial approach that includes pain education, physiotherapy, and psychological & medical treatments.

SUMMARY STATEMENTS:

1. Chronic pelvic pain is a common condition that can cause severe distress and considerable burden on the person affected as well as the health care system (*high*).
2. Etiology of chronic pelvic pain can be multifactorial and involve gynaecologic, urologic, gastrointestinal, myofascial/musculoskeletal, neuropathic, and psychosocial contributors as well as alterations in the central nervous system (*high*).
3. When pain becomes persistent and unresponsive to standard treatments, there is likely altered pain processing at the level of the central nervous system called central sensitization/nociplastic pain (*moderate*).
4. The therapeutic relationship between pain patients and their health care providers can impact their satisfaction with care and health outcomes (*moderate*).
5. Possible gynaecologic contributors to chronic pelvic pain include dysmenorrhea, endometriosis, adenomyosis, vulvodynia, and pelvic venous disorders (*moderate).*
6. Painful bladder syndrome and irritable bowel syndrome are chronic overlapping pain conditions that are common in the pelvic pain population and can be diagnosed based on clinical history (*high*).
7. Pelvic floor myofascial dysfunction is a frequent source of pain in women with chronic pelvic pain (*high*).
8. Myofascial abdominal pain, pelvic girdle pain, low back pain, referred hip pain, joint hypermobility, and postural imbalances may contribute to chronic pelvic pain (*Moderate*).
9. Pain, tingling, numbness following a dermatome may be suggestive of entrapment neuropathy (*moderate*).
10. Pain-specific psychosocial factors, such as pain catastrophizing and fear avoidance, and mental health conditions, including depression, anxiety, insomnia, and active trauma symptoms, contribute to perpetuating and intensifying chronic pelvic pain (*moderate*).

11. The presence of cutaneous allodynia, hyperalgesia, pelvic floor tenderness, and/or chronic overlapping pain conditions are suggestive of central sensitization/nociplastic pain (*moderate*).
12. Interdisciplinary management that includes psychological, physiotherapy, and medical treatment and is based on a biopsychosocial model offers more comprehensive and effective care than stand-alone treatments for individuals with chronic pelvic pain (*moderate*).

RECOMMENDATIONS:

1. Health care providers should have a systematic approach to identify and address nociceptive stimuli initiating and perpetuating pain, as well as clinical findings suggestive of central sensitization/nociplastic pain (*strong, moderate*).
2. Pelvic ultrasound should be offered as a low-cost investigation to identify some pelvic pathologies that may contribute to chronic pelvic pain (*strong, moderate*).
3. In the absence of studies specific to chronic pelvic pain, management interventions deemed effective for generalized chronic pain may be used for chronic pelvic pain when a centralized process is suspected (*conditional, low*).
4. Pain neuroscience education should be included as an important component of chronic pain management (*strong, moderate*).
5. Lifestyle changes including dietary modifications, exercise, and smoking cessation may be offered as part of chronic pain management (*conditional, low*).
6. Physiotherapy assessment and treatment should be offered for chronic pelvic pain management (*strong, high*).
7. Psychological treatments that should be included in management of chronic pain are cognitive behavioural therapy (*strong, high),* acceptance and commitment therapy (*strong, high*), and mindfulness meditation (*conditional, moderate*).
8. For insomnia symptoms, cognitive behavioural therapy for insomnia is the initial treatment of choice and is safer than prolonged use of sleeping medications (*strong, high*).
9. Nonsteroidal anti-inflammatory drugs should be recommended for the treatment of dysmenorrhea (*strong, moderate*), but are of unclear effectiveness for non-menstrual chronic pelvic pain (*conditional, low*).
10. Opioid medications are not recommended for long-term management of chronic pelvic pain (*strong, moderate*).
11. Tricyclic antidepressants should be considered for neuropathic pelvic pain (*moderate, strong*), painful bladder syndrome (*conditional, moderate*), and irritable bowel syndrome (*strong, moderate*).
12. Serotonin and norepinephrine reuptake inhibitors such as duloxetine (*conditional, moderate*) and venlafaxine (*conditional, low*) may be considered in chronic pain management.
13. Clinicians should consider alternative treatment options to gabapentin for the management of chronic pelvic pain without gynaecologic pathology; however, gabapentin may be an option in cases of neuropathic pain (*strong, moderate*).
14. There is currently insufficient evidence to recommend cannabinoids for chronic pelvic pain management (*conditional, low*).
15. Progestogens and gonadotropin-releasing hormone agonists or antagonists should be considered in the management of chronic pelvic pain, especially in the presence of cyclical exacerbation or endometriosis (*strong, moderate*).
16. Surgery for chronic pelvic pain may be offered after pain beliefs and treatment expectations are explored and with counselling about the rationale for surgery, the uncertainty of the evidence surrounding outcomes, and the possibility that pain could be unchanged or worse post-operatively (*strong, moderate*).
17. Targeted therapies such as trigger point injections and nerve blocks can be considered in specific clinical circumstances, if there is no response to modalities with higher evidence (*good practice point, low*).
18. Botox injection of the pelvic floor muscles may be considered in the presence of persistent spasm that has not responded to pelvic physiotherapy interventions (*conditional, low*).
19. Acupuncture may be considered as a complementary modality for chronic pelvic pain (*conditional, low*).
20. There is a need in Canada for more publicly funded programs where health care providers work together in an interdisciplinary approach to treat chronic pelvic pain (*strong, moderate*).

INTRODUCTION

Definition and Epidemiology

Chronic pelvic pain (CPP) is a common and often debilitating problem that is estimated to affect 15% of adult females worldwide.[1] In Canada each year, CPP is responsible for a considerable number of inpatient admissions and accounts for 10% of indications for hysterectomy.[2,3] Additional outpatient and indirect productivity costs are estimated to be much higher.[4] As such, CPP carries a large economic burden for those afflicted as well as for the health care system.

CPP has been defined as pain symptoms perceived to originate from pelvic organs/structures typically lasting more than 6 months, though the most recent International Association for the Study of Pain (IASP) classification of chronic pain has shortened the time criteria to 3 months.[5] CPP is associated with symptoms suggestive of lower urinary tract, sexual, bowel, myofascial, or gynaecologic dysfunction and can have negative cognitive, behavioural, sexual, and emotional consequences.[6] CPP is therefore complex, multifactorial, and can be challenging to treat. It can cause severe distress and impact all aspects of a person's life. This guideline was developed to provide clinical tools and evidence-based recommendations for the assessment and treatment of CPP. This document does not address genital/vulvar pain.

Summary Statement 1

Etiology of Chronic Pelvic Pain

The female pelvis contains many visceral, musculoskeletal, nerve, and soft tissue structures that are challenging to examine individually, and any of which can be involved as precipitators or ongoing contributors to CPP. Some of the most common contributors are listed in Table 1. Since there is often a suspicion of gynaecologic contributors in women with pelvic pain, the gynaecologist is often the first specialist to assess these patients. As such, gynaecologists should have a systematic approach to identifying the common contributors to CPP and providing a plan of management for this problem.

The traditional approach of focusing only on the identification and treatment of organ-related signs and symptoms, has been shown to be limited in scope when dealing with chronic pain. In most cases of CPP, the contributors to pain are multifactorial, and can include organ-specific causes, neuromusculoskeletal contributors, and psychosocial factors. Following the latest IASP classification, CPP may present as a chronic primary pain, when no other diagnosis accounts for the symptoms, or can also occur as a chronic secondary pain, where pain persists despite the presumed primary cause having been treated.[5] In many cases, the primary cause (or provoking pain) can be identified, such as a history of severe dysmenorrhea, persistent dyspareunia, or recurrent bladder infections.[5] Such conditions can be considered examples of peripheral nociception, leading to peripheral sensitization. Hyperalgesic priming from these repetitive painful experiences may contribute to wind-up of the central nervous system and a transition to a centralized chronic pain state.[7] Endometriosis-associated chronic pain is a good example of chronic secondary pain, where complex neural mechanisms can contribute to the persistence of pain beyond the site of the lesion, even with adequate treatment of the lesion.[8]

This classification of pain recognizes that while identifying and diminishing ongoing peripheral irritants is important, it should also be recognized that when pain becomes persistent and unresponsive to standard treatments there is likely altered pain processing at the level of the central nervous system (i.e., central sensitization/nociplastic pain). Both peripheral and central sensitization/nociplastic pain can occur with primary and secondary pain.

