Green-top Guideline No. 41
Peer Review Draft – June 2026

The Management of Chronic Pelvic Pain

Scott C Mackenzie | Una Pak | Katy Vincent | Rajvinder Khasriya | Andrew Horne | on behalf of the Royal College of Obstetricians and Gynaecologists

Correspondence: clincicaleffectiveness@rcog.org.uk

This is the 3rd edition of this guideline, which was previously published in 2005 and 2012 under the title *The Initial Management of Chronic Pelvic Pain*.

Key recommendations

- Clinicians should recognise chronic pelvic pain (CPP) is a multifactorial condition and aim to identify contributory factors rather than assign causality to a single pathology. [Grade C]
- Clinicians should recognise that many women with CPP have altered central nervous system function, with widespread implications including increased pain sensitivity, fatigue, visceral symptoms, and psychological impact. [Grade C]
- Clinicians should introduce and explain the multifactorial nature of CPP at the outset, working in partnership with the woman to develop a shared care plan. [Grade D}
- Clinicians should perform relevant investigations to rule out potential underlying pathologies. However, the likelihood of normal findings should be emphasised to the woman from the start. [Grade D]
- If the history suggests a specific non-gynaecological contributor to pain or if red flag features are present, clinicians should consider referral to, or collaboration with, relevant multidisciplinary team members. [Grade D]
- A multidisciplinary CPP team should provide expertise in gynaecology, pain medicine, physiotherapy and psychology [Grade D].
- Lifestyle factors (e.g. diet, physical activity, and sleep) should be addressed as part of a multimodal approach, and discussions should be broached sensitively [Grade D].
- Pain education should be included in the management of chronic pelvic pain. Clinicians should explain the biopsychosocial model of CPP to women, highlighting how biological, psychological, and social factors interact to influence the pain experience [Grade D].
- Opiates should not be prescribed by gynaecologists for CPP [Grade D].
- Women with CPP who undergo a diagnostic laparoscopy that does not reveal a cause for their symptoms should be offered follow-up care in gynaecology to explore alternative approaches [Grade D].

1. Purpose and scope

This guideline comprises recommendations on the care of women with chronic pelvic pain (CPP), including dysmenorrhoea and dyspareunia. It addresses contributing factors, clinical assessment and management strategies, emphasising informed decision-making and a multimodal approach. It is relevant to all healthcare professionals involved in the care of individuals with CPP, including those

in general practice, gynaecology, gastroenterology, urology, surgery, pain medicine, sexual health, physiotherapy, dietetics and mental health.

This guideline is intended to apply to women aged 16 and over, and to post-menarchal girls under 16. We primarily use the terms "woman" and "women" for simplicity; however, it applies to all individuals assigned female at birth, regardless of gender identity, acknowledging that gender identity may differ from sex assigned at birth.

2. Introduction and background epidemiology

Chronic pelvic pain (CPP) is a complex and often disabling condition that affects up to one in four women worldwide (1). It significantly impacts the daily functioning, productivity, and quality of life of women, contributing to psychological distress, social isolation, and increased use of healthcare services (2, 3).

Estimates of CPP prevalence vary globally from 5.7% to 26.6% due to differences in study definitions and methodologies (4). United Kingdom (UK) based studies from 2001 and 2016 reported rates of 24% and 14.8%, respectively (5, 6). CPP is thought to account for around 20% of gynaecology consultations resulting in a considerable demand on healthcare services (1, 7). There is currently limited epidemiological evidence to determine whether specific population subgroups are disproportionately affected by CPP.

A 2022 systematic review described the economic impact of CPP on both individuals and healthcare systems, with indirect costs contributing substantially (8). In Australia, the estimated annual cost per woman ranges from $16,970 to $20,898 AUD (approximately £8,500 to £10,500 GBP). Although UK-based estimates are lacking, the financial burden is likely comparable.

For the purposes of this guideline, in line with the International Classification of Diseases 11 (ICD-11 (9)), CPP is defined as cyclical, intermittent, or constant pain in the lower abdomen or pelvis typically lasting at least three months, including dysmenorrhoea and dyspareunia, and unrelated to pregnancy or cancer. Symptoms may affect the lower urinary tract, bowel, sexual function, pelvic floor, and/or gynaecological structures.

Despite its high prevalence and burden, CPP remains under-recognised, leading to delays in treatment and variable care. A 2020 survey found that 45% of UK gynaecologists believe CPP is poorly managed (10). Many women experience prolonged diagnostic delays, resulting in ongoing physical, emotional and financial strain (11, 12).

CPP is heterogeneous, involving multiple organ systems and biological, psychological, and social factors, making diagnosis and treatment challenging. Variability in study designs, definitions and patient populations has led to limited high-quality evidence on optimal management strategies. This guideline provides an up-to-date evidence-based framework for assessment and management of CPP, with a focus on multidisciplinary, patient-centred care.

3. Identification and assessment of evidence

This guideline was developed using the methodology described in the Royal College of Obstetricians and Gynaecologists (RCOG) handbook Developing a Green-top Guideline: Guidance for developers.

The Cochrane Library and electronic databases (DARE, EMBASE, Trip, MEDLINE) were searched looking for the following terms in the title or abstract 'pelvic pain', 'chronic pelvic pain', 'dysmenorrhea'. The search was limited to female humans and English language and restricted to articles published from July 2011 until May 2023. The full search strategy is available to view online as supporting information. Papers identified by peer reviewers and the developers which fall outside the literature searches and may be more recent have also been included in the evidence base for the guideline.

Where possible, recommendations are based on available evidence. In the absence of published evidence, these have been annotated as 'good practice points'. Further information about the assessment of evidence and the grading of recommendations may be found in Appendix 1.

4. Classification and terminology

Table 1. Nociceptive pain, neuropathic pain, nociplastic pain and central sensitisation definitions are derived from International Association for the Study of Pain (13).

Terminology	Definition	Examples
Chronic pelvic pain	CPP is defined as cyclical, intermittent, or constant pain in the lower abdomen or pelvis typically lasting at least three months, including dysmenorrhoea and dyspareunia, and unrelated to pregnancy or cancer.	
Chronic pelvic pain syndrome	CPPS is a diagnosis of exclusion, made when CPP is present and no underlying cause is identified, and symptoms are not better explained by another condition.	
Nociceptive pain	Pain that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors.	Inflammation (i.e. pelvic inflammatory disease, where inflammatory mediators activate pelvic nociceptors); tissue ischaemia (i.e. ovarian torsion, where ischaemia and capsular stretch generate acute pain); and peritoneal irritation (i.e. ruptured ovarian cyst or haemoperitoneum, where chemical irritation of the peritoneum causes pain).
Neuropathic pain	Pain caused by a lesion or disease of the somatosensory nervous system.	Peripheral nerve injury (i.e. ilioinguinal or genitofemoral nerve injury after pelvic surgery, due to direct surgical trauma); nerve involvement by disease (i.e. deep endometriosis or pelvic tumours infiltrating or compressing pelvic nerves); and pudendal neuralgia (i.e. pudendal nerve compression or entrapment causing burning or shooting pain).
Nociplastic pain	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	Examples may include bladder pain syndrome / IBS-related pelvic pain flares (i.e. visceral hypersensitivity without ongoing tissue injury); and fibromyalgia (i.e. widespread central sensitisation affecting multiple pain regions including the pelvis).

	the somatosensory system causing the pain.	
Central sensitisation	Increased responsiveness of nociceptive neurons in the central nervous system to their normal or subthreshold afferent input.	Disproportionate pain severity and/or distribution relative to tissue findings often with associated features including fatigue, sleep disturbance, mood symptoms, and overlapping pain conditions (e.g. fibromyalgia, irritable bowel syndrome, bladder pain syndrome).

CPP, chronic pelvic pain; CPPS, chronic pelvic pain syndrome; IBS, irritable bowel syndrome.

In this guideline, CPP is defined as cyclical, intermittent, or constant pain in the lower abdomen or pelvis typically lasting at least three months, including dysmenorrhoea and dyspareunia, and unrelated to pregnancy or cancer (see Table 1). It is often associated with emotional distress, cognitive changes and functional impairment, with biological, psychological, and social factors contributing to symptom persistence. Symptoms may affect the lower urinary tract, bowel, sexual function, pelvic floor or gynaecological structures.

This definition is updated from the previous RCOG guideline, which classified CPP as pain lasting at least six months, not occurring exclusively with menstruation or intercourse, and unrelated to pregnancy. Traditionally, pain was only considered chronic after six months, partly because pelvic pain is often cyclical or linked to functions such as urination, defecation and sexual activity, which may not occur daily. International Classification of Diseases 11 (ICD-11), adopted by the World Health Organization (WHO) in 2019, now defines pain as chronic after three months (14). This change supports earlier recognition and intervention, aiming to reduce delays in diagnosis and treatment. Additionally, this guideline explicitly includes dysmenorrhoea and dyspareunia, recognising them as a types of CPP in their own right, as supported by current evidence. By broadening the definition, we aim to improve access to care and ensure that cyclical and intercourse-related pain is appropriately identified, assessed and managed.

Chronic pelvic pain syndrome (CPPS) is a diagnosis of exclusion, made when no underlying cause is identified, and symptoms are not better explained by another condition. Notably, ICD-11 also introduces a distinction between chronic primary pain and chronic secondary pain. Chronic primary pain is defined as "*pain characterised by disability or emotional distress and not better accounted for by another diagnosis of chronic pain*." In contrast, chronic secondary pain is pain attributed to an underlying condition, such as endometriosis.

CPP can arise from a combination of nociceptive, neuropathic and nociplastic pain mechanisms. The International Association for the Study of Pain (IASP (14)) define nociceptive pain as pain that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors. In CPP, this may result from inflammation or tissue injury. Neuropathic pain is defined as pain caused by a lesion or disease of the somatosensory nervous system. In CPP, this may occur when pelvic nerves are compressed or damaged. Nociplastic pain refers to pain arising from altered nociception despite no clear evidence of peripheral tissue damage or nerve injury. This mechanism often involves central sensitisation, characterised increased responsiveness of nociceptive neurons in the central nervous system to their normal or subthreshold afferent input. Nociplastic pain is often associated with generalised sensory sensitivity and may be influenced by systemic immune or inflammatory changes. Importantly for pelvic pain, cross-sensitisation between viscera is common, where neuronal activity in one pelvic organ (e.g. bladder, uterus, bowel) leads to pain in another via

shared neural pathways in the spinal cord. These multiple mechanisms often coexist in individuals with CPP and may evolve over time, contributing to the complexity and persistence of symptoms.

5. To what extent are the following factors associated with development of pelvic pain?

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should recognise CPP is a multifactorial condition and aim to identify contributory factors rather than assign causality to a single pathology.	**2+**	**C**	**CPP is a complex and heterogeneous condition and frequently involves multiple overlapping conditions, including gynaecological, urological, gastrointestinal, neurological and musculoskeletal disorders. It may not be possible to confidently identify the cause of the pain.**

CPP often involves multiple overlapping symptoms and diagnoses. A UK postal survey found that half of women with CPP also had symptoms of irritable bowel syndrome (IBS) or genitourinary symptoms. Additionally, dysmenorrhoea and dyspareunia were significantly more common in women with CPP (81% and 41%, respectively) compared to those without CPP (58% and 14%) (15).

This symptom overlap frequently translates into co-occurring diagnoses. A US-based study analysed a health database of over 3 million individuals to investigate the co-occurrence of chronic overlapping pain conditions using ICD-10 codes (16). For example, compared to those without endometriosis, individuals with a diagnosis of endometriosis were 18.62 times more likely to have a diagnosis of bladder pain syndrome (BPS) / interstitial cystitis (IC), 15.56 times more likely to have a diagnosis of vulvodynia, 5.05 times more likely to have a diagnosis of IBS, and 6.07 times more likely to have a diagnosis of fibromyalgia.

The recent CPP classification system developed jointly by the International Federation of Gynecology and Obstetrics (FIGO) and the International Pelvic Pain Society (IPPS) emphasised the need to consider CPP as a symptom with numerous potential causes, adopting the system-based 'R U MOVVING SOMe' framework, which recognises that chronic pelvic pain commonly arises from multiple overlapping reproductive, urological, gastrointestinal, musculoskeletal, vulvovaginal, neurological, vascular, nociplastic, and psychosocial contributors (see Table 2) (17).

Table 2. Categories and example conditions associated with CPP, adapted from the FIGO-IPPS 'R U MOVVING SOMe' classification system (17).

Category	Example conditions associated with CPP
Reproductive	Endometriosis, adenomyosis
Urological	Bladder pain syndrome/interstitial cystitis
Musculoskeletal	Pelvic floor dysfunction
Other	Foreign body, adhesions
Vulvovaginal	Vulvodynia, vaginismus

Vascular	Pelvic congestion syndrome
Idiopathic	Unexplained despite investigation
Neurological	Iliohypogastric nerve injury
Gastrointestinal	Irritable bowel syndrome
Sensitisation/Nociplastic	Hyperalgesia, allodynia
Overlapping Pain Conditions	Fibromyalgia, migraine
Mental Health	Depression, anxiety, trauma, abuse

Further, pain severity in CPP can vary widely. A Canadian cross-sectional study of 656 women with CPP identified several factors independently associated with greater pain severity, including abdominal wall pain, pelvic floor tenderness, painful bladder syndrome, pain catastrophising (a negative cognitive emotional response to anticipated or actual pain characterised by rumination, amplification of pain, and feelings of helplessness), history of adult sexual assault, higher BMI, smoking, and family history of chronic pain (18). A cohort study of 785 women found that pain severity and quality of life differed across diagnostic subgroups. Women with co-existing endometriosis and bladder pain syndrome reported the most severe pain, along with greater pain interference and lower quality of life scores. Across all CPP groups, common co-existing conditions included anxiety, depression, and IBS, reinforcing the multifactorial nature of CPP and the need for holistic, multidisciplinary management (19). *[Evidence level 2+]*

6.1 Anatomical conditions

6.1.1 Endometriosis

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should consider the diagnosis of endometriosis in women with CPP, but should be aware that disease severity, lesion number and location often do not correlate with pain experience.	**2+**	**B**	**Endometriosis is commonly found in women with CPP but can be asymptomatic.**
Clinicians should recognise that endometriosis-associated pain is often multifactorial and therefore a pain-focused approach may be more appropriate.	**4**	**GPP**	**Endometriosis-associated pain can be influenced by a range of factors, such as hormonal, musculoskeletal, psychological, and central sensitisation processes. Pain in endometriosis is often multifactorial. Recognising contributors may support a more effective, pain-focused approach than relying solely on lesion extent.**

Endometriosis is a chronic, oestrogen-dependant inflammatory disease defined by endometrial-like tissue (lesions) outside the uterus (12). The relationship between endometriosis and CPP is complex

and not fully characterised. A meta-analysis of 5104 individuals with CPP estimated the prevalence of endometriosis (primarily identified via laparoscopy) at 28.1% (95% CI 26.9–29.4) (20). However, while some individuals with endometriosis experience pain, many remain asymptomatic. In the same study, the prevalence of endometriosis among those undergoing diagnostic laparoscopy for typically non-pain-related indications, such as tubal sterilisation and ovarian cancer, was 4.4% (n=4477; 95% CI 3.8–5.1) and 9.3% (n=1171; 95% CI 7.7–11.1), respectively. Further, the number, location and subtype of endometriosis lesions correlate poorly with pain symptoms; notably excluding deep endometriosis in the posterior cul-de-sac which correlates with dyspareunia (18, 21).

A cross-sectional study investigating biopsychosocial factors associated with persistent postsurgical pain in women with endometriosis found that pain persistence was not predicted by disease severity. Instead, younger age (p=0.002) and higher catastrophising scores (p=0.01) were significantly associated with greater pain intensity (22).

The relationship between ovarian endometriomas and CPP remains unclear. A retrospective cohort study in Japan of 2988 cases undergoing diagnostic laparoscopy for ectopic pregnancy, tubal infertility and other benign gynaecologic diseases found that only 38.3% of women with ovarian endometriomas reported pain, compared with 85.4% of those with both ovarian endometriomas and concurrent peritoneal endometriosis (23). Similarly, a study in France of 300 women with histologically confirmed ovarian endometriomas found that pain severity correlated with the presence of deep infiltrating endometriosis but not with endometrioma size (24). Both studies found there was no statistical difference in severity of pain based on the size of the endometrioma.

Pain associated with endometriosis can arise through nociceptive, neuropathic and nociplastic mechanisms (defined in Section 5) (12, 25). Nociceptive pain may be driven by inflammation and immune cell infiltration or fibrosis around endometriosis lesions. Neuropathic-like pain is likely generated by sensitisation of pelvic nerves (including neoinnervation of endometriosis lesions) from localised inflammatory mediators or surgery; infiltration by deep lesions into pelvic nerves may also play a role (e.g. sciatic endometriosis). Nociplastic mechanisms are suggested by the presence of widespread pain, fatigue, and sensory hypersensitivity, and may underpin persistent pain despite effective lesion suppression or surgical removal. *[Evidence level 2+]*

6.1.2 Adenomyosis

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should consider adenomyosis as a differential diagnosis in women of all ages presenting with CPP, particularly in those with prominent dysmenorrhoea or heavy menstrual bleeding.	**2+**	**C**	**An estimated 27–34% of women with dysmenorrhoea have sonographic feautures of adenomyosis.**

Adenomyosis, characterised by the presence of endometrial glands and stroma within the myometrium, is an important differential diagnosis in women presenting with CPP. Dysmenorrhoea (occurring in 15–30%) and heavy menstrual bleeding (occurring in 40–60%) are important clinical features (26). Endometriosis and adenomyosis share features such as oestrogen-dependence and partially overlapping genetic susceptibility, and are often reported to co-occur, although robust

epidemiological evidence remains limited (27). Historically, adenomyosis was only diagnosed through histopathological examination after hysterectomy, leading to a limited and biased understanding of its prevalence and association with CPP. The estimated prevalence of adenomyosis among women undergoing hysterectomy has ranged from 8.8% to 61.5% (28).

Improvements in ultrasound and magnetic resonance imaging (MRI) identification of adenomyosis features are improving diagnosis, despite the lack of a validated, universally accepted diagnostic criteria (29). A prospective observational study of 718 premenopausal women attending a general gynaecology clinic investigated the association between ultrasound features of adenomyosis and menstrual pain severity (30). Adenomyosis was diagnosed in 21.9% (95% CI 18.8–24.9) of participants via transvaginal ultrasound. Multiple linear regression analysis found a significant association between an ultrasound diagnosis of adenomyosis and increased menstrual pain. Additionally, pain severity correlated positively with the number of ultrasound features of adenomyosis observed.

A prospective observational study of 156 nulligravid women without endometriosis aged between 18 and 30 years found diffuse sonographic features of adenomyosis in 34% of women and was associated with dysmenorrhoea (31). Another observational cohort study of 95 women and girls aged between 13 and 25 years found 27.4% had sonographic features of adenomyosis and the presence of dysmenorrhoea and heavy menstrual bleeding was significantly associated with adenomyosis (OR 5.68; 95% CI 1.65–19.5) (32). *[Evidence level 2+]*

6.1.3 Adhesions

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should not offer laparoscopy to women with CPP to identify adhesions as a cause for pain.	**1+**	**B**	**There is limited evidence linking adhesions to CPP or supporting adhesiolysis as a treatment.**
Clinicians should counsel women with CPP and adhesions that the relationship between adhesions and pain is unclear.	**2-**	**D**	**Epidemiological data is limited supporting the association between adhesions and CPP.**
When adhesions are identified at laparoscopy, clinicians should not routinely perform adhesiolysis as a treatment for CPP, given the lack of consistent evidence of long-term benefit.	**1+**	**B**	**Evidence has shown no sustained improvement in pain following adhesiolysis compared with diagnostic laparoscopy alone, and potential for harm has been noted.**
In women with dense, vascular, bowel-involving adhesions, adhesiolysis may be considered	**3**	**D**	**Subgroup analysis suggests that patients with severe adhesions involving the bowel may experience**

in selected cases following multidisciplinary review.	**symptomatic benefit from adhesiolysis, although the data are not based on a laparoscopic surgical approach.**

Adhesions, which are bands of scar-like tissue connecting otherwise unconnected structures, are identified in up to 50% of women with CPP during diagnostic laparoscopy, and can originate from previous surgery, pelvic infection, endometriosis, or inflammation. The relationship between adhesions and CPP remains uncertain, and the clinical benefit of adhesiolysis (surgical division of adhesions) in this context is controversial (33-35). A small cluster analysis of 62 women who underwent laparoscopy for investigation of CPP found there was no correlation between the presence or severity of adhesions (found in 37 women) and patient-reported pain, physical, emotional or functional characteristics, when compared to women without adhesions (36). *[Evidence level 2–]*

Very limited evidence exists assessing the role of adhesiolysis in women with CPP. A systematic review and meta-analysis of 13 studies including two randomised controlled trials (RCTs), involving 533 patients (~90% women) evaluated adhesiolysis for chronic post-operative abdominal and pelvic pain (37). While pooled data from trials and non-randomised studies suggest short-term pain improvement following adhesiolysis, with 74% (95% CI 60–88) of 291 patients reporting improvement of pain at any follow-up; high-quality randomised evidence does not support its long-term efficacy.

In a RCT of 100 patients (87% female) with chronic abdominal pain attributed to adhesions, laparoscopic adhesiolysis showed no significant benefit over diagnostic laparoscopy alone at 12 months (mean 0-100 visual analogue scale (VAS) difference: 3 points), with both groups reporting substantial pain relief and improved quality of life (38). A 12-year follow-up of this cohort (73% retention) reported that patients who underwent adhesiolysis were significantly less likely to be pain-free, required more analgesia, had more medical consultations, and were more likely to undergo further surgery, suggesting not only a lack of sustained benefit, but also potential long-term harm (39). These findings highlight a strong and durable placebo effect of diagnostic laparoscopy and support a cautious, evidence-based approach to adhesiolysis for CPP.

A double-blinded RCT conducted in two UK tertiary hospitals investigated the effect of laparoscopic adhesiolysis on pain scores and quality of life in women with CPP, however, the study was terminated early due to recruitment challenges (40). Among 50 randomised participants, those undergoing adhesiolysis showed significant improvement in pain scores measured on a 100-point VAS at six months (n=26, -17.5 (95% CI -36.0– -5.0) compared to laparoscopy alone group (n=24, -1.5 (95% CI, -15.0–4.5); p=0.048). Although an improvement in pain was observed in the adhesiolysis group, the study was underpowered, did not include long-term follow-up and the group undergoing adhesiolysis had significantly higher adhesion scores than those undergoing laparoscopy alone.

A randomised trial of 48 women with pelvic adhesions found that adhesiolysis did not significantly improve pelvic pain after 9–12 months (41). A subgroup analysis of 15 women with stage IV adhesions, defined as dense, vascularised adhesions involving the serosa of the bowel and fixed to the peritoneum, experienced significant pain improvement, suggesting potential benefit in this select group. Notably, the procedure was performed via midline laparotomy, limiting the

generalisability of these findings to current laparoscopic practice, which is now standard when feasible.

In a case series of 10 women with post-caesarean section pain, dense adhesions were found fixing the uterus on the anterior abdominal wall coined “captive uterus syndrome” (42). After laparoscopic adhesiolysis or hysterectomy, all patients had complete resolution of their pain that lasted at five years follow-up. *[Evidence level 1+ to 3]*

6.1.4 Pelvic venous congestion

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should not routinely diagnose or manage pelvic venous congestion as a cause of CPP.	**4**	**GPP**	**There is an absence of validated diagnostic criteria and limited high-quality evidence supporting a causal relationship between pelvic venous congestion and CPP.**

A systematic review published in 2016 assessed the evidence relating to pelvic congestion syndrome, which is described as CPP arising from dilated and refluxing pelvic veins (43). The review aimed to evaluate diagnostic criteria, associations between pelvic vein incompetence and CPP, diagnostic accuracy of imaging, and effectiveness of embolization as a therapeutic approach. Across 40 studies including association studies, imaging evaluations, case series and one poor-quality RCT, the review found no consistent diagnostic criteria for pelvic congestion syndrome and only limited, heterogeneous evidence linking pelvic vein incompetence to CPP. Although some studies suggested statistically significant associations, methodological limitations and variability in pelvic vein incompetence definitions undermined the findings. Non-invasive imaging techniques such as Doppler ultrasound and magnetic resonance venography were commonly used, but data on their diagnostic accuracy remain sparse. Embolisation appeared to provide short and medium term pain relief in some cases, with low complication rates; but the evidence was primarily based on small, uncontrolled case series, and the single RCT was at risk of bias. The authors concluded that while embolisation is safe and may be effective, robust evidence is lacking, and well-designed, adequately powered studies are needed to establish diagnostic standards and determine treatment efficacy. Given the absence of robust evidence and agreed diagnostic criteria, clinicians are not currently advised to routinely diagnose or manage pelvic venous congestion as a cause of CPP; this recommendation is based on expert consensus. *[Evidence level 2++]*

6.1.5 Nerve entrapment

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should consider nerve entrapment in the differential diagnosis of chronic pelvic or incision site pain, particularly when symptoms are localised and follow a specific nerve distribution.	**3**	**D**	**Retrospective and prospective studies have identified it as a treatable cause in patients with localised, neuropathic symptoms.**

Nerve entrapment can cause localised neuropathic pain, typically described as sharp, stabbing or aching. It may arise from scar tissue, fascia or compression at a narrow anatomical foramen. In a study of 866 women who had undergone a Pfannenstiel incision for caesarean delivery or abdominal hysterectomy found that approximately one-third reported chronic pain at the incision site (44). Iliohypogastric or ilioinguinal nerve entrapment was diagnosed in 17 cases. In a prospective cohort study of 213 women with sacral radiculopathy, laparoscopic evaluation identified sciatic nerve and sacral plexus entrapment due to isolated endometriosis, deep infiltrating parametric endometriosis, or vascular compression. Laparoscopic surgical treatment with nerve release resulted in symptom improvement (45). *[Evidence level 3]*

6.1.6 Congenital anomalies of the reproductive tract

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should consider congenital anomalies in the differential diagnosis of CPP, particularly in adolescents and young women.	**3**	**D**	**Evidence from scoping reviews and retrospective cohort studies highlights their association with obstructive symptoms, diagnostic delays, and chronic pain, especially in cases of reproductive tract anomalies and uterine remnants.**
Clinicians should consider accessory cavitated uterine malformations (ACUMs) in the differential diagnosis of CPP, particularly in adolescents and young women.	**3**	**D**	**ACUMs are a rare cause of CPP.**
In women with Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome and pelvic pain, obstructed uterine remnants and endometriosis should be considered.	**2–**	**D**	**Low-quality evidence suggests an association between uterine remnants with functional endometrium and increased likelihood of pain.**

Obstructive reproductive tract anomalies should be considered in adolescents and young women with CPP, particularly when symptoms begin around menarche (46). Approximately 7% of girls will have an anatomical abnormality of their reproductive tract, diagnosed before or after puberty (47). An imperforate hymen, transverse vaginal septum or vaginal agenesis causing obstructed menstrual outflow typically presents with primary amenorrhoea and progressive cyclical pain, which may evolve into non-cyclical pain due to hematocolpos or hematometrocolpos. In contrast, a blind-ending uterine horn may present with regular menstruation alongside cyclical pelvic pain due to unilateral outflow obstruction. These anomalies are often misattributed to dysmenorrhoea, leading to diagnostic delays and an increased risk of complications such as endometriosis. Early imaging, typically with pelvic ultrasound followed by MRI, is essential for accurate diagnosis and timely surgical management. *[Evidence level 3]*