Summary Statement 2

Central Sensitization/Nociplastic Pain

The abnormal pain processing associated with chronic pain has been called central sensitization/nociplastic pain, which is a condition of the nervous system associated with the development and maintenance of chronic pain related to a process called wind-up and a persistent state of high reactivity.[9] This in turn lowers the threshold for what causes pain and may result in the persistence of pain even after the initial injury has healed.[9]

ABBREVIATIONS

CPP	Chronic pelvic pain
IASP	International Association for the Study of Pain
IBS	Irritable bowel syndrome
MTrP	Myofascial trigger point
NSAID	Nonsteroidal anti-inflammatory drug
PBS/IC	Painful bladder syndrome/Interstitial cystitis
PNE	Pain neuroscience education
SNRI	Serotonin and norepinephrine reuptake inhibitor
SSRI	Selective serotonin reuptake inhibitors
TCA	Tricyclic antidepressant

Table 1. Pain contributors in chronic pelvic pain

Category	Possible Pain Contributors
Gynaecologic	Endometriosis, adenomyosis, vulvo/vestibulodynia, pelvic venous disorders, chronic pelvic inflammatory disease, pelvic adhesions, adnexal masses, uterine fibroids, and/or ovarian remnant
Gastrointestinal	Irritable bowel syndrome, chronic constipation, chronic appendicitis, and/or inflammatory bowel disease
Urological	Painful bladder syndrome/interstitial cystitis, recurrent urinary tract infection, and/or chronic renal/bladder stones
Musculoskeletal and myofascial	Pelvic floor, abdominal wall, pelvic girdle, hip, and lumbosacral disorders, hernias, and/or hypermobility syndromes
Neuropathic	Ilioinguinal/iliohypogastric neuropathy, pudendal neuropathy, radiculopathy, and/or entrapment/neuromas
Psychosocial and mental health factors	Active trauma symptoms, depression, anxiety, insomnia, and/or pain catastrophizing

This has also been recently described by the IASP as *nociplastic* pain, a type of pain different from the currently recognized nociceptive (tissue injury) and neuropathic (nerve injury) pain. It is defined as "pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain."[10] Large-scale genetic and epigenetic studies have concluded that a predisposition to developing chronic pain is heritable. However, there are likely many other factors that influence this process that have yet to be fully identified.[11,12] These include endocrine, neural, immune, and hormonal influences. Adolescents may be particularly susceptible to altered neural development, and thus adolescence may be a critical window for intervention to prevent the development of long-term pain morbidity in adulthood.[13]

The transition from peripheral nociception to central sensitization/nociplastic pain can be identified by the presence of two clinical signs: (1) allodynia, which is the presence of pain from a non-painful source, such as a cotton swab drawn across the skin; and (2) hyperalgesia, which is the representation of excess pain from a painful stimulus.[14] When these are present, other organ systems may also be affected through cross-sensitization.

CPP can be associated with chronic overlapping pain conditions, a cluster of conditions occurring mostly in females, that is believed to reflect cross-sensitization and a centralized pain process.[15] The conditions included in the spectrum of chronic overlapping pain conditions are endometriosis, vulvodynia/vestibulodynia, painful bladder syndrome/interstitial cystitis (PBS/IC), irritable bowel syndrome (IBS), chronic low back pain, chronic tension or migraine headaches, temporomandibular disorder, fibromyalgia, and chronic fatigue syndrome/myalgic encephalomyelitis.

When central sensitization/nociplastic pain is present, CPP becomes systemic disease process that must be managed as a chronic illness.[16] Physicians managing CPP not only need to reduce the peripheral irritants or nociceptive stimuli initiating and perpetuating the pain but also be aware of the complex presentation from other parts of the pain system. This complex system disease therefore requires a comprehensive and multimodal approach which can benefit from input from many specialties and allied health professionals.

Summary Statement 3

APPROACH TO EVALUATION OF PATIENTS WITH CHRONIC PELVIC PAIN

Based on our current understanding of CPP, the aims of the evaluation should be to establish a therapeutic relationship, identify pain contributors, and look for clinical findings suggestive of central sensitization/nociplastic pain. The systematic assessment of patients with CPP is outlined in Table 2 and Table 3 (history and physical exam).

Recommendation 1

Therapeutic Relationship

The therapeutic relationship between pain patients and their health care providers can impact their satisfaction with care and their health outcomes.[17] As such, the evaluation of patients with CPP requires a balance between

being thorough and systematic while conducting an empathetic inquiry into the patient's lived experience and goals of therapy. It is important to validate the patient's experience while eliciting their beliefs about their pain and their treatment expectations to ensure that these are addressed during the evaluation process (Table 2).

Specific questions about the impact of CPP on quality of life can help a patient feel validated and reassured that their pain will be taken seriously and not dismissed and stigmatized,[18] which in turn builds the therapeutic alliance, provides the rationale for a multidisciplinary approach, and contributes to improved treatment satisfaction.

A trauma-informed care approach[19] is a strengths-based framework that is responsive to the impact of trauma and assumes an individual is more likely to have a history of trauma.[20] The approach acknowledges the importance of physical, psychological, and emotional safety in the relationship between provider and survivor, and promotes trust, choices, collaboration, and empowerment (see Appendix B).

Diversity and inclusion practices, including gender-inclusive language, also contribute to safer medical encounters for all patients, including those with chronic pain.

Summary Statement 4

History and Physical Examination

In taking a thorough intake history for CPP, clinicians may find it useful to request that patients fill out a pre-appointment questionnaire. The International Pelvic Pain Society patient history questionnaire may be useful for this purpose and is available in several languages.[21] Several questionnaires and physician tools for evaluation of patient history and psychosocial factors are available and listed in Appendix B.

The history and physical examination are important tools to help identify peripheral nociceptors as well as clinical findings suggestive of central sensitization/nociplastic pain. Tables 2 and 3 outline the approach to clinical history taking and physical examination of patients with CPP. Physical exams, in particular pelvic examinations, may be a source of apprehension for many patients with pelvic pain. It is helpful to reassure patients early in the encounter that an examination will only be performed as tolerated, that they will be in control of the examination at all times, and that a pelvic examination will be performed only if it will contribute to the management plan and only when the patient feels ready, with a chaperone should they want one.

Investigations

There are a limited number of investigations that may be useful in evaluating patients with CPP. A pelvic ultrasound is a low-cost investigation that will identify any uterine pathology, such as fibroids or adenomyosis, as well as adnexal pathology, such as endometriomas or other ovarian cysts. In most cases, MRI does not provide additional information and should not be ordered routinely but may have a role in cases where deep endometriosis is suspected.[22] If bladder symptoms are predominant, a urinalysis with or without culture should be done. Cervical swabs may be appropriate if pelvic infection is suspected as a contributor.

The role of laparoscopy as an investigation tool is discussed under the therapeutic section of this document.

Recommendation 2

PAIN CONTRIBUTORS

While there are many possible precipitators and perpetuators of CPP, some of the more commonly encountered pain contributors are described in this section. A detailed literature review of all these contributors is beyond the scope of this guideline.

Gynaecologic Contributors

Dysmenorrhea

Dysmenorrhea is a common symptom, experienced by over 90% of females at some point in their lives, and reported as moderate to severe in one-third to one-half.[23,24] Severe dysmenorrhea, especially if untreated, is a potential risk factor for the development of CPP, due to hyperalgesic priming and wind-up of the nervous system. Dysmenorrhea can also be a perpetuator or aggravator of CPP through cross-sensitization. A recent meta-analysis of the relationship between primary dysmenorrhea and chronic pain found 32 eligible studies and concluded that, overall, dysmenorrhea was associated with 2.50 (95% confidence interval, 2.02−3.10) times the odds of chronic pain, which did not differ by chronic pelvic versus chronic non-pelvic pain, community versus clinical populations, or different geographical regions.[13] Given that adolescence is a sensitive period for neurodevelopment, addressing dysmenorrhea with effective treatments may help in