Accessory cavitated uterine malformations (ACUMs) are a rare form of uterine anomaly, characterised by non-communicating cavitated lesions typically located within the lateral aspect of

the myometrium, inferior to the round ligament attachment (48). Although their pathogenesis remains unclear, ACUMs are recognised as a cause of CPP, particularly in adolescents and young women. A 2024 scoping review of 115 cases of ACUMs reported a median age of symptom onset of 17 years and a median age at diagnosis of 21.5 years. The most frequently reported symptoms were dysmenorrhoea (88/115, 76.5%) and non-cyclical pelvic pain (65/115, 56.5%). Although ultrasound and MRI are the most used imaging modalities, diagnosis remains challenging due to the absence of standardised classification and variability in terminology. ACUMs may be underdiagnosed and may be misinterpreted as other pathology, such as fibroids (48, 49). Although 83% of reported cases underwent surgical management, there is limited evidence on the effectiveness of any therapeutic strategy in relation to pain, impact on fertility or subsequent pregnancy outcomes (49). *[Evidence level 3]*

Low-quality evidence suggests pelvic pain may be associated with Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome, despite the absence of a functional uterus. A retrospective cohort study of 117 women with MRKH syndrome found that persistent abdominopelvic pain was reported by 43 patients (36.8%), and cyclic abdominopelvic pain by 24 patients (20.5%) (50). The presence of uterine remnants was significantly associated with both persistent pain and cyclic pain. In a single centre retrospective cohort study of 48 women with MRKH syndrome, 22 patients reported pelvic pain. 14 had uterine remnants, 9 with and 5 without functional endometrium (51). Patients with uterine remnants with functional endometrium were 3.6 times more likely to have pelvic pain compare with those without uterine remnants (RR 3.6, 95% CI 1.9–6.7). Endometriosis was also found in five patients with uterine remnants with functional endometrium. Although the overall prevalence and mechanisms of pelvic pain in MRKH syndrome remain unclear, obstructed uterine remnants and/or endometriosis should be considered (52). *[Evidence level 2– to 3]*

6.1.7 Vulval and vaginal conditions

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should recognise vaginismus as a cause of CPP, particularly in women presenting with superficial dyspareunia or pain with tampon use or speculum examination .	**4**	**GPP**	**Recent literature highlights its complex biopsychosocial etiology beyond involuntary muscle contraction.**
Women with vaginismus should be offered multidisciplinary care that addresses physical and psychological factors.	**4**	**GPP**	**Multidisciplinary care is supported by a biopsychosocial framework, which accounts for the psychological, relational, and cultural contributors to vaginismus and is essential for effective, holistic treatment.**

Vaginismus is a recognised cause of CPP, particularly associated with superficial dyspareunia (pain at the vulva or introitus during attempted penetration). It is characterised by involuntary contraction of the pelvic floor muscles, which can make vaginal penetration painful or impossible, affecting sexual function, relationships, and complicating medical procedures such as vaginal examination. While earlier definitions emphasised muscle spasm, more recent descriptions recognise pain, fear of pain

and avoidance behaviours as central features (53). Vaginismus often overlaps with conditions such as vulvodynia and may present with similar pain profiles. A biopsychosocial framework is essential for understanding its aetiology, as psychological, relational, and cultural factors frequently contribute to its onset and persistence. *[Evidence level 4]*

Vulvodynia is defined as vulvar pain of at least three months' duration, without a clear identifiable cause, which may have potential associated factors (54). It is estimated to affect 8% of women and can be classified by location, as either localised (affecting a specific area such as the vestibule or clitoris) or generalised (involving the entire vulva), and by trigger as either provoked by touch, intercourse or tampon use, or spontaneous (55). Many women experience a combination of both types. Vulvodynia should be managed per the British Association Sexual Health and HIV (BASHH) National Guideline on the Management of Vulval Conditions (56). *[Evidence level 2+]*

1.1. Visceral functional pain conditions

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should assess for coexisting visceral pain conditions, as management of one condition may lead to improvement in symptoms of another.	**4**	**GPP**	**CPP frequently coexists with other visceral pain conditions. Shared sensitisation and referral pathways may contribute to symptom amplification and worsening of pain.**

Visceral functional conditions are chronic pain disorders that occur in the absence of identifiable structural, infectious, inflammatory or neoplastic pathology and are associated with organ function (e.g. defecation, urination, menstruation). CPP frequently coexists with other visceral functional conditions, such as fibromyalgia, bladder pain syndrome/interstitial cystitis, and irritable bowel syndrome (57-59). This overlap may be explained by shared neural pathways, where pain signals from one organ can increase the sensitivity of other internal organs (viscero-visceral hyperalgesia) or nearby somatic structures such as muscles and fascia (viscero-somatic hyperalgesia) (60). These mechanisms contribute to a process known as central sensitisation, which refers to an amplification of pain signalling within the central nervous system, resulting in heightened pain sensitivity. This can cause disproportionately severe, widespread or persistent pain, even in the absence of ongoing tissue damage (61). This can result in higher levels of pelvic pain resulting in greater impact on daily activities. In women with CPP, those with coexisting endometriosis and bladder pain symptoms scored higher for all types of pelvic pain with poorer quality of life (19). There is increasing evidence that treatment of coexisting visceral pain conditions can reduce overall pain burden and central sensitisation. In a non-randomised study of patients with fibromyalgia and visceral co-existing conditions (e.g. IBS, endometriosis, dysmenorrhoea), managing the visceral condition led to improvements in fibromyalgia-related pain and hyperalgesia, supporting an integrated treatment approach in women with CPP and overlapping pain syndromes (62). *[Evidence level 2– to 4]*

1.1.1. Irritable bowel syndrome and other disorders of gut-brain interaction

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should consider if irritable bowel syndrome or other disorders of gut-brain interaction are present in women presenting with CPP.	**2–**	**GPP**	**Bowel symptoms frequently co-occur with CPP, and recognition of coexisting conditions may aid diagnosis and management.**

Population-based studies have demonstrated an association between IBS and CPP. In one postal survey of 479 women, 38.5% (95% CI 34.2–42.8) met diagnostic criteria for IBS (15). In a separate UK survey of 2,088 women, IBS was the most common diagnosis among those with CPP, reported in 31.1% of pre-menopausal and 55.9% of post-menopausal women (6). Other disorders of gut–brain interaction (previously termed functional gastrointestinal disorders), such as functional constipation and functional dyspepsia, may share overlapping mechanisms with CPP, including visceral hypersensitivity and central sensitisation (63). Although epidemiological data on their co-occurrence with CPP are currently lacking, these conditions often coexist in clinical practice and may contribute to symptom burden. *[Evidence level 2–]*

Clinicians can screen for IBS using the Rome IV criteria (64).

1.1.2. Bladder pain syndrome/interstitial cystitis

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should consider bladder pain syndrome/interstitial cystitis in the differential diagnosis of CPP, particularly when bladder symptoms are present or in the context of coexisting endometriosis.	**2+**	**GPP**	**Bladder pain syndrome/interstitial cystitis is often present in women with CPP.**

Overlap exists between CPP and urinary symptoms. Bladder pain syndrome (BPS), as defined by the European Society for the Study of Interstitial Cystitis, is CPP, pressure, or discomfort perceived to be related to the urinary bladder, accompanied by at least one other urinary symptom such as persistent urge to void or increased urinary frequency (65). A 2013 systematic review of nine studies involving 1,016 women with CPP reported a pooled prevalence of BPS of 61% (95% CI 58–64) and coexistent BPS and endometriosis in 48% (95% CI 44–51) (58).

Baseline data from a large cohort (n=424; 55% female) with urological CPPS (including IC/BPS in men and women, and chronic prostatitis/CPPS in men) showed that coexisting non-urological conditions were common: 22% had IBS, 4% had fibromyalgia, 3% had chronic fatigue syndrome, and 10% had multiple syndromes (66). In the same cohort, the largest body pain mapping study of urological CPPS to date found that 75% of participants reported pain beyond the pelvis, of whom 38% reported widespread pain (67). Widespread pain was associated with poorer mental health, greater nonpelvic pain severity, and worse quality of life.

Further, neuroimaging within this cohort also identified a distinct subgroup with bladder-filling pain, associated with increased symptom severity, greater healthcare utilisation and more frequent

symptom flares (68). Individuals with bladder-filling pain had altered brain structure and function; neuroimaging revealed reduced cortical surface area and increased activity in regions involved in pain, suggesting evidence of central sensitisation. Quantitative sensory testing in this cohort further supports the presence of a centralised pain phenotype. Participants with urologic CPPS demonstrated significantly increased pressure pain sensitivity compared to healthy controls, along with heightened sensitivity to unpleasant auditory stimuli. These findings reflect global multisensory hypersensitivity, a key feature of central sensitisation (69). A 2025 consensus review on IC/BPS phenotyping emphasised the importance of distinguishing bladder-centric versus systemic (widespread pain) phenotypes, which has significant implications for subsequent diagnosis and treatment approaches (70). *[Evidence level 2+]*

1.1.3. Dysmenorrhoea

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should recognise that dysmenorrhoea is a significant cause of CPP in women of reproductive age.	**2+**	**GPP**	**Dysmenorrhoea is common and is associated with impacts on quality of life, including school and work absenteeism, reduced educational attainment, and poorer mental health outcomes.**
Clinicians should recognise that dysmenorrhoea may contribute to increased pain sensitivity.	**2+**	**B**	**Primary dysmenorrhoea may contribute to development of central sensitisation and be a precursor to other chronic conditions.**

Dysmenorrhoea, defined as painful, spasmodic cramping in the lower abdomen just before and/or during menstruation, in the absence of any identifiable macroscopic pelvic pathology, is one of the most common gynaecological pain conditions in women of reproductive age (71). It affects an estimated 45–97% of menstruating women, with 10–25% experiencing severe pain that significantly impacts quality of life (71). Dysmenorrhoea is associated with school and work absenteeism, and poorer educational attainment in young women. A longitudinal study of 1600 Australian adolescents found that those with dysmenorrhoea were significantly more likely to experience symptoms of anxiety (OR 1.75–1.82) and depression (OR 2.03–2.89) in early adulthood compared to peers without dysmenorrhoea (72). Notably, dysmenorrhoea predated the development of these mental health diagnoses, rather than the reverse. Despite the availability of effective treatments (See Section 9), including non-steroidal anti-inflammatory drugs and hormonal therapies that induce amenorrhoea, dysmenorrhoea is frequently normalised, and many women do not seek medical care (73). Persistent, severe dysmenorrhoea has been associated with increased pain sensitivity, both during menstruation and at non-pelvic sites, suggestive of central sensitisation (71). Whether this heightened pain sensitivity is a cause or consequence of dysmenorrhoea remains unclear. In a prospective study of 181 women with CPP, abdominal allodynia (pain due to a stimulus such as light touch that does not normally provoke pain) was identified in 62.1% of participants (74). Women with allodynia were significantly more likely to report severe dysmenorrhoea (89% vs 63%) and had a longer average duration of dysmenorrhoea (12.6 vs 10.7 years). An Australian observational study

of 100 patients referred to a private gynaecologist found 52.8% of women had developed CPP within 12 years of the onset of dysmenorrhoea (75).

Dysmenorrhoea may also precede or contribute to the development of other chronic pain conditions, such as fibromyalgia (71). In a UK longitudinal birth cohort, adolescent dysmenorrhoea showed a dose–response relationship with adult chronic pain: mild, moderate, and severe dysmenorrhoea at age 15 were associated with approximately 23%, 65%, and 76% increased risk of chronic pain at age 26 (76). Central nervous system changes have been observed in women with dysmenorrhoea, including reduced grey matter volume, dysregulation of the hypothalamic-pituitary-adrenal axis, visceral hypersensitivity and altered functional connectivity, features consistent with other chronic pain states (77-79). A survey-based cross-sectional study of 10,402 Brazilian women used the Central Sensitisation Inventory to assess symptoms of central sensitisation in relation to menstrual characteristics (80). Dysmenorrhoea was reported by 67.3% of respondents, and central sensitivity symptoms were identified in approximately 50% of participants. Greater menstrual pain intensity (OR 1.12), onset of dysmenorrhoea in adolescence (OR 1.20), irregular menstrual cycles (OR 1.47), and the presence of gynaecological co-existing conditions (OR 1.51) were all independently associated with increased odds of central sensitisation. These findings highlight the potential importance of early and effective management of dysmenorrhoea to reduce the risk of central sens itisation and progression to CPP or other chronic pain syndromes. *[Evidence level 2+]*

1.2. Altered nervous system function

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should consider the role of the nervous system in the generation and maintenance of CPP.	**2+**	**C**	**There is increasing evidence for the presence of both neuropathic and nociplastic pain mechanisms in many women with CPP.**
Clinicians should recognise that many women with CPP have altered central nervous system function, with widespread implications including increased pain sensitivity, fatigue, visceral symptoms, and psychological impact.	**2++**	**C**	**Emerging evidence links CPP to central sensitisation.**
Clinicians should consider that many women with CPP have altered function of their peripheral nerves contributing to both chronic pain and associated functional symptoms.	**2+**	**C**	**Clinical studies demonstrate that altered peripheral nerve function, including mechanosensitivity and somatosensory changes, is prevalent among women with CPP.**

Altered function of the central and peripheral nervous systems may contribute to pain generation and maintenance in women with CPP, often in the absence of identifiable pathology. These mechanisms can sometimes be inferred from the characteristics of the pain described and the response to treatment. Pain can be broadly categorised as nociceptive, neuropathic or nociplastic (See Section 5 for definitions). It is important to identify the likely pain mechanism(s) involved as the treatment approaches for neuropathic and nociplastic pain differ from those targeting nociceptive mechanisms.

Peripheral pain mechanisms involve increased sensitivity or dysfunction of peripheral nociceptors or nerves, typically following inflammation, injury, or repeated nociceptive input. This may present as localised pain with mechanical sensitivity, altered sensation, or nerve-related symptoms. Neuropathic pain, a subset of peripheral pain commonly reported in CPP, can be suggested by the words used to describe pain (e.g. electric shock-like, burning or tingling) and is commonly associated with altered sensation in a clearly defined area, either increased sensation (allodynia) or loss of sensation (numbness) (81).

Central pain mechanisms reflect altered processing within the central nervous system, resulting in pain amplification, persistence, and reduced inhibitory control. Nociplastic pain, commonly seen in associated with central sensitisation, is frequently accompanied by fatigue, sleep disturbance, cognitive or mood changes, and is typically unresponsive to treatment such as surgery, NSAIDs or opiates (82).

Observational studies demonstrate that CPP is mechanistically heterogeneous, with frequent overlap between pain types. In a prospective observational cohort study of 568 participants (n=378 women) with urologic CPPS, mixed nociceptive-nociplastic and neuropathic-nociplastic phenotypes were common, highlighting the contribution of central pain mechanisms (83). Further, a prospective cross-sectional study of 303 women at a tertiary referral centre for CPP and endometriosis found that higher levels of nociplastic pain, were associated with greater pain severity, frequency, interference and pelvic myofascial tenderness, regardless of endometriosis diagnosis or previous surgery (84). The presence of nociplastic pain showed a stronger association with pain outcomes than the presence of endometriosis and likelihood of high-impact pain increased by 7% for each additional point on the Fibromyalgia Survey Score.

Evidence supports both peripheral and central sensitisation in CPP. Quantitative sensory testing and clinical studies demonstrate altered somatosensory processing at pelvic and distant sites, with features of heightened peripheral nerve sensitivity and reduced pain thresholds alongside central pain amplification (85-88). Systematic reviews and cohort studies further demonstrate features of central sensitisation, including widespread hyperalgesia and altered central pain processing, although causality remains uncertain (89-92) (Evidence level 2++ to 4). These findings indicate that peripheral nerve dysfunction may coexist with central mechanisms in many women with CPP, and support a mechanism-informed approach to the assessment of CPP, recognising that peripheral and central nervous system contributions frequently coexist and that persistent symptoms may not reflect ongoing peripheral pathology alone.

1.2.1. Autonomic nervous system

Recommendation	Evidence quality	Strength	Rationale for the recommendation

Clinicians should consider that symptoms generated by the autonomic nervous system, such as sweating and dizziness, are common in association with CPP.	**2-**	**C**	**There is increasing evidence associating autonomic dysregulation and chronic pain conditions.**

Several studies suggest that autonomic nervous system dysfunction may contribute to symptom burden in CPP. In a prospective study of 170 women, those with CPP showed signs of autonomic neuropathy, increased orthostatic intolerance, and elevated resting heart rates, findings particularly pronounced in women with myofascial pelvic pain (93). A smaller case-control study also identified higher levels of autonomic symptoms in women with CPP compared with healthy controls, including vasomotor instability, gastrointestinal dysmotility, orthostatic intolerance, and bladder dysfunction (94). Similar features have been described in other specific forms of CPP: in vulvodynia, women displayed higher resting heart rates, lower blood pressure and exaggerated pain responses, suggesting chronic sympathetic activation (95) and in endometriosis, a shift toward sympathetic dominance and reduced vagal tone has been reported (96).

These findings mirror those observed in IBS, where altered autonomic nervous system activity, particularly increased sympathetic and reduced parasympathetic tone, has been implicated in symptom generation (97). While it remains unclear whether such autonomic changes are specific to individual CPP subtypes or reflect a broader shared pathophysiology, autonomic nervous system dysregulation may play an important role in CPP. *[Evidence level 2–]*

1.3. Musculoskeletal dysfunction

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should consider musculoskeletal dysfunction as a potential primary cause of CPP, or as a secondary consequence.	**2+**	**C**	**Musculoskeletal dysfunction is common in women with CPP and may result from or contribute to pain-related impairment.**
Pelvic floor dysfunction should be recognised as a treatable cause of CPP.	**2+**	**C**	**Pelvic floor muscle dysfunction is frequently identified in these conditions and may represent a treatable component of CPP.**

Musculoskeletal dysfunction may contribute to CPP either as a primary driver, such as pelvic floor muscle spasm, levator ani or piriformis syndromes, or sacroiliac joint dysfunction, or as a secondary consequence of other conditions. These conditions can lead to postural and functional adaptations, resulting in muscle hypertonicity and associated pain (98). Symptoms are often flared or triggered by physical activity and many women with musculoskeletal pelvic floor dysfunction experience dyspareunia and painful vaginal speculum examination or tampon insertion.

A musculoskeletal component to CPP is highly prevalent and has been reported in numerous, mostly small observational studies. In a retrospective cohort of 353 women undergoing hysterectomy, myofascial pain was identified in 86% of those with CPP, compared with 13.7% of those without (99). In a cross-sectional study of 94 women referred for laparoscopy, 51% were found to have musculoskeletal dysfunction of the lumbar or pelvic region on clinical examination (100). Additional cross-sectional studies comparing women with CPP to pain-free controls have demonstrated significantly higher rates of pelvic floor muscle tenderness, increased resting tone, reduced strength and relaxation capacity, and lower pressure-pain thresholds in the CPP group (101, 102). These findings are further supported by a case-control study of 149 women with CPP, which identified increased stiffness in five of 11 lumbopelvic muscles and reduced pressure-pain thresholds across all muscles assessed (103). In another cross-sectional study of 49 women with pelvic pain and 20 healthy controls, sensitisation (defined as allodynia or hyperalgesia) was found in 83% of those with endometriosis and 82% of those with CPP without endometriosis, compared to 15% of pain-free controls (104). Myofascial trigger points were present in 94% and 91% of the pain groups, respectively. Sensitisation was more likely in those with higher anxiety and depression scores and was strongly associated with the presence of myofascial trigger points (OR 9.41, 95% CI 1.77–50.08).

Pelvic floor dysfunction is a common finding among women with dyspareunia, BPS/IC, vulval pain and bowel dysfunction. It may arise from pelvic floor muscle spasm secondary to visceral-somatic referral patterns, but other mechanisms such as biomechanical strain, trauma, or muscular overuse can also contribute (105). In a study of 70 women with CPP and 35 pain-free women, musculoskeletal trigger point pain scores were higher in women with CPP compared with healthy women with concurrent bladder pain resulting in increased pain scores compared with BPS alone (106).

In a cohort of 562 individuals (n=375; 67% female) and 69 controls with urologic CPPS, 81% had pelvic floor muscle tenderness on standardised pelvic examination, compared with only 9% of controls (107). Greater pelvic floor muscle tenderness severity was associated with worse pelvic pain and urinary symptoms, poorer quality of life, and more widespread pain, suggesting a phenotype with overlapping central sensitisation features. These findings support the need for routine clinical evaluation of pelvic floor muscle tenderness, as its presence correlates with symptom severity in a dose-dependent manner. Individuals with more focal muscle tenderness may benefit from pelvic health physiotherapy, whereas those with both pelvic floor muscle tenderness and widespread pain may require additional systemic treatments targeting central sensitisation. *[Evidence level 2+]*

1.4. Psychology and trauma

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should be aware that psychological distress is common in women with CPP and may influence their presentation and experience.	**2+**	**GPP**	**Psychological symptoms are prevalent in women with CPP and are associated with increased symptom burden and poorer outcomes.**
Clinicians should sensitively enquire about a history of trauma or sexual	**2+**	**GPP**	**There is evidence linking trauma, particularly cumulative**

assault, as this may contribute to the development or persistence of CPP.	and early life trauma, with CPP and psychological distress.

Living with chronic pain, particularly unexplained pain, and associated symptoms is challenging. This is especially true for CPP where symptoms may have been minimised or dismissed by healthcare professionals, family or friends for many years. For many individuals, this experience of invalidation can contribute to psychological distress and reinforce a cycle of suffering. It is therefore unsurprising that, like other chronic pain conditions, CPP is associated with a disproportionately high burden of coexisting mental health conditions, particularly anxiety and depression. In women with CPP, the prevalence of depression is estimated at 26–52% (compared to 5–10% in the general population), and anxiety at 39–73% (compared to 12% in the general population) (108).

Co-occurring psychological factors can also contribute to the development and maintenance of a chronic pain cycle and may impact on the ability to engage with therapeutic approaches. There is a well-recognised bidirectional relationship between pain and psychological distress: poor mental health can exacerbate pain perception and interfere with coping, while chronic pain itself may lead to or worsen psychological symptoms (109). A cross-sectional study of 175 women attending a specialist CPP clinic found 53% of women reported moderate or severe anxiety and 26.7% reported moderate-to-severe depression (110). Depressive symptoms were strongly correlated with pain interference (the degree to which pain impacts an individual's daily activities) and pain catastrophising (108, 111).

Pain catastrophising is highly prevalent in women with CPP, with 43% (n=50/115) of women with endometriosis-associated CPP in one prospective study reporting moderate-to-severe levels (112). A systematic review and meta-analysis of 25 studies of woman with CPP (n=4540) found a small but significant positive association between catastrophising and pain severity (107). Furthermore, in a cross-sectional study of 236 women with endometriosis-associated CPP, higher levels of pain catastrophising were independently associated with worse pain-related quality of life, even after adjusting for pain severity (113). *[Evidence level 2+]*

Many women with CPP report a history of sexual, physical, or emotional abuse; however, the nature of this relationship is complex and conclusions about causality cannot be made. A cross-sectional study of 271 women found that presence of severe pelvic pain was significantly associated with such a history of childhood or adolescent sexual abuse (OR 3.6, 95% CI 1.2–10.4) (114). Further, in a cross-sectional study of 273 women with CPP, a history of adolescent or adult sexual abuse was associated with greater pain-related disability (OR 2.39; 95% CI 1.05–5.40) but not increased pain severity (115). In a case-control study of 120 women, those with CPP had significantly higher rates of adverse childhood experiences (ACEs) than non-CPP controls, including physical, sexual, and emotional abuse, and witnessing domestic violence. Over half (53%) of the CPP group reported four or more ACEs, compared to 27% of controls (OR 3.14, 95% CI 1.46–6.75) (116). In view of these associations, trauma-informed care is essential in the assessment and management of CPP. This approach promotes sensitive enquiry, minimises the risk of retraumatisation, and supports a holistic, multidisciplinary model of care (117). *[Evidence level 2+]*

1.5. Lifestyle and socio-cultural factors

Recommendation	Evidence quality	Strength	Rationale for the recommendation

Clinicians should consider social, cultural and lifestyle factors when assessing and managing CPP.	**4**	**GPP**	**Socio-cultural and lifestyle factors may contribute to the development of chronic pain, impact on the ability to engage with therapeutic options and influence health-seeking behaviour.**

Socio-cultural and lifestyle factors may contribute to the development and persistence of chronic pain, influence beliefs about its cause and prognosis, and affect engagement with care. These factors can also modulate pain perception and coping strategies. Multiple lifestyle elements such as sleep, diet, physical activity, weight, stress and smoking impact chronic pain and quality of life. While high-quality evidence specific to CPP is limited, general chronic pain data support addressing these domains as part of a multimodal approach. These topics are discussed further in Section 9.6.
The role of social media in influencing treatment choices should not be underestimated. A 2024 content analysis of 515 Instagram posts about endometriosis found that many educational claims, particularly those related to surgery, were not evidence-based, highlighting the potential for misinformation to influence real-life decision-making (118). A similar analysis found most endometriosis posts centred around social support and personal experience (119). Objective educational posts, particularly those shared by healthcare providers, were found to be largely accurate. Clinicians should be prepared to discuss and contextualise information patients encounter online.

Social and cultural influences also shape pain understanding and engagement with treatment. For example, cultural views on menstruation, pain expression or the use of hormonal treatments may affect care preferences and acceptance of therapeutic options. Family, partner, or community support may significantly affect a patient's ability to engage in lifestyle changes such as regular physical activity, dietary modification or smoking cessation. A sensitive and individualised approach to care that acknowledges the woman's cultural background, beliefs and social context is essential to building trust and optimising outcomes in CPP management. *[Evidence level 3 to 4]*

1.6. Iatrogenic

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should consider iatrogenic factors, including previous surgical interventions and obstetric events, in the evaluation of CPP.	**2-**	**D**	**Surgical and procedural interventions can be associated with persistent pelvic pain.**
Women with suspected mesh or device-related pelvic pain should be managed in conjunction with an appropriate specialist centre.	**4**	**GPP**	**Specialist centres aim to offer specialist assessment and multidisciplinary care, enabling clearer diagnosis and more effective, tailored treatment.**

Chronic post-surgical pain (CPSP) is one of the most common complications following surgery, affecting 20–30% of patients at 6–12 months postoperatively, with wide variation depending on the type of surgery (120). Risk factors for CPSP include pre-existing chronic pain, high levels of acute postoperative pain, psychological factors (such as anxiety or catastrophising), and surgical elements including nerve injury, complications and repeated procedures (120). Caesarean birth carries a CPSP incidence of 6–55%; 5–10% of women report this pain as severe (120). A 2025 cross-sectional study of 2160 Brazilian women reported a 12.7% incidence of CPP 12–24 months postpartum (121). Caesarean birth was significantly associated with increased odds of CPP (OR 1.94, 95% CI 1.45–2.58). In a Finnish survey of 438 women, persistent pain one year after birth was significantly more common following caesarean birth than vaginal birth (18% versus 10%; OR 2.1, 95% CI 1.2–3.7) (122). Similarly, a cross-sectional study of 1456 primiparous women in China found that 9.1% developed CPP following childbirth (123). CPP was more frequently reported after caesarean birth compared to vaginal birth (11.2% versus 6.9%). Mechanisms that may contribute include nerve entrapment at the incision site (particularly Pfannenstiel), formation of postoperative adhesions, the development of adenomyosis, isthmocele (niche) formation, and abdominal wall endometriosis.