Table 2. Assessment of patients with chronic pelvic pain: clinical history

Assessment Techniques	Description
Building a therapeutic relationship	• Avoid pain stigma (e.g., pain is "all in the head" or caused by past sexual abuse) • Validate the pain (e.g., rather than saying "there's nothing there," provide a fuller explanation of the pain) • Active listening (i.e., let the patient tell her story then summarize back) • Empathy (e.g., "That must be very difficult"; "I am sorry you have experienced that")
Pain History (OPQRST)	• Onset: Initiating event (e.g., surgery, cyst rupture, miscarriage), duration and frequency • Provoking/palliating: Movement, intercourse, menstrual cycle, and medications • Quality: Sharp, cramping, and/or burning • Region/Radiation: Diffuse, localized, unilateral, and/or radiates to back, hip, groin, or vulva • Severity: Elicit numeric rating (0−10) for pain at specific times. • Treatment: What medications/surgeries/treatments have been tried and/or are currently using? What is the effectiveness and are there side effects? • History of pelvic surgeries: Targeted to pain (laparoscopy, -ectomies), duration of any relief; other pelvic surgeries and any temporal relation to pain onset
Psychosocial factors questions (FIFE)	• Function: Ask about pain impact (e.g., "How has this pain impacted your life?") • Ideas: Elicit beliefs about the pain (e.g., "What do you believe is causing this pain?") • Fears: Explore pain catastrophizing (e.g., "Do you have any anxious or worrisome thoughts about this pain?") • Expectations: Explore the hopes for this visit and treatment expectations (e.g., "What kind of results are you hoping to get from this treatment?")
Assessment of pain contributors: gynaecologic	• Dysmenorrhea: Primary (at menarche) or secondary, response to suppression • Dyspareunia: (introital and/or deep), pain with arousal or orgasm, pain with tampon insertion, unprovoked vulvar pain (dermatomal pattern, improved by sitting on toilet?), vaginal discharge/pruritis, clitoral pain • Other: Infertility, pregnancies (relationship to pain), history of sexually transmitted infections, abnormal Pap tests

Table 3. Assessment of patients with chronic pelvic pain: physical examination

Assessment Techniques	Description
Overall	• Affect, gait, mobility, and pain behaviour
If tender area found, ask if it reproduces usual pain	
Back/pelvis	• Screen for pelvic girdle pain*, pelvic asymmetry, hip exam (e.g., FABER, FAIR)
Abdomen	• Scars (tender?), allodynia test with cotton swab* • Localized tender areas (Carnett's test to identify abdominal wall pain* • Look for masses or hernias
Pelvic exam	• Offer chaperone • Obtain permission (e.g., "Are you okay to have an internal exam now or at a future appointment"?) • Frequent check-ins during exam (e.g., "Are you okay? Can we continue with the exam?") • Vulvar skin examination for lesions or skin conditions • Cotton swab test for provoked vestibulodynia and dermatomal pain S2−S4 (vulvodynia) • Single digit vaginal exam of the pelvic floor muscles (Figure 1)*, anterior vagina for bladder tenderness, cervix, adnexae, cul-de-sac tenderness and/or nodularity • Bimanual exam of uterine size, position, tenderness, mobility, adnexal masses/tenderness • Rectovaginal exam if deep endometriosis of bowel is suspected or to determine extent of any cul-de-sac nodule palpated • Speculum exam (if bimanual exam tolerated) for the examination of the vagina, cervix, cuff, look for discharge, prolapse
Basic investigations	• Pelvic ultrasound (as an initial evaluation for pelvic pathology) • Urinalysis (if bladder symptoms) • Vaginal wet prep and cervical swabs (if infection suspected)

FABER: flexion, abduction, and external rotation; FAIR: flexion, adduction, internal rotation.
*see Supplementary videos in references 59 and 60.

preventing both chronic pelvic and non-pelvic pain. Management of dysmenorrhea is well described in the 2017 SOGC guidelines.[24]

Endometriosis

Endometriosis is the most common diagnosis associated with CPP and is defined as the presence of endometrial-like tissue outside of the uterus.[25] A history of severe dysmenorrhea, deep dyspareunia, cyclical pelvic pain, and infertility are suggestive of this condition. Relief of pain with cycle suppression or pregnancy may also suggest endometriosis. Cyclical dysuria and/or hematuria, dyschezia, and/or rectal bleeding may suggest more advanced disease. In such cases, the clinical examination may reveal tender nodularity in the cul-de-sac or a fixed, retroverted uterus, and pelvic ultrasound may show an endometrioma. A specialized ultrasound or MRI scan may be indicated to detect the extent of deep disease. If endometriosis is suspected or identified, hormonal suppression is usually offered as a first-line treatment, according to the current SOGC guidelines, but if this treatment fails or is contraindicated, there is a role for laparoscopic evaluation and treatment.[25]

Adenomyosis

Adenomyosis is another commonly diagnosed condition associated with pelvic pain. It is described as endometrial-like tissue that is found to infiltrate within the myometrium. Adenomyosis may present as severe dysmenorrhea and heavy menstrual bleeding and a tender bulky uterus on examination. It is frequently associated with endometriosis (20%−80%) but also occurs in isolation, typically presenting later in life, often after pregnancy or uterine surgery, but it can also present in younger patients.[26] Diagnosis can be made with sonography or MRI. Treatment is usually suppressive medical therapy, and hysterectomy cures uterine symptoms.[27]

Uterine Fibroids

Uterine fibroids (leiomyomas) are very common benign tumours, estimated to occur in 70%−80% of people with a uterus by age 50.[28] They are often asymptomatic, but can cause pain due to degeneration, torsion, and, when large, may lead to pressure symptoms as well as bowel and bladder dysfunction. Fibroids frequently coexist with endometriosis and adenomyosis, both causes of pelvic pain, rendering it difficult at times to determine the contribution of fibroids to pain symptoms. When fibroids are detected by ultrasound, the clinicians should be prompted to consider the possibility that these other conditions may also be present.

Pelvic Venous Disorders

Pelvic venous disorders have been associated with CPP and are thought to be secondary to venous hypertension caused by either reflux in the pelvic veins (pelvic congestion) or obstruction of the pelvic veins (May-Thurner syndrome, Nutcracker syndrome) .[29] Symptoms suggestive of venous hypertension include pelvic heaviness or pain, often aggravated by prolonged standing or activity, relieved by recumbency, exacerbated during the premenstrual period, and often associated with prolonged post-coital ache.[30] Physical findings are scarce but may include perineal and vulvar varicosities, cervical and uterine tenderness, and ovarian point tenderness. When the renal vein is involved, there may be left flank pain and hematuria[29]. Diagnostic tests include dynamic/Doppler ultrasound, CT, MRI, or venography scans. Criteria for diagnosis are evolving and under active review by an international working group.[29] Treatment options include medication for ovarian suppression, hysterectomy, and interventional radiology procedures.[31,32] The literature on treatment outcomes is mostly composed of case series and therefore considered of low quality.

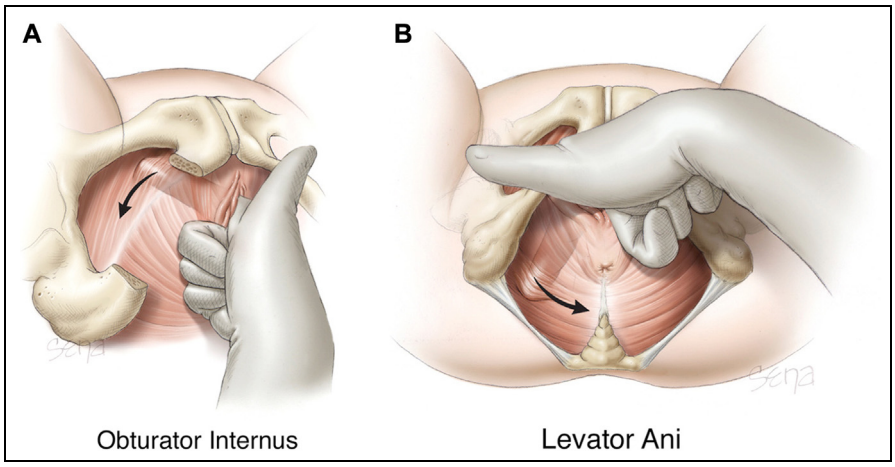
Vulvodynia

Vulvodynia is a common overlapping pain condition defined as vulvar pain lasting longer than 3 months, without clear identifiable cause, which may have potential associated factors.[33] Localized provoked vestibulodynia is a subtype of vulvodynia that is the most commonly associated with CPP.[34,35] It presents with pain at the vestibule, with introital pain on contact with clothing, during intercourse, tampon insertion, pelvic examination, sitting, or bike riding. The pain is often described as burning, stinging, irritation, rawness. or itching.[36,37] Typically, a cotton swab test is performed to identify areas of point tenderness corresponding to dysesthesia or allodynia[38]. Vulvodynia can also be spontaneous or unprovoked, and generalized to other areas of the vulva.[39] This challenging problem is outside the scope of the current guideline.

Pelvic Adhesions

Pelvic adhesions may be suspected in a patient who has had recurrent episodes of sexually transmitted infection or pelvic inflammatory disease or extensive pelvic surgery. Uterine or adnexal fixation on examination or dynamic

Figure 1. Internal palpation of the right obturator internus and levator ani performed with the index finger of the dominant hand.



Note: Obturator internus palpation is performed with the index finger of the dominant hand, which is placed on the centre of the muscle belly. Then use a sweeping motion along the length of the muscle following the orientation of the muscle proceeding counterclockwise towards the levator ani. Similar techniques are used on the left side. Permission to reproduce was granted by Elsevier; license number 5593641311178.

ultrasound may be suggestive of adhesions. The link between adhesions and CPP remains unclear, as surgery targeting adhesions has been shown to be ineffective in relieving pelvic pain in a systematic review and is not recommended.[40]

Summary Statement 5

Urinary Contributors

PBS/IC is generally accompanied by irritative symptoms, which include frequency, urgency, and nocturia. While other etiologies should be considered when patients present with these symptoms, PBS is one of the chronic overlapping pain conditions that can coexist with endometriosis and has been noted to be present in about half of patients in the gynaecologic pelvic pain population.[41] Patients with PBS often have bladder base tenderness (i.e., anterior vaginal wall tenderness) on pelvic examination and can have concomitant pelvic floor dysfunction. Though a diagnosis of IC was previously made based on the presence of cystoscopic findings (e.g., Hunner's ulcers and glomerulations), a clinical diagnosis of PBS without cystoscopy can be made based on an unpleasant sensation (pain, pressure, discomfort) perceived from the urinary bladder and associated with lower urinary tract symptoms for at least 6 weeks, in the absence of infectious or other causes.[42] While the 2016 Canadian Urological Association (CUA) guideline recommends cystoscopy to rule out other conditions, it is considered optional in young female patients with PBS without risk factors for other conditions or malignancy (e.g., hematuria, smoking) .[43] Objective testing (e.g., hydrodistension) is also considered optional, and urodynamics are not recommended as part of routine evaluation.[43]

Patients with mostly obstructive urinary symptoms may have underlying pelvic floor dysfunction, which is discussed in the section on musculoskeletal contributors.

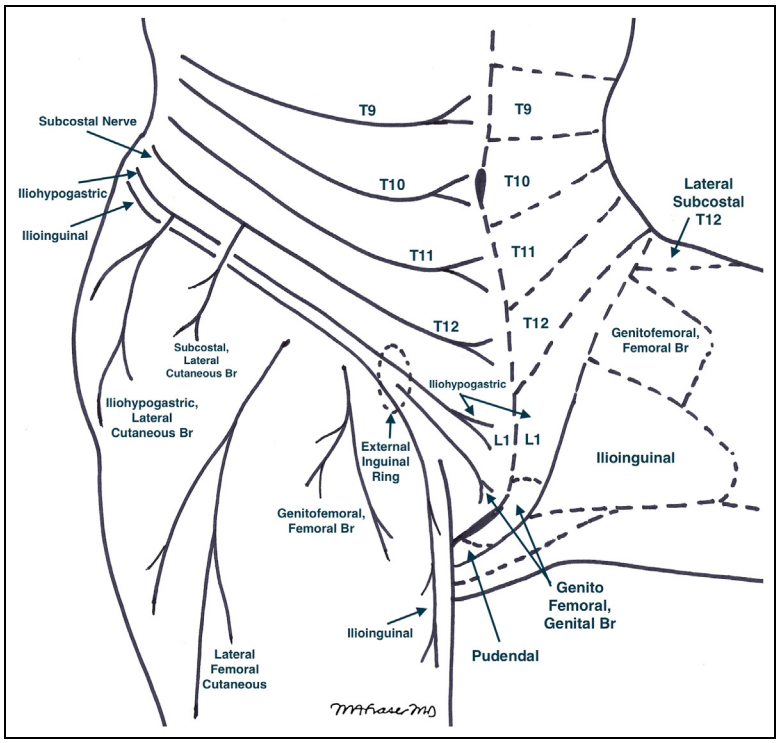
Gastrointestinal Contributors

IBS is a disorder of gut–brain interaction that can be present in nearly half of patients with CPP. It can coexist with endometriosis and many of the other chronic overlapping pain conditions via central sensitization/nociplastic pain or cross-organ sensitization.[41] The diagnosis of IBS is based on clinical history.

In 2019, the Canadian Association of Gastroenterology (CAG) released guidelines for IBS.[44] IBS is diagnosed using Rome IV criteria as recurrent abdominal pain, on average, at least 1 day per week in the last 3 months (with onset >6 months), associated with at least 2 of the following criteria: 1) related to defecation; 2) associated with a change in stool frequency; 3) associated with a change in stool form.[45] IBS can be further subtyped into "predominant constipation," "predominant diarrhea," "mixed bowel habits," or "unclassified."[45] There are no specific findings on physical examination.

While colonoscopy is not required for diagnosis, it may be warranted in patients with risk factors for malignancy (i.e., alarm features), such as persistent non-cyclical

Figure 2. Dermatomes of Ilioinguinal, iliohypogastric, and genitofemoral nerves. (Art by Dr Margaret Fraser MDCM, FRCPC and permission granted to reproduce)



hematochezia or family history of gastrointestinal cancer, although the CAG suggests against routine colonoscopy in IBS patients aged <50 years.[44]

Summary Statement 6

Musculoskeletal Contributors

The musculoskeletal system is a recognized contributor to pelvic pain and includes muscle, fascia, ligaments, joints, or bones.[46]

Pelvic Floor Myofascial Pain

Pelvic floor myofascial pain is a frequent contributor to CPP, with studies finding the presence of pelvic floor tenderness in 58%–79% of the CPP population[47–49]. It is therefore very important that the pelvic floor muscles be assessed routinely.[50,] Meister et al.[51] describe a standardized approach to pelvic floor assessment that had good inter-observer validity. With a single digit inserted intra-vaginally, mild pressure is exerted in the centre of the muscle then swept along its length, proceeding along the obturator internus and levator ani in a counterclockwise fashion (Figure 1). The patient is asked to report any pain and rate its intensity and whether it reproduces usual symptoms.[50,51] The assessment of tenderness should also be extended to the piriformis and internal obturator muscle.[51] Higher sensitivity with palpation is significantly associated with pain severity in CPP[41,52] and with indicators of central sensitization/nociplastic pain.[53,54] Increased pelvic floor muscle tone and elevated resting activity are also found in women with CPP as assessed with palpation and electromyography, respectively.[47,48] Pelvic floor muscle contractility and ability to relax should also be examined, as difficulties in contracting, controlling, and relaxing this musculature are common in women with CPP.[47,48]

Overall, the pelvic floor muscles are directly and indirectly related to CPP; directly, as the muscle can be a source of pain due to sustained increased tension and consequent adaptive modifications in viscoelastic properties; and indirectly, because, by becoming chronic, pain may lead to the onset of viscero–visceral or viscero–muscular reflexes (i.e., cross-sensitization), affecting structures that share the same nervous segmentation.[55]

Summary Statement 7

Abdominal Myofascial Pain

Abdominal myofascial pain can manifest as tenderness in multiple points of the abdominal muscles and could be

highlighted with the Carnett's test (i.e., palpation of point of tenderness worsens pain with abdominal wall contraction)[56].

In addition to tenderness, examination of the symptomatic region can reveal myofascial trigger points (MTrPs). They consist of small hyperirritable nodules located on taut bands of skeletal muscle that are in a sustained state of contracture.[57] They may be painful on their own or with provocation. They may be found along the abdominal wall or pelvic floor in patients with CPP.

Examination of the abdominal wall can also identify hernias or tender scars which may be pain contributors.

Pelvic Girdle and Hip Pain

Pelvic girdle pain such as sacroiliac joint dysfunction and pubic pain may be sources of CPP. The following signs can be observed in women with CPP: asymmetry in iliac crest and pubic symphysis height,[47,56] positive findings on sacroiliac joint provocative tests such as the flexion, abduction and external rotation test (Table 3) .[47,56] Conditions affecting the hip joint such as labral tears, femoroacetabular impingement, hip deformity, arthritis, synovitis, stress fractures, osteoarthritis, congenital hip dysplasia, and osteonecrosis may cause local or referred pain in women with CPP.[58] The pain is usually unilateral and worse with lying on the affected side, weight-bearing activities, walking, running, stair climbing, sitting, pivoting, and squatting. It may be relieved by lying on the opposite side with a pillow between legs or on lying on the back with knees supported. The hip can be assessed with flexion, abduction and external rotation and flexion, adduction, and internal rotation tests.