While there is a common misconception that minimally invasive approaches carry little long-term risk, a systematic review found that the prevalence of CPSP after laparoscopic surgery still ranged from 5.6–17% (124). These included procedures performed for non-pain indications such as laparoscopic donor nephrectomy, adrenalectomy and prostatectomy. Given these associations, a detailed surgical history is important when assessing women with CPP. *[Evidence level 2–]*

Iatrogenic causes of chronic pelvic pain may include retained or migrated foreign bodies, such as gynaecological devices (e.g. Filshie clips or intrauterine systems), which should be considered where pain is temporally related to insertion or surgery, is focal in nature, or where relevant findings are identified on imaging. Synthetic vaginal mesh was introduced in the UK in the mid-1990s for the treatment of stress urinary incontinence and pelvic organ prolapse. However, due to concerns about complications, including persistent pelvic pain, its use was suspended in July 2018 (125). In an RCT of 865 patients, 12% (51/434) of those who received synthetic mesh experienced complications within two years, with 9% (37/434) requiring surgical removal (125, 126). Vaginal mesh is now recognised as a potential cause of CPP, and referral to a Mesh Complications Specialist Centre is recommended for women with suspected mesh-related complications. *[Evidence level 1+]*

Essure was a microinsert used in hysteroscopic sterilisation associated with CPP that was withdrawn from the market in 2018. In a population-based cohort study of 2474 patients in Spain, 0.16% of women developed CPP with resolution of pain after Essure removal (127). A Canadian study of 458 patients reported 4.2% had persistent pain at three months after hysteroscopic sterilisation with Essure, with a six-fold increase in both acute and chronic pain if the patient had a history of chronic pain condition (128). Removal should be considered in women with CPP persisting after insertion. *[Evidence level 2–]*

2. How should pelvic pain be clinically assessed?

Recommendation	Evidence quality	Strength	Rationale for the recommendation

Clinicians should allocate sufficient time for the initial assessment to allow women with CPP to share their experiences, ensuring they feel heard, validated, and believed.	**4**	**GPP**	**Qualitative studies consistently show that women with CPP often feel dismissed or disbelieved during brief, poorly structured consultations, underscoring the need for adequate time to build trust, validate their experiences, and explore their concerns empathetically.**
Clinicians should explore the woman's own understanding, concerns, and beliefs about their pain during the initial consultation.	**4**	**GPP**	**Many women present with theories or anxieties regarding the cause of their pain that warrant early discussion.**
Clinicians should use empathetic and clear communication, recognising that judgmental language can exacerbate the experience of CPP and reinforce unhelpful beliefs.	**4**	**GPP**	**Language and communication can influence pain perception and psychological outcomes.**
Clinicians should introduce and explain the multifactorial nature of CPP at the outset, working in partnership with the patient to develop a shared management plan.	**4**	**GPP**	**Introducing the multifactorial nature of CPP early fosters shared understanding, dispels misconceptions, and supports trauma-informed, collaborative care - key elements shown to improve patient satisfaction and engagement.**
Clinicians should screen for previous trauma and approach clinical assessment of CPP, including history and physical examination, with a trauma-informed approach.	**4**	**GPP**	**Many women, particularly those with a history of trauma, may find clinical assessments distressing.**

An overview of the clinical assessment of CPP is provided in Appendix 2. Multiple qualitative analyses demonstrate women with CPP frequently experience negative interactions with healthcare systems, characterised by clinician dismissiveness, insufficient empathy and invalidation of their pain experiences (129-131). Common themes emerging from patient feedback include feeling unheard, minimised, or disbelieved, often due to time constraints during consultations, inadequate exploration of patient concerns, and insensitive communication practices. As such, it is critical that gynaecology services allocate adequate consultation time for women with CPP. Initial appointments may require extended durations, and a single appointment may not always suffice for both history-taking and examination. Clinicians should explore patients' own understanding, beliefs and concerns regarding their pain, including discussing the multifactorial nature of CPP. Early identification and discussion of patient ideas or concerns about CPP are essential to dispel misconceptions. Clear,

empathetic and non-judgmental communication is essential. Judgmental or dismissive language can significantly exacerbate the psychological impact of chronic pain, perpetuate unhelpful beliefs, and lead to distrust and avoidance of care. Thus, clinicians should communicate using easily understood language, creating an environment where patients feel comfortable, respected and supported. *[Evidence level 4]*

Clinical assessment of CPP should follow a trauma-informed approach that acknowledges the impact of trauma, recognises its signs and symptoms, and aims to prevent re-traumatisation (132). Many women may have a history of trauma, abuse or psychological distress that can affect how symptoms are experienced, reported and explored during consultations. Universal screening for past trauma is recommended, and can be approached through a framing statement, such as "*Many people living with chronic pelvic pain have experienced difficult or traumatic events in the past. These experiences can sometimes affect both physical and mental health, as well as how someone responds to pain and medical care. For this reason, I ask all patients about this. Is there anything in your past that you feel it would be helpful to share today?*" If trauma is disclosed, it is not necessary to ask for the type or details of the trauma. Instead, sensitively explore with the woman whether she feels comfortable having this documented and to what extent. Avoid repeated requests for disclosure, to prevent unnecessary distress or re-living of traumatic experiences.

Trauma-informed principles should underpin both history-taking and physical examination in the assessment of CPP. Sensitivity, respect for personal boundaries, and the promotion of trust are essential throughout. The consultation should, where possible, be conducted with the patient clothed, with privacy provided for dressing and undressing. Each step of the examination should be clearly explained in advance, and patients should be offered choices about what is done and when. Consent should be active and ongoing. A chaperone should be offered for physical examination, and patients should be reassured of their autonomy to pause or decline any part of the assessment at any time. *[Evidence level 4]*

2.1. History

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should take a comprehensive initial history that extends beyond a standard gynaecological history.	**4**	**GPP**	**A broad history allows identification of musculoskeletal, gastrointestinal, urological, psychological and social contributors to pain.**
The presence of others during the consultation should be guided by the patient's preferences. Clinicians should remain alert to signs of domestic violence and coercive control.	**4**	**GPP**	**A private, safe environment facilitates disclosure of sensitive information.**

If the history suggests a specific non-gynaecological contributor to pain or if red flag features are present, clinicians should consider referral to, or collaboration with, relevant multidisciplinary team members.	**4**	**GPP**	**Referral ensures appropriate investigation and holistic management.**
Validated tools such as pain diaries, standardised questionnaires or mobile applications may support assessment in time-limited settings, but clinicians should be aware of their limitations.	**4**	**GPP**	**These tools can support symptom monitoring but are rarely diagnostic.**

A comprehensive clinical history is fundamental to the assessment of women with CPP and should extend beyond a standard gynaecological history. A comprehensive history-taking proforma that can be completed by patients in advance of the consultation, or completed jointly with the clinician, is provided in Appendix 3. History-taking should be conducted in a private setting with the patient fully dressed and with accompanying individuals present per the patient's wishes. Clinicians should remain vigilant for signs of domestic abuse or coercive control and speak to the patient privately if concerns are present.

Clinicians should elicit the nature, onset, duration and distribution of painful symptoms, along with any cyclical variation that may suggest hormonal influences. Gynaecological symptoms should also be explored, including abnormal uterine bleeding (e.g. heavy, intermenstrual, postcoital or postmenopausal bleeding), abnormal vaginal discharge, dysmenorrhoea and dyspareunia. Associated gastrointestinal, urological, musculoskeletal and psychosocial features should be reviewed, with particular attention to symptoms suggestive of chronic overlapping pain conditions. Exacerbating factors such as menstruation, sexual intercourse, physical activity, urination, defaecation and stress should be identified. The impact of symptoms on daily life, including mood, sleep, fatigue, relationships, employment and physical activity, should be assessed. The history should include prior surgical interventions and relevant medical diagnoses. Screening for IBS (via Rome IV (64)) and for interstitial cystitis/bladder pain syndrome (IC/BPS) using the O'Leary-Sant Interstitial Cystitis Symptom Index (133) is recommended, as these conditions frequently coexist with CPP.

Where the history suggests a non-gynaecological cause or contributor to CPP, or red flag features are present (See Table 3: Red flag symptoms and signs), further investigation or referral to appropriate specialties (such as urology or gastroenterology) should be considered. Discussion should include current and previously trialled management strategies, with attention to the patient's perceived effectiveness. This should encompass pharmacological therapies (e.g. analgesics, neuromodulators, hormonal treatments), surgical interventions, psychological or physical therapies, and self-management approaches (see Section 9. What therapeutic options are available?). Finally, it is helpful to explore the patient's ideas, concerns, expectations and goals regarding their pain to facilitate a collaborative and person-centred management plan. *[Evidence level 4]*

Tools such as pain diaries, pain maps, symptom trackers and questionnaires may support symptom documentation and monitoring. They may also provide insight into the functional impact of symptoms, including effects on quality of life, daily activities, mood, and sleep (134). Current evidence does not support the use of symptom-based questionnaires or diaries as diagnostic tools for endometriosis (135). Individual instruments have limited diagnostic accuracy, and studies highlight variability in their reliability, consistency and applicability across different populations (136, 137). These tools are best used as adjuncts to, rather than substitutes for, thorough clinical history-taking. When used appropriately, they can support patient-clinician communication, help identify symptom patterns and assist patients in articulating their pain experience, particularly in time-limited consultations. *[Evidence level 4]*

Table 3. Red flag symptoms and signs

- New pain after the menopause
- Pelvic mass
- Suicidal ideation
- Irregular vaginal bleeding over 40 years of age
- Postcoital bleeding
- Bleeding per rectum
- New bowel symptoms over 50 years of age
- Excessive weight loss

2.2. External examination

Recommendation	Evidence quality	Strength	Rationale for the recommendation
External examination should proceed in a stepwise manner, starting with the least invasive components and guided by patient consent.	**4**	**GPP**	**CPP is often associated with other conditions which may manifest signs elsewhere.**

Physical examination should proceed in a stepwise manner, beginning with the least invasive components (138). Observation of the patient seated and transferring to the examination bed allows for assessment of posture and gait, which may reveal musculoskeletal dysfunction. CPP is frequently associated with musculoskeletal abnormalities, including tenderness of the sacroiliac joints, coccyx and paraspinal musculature.

Where pain is associated with or flared by activities or if associated features such as scoliosis or lower limb pathology coexist, then examination in standing may be appropriate. This allows identification of tenderness over lower back muscles; pain/restriction with forward, back and side flexion; and pain/instability with single leg standing.

Examination should then proceed in a supine position, where the abdomen should be assessed for focal tenderness, surgical scars or abdominal wall trigger points suggestive of myofascial pain. Abdominal wall endometriosis may present as a tender umbilical or incisional nodule. Skin changes such as erythema abigne may also be noted, reflecting repeated application of heat for analgesia. Assessment for features of neuropathic or nociplastic pain may be incorporated into the physical examination. Allodynia may be identified when light touch (e.g. cotton wool or fingertip or mild thermal stimuli) provoke pain that would not normally be painful. Clinicians may also assess for

altered pinprick or cold sensation, which may indicate peripheral or central sensitisation. Temporal summation ('wind-up'), a bedside marker of central sensitisation, can be evaluated by comparing the patient's pain response to a single pin-prick stimulus with their response to a series of repeated stimuli of identical intensity. A progressive increase in perceived pain with repetition is consistent with temporal summation (139, 140).

Single leg raises and lifting the head and neck off the couch assesses movement related pain. Given the prevalence of chronic overlapping pain conditions with CPP, findings outside the pelvis, such as pain in other joints or myofascial trigger points, should be documented, and further systemic examination may be warranted based on history and symptom distribution, *[Evidence level 4]*

2.3. Internal examination

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should be aware that internal examination is most usefully undertaken when there is sufficient time to address any fears or anxieties, particularly in the context of previous trauma.	**4**	**GPP**	**Internal examinations can provoke anxiety or retraumatisation, especially in women with a history of trauma, so they should only be performed when adequate time and sensitivity can be provided to ensure patient comfort, consent, and emotional safety.**
Clinicians should be prepared for additional parts of the history to be revealed at the time of internal examination, including information relating to self-esteem/body confidence, sexual function or previous traumatic events.	**4**	**GPP**	**Internal examinations may prompt unanticipated disclosures about self-image, sexual function, or past trauma, making it essential for clinicians to approach them with openness and flexibility to adapt the consultation accordingly.**
When discussing physical examinations, clinicians should use specific anatomical terms, such as vulval, vaginal or cervical examination, as well as explain the indications.	**4**	**GPP**	**Clear terminology enables patients to understand the nature of and reason for the examination which may aggravate their pain, therefore provide meaningful consent.**

An internal examination may not be appropriate at the first consultation and is not always necessary; its value depends on the clinical history, prior examinations and available investigations. When an internal examination is indicated, it should only be undertaken when there is sufficient time to address any fears or anxieties, particularly in the context of previous trauma and clinicians should be prepared that patients may also disclose further relevant information during the

examination itself. A chaperone should be present for all internal examinations. Clinicians should be aware that pelvic examination can provoke anxiety, fear, pain and traumatisation, particularly among women who have experienced previous sexual violence (141).

Where an examination is proposed, clinicians should clearly name the specific anatomical areas involved and the reason for the examination to support patient understanding. For example, a vaginal examination, cervical examination, or rectal examination i.e. "*this involves a physical examination of your vagina to assess for pelvic floor overactivity*"). Explicit terminology helps ensure patients are fully informed and can consent meaningfully to the examination. The use of a mirror may be helpful for some patients to visualise and understand their anatomy.

Assessment of the perineum and vulva may identify neuropathic features, hyperalgesia or allodynia, which are relevant to pain syndromes such as vulvodynia or pelvic floor myofascial pain. Dermatological vulval pathologies may be evident, and if female genital mutilation is present, this should be managed as per the RCOG GTG No. 53 (142).

When performed, the examination should begin in a supine position with a single-digit assessment of the vagina and pelvic floor muscles to evaluate for signs of pelvic floor myalgia. Pelvic floor tenderness is a common finding in women with CPP and may warrant further evaluation by a specialist pelvic physiotherapist, particularly if myofascial pain is suspected. Findings should be interpreted within the broader clinical context, recognising that tenderness alone does not imply pathology, and that symptom overlap with conditions such as bladder pain syndrome, irritable bowel syndrome and fibromyalgia is frequent.