For supplementary videos for these components of the musculoskeletal exam, see references.[59,60]

Other Conditions

Joint hypermobility can be related to CPP as it may lead to instability of the pelvic girdle and, consequently, the pelvic floor muscles may compensate with abnormally increased tone becoming a source of pain.[61] Systemic conditions affecting connective tissue, such as hypermobility syndromes, can contribute to pain. Postural imbalances, including spine misalignments, as well as a more pronounced kyphosis and lumbar lordosis may also be found in women with CPP.[62] Low back pain, such as discopathy, radicular pain, or non-specific low back pain, are often found in those with CPP and may also cause referred pain to the pelvic area and, as such, need to be considered as a possible contributor to CPP.[63]

Summary Statement 8

Neuropathic Pain

The pelvis and abdominal wall are richly innervated (Figure 2). Trauma or damage to any of these nerves can generate a neuropathic pain contributor. Neuropathic pain is pain caused by a lesion or disease of the somatosensory nervous system.[64] This distinguishes it from pain thought to be due to a nociplastic process and avoids the confusion and overlap that has been associated with the term *neuropathic* in the past.

The pain quality from neuropathy is often described as burning, electric shocks, or painful cold and is associated with tingling, pins and needles, numbness, and itching (DN4 criteria). Possible causative factors include pelvic or abdominal surgery, prolonged sitting, high intensity sports, diabetes, or herpetic infections.[65]

Nerve entrapment syndrome, or compression neuropathy, is a clinical condition caused by compression of a single nerve or nerve root.[66] Since nerve fibers crossing the pelvis are branches of the lumbosacral plexus, most intrapelvic nerve entrapments will produce symptoms in the lumbosacral dermatomes, such as sciatica or pudendal and gluteal pain. Since the sacral nerve roots give origin to both somatic and parasympathetic nerves, entrapments of such roots will produce pain on their somatic dermatomes, along with urinary and bowel dysfunction.[67] In such entrapments, pelvic pain can be a secondary symptom, associated with hyperactivity of the pelvic floor muscles, also innervated by these nerve roots. Entrapments of the pudendal nerve and its roots will cause genital, gluteal and/or sciatic pain and are outside the scope of the current guideline.

The most common nerves of the lumbosacral plexus that produce CPP as a primary symptom are the ilioinguinal and iliohypogastric nerves. In some cases, genitofemoral neuropathies will produce groin pain with some pelvic component (Figure 2). These entrapments can be caused by transverse abdominal incisions or laparoscopic port incisions,[68] inguinal or spigelian hernias or surgeries to correct them,[69] inguinal endometriosis,[70] or neoplasms.[71] Since entrapments are unilateral in most cases, physical examination will reveal a difference in sensation in the

nerve dermatomes on each side, as well as hyper/hypoesthesia and/or hyper/hypoalgesia and/or allodynia on the affected nerve dermatome. After a topographical hypothesis has been formulated, a confirmatory diagnostic block is recommended. The block should be performed with low-volume anesthetics, specifically directed at the suspected point of entrapment. Bupivacaine or ropivacaine should be used because of their longer duration anesthetic effects. A positive block is characterized by the complete resolution of the radicular pain for the duration of the anesthetic effect.[72]

Interventional protocol for ilioinguinal, iliohypogastric, and genitofemoral neuropathies may start with a sequence of nerve blocks,[73] followed by pulsed radiofrequency,[74] radiofrequency ablation,[75] and, finally, surgical transection (laparoscopic or open) if none of the previous conservative therapies have worked.[63] The evidence for these interventions is of very low quality with findings mostly derived from case series.[74,76]

Medications such as gabapentinoids, tricyclic antidepressants (TCAs), and serotonin and norepinephrine reuptake inhibitors (SNRIs) are also helpful for neuropathic pain and discussed further under pharmacologic interventions.[77]

Summary Statement 9

Psychosocial Contributors

There are reciprocal and complex relationships between trauma, pain-specific factors, mental health, and CPP.

Post-Traumatic Symptoms

While a history of childhood sexual abuse has not been established as a cause of CPP in adulthood,[20] adverse childhood experiences (ACEs) are associated with numerous negative health outcomes in adulthood including widespread pain and pelvic pain.[78] Specifically, the presence of active post-traumatic symptoms appears to be the risk factor for developing and maintaining chronic pain.[78,79] Moreover, in chronic pain, adults with exposure to ACEs show improvement in pain and function in interdisciplinary pain treatment programs.[80] It is incumbent upon the provider not to assume past abuse has caused CPP or that past trauma and its impact have not been treated. Rather, by adopting a trauma-informed care approach, the provider can collaboratively engage with the patient on their view of the role of past trauma on current pelvic pain (See Table 2).

Pain-Specific Factors

Often, patients are most motivated to address the current aspects of their pain experience. Pain-specific factors that have been implicated in pelvic pain include pain stigma,[81] pain catastrophizing,[82,83] fear avoidance,[84] pain coping,[85] lack of pain self-efficacy,[86] perceived injustice,[87] and spousal responses to pain.[88] Pain catastrophizing is a cognitive response to pain that involves three components: rumination about the pain, magnification about the cause and future consequences of the pain, and helplessness about the pain. Fear avoidance is a behavioural response for dealing with pain that involves fear of movement and fear of pain, leading to disuse, deconditioning, and avoidance of day-to-day valued activities. As such, it may be helpful to discuss pain beliefs[89] and treatment expectations,[90] and later, the potential aim of the person's self-management of CPP.[65]

Mental Health

In addition to considering active post-traumatic symptoms, depression and suicidal ideation, anxiety, and insomnia are common consequences of pain, including CPP[91-93] and thereafter, can become pain perpetuating factors. Personality disorder features (e.g., emotional dysregulation) can significantly increase the complexity of caring for patients with chronic pain.[94] Safety issues such as housing, food security, and domestic violence may also be limiting factors in recovery from CPP. More resources for screening and managing mental health contributors can be found in Appendix B.

Summary Statement 10

Diagnosing Central Sensitization /Nociplastic Pain

Though there are many research tools used to demonstrate evidence of central sensitization/nociplastic pain (e.g., functional MRI, qualitative sensory testing) there are currently no definite clinical tools to diagnose this condition. The presence of allodynia and reduced pressure pain thresholds are accepted as strongly suggestive of pain sensitization.[95,96] A recent study also showed that presence of pelvic floor tenderness was highly correlated with lower pressure pain thresholds in CPP patients, suggesting this could be a marker for a centralized pain process.[53] Central sensitization/nociplastic pain is an accepted explanation for chronic pain when certain conditions are met. That is, when peripheral nociception is unlikely to explain the pain after a comprehensive diagnostic research or when a non-painful stimulation of the affected parts of the body leads to pain and hyperalgesia.[95,97] Generalized hyperalgesia and

the reduction of pain threshold in zones remote to pelvic cavity are often present and may have important implications for clinical management of women with CPP.[56,58] The sensitization of neighbouring structures resulting in dysfunctional responses from the unaffected organs, termed *cross-sensitization*, is often a feature as well.[98] This cross-sensitization and central sensitization/nociplastic pain manifests as the presence of multiple comorbidities, as previously mentioned, and now recognized as chronic overlapping pain conditions.[15]

Recently, it has been reported that the Central Sensitization Inventory (CSI) questionnaire was associated with the presence of 3 or more pain comorbidities (e.g., chronic overlapping pain conditions) with 78% sensitivity and 80% specificity in patients with endometriosis.[99] This tool may prove useful in a clinical setting, though further research is required to validate it.

Summary Statement 11

MANAGEMENT OF CHRONIC PELVIC PAIN

In this section, we review the management of CPP from the perspective of a general practitioner and/or gynaecologist and their patient. Once a therapeutic alliance is formed, a patient-centred management plan can be created to decrease disability by aiming to improve emotional, physical, and social functioning, rather than focus strictly on pain reduction. The final treatment plan will likely consist of a combination of organ-specific treatments for peripheral contributors and treatments directed to address the psychosocial aspects of pain and central sensitization/nociplastic pain. The management plan may require collaboration with allied health professionals qualified to deliver some of the treatments mentioned. As evidence specific to CPP is often lacking, management interventions deemed effective for generalized chronic pain may be used for CPP when a centralized/nociplastic process is suspected, when there is no evidence specific to CPP available.

Recommendation 3

Lifestyle and Participatory Interventions

Pain Neuroscience Education

Pain neuroscience education (PNE) is an educational strategy that adheres to the biopsychosocial model of pain and helps people redefine persistent pain. In chronic pain, PNE has demonstrated outcomes of reduced pain, improved patient knowledge, improved function, lower disability, reduced psychosocial factors, enhanced movement, and decreased health care utilization.[100,101] In CPP, women show significant improvement in neurophysiology knowledge with PNE[101] (see Appendix B).

Physicians are well positioned to provide PNE concerning pain beliefs, pain catastrophizing, and treatment expectations. They can move away from solely visceral or tissue-based explanations for pain, avoid using alarmist language about investigations, validate the patient's pain even in the absence of positive test findings, reassure them that something sinister has not been missed, and explain the neurophysiological influence of the psychological and social aspects of the pain experience.

Recommendation 4

Health Behaviour Change

The health behaviour changes required to recover from persistent pain are numerous and complex.[102] The provider can encourage their patient to make changes or adopt new health behaviours by supporting patient-led goals.[103] To be successful, the patient must view their goals as important and meaningful[103] to their recovery and have the confidence to carry out the goals. Providers can use motivational interviewing or other communication and goal-setting strategies to support patient behaviour change (See Appendix B).

There are many lifestyle factors that have shown to be associated with chronic pain; therefore, some suggested health behaviour changes may include dietary modifications, exercise, and smoking cessation.[104,105] Dietary changes that may promote improvement in chronic pain include a whole foods diet,[106,107] or dietary interventions specific to pain contributors, such as the low-FODMAP diet for IBS and IC diet for painful bladder syndrome.[41,44] Exercise and movement have been shown to have some benefit for pain severity and physical function in chronic pain, though the quality of evidence is low[108].

Recommendation 5

Physiotherapy Treatments

Physiotherapy management is embedded in a biopsychosocial framework in which education is a

component.[109] The impact of physiotherapy, therefore, goes beyond the musculoskeletal system, as significant changes in psychological factors (e.g., fear of pain, depressive symptoms, catastrophizing), blood flow, and pain sensitivity have been seen after treatment.[110,111]

Pelvic physiotherapy in particular entails several modalities and approaches, of which myofascial manual therapy has the highest level of evidence. Myofascial manual therapy, consisting of internal and external techniques applied to the pelvic floor and surrounding myofascial structures, has shown a significant effect on global response assessment, pain intensity, quality of life, fear avoidance, and disability in women with CPP.[111–115] Even though manual techniques are performed by the pelvic physiotherapist during the treatment sessions, it should be highlighted that patients actively take part in therapy by performing home stretching exercises and following recommendations. Importantly, this approach has shown not only clinical effects on pain but also significant systemic effects on central sensitization/nociplastic pain, as demonstrated with objective physiological testing.[116]

To further target pelvic floor muscle trigger points or tenderness, the use of a massage wand was shown to provide benefits in women with CPP.[117,118]

Pelvic floor muscle exercises assisted with biofeedback also provide benefit to women with CPP.[115,119]

Multimodal body awareness and movement therapy including movement patterns, posture, relaxation, and functional respiration has shown a significant effect on pain in women with CPP, when compared with usual care.[120,121]

Electrotherapy, including intravaginal electrical stimulation[122] and percutaneous tibial nerve stimulation,[123] may be helpful in reducing pain in women with CPP.

Recommendation 6

Psychological Treatments

Given the significant impact of CPP on quality of life and the contribution of psychosocial factors and mental health in the maintenance of CPP, psychological treatments are an essential and well-established component of treatment for CPP.[124,125]

Effective psychological treatments for chronic pain are cognitive behavioural therapy,[126] acceptance and commitment therapy,[127] and mindfulness meditation.[128] These interventions can result in improved mood, decreased catastrophizing, improved function, increased self-efficacy, and reduced pain. For impact of pain on sexual function, there are specific and effective psychological treatments that draw upon cognitive behavioural therapy, mindfulness, and sex therapy strategies.[129] For insomnia symptoms, cognitive behavioural therapy for insomnia is the initial treatment of choice and is safer than prolonged use of sleeping medications.[130]

Recommendations 7 and 8

Pharmacologic Interventions

Analgesics

Traditional analgesics may be useful in the management of CPP, especially in the setting of acute pain flares. Nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen are often used as foundational analgesia, while opioid receptor agonists/antagonists should be used with caution in chronic pain management.

Nonsteroidal anti-inflammatory drugs

A systematic review by Latthe et al. found that NSAIDs can reduce moderate and severe pain associated with dysmenorrhea when compared with placebo.[131] The risks of scheduled use include adverse effects on the renal, hepatic, gastrointestinal, and cardiovascular systems, with increased risk of bleeding, hypertension, gastrointestinal upset. and constipation.[132] The evidence for NSAIDs for non-menstrual CPP is unclear.[133]

Recommendation 9

Acetaminophen

While the exact mechanism of action is unknown, acetaminophen is believed to inhibit cyclooxygenase pathways and the synthesis of prostaglandins in the central nervous system. Like NSAIDs, acetaminophen has analgesic and antipyretic properties but lacks peripheral anti-inflammatory properties.[134] With chronic use (>7 days), the maximum daily dose should not exceed 3000 mg. With short-term use (3–4 days) doses of up to 4000 mg per day

are generally well tolerated, though dose-related hepatotoxicity is a major known risk of acetaminophen.[135] There are no studies on acetaminophen effectiveness for CPP. A systematic review on its use in chronic pain found no or little efficacy.[135]

Opioid medications

Many opioid medications, both synthetic and natural derivatives of the opium poppy, are widely used to treat pain. Opioids work by binding to opioid receptors in the central and peripheral nervous system.[136] Opioid medications are effective for acute pain and malignant pain but their use in the treatment of chronic non-cancer pain remains highly controversial and recent research shows evidence against chronic opioid therapy for CPP.[137] Patients who use long-term opioids are at risk of developing tolerance, physical dependence, addiction, and pain sensitization.

The 2017 Canadian Guideline for Opioids for Chronic Non-Cancer Pain provides recommendations, best practice statements, expert guidance, and risk mitigation strategies for prescribing opioids to patients with chronic non-cancer pain.[137] In this document, important information can be found outlining considerations for prescribing opioids to any patient including, first, optimizing use of non-opioid medications for the management of chronic pain; screening for the risk of misuse; following a framework for a trial of opioids (initiation, monitoring of response, and discontinuation if improvement in pain and function is not achieved); recommended "watchful dose" limits; and opioid tapering/discontinuation strategies for an unsuccessful trial of opioid medications. Tramadol became available for use in Canada in 2005 and has quickly become a frequently prescribed analgesic medication because providers believe it has a low addiction liability and favorable safety profile.[138] Tramadol is a synthetic opioid and produces dual-analgesic effects, acting synergistically as a weak μ-opioid receptor agonist and also as a SNRI. It produces analgesia by affecting the nociceptive process and boosting the central modulation of pain. Cochrane meta-analyses have found tramadol to be efficacious in neuropathic pain (low-quality evidence). Coadministration of tramadol with proserotonergic medications can result in a hyperserotonergic state that develops soon after initiation or dosage changes. Serotonin syndrome can be subacute or chronic and range from mild to severe. Some of the serotonergic medications that can interact with tramadol include selective serotonin reuptake inhibitors (SSRIs), SNRIs, and TCAs, which are all commonly prescribed for the treatment of chronic pain conditions. Finally, despite assumptions among some prescribers that tramadol does not have an addiction liability, it can produce euphoric, stimulant, and relaxing effects that increase its abuse potential.

Naltrexone is a competitive opioid antagonist, with high affinity for the mu receptor. At doses of 50−100 mg daily, naltrexone is used for alcohol and opioid use disorders. Low-dose naltrexone has been used for the treatment of chronic pain conditions, such as fibromyalgia and complex regional pain syndrome but there are no studies on its effectiveness for CPP.[139]

Recommendation 10

Neuromodulators

Central sensitization/nociplastic pain and maintained chronic pain pathways between the periphery and the brain support the use of multimodal therapy and centrally acting medications to modulate nociplastic pain and treat chronic pain.[140] Most evidence for cited in this section is extrapolated from the chronic pain literature, as there is limited evidence specific to CPP.

Antidepressants

Although antidepressants may indirectly improve the pain experience by enhancing mood, they also have a direct analgesic effect, as both depressive symptoms and pain are modulated by the neurotransmitters serotonin and norepinephrine.[141]

Tricyclic antidepressants

Tricyclic antidepressants (TCAs) are a first-line treatment for neuropathic chronic pain, in part, because they increase the amount of norepinephrine in the descending modulatory pain pathways.[77] A Cochrane systematic review found TCAs to be effective with a number needed to treat of 3.6 and relative risk of 2.1 for the achievement of at least moderate pain relief.[142]There is also good evidence for using TCAs for the management of IBS (diarrhea predominant) and PBS/IC symptoms.