For many women with CPP even a single digit examination will be painful, particularly when significant pelvic floor tension is present. Clinicians should therefore carefully consider whether the additional information gained from speculum or bimanual examination is required unless there are red flag symptoms or concerns about bleeding, e.g. post-coital bleeding,. A bimanual examination may identify adnexal masses, restricted uterine mobility, or tenderness or nodules in the posterior fornix suggestive of deep endometriosis. However, this information may also be gained by non-invasive imaging. Single digit palpation of the cervix can be helpful to determine tenderness that supports a visceral component to pain. *[Evidence level 4]*

3. Which investigations may be required?

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should perform relevant investigations to rule out potential underlying pathologies. However, the likelihood of normal findings should be emphasised to the patient from the start.	**4**	**GPP**	**Many women with CPP will have normal investigations.**

Women with CPP often have a protracted course in their journey to obtaining help. A diagnosis can provide relief validation of their experience. However, investigations frequently show normal results, even in the presence of significant symptoms. When discussing investigations with women with CPP, the likelihood of otherwise normal findings should be emphasised from the outset and

reassurance provided that their experience, symptoms and concerns are still valid. Selection of investigations should be proportionate to the impact of symptoms, and clinicians should highlight the risks and benefits of each investigation. *[Evidence level 4]*

3.1. Imaging

Recommendation	Evidence quality	Strength	Rationale for the recommendation
All women with CPP should have a pelvic ultrasound prior to referral to secondary care.	**4**	**GPP**	**Ultrasound may detect common gynaecological causes of pelvic pain, such as ovarian cysts or adenomyosis, and helps guide initial management.**
Where appropriate, a transvaginal ultrasound is preferable to a transabdominal ultrasound.	**4**	**GPP**	**Transvaginal ultrasound offers superior anatomical resolution and tissue characterisation of the uterus and adnexa.**
Transvaginal ultrasound and magnetic resonance imaging (MRI) are useful tests to diagnose adenomyosis.	**2+**	**C**	**Both modalities demonstrate moderate diagnostic accuracy and are the most effective non-invasive tests currently available for adenomyosis.**
Transvaginal ultrasound and magnetic resonance imaging (MRI) may be useful to determine the extent and location of endometriosis and inform discussion of further management.	**2+**	**C**	**Imaging may aid surgical planning and informed decision-making, though diagnostic sensitivity is limited, particularly for superficial peritoneal disease.**
Augmented pelvic ultrasound should be available to aid in the detection of endometriosis.	**2+**	**C**	**Augmented pelvic ultrasound, including targeted assessment of the posterior compartment, ovarian position and uterine sliding sign, has superior sensitivity and specificity for the identification of deep endometriosis.**
Non-gynaecologic imaging (e.g. hip, lumbar spine, bony pelvis etc) may be required as indicated by history and examination findings.	**4**	**GPP**	**Alternative causes of pelvic pain, including musculoskeletal and spinal pathology, may be detected with targeted imaging where indicated.**

Pelvic ultrasound is recommended as the first-line imaging investigation for women with CPP and should be performed prior to referral to secondary care. Where feasible, transvaginal ultrasound is preferred over transabdominal ultrasound due to its superior anatomical resolution and ability to assess the uterus, adnexa and rectouterine pouch. MRI can provide superior soft tissue contrast and is particularly useful in assessing adenomyosis, deep endometriosis and disease involving the bowel, bladder or uterosacral ligaments. For some individuals, particularly those with a history of sexual trauma, significant anxiety or marked dyspareunia, transvaginal ultrasound may be distressingly painful or unacceptable. In such cases, an informed decision-making approach should be taken, balancing diagnostic benefit with patient comfort and consent. Transvaginal ultrasound is not routinely appropriate (though not strictly contraindicated) in non-sexually active adolescents, for whom transabdominal imaging is typically preferred. Importantly, the absence of a transvaginal scan should not be used to deny or delay a working diagnosis of endometriosis, particularly when supported by a clear clinical history. *[Evidence level 4]*

A meta-analysis of 10 studies (827 women undergoing transvaginal ultrasound; 317 undergoing MRI) reported that transvaginal ultrasound had a sensitivity of 78% and specificity of 78% for the diagnosis of adenomyosis, whereas MRI had a sensitivity of 78% and specificity of 88% (143). *[Evidence level 2+]*

Both transvaginal ultrasound and MRI have limited utility in detecting endometriosis at all pelvic sites. A 2016 Cochrane review found that for pelvic endometriosis, transvaginal ultrasound had 65% sensitivity and 95% specificity, while MRI had 79% sensitivity and 72% specificity (144). Diagnostic performance is higher for some subtypes. For ovarian endometriomas, transvaginal ultrasound has 93% sensitivity and 96% specificity, and MRI 95% and 91%. For deep endometriosis, ultrasound reaches 79% sensitivity and 94% specificity, while MRI achieves 94% and 77%. Therefore, both transvaginal ultrasound and MRI can help define disease extent, but their diagnostic utility is limited, particularly for superficial peritoneal disease, and normal imaging does not exclude endometriosis. *[Evidence level 2+]*

In 2024, the Society of Radiologists in Ultrasound published a consensus statement recommending augmented pelvic ultrasound to improve the detection of deep endometriosis (145). This approach, supported by European Society of Human Reproduction and Embryology, the Society of Obstetricians and Gynecologists of Canada, and the National Institute of Health and Care Excellence (NICE), includes targeted assessment of the posterior compartment, evaluation of ovarian position (e.g. “kissing ovaries”), and the uterine sliding sign, which may indicate rectouterine deep endometriosis or adhesions ((135, 145-147). In a prospective observational study of 273 patients undergoing laparoscopy, augmented pelvic ultrasound demonstrated a sensitivity of 88.4% and specificity of 78.8% for the detection of deep endometriosis (148). *[Evidence level 2+]*

Non-invasive imaging has been evaluated for detecting pelvic and abdominal adhesions, but the evidence base is limited and inconsistent. Ultrasound using the visceral slide technique can identify adhesions between bowel and the abdominal wall, but reported sensitivity and specificity vary widely across heterogeneous studies, and many carry a high risk of bias (149). Cine MRI can visualise some adhesions directly and may delineate associated anatomical distortion, yet published studies report variable diagnostic performance (sensitivity 73–93% specificity 87–93%), and MRI is further constrained by limited availability, and poor visualisation of the posterior pelvic cul-de-sac (150). Findings such as a negative sliding sign on ultrasound or a normal MRI may be associated with a lower likelihood of significant adhesions; however, neither modality can reliably exclude their

presence. Overall, current evidence is insufficient to recommend imaging for the diagnosis of pelvic or abdominal adhesions in the evaluation of chronic pelvic pain. Nevertheless, if adhesions are suspected upon imaging, clinicians may consider discussing this during the informed consent process.

Imaging of non-gynaecological structures, such as the lumbar spine, hips, or sacroiliac joints, may be warranted where history or examination suggests musculoskeletal or neurological causes of CPP. *[Evidence level 4]*

3.2. Diagnostic laparoscopy

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Diagnostic laparoscopy should not be considered an essential investigation for women with CPP. It is more appropriately offered as a second-line option if empirical or non-invasive treatments have failed.	**4**	**GPP**	**Laparoscopy offers limited diagnostic benefit in many cases and carries inherent procedural risks.**
Imaging findings should be taken into consideration when discussing laparoscopy (e.g. for presence of adenomyosis, deep endometriosis, etc.)	**4**	**GPP**	**Given that nearly half of women with CPP have no visible pathology at laparoscopy, incorporating imaging findings, especially for conditions like deep endometriosis or adenomyosis, helps to better target surgical intervention and avoid unnecessary procedures.**
Where appropriate, women should also be offered opportunistic insertion of a levonorgestrel intrauterine system (LNG-IUS) under anaesthetic at the time of laparoscopy, if this aligns with their treatment preferences and contraceptive needs.	**4**	**GPP**	**Offering LNG-IUS insertion during laparoscopy allows for pain-free delivery of a first-line treatment for dysmenorrhoea and endometriosis-related symptoms, aligning with patient-centred care and optimising use of anaesthesia.**
Women with CPP who undergo a diagnostic laparoscopy that does not reveal a cause for their symptoms should be offered follow-up care in gynaecology to explore alternative approaches.	**4**	**GPP**	**As approximately 1 in 2 women with CPP have no pathology identified at laparoscopy, structured gynaecological follow-up is essential to address ongoing symptoms, provide reassurance, and initiate further**

multidisciplinary pain management.

Diagnostic laparoscopy, involving systematic inspection of the abdomen and pelvis using a laparoscope, has long been considered the cornerstone investigation for women with CPP. It enables visualisation and, where pre-planned, treatment of pathology such as endometriosis, adhesions, or ovarian lesions, as well as biopsy for histopathological confirmation where necessary. Notably, diagnostic laparoscopy remains the only method currently capable of diagnosing superficial peritoneal endometriosis and pelvic adhesions, as these are not reliably detectable with imaging.

However, laparoscopy has important limitations: it cannot diagnose conditions that do not produce macroscopic changes within the peritoneal cavity. These include adenomyosis, which is confined to the uterine myometrium; irritable bowel syndrome and bladder pain syndrome/interstitial cystitis, which lack peritoneal manifestations; and central sensitisation syndromes, such as fibromyalgia or persistent post-surgical pain, which involve altered neural processing. Musculoskeletal causes of pain, including pelvic floor myofascial dysfunction, sacroiliac joint dysfunction and nerve entrapment syndromes, are similarly beyond the diagnostic scope of laparoscopy.

The overall rate of serious complications such as visceral injury is low. Bowel injury occurs in approximately 1 in 769 gynaecological laparoscopies in systematic review data (151). The risk of complications is increased in women with obesity, prior abdominal surgery or significant intra-abdominal pathology. Persistent post-surgical pain associated with laparoscopy presents an additional risk (See Section 6.7). Risks should be discussed in line with the Diagnostic laparoscopy consent form produced by NHS England in collaboration with RCOG (152).

Importantly, laparoscopy may not provide a diagnosis. Approximately 50% of women with CPP have no obvious macroscopic pathology identified at diagnostic laparoscopy, based on data from 17 studies including 2310 participants (range 2% to 73%) (153-159). The most robust data come from a large UK multicentre randomised controlled trial, which found that 54% (n=266/487) of women with CPP had no visible pelvic pathology at diagnostic laparoscopy (153). Further, a 1993 review of 1318 cases reported 39% (n=516) had no identifiable pathology (154). Given these diagnostic limitations, laparoscopy may be more appropriately considered as a second-line investigation, particularly when empirical treatments have failed. However, if there is a high clinical suspicion of pathology only diagnosable via laparoscopy, such as superficial peritoneal endometriosis or pelvic adhesions, or if imaging findings suggest conditions amenable to surgical treatment (e.g. deep endometriosis or ovarian cysts), earlier use of laparoscopy may be warranted. Imaging findings and overall clinical context should guide decision-making. It is recommended that women should be offered insertion of a levonorgestrel intrauterine system (LNG-IUS) under anaesthetic at the time of laparoscopy, if this aligns with their treatment preferences and contraceptive needs, as pain during LNG-IUS insertion may otherwise be a barrier to use. *[Evidence level 2++]*

3.3. Other investigations

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Take appropriate microbiological samples, including for *Chlamydia trachomatis* and *Neisseria gonorrhoeae*, where pelvic inflammatory disease is suspected.	4	GPP	Targeted microbiological sampling supports accurate diagnosis and treatment, helping to prevent long-term sequelae such as tubal damage and exacerbation of CPP.
Consider referral to urogynaecology/urology if chronic/recurrent urinary tract infection has not been explored with the patient and symptoms/history suggest urinary tract infection (urinary symptoms with response to antibiotics).	4	GPP	Mid-stream urine cultures and urinary dipsticks can lack sensitivity (true positive rate) in patients with chronic urinary symptoms.
Clinicians should not routinely conduct blood tests in the assessment of CPP, consider only if indicated by clinical history (e.g. CA-125, inflammatory arthropathy screen, FSH, estradiol).	4	GPP	There are currently no validated blood tests or biomarkers for diagnosing CPP.
Perform other invasive investigations (e.g. cystoscopy, colonoscopy, sigmoidoscopy) only where clearly indicated by clinical history or symptoms.	4	GPP	These investigations have no routine diagnostic role in CPP, but may be needed where bladder, bowel, or malignancy-related symptoms are present.