While their effectiveness in many chronic pain conditions is well established, the anticholinergic side effect profile does limit the tolerability of TCAs for many patients. Generally, the rule "start low and go slow" should be applied to TCAs. This approach will reduce early discontinuation and improve compliance, as most TCAs need to

be given at a moderate dose for at least 8 weeks before they are declared ineffective.

Amitriptyline is the most well studied TCA for chronic pain. However, nortriptyline and desipramine may have slightly better tolerability.

Recommendation 11

Serotonin and norepinephrine reuptake inhibitors

Duloxetine and venlafaxine increase norepinephrine in the descending modulating pathways. Duloxetine has been identified as an effective pain modulator in several pain syndromes, including diabetic peripheral neuropathy, fibromyalgia, chronic musculoskeletal pain, and urologic pelvic pain.[143] Venlafaxine has also been found to be effective for chronic pain with a number needed to treat of 3.1 and relative risk of 2.2 for moderate pain relief.

There is limited evidence for the use of SSRIs (e.g., sertraline), noradrenergic and dopaminergic pump inhibitors (e.g., buproprion), and serotonin-2 antagonist/reuptake inhibitors (e.g., trazadone) in CPP.

Recommendation 12

Gabapentinoids

Gabapentin and pregabalin are both calcium channel blockers that decrease the reuptake of glutamate, norepinephrine, and substance P and operate as membrane stabilizers peripherally and centrally. Side effects are common, notably drowsiness, dizziness, and peripheral edema. We have found no studies looking at pregabalin use in CPP.

One study of women with CPP has suggested that gabapentin alone or in combination with amitriptyline provides better pain relief than amitriptyline alone.[144] In women with CPP without known gynaecologic pathology, gabapentin had been shown to improve pain symptoms in one pilot RCT, which prompted a larger trial.[136] This adequately powered, multicentre, randomized, double-blind, placebo-controlled trial in 39 UK hospital centres with 306 participants, showed that treatment with gabapentin did not result in significantly lower pain scores in women with CPP and no obvious pelvic pathology and was associated with higher rates of side effects than placebo.[136] Clinicians should therefore consider alternative treatment options to gabapentin for the management of CPP.

Recommendation 13

Cannabinoids

Cannabinoids have been playing an increasing role in the treatment of pain. The legalization of recreational cannabis in Canada, in October 2018, has increased its accessibility and use in the general population. Cannabis acts on the endocannabinoid system, which is involved in pain sensation, appetite, mood, and memory.[145] The uterus has many CB-1 receptors, which are increased by the elevated progesterone levels in the luteal phase.[146] Patients with endometriosis have reduced CB-1 expression and higher endocannabinoid ligand levels. Elevated endocannabinoid ligand levels are also noted in patients with dysmenorrhea and dyspareunia,[145] suggesting a role of the endocannabinoid system in the development of endometriosis and related pain disorders.

Nabilone is a synthetic cannabinoid approved in Canada for use as an antiemetic for chemotherapy recipients and used off-label as an adjunct for chronic pain. A recent review of the evidence for use of nabilone in chronic pain showed no effectiveness over placebo, but one guideline had a weak recommendation as a third-line agent for chronic neuropathic pain.[147]

There is considerable variation in the types of cannabis products, levels of active compounds, and routes of administration, creating challenges in determining effectiveness of specific treatment regimens. There is low-quality evidence for their use in chronic pain conditions, but there is no information on their use in CPP. The Canadian Pain Society included cannabinoids as third-line treatments for chronic neuropathic pain behind first-line agents such as gabapentinoids, TCAs, and SNRIs, and second-line agents, including tramadol and controlled-release opioids.[148] However, the IASP recently published a position statement recommending against the use of cannabinoids for pain relief because of the current lack of high-quality clinical evidence.[149] There is a pressing need for preclinical and clinical studies to better

understand the pharmacokinetics and efficacy of these compounds.

Recommendation 14

Topical Therapies

Capsaicin's proposed mechanism of action is basic desensitization of the unmyelinated C nerve fibers found to contribute to some pelvic pain syndromes. A systematic review has suggested capsaicin may be beneficial for some patients in the areas of hyperaesthesia or allodynia, though long-term efficacy requires further evaluation. A Cochrane review of high concentration topical capsaicin (5% or more) in neuropathic pain patients (N = 2488) found the number needed to treat to be 8.8−12, with 10% of patients showing improvement.[150] Authors of the review found high susceptibility to publication bias. Local skin reactions were more common with capsaicin, which were usually tolerable and attenuated with time.[150] Other compounded topical preparations that include medications such as lidocaine, amitriptyline, gabapentin, diclofenac, and ketamine have limited evidence for use.[151]

Targeted Treatments

Gynaecologic Contributors

Hormonal medication. For patients who have a gynaecologic contributor to their chronic pain, such as dysmenorrhea, endometriosis, or adenomyosis, menstrual cycle suppression can be an important treatment strategy in decreasing the overall pain experience. In patients with cyclical exacerbations of pain, first-line therapy is a combined hormonal contraceptive (CHC),[24,25,30] while progestins are also commonly used. Continuous CHC treatment for dysmenorrhea decreases pain duration when compared with cyclic use.[152] A review of non-surgical treatments for CPP by the Cochrane group in 2014 found 2 RCTs that showed progestogen (medroxyprogesterone acetate) was more effective than placebo, achieving a greater than 50% reduction in visual analog pain scores immediately after treatment and effectiveness up to 9 months after treatment.[153]

For women unable to achieve benefit from CHCs or progestins, gonadotropin hormone-releasing hormone (GnRH) agonist therapy may be an effective option for menstrual suppression. Head-to-head comparisons showed that women treated with goserelin (a GnRH agonist) had greater improvements in pelvic pain scores at one year than those taking progestogens.[153] Mood and sexual function were also improved with goserelin. Randomized controlled trials have also demonstrated GnRH agonist therapy to be effective for endometriosis-associated pelvic pain.[154] Using add-back hormone replacement therapy along with GnRH agonists diminishes adverse effects without impacting effectiveness. GnRH antagonists have also been shown to improve endometriosis-associated pelvic pain scores in an RCT when compared with placebo.[155]

Recommendations 15

Surgery

In case series of CPP patients with normal preoperative evaluation (e.g., examination with or without pelvic ultrasound), laparoscopy revealed pathology in 27%−58%, with endometriosis and adhesions being common findings.[156,157] A potential advantage of laparoscopy is to "see and treat" at the same time as diagnosis. However, the therapeutic benefit of surgery for CPP is contentious. In a 2014 Cochrane meta-analysis, the authors found that laparoscopic ablation/excision of endometriosis was associated with decreased overall pain (i.e., pain not specific to CPP) compared with diagnostic laparoscopy.[158] However, an updated 2020 Cochrane review found uncertain benefit regarding the effect of laparoscopic excision/ablation of endometriosis versus diagnostic laparoscopy for overall pain.[159] Furthermore, the issue of endometriosis excision versus ablation[160] and the role of adhesiolysis[40] or presacral neurectomy[161] remain controversial in the management of CPP. Meta-analyses have concluded that the evidence for laparoscopic uterosacral nerve ablation has an uncertain or no effect on pelvic pain.[162,163] Hysterectomy can be helpful in some cases of CPP; that is, if a uterine source of pain is suspected or to eliminate hyperalgesic priming from recurrent dysmenorrhea. However, the co-existence of other pain generators (e.g., myofascial) may be associated with persistent pain post hysterectomy.[164,165]

A systematic review of the literature showed no effectiveness of lysis of adhesions for chronic pelvic and abdominal pain.[40] In general, the therapeutic role of surgery is unclear in cases where a patient may have a gynaecologic pathology (e.g., endometriosis) in addition to central sensitization/nociplastic pain and non-gynaecologic pain generators. It is possible that surgical treatment of a peripheral gynaecologic nociceptive source may secondarily reduce central sensitization/nociplastic pain.[166] However, it is also possible for central sensitization/nociplastic pain to become

independent or autonomous of the original peripheral nociceptive source, such that treatment of the latter does not modify central pain.[167] There is also a risk that post-surgical chronic pain may developing following the intervention.[168] Higher levels of catastrophizing also seems to predict persistent pain after surgery.[169]

In the absence of clear data from the literature, we recommend that surgery for CPP be used with caution. In the counselling process, the rationale for surgically treating gynaecologic pain (i.e., to reduce peripheral input to the central nervous system and thus perhaps also reduce central sensitization/nociplastic pain) can be discussed. Yet, patients should also be aware of the uncertain impact of such surgery on CPP and that it is possible that pain may not change or even worsen when central sensitization/nociplastic pain has become autonomous from peripheral pain sources.

Recommendation 16

Painful Bladder Syndrome/Interstitial Cystitis

The health care provider can make the diagnosis of painful bladder syndrome and can make appropriate referrals or initiate therapy. As per the CUA, the mainstay of therapy includes education and support as well as physiotherapy in those with pelvic floor dysfunction (Grade A) in addition to avoidance of dietary triggers, bladder retraining, and/or psychological treatment (Grade B). In the 2016 CUA document, a range of pharmacologic, intravesical, and other treatments are discussed and are beyond the scope of this guideline.[43] Among these treatments, gynaecologists should be aware of the risk of maculopathy with pentosan polysulfate sodium.[170,171] However, pharmacological options (Grade B) that a gynaecologist may be comfortable initiating are TCAs (amitriptyline) and cimetidine.[43]

Irritable Bowel Syndrome

The health care provider can make the diagnosis of IBS and can make appropriate referrals or initiate therapy. As per the CAG, initial management may involve a low-FODMAP diet, psyllium soluble fibre, peppermint oil, and probiotics.[44] Referral for cognitive behavioural therapy or hypnotherapy may also be made.[44] Pharmacological options that a gynaecologist may be comfortable initiating include antispasmodics, low-dose TCAs, and SSRIs.[44] Intermittent use of loperamide may be an option for diarrhea-predominant IBS, but it should not be used continuously.[44] Linaclotide is an option for constipation-predominant IBS.[44] The CAG gave conditional recommendations for these treatments, except for psyllium, low-dose TCAs, and linaclotide (strong recommendations).[44] Physiotherapy with biofeedback training has also been recommended (Grade A) for patients with defecatory dysfunction (constipation).[172]

Musculoskeletal Contributors

Physiotherapy is the cornerstone therapy for musculoskeletal pain contributors. Because of its wide benefit for individuals with CPP and requirement of active participation, it is described in the section on lifestyle and participatory intervention.

Injection Therapies

Trigger Point Injections

Manual therapy, dry needling, and direct infiltration with saline, anesthetics and/or steroids are commonly used methods for managing MTrPs.

Trigger point injections can be considered in patients with MTrPs after manual therapy has not been successful or the patient is not able to tolerate other therapies because of pain. However, a small (n = 30) RCT by Montenegro et al. demonstrated superiority of trigger point injection with local anesthetic compared with ischemic compression by physical therapy.[173] In this study, women with other underlying pain conditions such as endometriosis were excluded. Injection of local anesthetic into the affected area is thought to reduce the transmission of nerve signals along these hyperactive networks and provide some relief. This may allow time for the management of the original pain generator (e.g., dysmenorrhea) to be treated and ultimately lead to longer term pain control as well.

Pelvic floor trigger point injections were studied in a small series of 18 women with a success rate of 72% when combined with pelvic floor education for CPP with levator ani trigger points.[174]

Injections may be done in the office setting after appropriate training. However, a detailed description of techniques is beyond the scope of this guideline.[175] However, key principles include:

- Understanding the indications and contraindications for trigger point injections;
- Understanding the principles, dosing, and potential toxicities of local anesthetics; and
- Avoiding the use of epinephrine in trigger point injections (to avoid potential of muscle necrosis).

Nerve Blocks

Nerve blocks for chronic pain are well established and utilized for a multitude of pain conditions.

Nerve blocks may be surface landmark based or image guided (i.e., ultrasound, fluoroscopy, or CT guided). Anesthetics such as lidocaine are most commonly used, although the application of steroids have also been described. The evidence among gynaecology patients is limited to case series, but a few randomized controlled trials exist for pudendal neuralgia management,[176] which is beyond the scope of this guideline.

Ultimately, the risks and benefits of nerve blocks will continue to be based on an individual assessment after having trialed conservative options.

Common nerve blocks that are used in patients with CPP are described here.

Nerves of the abdominal wall include ilioinguinal, iliohypogastric, and genitofemoral nerves, as these provide sensory innervation to the skin bordering the thigh and abdomen. They are also known as *border nerves*.

There is limited data to support the use of these approaches in the gynaecologic population. A Canadian case series of 131 patients seen for CPP in a gynaecology clinic and treated with ilioinguinal/iliohypogastric nerve blocks for abdominal wall pain, found that over 80% of patients reported pain reduction lasting on average 3.5 weeks.[73] There were no major adverse events reported, and all patients with underlying conditions such as endometriosis were included.

Superior hypogastric plexus and ganglion impar blocks are usually reserved for cases where other treatment modalities have failed and offered under fluoroscopic guidance by experienced providers, usually anesthesiologists or radiologists.

Recommendations 17

Botulinum Toxin Injection

Botulinum toxin A significantly inhibits the release of the neurotransmitter acetylcholine from nerve fibers, resulting in short-term paralysis. Botulinum toxin A has been used to treat pelvic pain disorders by interrupting persistent muscle contractions. In addition to the inhibition of acetylcholine and subsequent muscle contraction, botulinum toxin A also blocks other neurotransmitters that regulate pain, including glutamate, substance P, and calcitonin gene-related peptide, and may contribute to its analgesic effect.

A systematic review of the effect of botulinum toxin injection of pelvic floor muscles on CPP found 11 eligible studies out of 20 that were screened, 2 were RCTs, involving 62 women, and 9 were observational studies, involving 466 women.[132] Great heterogeneity existed in the definition of CPP, the dose of botulinum toxin used, and the outcome measures. This systematic review showed that there is insufficient evidence to recommend pelvic floor muscle botulinum toxin injection as a cost-effective treatment for CPP; however, its safety can be assured. Future studies of higher quality and with higher dosages along with pelvic physiotherapy are indicated.

Recommendation 18

Acupuncture

There has been one systematic review of acupuncture for female CPP (4 randomized trials) .[177] Meta-analysis of 2 of the trials was performed for acupuncture combined with conventional treatment, which showed reduced pain compared with conventional treatment alone. However, the trials were rated as low quality.

Recommendation 19

Multidisciplinary and Interdisciplinary Care

Considering the complexity of CPP and its many contributors, a multimodal approach to care is advisable. The superiority of multidisciplinary care for CPP compared with standard care has been studied and demonstrated in a randomized controlled trial.[178] Interdisciplinary care, defined as integrated care being delivered at a single site by a collaborative team of care providers from multiple disciplines, is likely preferable for management of complex pain problems.[179] If not available in their area, the family doctor or gynaecologist can initiate multidisciplinary care by identifying allied health care providers in their community, particularly pelvic physiotherapists, psychologists/counsellors, and dieticians, who have an interest in CPP. Communication between gynaecologists, family physicians and these allied health care providers helps to promote integration of care. Gynecologists should also seek

collaboration with other specialists with pain-focused practices, where available, such as urologists, gastroenterologists, physiatrists, interventional radiologists, anesthesiologists, and pain medicine specialists and pain clinics.[65]

Based on several decades of chronic pain research, an interdisciplinary program that includes PNE, psychological treatment, physiotherapy treatment, and medical care and is based on a biopsychosocial model[180] can offer more comprehensive and effective care than stand-alone conventional medical treatment for individuals with chronic pain,[181–183] including CPP.[184–186] Considering the complexity of the problem, its multifactorial nature and the potential usefulness of various treatments outlined above, there is a need in Canada[187] for more programs where health care providers work together in an interdisciplinary approach to treat CPP.

Summary Statement 12 and Recommendation 20

SUMMARY

The management of chronic pelvic pain has been improved by a better understanding of the mechanisms of this pain and an optimized integrated approach to patient care. The concept of organic lesions responsible for persistent nociceptive input has gradually been replaced by that of dysregulation of nociceptive messages derived from the pelvis and perineum. CPP is generally no longer derived from the organs but is expressed via these organs. CPP with dysfunctional sensory and central processing, is recognized as a systemic disease process that, like other chronic diseases, is to be managed as opposed to a local problem with a simple fix.

A biopsychosocial perspective that considers the ways in which biological, psychological, and social factors work independently and jointly to affect a person's experience is the most effective conceptualization and guide for effective treatment. A multidisciplinary or interdisciplinary approach to assessment and treatment with a focus on improving emotional, physical, and social functioning instead of focusing strictly on pain reduction is more effective in decreasing disability. This is best achieved by determining the patient's needs and perspective through a patient-centred approach.

There is robust literature on the management of chronic pain but most studies on CPP specifically are observational or of low quality. Further research in this area, with additional focus on prevention or progression of disease, is urgently needed .

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.jogc.2023.102283

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