Other investigations in the assessment of CPP should be guided by clinical history and symptomatology. Microbiological samples, including testing for *Chlamydia trachomatis* and *Neisseria gonorrhoeae*, should be taken where pelvic inflammatory disease is suspected. Mid-stream urine samples to exclude urinary tract infection can also be considered. In a retrospective study of women acutely admitted to a tertiary gynaecology centre, among the 100 women admitted with pelvic pain who had a pre-existing diagnosis of CPP, 24% (n=11/46) had a positive mid-stream urine culture, and no women (n=0/72) had positive vaginal swab cultures (160). Routine blood tests are not recommended, as no validated biomarkers currently exist to aid in the diagnosis of CPP or endometriosis (161). However, selected tests such as CA-125, inflammatory markers, or hormonal profiles (FSH, oestradiol) may be considered where clinically indicated. Invasive diagnostic procedures such as cystoscopy, colonoscopy or sigmoidoscopy should not be performed routinely but may be warranted where symptoms suggest an alternative diagnosis, or to exclude malignancy. While cystoscopy cannot confirm or exclude bladder pain syndrome, it may be necessary to rule out other bladder pathology in selected cases (162). *[Evidence level 4]*

4. What therapeutic options are available?

4.1. Multimodal and person-centred approach

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should avoid relying on a single therapeutic approach, as it is unlikely to be effective due to the multifactorial nature of CPP.	**4**	**GPP**	**Multidisciplinary and multimodal approaches may improve pain, psychological distress, and functional impairment in CPP.**
The therapeutic approach should be tailored to fit the patient's life stage, lifestyle and beliefs.	**4**	**GPP**	**Individualised treatment enhances patient engagement, aiming to improve effectiveness of CPP management.**
Clinicians should inform patients about the available management approaches and ensure they have the opportunity to discuss all treatment options, including the choice of no treatment.	**4**	**GPP**	**This aims to support informed-decision making.**
Pain education should be included in the management of CPP. Clinicians should explain the biopsychosocial model of CPP to women, highlighting how biological, psychological, and social factors interact to influence the pain experience.	**4**	**GPP**	**Pain education helps patients understand their condition, reframe misconceptions, and adopt practical self-management strategies.**
A multidisciplinary CPP team should provide expertise in gynaecology, pain medicine, physiotherapy and psychology.	**4**	**GPP**	**A multidisciplinary team addresses the diverse contributors to CPP and allows for integrated care.**

Clinicians should focus on identifying factors (as outlined in Section 6) that may contribute to CPP and how it is experienced, and aim to address these factors individually, viewing management through a biopsychosocial framework (see Figure 1). A multidisciplinary and multimodal approach is often required to address pain, psychological distress and functional impairment. A prospective cohort study of 296 women with CPP managed in a tertiary referral centre demonstrated that a multidisciplinary, multimodal approach combining minimally invasive surgery, medical management, pain education, physiotherapy and psychological therapy significantly reduced CPP severity, improved quality of life and decreased healthcare utilization (163). At one-year, median pain scores significantly decreased (from 6 to 4 out of 10), functional quality of life improved, and the proportion of participants requiring physician visits (73% to 36%) or emergency visits (24% to 11%) was reduced. Further supporting this multidisciplinary model, a retrospective review of 107 women with CPP attending a multidisciplinary clinic found that psychological distress and functional impairment were common and closely interrelated. Notably, functional limitations in daily activities, including sleep, social participation and employment, were strongly associated with anxiety and depression, rather than pain severity alone. This highlights the importance of addressing psychological symptoms to effectively improve overall functioning and quality of life (164). These data support the use of multimodal and multidisciplinary approaches to address CPP, psychological distress and functional impairment. While interdisciplinary CPP pain management programmes have been developed and implemented (165), there remains very limited high-quality evidence evaluating their effectiveness. Regardless of the specific combination of treatments used, it remains crucial to adapt treatment plans to each patient's unique circumstances, including their lifestyle, personal goals, life stage, beliefs and preferences, to promote patient engagement, and ultimately, treatment effectiveness. *[Evidence level 4]*

Clinicians should be clear that multiple management approaches exist and that evidence for the optimal strategy is often limited. They should provide clear information, ensure that women or people being cared for can discuss all available options (including tests, investigations, treatment and no treatment) and recognise that in some circumstances, joint review with the wider multidisciplinary team may be beneficial.

Pain science education, grounded in a biopsychosocial model, aims to help individuals better understand and manage their pain. This concept should be integrated into care for all women with CPP, although evidence on efficacy on pain outcomes or on optimal content and delivery methods remains limited (166). A qualitative thematic analysis (167) exploring the views of 20 women with CPP identified four core concepts they considered important in relation to pain education:

(1) a sensitised nervous system leads to overprotective pain,
(2) pain does not always mean my pelvis is damaged (although sometimes it does),
(3) how I think, feel, and "see" my pain can make it worse, and
(4) I can change my pain… slowly.

These themes underscore the importance of validating lived experiences of CPP, reframing misconceptions about pain, and empowering them with practical strategies to gradually improve their condition. In parallel, a Delphi consensus of expert clinicians developed a structured set of 125 pain science education concepts specific to pelvic pain (168). These included understanding that persistent pain may not reflect ongoing tissue damage, the role of the brain and nervous system in modulating pain, and the importance of individualised, contextual education to support pain-related

behavioural change. Taken together, these findings emphasise that clinicians should discuss the complex nature of pain, communicate that persistent pain does not always signify ongoing tissue damage, and explore individualised pain self-management strategies. Online resources such as Live Well with Pain (https://livewellwithpain.co.uk/) can support education by providing skills and knowledge for clinicians and self-management resources for people living with pain. *[Evidence level 4]*

4.2. Pharmacological treatments

4.2.1. Hormonal treatments

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Women with cyclical pain should be offered a therapeutic trial using hormonal treatment (e.g. a combined hormonal contraceptive or progestogen) for a period of three to six months.	**1–**	**C**	**Hormonal treatment may improve CPP with cyclical features.**
Where hormone-responsive pain is present (endometriosis, adenomyosis, or clinical description) but first line hormonal therapies are ineffective or poorly tolerated, a trial of a GnRH agonist or antagonist may be considered.	**2+**	**C**	**GnRH analogues can provide further symptom relief by inducing ovarian suppression, with evidence supporting their efficacy particularly in endometriosis, although use is typically limited by side effects and the need for add-back therapy.**
Dyspareunia, vulvovaginal and lower urinary tract symptoms in peri-menopausal and post-menopausal women may benefit from hormone replacement therapy (HRT) and/or vaginal oestrogen.	**1–**	**C**	**Low oestrogen states in peri- and post-menopausal women can contribute to urogenital and sexual pain symptoms, and although evidence is limited, local therapies are supported for symptom relief in vulvovaginal atrophy.**

Most evidence for hormonal treatment in women with CPP relates specifically to endometriosis-associated pain, and generalisability beyond this group is limited. As such, no high-quality placebo-controlled trials are available to guide hormonal treatment in woman with CPPS. However, any pain and associated symptoms with a cyclical component or exacerbated by menstruation may benefit from hormonal therapy that results in amenorrhoea.

Ovarian suppression using a combined hormonal contraceptive or a progestogen can be an effective treatment for CPP with cyclical features, such as some endometriosis-associated pain, as well as pain

arising from other hormone-responsive conditions including adenomyosis and dysmenorrhoea. A 2014 Cochrane review identified moderate-quality evidence (n=204; 2 trials) that progestogen treatment (medroxyprogesterone acetate) is more effective than placebo for reducing CPP in women without a specific gynaecological diagnosis (169). Women receiving progestogen were more likely to report a >50% reduction in pain on a visual analogue scale both at the end of treatment (OR 3.00, 95% CI 1.70–5.31) and up to nine months later (OR 2.09, 95% CI 1.18–3.71). In a small, randomised trial of 60 women with idiopathic CPP, treatment with a combined hormonal contraceptive, either vaginal ring or orally, significantly improved pain after 84 days of treatment, with mean 0-10 visual analogue scale reductions of 6.23 (95% CI 5.45–7.01) and 5.53 (95% CI 4.83–6.23), respectively (170). *[Evidence level 1–]*

Further, a meta-analysis of 15 randomised controlled trials (n=1680 women with endometriosis) found that hormonal treatments including combined oral contraceptives, oral, intrauterine and intramuscular progestogens, and intramuscular GnRH agonists, were associated with a similar, clinically significant reduction in pelvic pain (12, 147). Pain was measured using a 0–100 visual analogue scale, with pain reductions ranging from 12.6 points (95% CI -15.3– -9.8) with oral progestogens to 17.7 points (95% CI -25.5– -9.8) with intrauterine progestogens (minimum clinically important difference=10 points). When endometriosis is suspected, treatment is often initiated based on a clinical diagnosis without surgical confirmation, in line with current endometriosis guidelines (135). In this context, a three-to-six-month trial of hormonal therapy (using a combined hormonal contraceptive or a progestogen) is recommended to suppress ovulation and hormone-responsive pathology, provided pregnancy is not immediately planned. Achieving amenorrhoea is preferable, and for those using a combined oral contraceptive, this can be facilitated by continuous use of a monophasic preparation (i.e. omitting the pill-free interval). *[Evidence level 1+]*

A 2025 network meta-analysis of six RCTs (n=563) found that dienogest significantly reduced adenomyosis-associated pelvic pain compared to placebo (mean difference [MD] 4.10, 95% CI 0.49–7.71) and intrauterine progestogen (MD 3.05, 95% CI 0.45–5.65) at three months, using a 0–10 visual analogue scale (171). It also remained superior to combined oral contraceptives at six months (MD –2.85, 95% CI -5.30– -0.39). Although dienogest was the most effective treatment, it was associated with more adverse effects, including irregular uterine bleeding, hot flushes and breast tenderness. Combined oral contraceptives and the levonorgestrel-releasing intrauterine system (LNG-IUS) may be better-tolerated alternatives. *[Evidence level 1+]*

Migraine, particularly with aura, is more common in individuals with CPP and may contraindicate the use of oestrogen-containing contraceptive preparations in premenopausal women and girls (172). A careful headache history should be taken before prescribing, in line with UK Medical Eligibility Criteria for Contraceptive Use (UKMEC) guidance (173), as oestrogen may need to be avoided in some cases. Non-oral oestrogen HRT should be used for post-menopausal women with CPP and migraines with or without aura (174). *[Evidence level 4]*

Gonadotropin-releasing hormone (GnRH) analogues may also be considered for the management of cyclical or hormone-responsive CPP, particularly if symptoms are refractory to first-line hormonal treatments. Their use can provide therapeutic benefit by inducing a hypo-oestrogenic state but is typically limited to short-term courses due to side effects including vasomotor symptoms and bone mineral density loss. GnRH analogues are available as injectable preparations and, more recently, as oral formulations, including combination preparations (e.g. relugolix/estradiol/norethisterone acetate) that incorporate hormonal add-back therapy within a single tablet. Current efficacy

evidence is limited to women with endometriosis (12). Add-back hormone therapy mitigates some of these adverse effects but the long-term impacts are unknown. GnRH analogues may also serve as a diagnostic tool, helping to clarify the extent to which symptoms are hormonally driven, potentially supporting a working diagnosis of endometriosis or adenomyosis, and illustrate the presence of other likely pain mechanisms where pain persists despite ovarian suppression. Although evidence is limited, their use may offer an alternative to hysterectomy or oophorectomy for premenopausal women seeking symptom relief while preserving fertility and avoiding the risks of surgery. *[Evidence level 2++]*

Topical vaginal oestrogen should be offered to peri- and post-menopausal women, or those in hypo-oestrogenic states such as when on GnRH analogues or long-term injectable Medroxyprogesterone contraception with vulvovaginal atrophy. Low-quality evidence from a Cochrane review supports its effectiveness in relieving symptoms compared to placebo (175). *[Evidence level 1–]*

There is no contraindication to the use of systemic HRT for postmenopausal women with CPP. However, for women with CPP who have had a hysterectomy and a history of endometriosis, combined continuous HRT should be considered because unopposed oestrogen may increase the risk of recurrence of endometriosis-associated pain and malignant transformation of residual or microscopic endometriosis (135).

4.2.2. Non-hormonal treatments

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Simple analgesia, such as paracetamol or non-steroidal anti-inflammatory drugs, are recommended for regular use.	**4**	**GPP**	**NSAIDs are safe and may be helpful due to the inflammatory component of many CPP conditions and are effective for primary dysmenorrhoea.**
Women should be offered analgesia whilst investigations are ongoing.	**4**	**GPP**	**Offering analgesia during investigations may help maintain quality of life and alleviate distress while awaiting diagnosis.**
Opiates should not be prescribed by gynaecologists for CPP.	**4**	**GPP**	**Opioids offer no long-term benefit in CPP and pose risks of dependence and side effects.**
Medication targeting the nervous system such as amitriptyline, nortriptyline, gabapentin, pregabalin and duloxetine may be considered where appropriate in line with NICE guidance on neuropathic and chronic	**4**	**GPP**	**These agents may be helpful for neuropathic components of pain but evidence in CPP is limited.**

primary pain conditions (CG173 and NG193).			
A trial of topical lidocaine may be considered for some women with dyspareunia, urethral and vulval pain.	**4**	**GPP**	**Topical lidocaine may provide symptom relief for selected individuals with localised vulval or vestibular pain, although evidence is inconsistent and primarily based on observational data.**
In individuals with CPP and coexisting symptoms of BPS/IC or IBS, particularly those with multiple allergies, a trial of combined H1 and H2 receptor antagonists (antihistamines) may be considered.	**4**	**GPP**	**This approach should be individualised, as evidence remains limited, and monotherapy is unlikely to be sufficient.**

There is limited evidence to guide the use of analgesics in CPP, and the heterogeneity of CPP necessitates an individualised approach to non-hormonal treatments. Many patients will reasonably request analgesia, and decisions should consider individual preferences, side effects and the likely underlying pathophysiology.

Simple analgesics such as paracetamol and non-steroidal anti-inflammatory drugs (NSAIDs) are generally safe and may be helpful when used regularly or during pain flares. A 2015 Cochrane review of 80 trials (n=5820) found that NSAIDs are effective for the treatment of primary dysmenorrhoea. Compared to placebo, NSAIDs significantly improved pain relief (OR 4.37, 95% CI 3.76–5.09; 35 RCTs, low-quality evidence), and appeared more effective than paracetamol (OR 1.89, 95% CI 1.05–3.43; three RCTs, low-quality evidence) (176). Given the inflammatory processes often implicated in CPP, such as dysmenorrhoea and endometriosis, NSAIDs should be considered a first-line option. *[Evidence level 4]*

There is no evidence to support the long-term use of opioids for CPP, and their use is not recommended by NICE for the management of chronic primary pain conditions (177). Opiates should not be initiated by gynaecologists due to the risks of tolerance, dependence, and lack of demonstrated benefit. Where they are required in episodes for acute pain, they should be used for a limited period and a plan for dose de-escalation made upon initiation. *[Evidence level 4]*

Medications targeting the nervous system such as amitriptyline, nortriptyline, gabapentin, pregabalin and duloxetine may be beneficial for those with a neuropathic component of their pain, although evidence specific to women with CPP is limited. In a large clinical trial of 306 women with CPP and no pathology at laparoscopy, gabapentin did not significantly reduce pain over 16 weeks compared to placebo and was associated with more adverse effects such as dizziness and drowsiness (178). Therefore, these medications should not be routinely used but may be considered in line with NICE guidance on neuropathic and chronic primary pain conditions (177, 179).

A recent meta-analysis included 313 double-blind randomised controlled trials (284 pharmacological, 29 neuromodulation) involving 48,789 adults with neuropathic pain (180). Tricyclic antidepressants were recommended as a first-line treatment, with a number needed to treat (NNT)

of 4.6 and a number needed to harm (NNH) of 17.1. α2δ-ligands such as gabapentin and pregabalin were also first-line treatments (NNT 8.9; NNH 26.2), as were serotonin-norepinephrine reuptake inhibitors (SNRIs; NNT 7.4; NNH 13.9). Although several treatments showed reasonable efficacy, all had limitations either in effect size or tolerability, underscoring the importance of tailoring treatments to individual patients and monitoring for adverse events. This meta-analysis did not include studies specific to CPP, and recommendations were based on broader neuropathic pain populations. However, in individuals with CPP where a clear neuropathic component is suspected, treatment may be considered in line with these recommendations. *[Evidence level 4]*

Topical lidocaine may offer symptomatic relief for selected women with dyspareunia, urethral, or vulval pain, particularly those with localised vestibulodynia. A small observational study found that nightly application of 5% lidocaine ointment led to a significant reduction in pain during intercourse and improved ability to engage in sexual activity (181). However, findings from a 12-week randomised controlled trial of 133 women with vulvodynia suggest that lidocaine cream does not perform significantly better than placebo (182). Given these mixed results, lidocaine can be considered on a trial basis in selected cases, with a clear understanding of its limitations and the importance of individualised response. *[Evidence level 4]*

Emerging evidence implicates histamine signalling in the pathophysiology of BPS/IS and IBS, with studies showing overexpression of histamine receptors and mast cell activation contributing to visceral hypersensitivity (183-185). Given this, a trial of combined H1 and H2 receptor antagonists may be considered in individuals with CPP who exhibit coexisting BPS/IC or IBS symptoms and multiple allergies, although this should be limited to a specialist multidisciplinary setting involving gastroenterology and/or urology. While these medications are generally well tolerated, the evidence base remains limited, and their use should be individualised. Importantly, targeting both H1 receptors (using e.g. cetirizine or fexofenadine) and H2 receptors (using e.g. ranitidine or cimetidine) is likely necessary, as monotherapy may be insufficient. *[Evidence level 4]*

4.3. Psychological interventions

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should offer specialist pain psychology to women with CPP they feel would benefit, but it needs to be approached sensitively with the patient.	**4**	**GPP**	**Psychological factors are central to CPP, and psychological therapies may improve both pain and psychological outcomes.**
Community mental health services may be beneficial for those with co-existing mental health conditions.	**4**	**GPP**	**Many women with CPP experience anxiety or depression, and referral to community mental health services may support broader wellbeing.**
Clinicians should be aware that dedicated trauma services may be required for those with PTSD.	**4**	**GPP**	**A history of trauma or PTSD is common women with CPP; dedicated trauma care may be necessary with ongoing symptoms.**

Psychological factors are central to the experience of CPP and form a core component of effective management. Psychological therapies, grounded in the biopsychosocial model of pain, aim to address the cognitive, emotional, and behavioural factors that contribute to symptom persistence. As outlined in Section 6, many individuals with CPP also experience stress, anxiety or depression, which can exacerbate pain and impair quality of life. It is important to clarify the difference between pain psychology and mental health psychology when discussing this with women with CPP.

A 2011 systematic review (four small RCTs, n=312) found that psychological therapy significantly reduced pain at both three months (standardised mean difference –3.27, 95% CI -4.52– -2.02) and six months or longer (-3.95, 95% CI -5.35– -2.55), on a 0–10 visual analogue scale (186). However, the authors concluded that the current evidence did not allow firm conclusions about the effect of psychological interventions on self-reported pain scores in women with CPP due to study limitations.

A 2024 systematic review (14 RCTs; n=871) assessed the effectiveness of biopsychosocial interventions for the management of CPP (187). Cognitive behavioural therapy (CBT) and acceptance and commitment therapy (ACT)-based approaches were found to be effective in reducing pain and improving psychometric outcomes. All studies reported improvements in pain intensity, and 13 out of 14 also demonstrated improvements in psychological outcomes. While findings support the use of psychological therapies in CPP, the review noted considerable heterogeneity in interventions and outcome measures, with several studies at high risk of bias. Pain science education (See Section 9.1) and support for engagement with valued activities were common features of effective interventions. In contrast, a 2025 systematic review and meta-analysis of 10 RCTs (n=547) reported no clinically important short-term effect of predominantly psychological approaches on pain intensity compared with control (mean difference of -0.31 (95% CI -0.95–0.34) on a 0–10 pain scale) (188). It should be noted that reduction in pain intensity might not be the best primary outcome to assess for psychosocial interventions as many aim to reduce the impact of pain rather than the intensity per se.

Overall, the evidence base is limited by small study sizes, clinical heterogeneity and variation in psychological treatment modalities. While psychological interventions may not produce large reductions in pain in isolation, they remain an important component of multidisciplinary care. Referral to community services such as may be appropriate for those with psychological distress or overlapping psychiatric diagnoses, and referral to dedicated trauma services should be considered for individuals with a history of trauma or post-traumatic stress disorder (PTSD). Women with symptoms of active PTSD such as re-experiencing should be offered referral to a dedicated trauma service. *[Evidence level 2++]*

4.4. Physiotherapy

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Physiotherapy should be made available for woman with CPP and may be provided by community services or within urogynaecology, chronic pain or CPP teams.	**1–**	**C**	**Systematic review of RCTs showed benefit but included studies has methodological concerns.**

Women with CPP and clinical features suggestive of pelvic floor dysfunction (i.e. dyspareunia, difficulty with speculum examination or tampon insertion, pelvic floor muscle tenderness or overactivity) should be offered early referral to a pelvic floor physiotherapist with experience managing pelvic pain.	**4**	**GPP**	**Physiotherapy interventions are heterogenous and often involve time-dependent goals. Early referral will reduce delays and can be offered concurrently with other treatment options.**

Physiotherapy is increasingly recognised as a key component of multidisciplinary management of CPP, although supporting evidence remains limited. Physiotherapy interventions are heterogeneous and typically tailored to the individual, addressing musculoskeletal dysfunction, pain-related behaviours, and avoidance of activity. Viscero-somatic referral has been repeatedly reported in women with CPP (See Section 6.4), contributing to altered muscle sensitivity and the development of secondary musculoskeletal dysfunction. Pelvic floor muscle dysfunction is a common finding and may contribute both to pelvic pain (including dyspareunia) but also to associated symptoms such as voiding and defecatory dysfunction.

A 2024 systematic review of 14 randomised controlled trials evaluating biopsychosocial treatments for women with CPP included four physiotherapy-based interventions (187). Three studies investigated one-to-one physiotherapy, and one assessed a group-based programme. All four demonstrated statistically significant benefit compared with control groups. Core elements across interventions included pain science education (covering information about CPP, fear of movement, beliefs about activity, lifestyle, and behavioural advice) as well as goal setting and graded exercise (189-192). However, the review authors identified methodological concerns in all four studies, and one was deemed to be at high risk of bias.

In a 2025 systematic review and meta-analysis of non-pharmacological conservative therapies for CPP without a defined pathology, seven trials (n=476) specifically evaluated multimodal physiotherapy interventions, which typically combined pelvic floor muscle training, manual therapy, education and self-management strategies (188). Physiotherapy was associated with a clinically meaningful reduction in pain, with a mean difference of -2.87 (95% CI -4.32– -1.45) on a 0–10 pain scale immediately after treatment, and -3.09 (95% CI -5.32– -0.88) at 12 to 36 weeks. The evidence was rated as high certainty for short-term effects and moderate for intermediate outcomes, supporting the role of multimodal physiotherapy as an effective treatment for pain reduction in women with CPP.

Although concerns remain regarding study quality and heterogeneity, the findings support physiotherapy's role within a biopsychosocial model of care. To ensure treatments are appropriately individualised, physiotherapy should be delivered by clinicians with specific expertise in chronic pelvic pain and women's health. It should be made available to women with CPP and may be delivered through community services, urogynaecology, chronic pain or dedicated pelvic pain teams. *[Evidence level 1–]*

4.5. Other invasive therapeutic procedures

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Repeated surgical treatment of endometriosis should be carefully considered, taking into account previous response to surgery.	4	C	There is limited evidence on the benefit of repeat surgery for persisting pelvic pain in women with endometriosis.
Hysterectomy is not routinely recommended for treatment of CPP. However, it may be appropriate in selected individuals, particularly those with confirmed adenomyosis, or with associated coexisting conditions such as heavy menstrual bleeding or pain that is unresponsive to medical therapies.	4	GPP	While hysterectomy may offer symptom relief for selected women with identifiable uterine pathology, large cohort and prospective studies show a significant proportion of patients continue to experience persistent or new pain postoperatively, and the procedure carries long-term risks including cardiovascular and metabolic complications.
Nerve blocks and spinal cord stimulators are not recommended in CPP, and these procedures carry risks including altered visceral functions.	2–	D	Systematic reviews and case series indicate weak, low-quality evidence for nerve blocks and neuromodulation in CPP, with inconsistent outcomes, high risk of bias and potential adverse effects including disruption of visceral function, making these interventions unsuitable for routine use.

A 2020 Cochrane review of 14 randomised controlled trials evaluated the effectiveness and safety of laparoscopic surgery for endometriosis (193). The review found very low-quality evidence suggesting uncertainty about the benefit of laparoscopic treatment compared to diagnostic laparoscopy alone for improving pain at six months (MD 0.90, 95% CI 0.31–1.49) and at 12 months (MD 1.65, 95% CI 1.11–2.19), based on a single small study (n=16). Similarly, the effect of laparoscopic treatment on quality-of-life measures was unclear, with low-quality evidence from one study (n=39) reporting non-significant differences in EuroQol-5D scores and SF-12 physical and mental health components.

In addition, a systematic review of 25 studies involving 2652 participants (including nine RCTs) reported recurrent pain in 15.8% of women who underwent surgical excision of endometriosis lesions but did not receive postoperative hormonal therapy (194). Currently, no evidence supports repeat surgery over medical management for women with previously treated endometriosis who experience recurrent symptoms. A retrospective study of 595 women undergoing repeat surgery reported higher rates of intraoperative complications, including longer operating time, extensive adhesiolysis, and increased risk of ureteric or visceral injury (195). Therefore, decisions regarding repeat surgical intervention should be individualised and based on the response to previous

treatment and consideration of factors such as central and peripheral sensitisation, which may contribute to ongoing pain even in the absence of disease progression. *[Evidence level 1– to 2–]*

There is limited evidence to recommend hysterectomy as a treatment option for CPP, particularly in the absence of clearly defined gynaecological pathology. A retrospective population-based study in Sweden of 16,694 women who underwent hysterectomy for benign conditions found 22% of women with pre-operative pelvic pain had persistent pelvic pain and 8% of women experience new postoperative pain (196-198). Hysterectomy may benefit select individuals, with symptoms directly attributable to uterine pathology, such as adenomyosis or fibroids, with co-existing heavy menstrual bleeding or pain that is unresponsive to medical therapies. In these cases, hysterectomy eliminates menstrual bleeding and dysmenorrhoea, providing symptomatic relief. Decisions should be individualised, considering age, fertility desires, symptom burden and the likely pathology. In women with extensive adenomyosis, heavy menstrual bleeding and CPP who have completed their family, hysterectomy is a reasonable option. A multidisciplinary approach is advised, particularly for younger patients or when pain symptoms are not clearly attributable to the uterus.

Hysterectomy, with or without oophorectomy, is associated with an increased incidence of cardiovascular disease, metabolic disease and mental health disorders, which should be communicated to patients and inform decision-making where hysterectomy is being considered as a pain treatment. A cohort study of 144,260 postmenopausal women found that hysterectomy with oophorectomy before age 40 was associated with an increased risk of cardiovascular diseases (HR 1.87, 95% CI 1.36–2.58) compared to those without premature menopause (199). Further, cohort studies of 2,094 women who underwent hysterectomy with ovarian conservation showed increased long-term risks of de novo hypertension (HR 1.13, 95% CI 1.03–1.25), obesity (HR 1.18, 95% CI 1.04–1.35), coronary artery disease (HR 1.33, 95% CI 1.12–1.58), and depression (HR 1.26, 95% CI 1.12–1.41), compared to age-matched controls after adjustment for 20 pre-existing chronic conditions (200). *[Evidence level 2–]*

Case series investigating pudendal and ganglion impar nerve blocks suggest limited evidence of benefit for women with pain localised to these nerve distributions (201, 202). A systematic review of 36 studies, including 8 randomised controlled trials, evaluated neuromodulation techniques such as sacral nerve stimulation (SNS), percutaneous tibial nerve stimulation (PTNS), transcutaneous electrical nerve stimulation (TENS), intravaginal stimulation, peripheral nerve stimulation (PNS), and spinal cord stimulation. While most studies reported reductions in pain scores, the overall evidence was poor in quality, with high risk of bias and limited reporting on quality-of-life outcomes and adverse event . Acupuncture and other complementary therapies may offer relief for some women, although supporting evidence is limited (203, 204). There is no evidence to support the use of botulinum toxin injections for CPP (205, 206). *[Evidence level 1– to 3]*

Laparoscopic uterosacral nerve ablation (LUNA) has been shown, via a large clinical trial, to be ineffective in the management of CPP (153). *[Evidence level 1++]*

4.6. Lifestyle interventions

Recommendation	Evidence quality	Strength	Rationale for the recommendation

Lifestyle factors (e.g. diet, physical activity, and sleep) should be addressed as part of a multimodal approach, and discussions should be broached sensitively.	**4**	**GPP**	**Lifestyle factors may contribute to or result from CPP and addressing them can improve overall well-being, though evidence specific to CPP remains limited.**
Dietary advice should ideally be delivered with the support of a dietitian.	**4**	**GPP**	**While evidence for dietary interventions in CPP is limited, patients with coexisting IBS or BPS may benefit from identifying and avoiding dietary triggers.**
Clinicians should be aware that obesity can exacerbate many of the factors associated with CPP, but weight loss is challenging and needs to be broached sensitively.	**4**	**GPP**	**Although obesity is not clearly linked to CPP severity, it may exacerbate coexisting conditions such as poor sleep or back pain and complicate medical and surgical treatment.**

Multiple lifestyle factors such as sleep, diet, physical activity, and stress are known to influence chronic pain and impact quality of life (207). While high-quality evidence specific to CPP is limited, there is no reason to assume these factors are not relevant. Addressing lifestyle factors can empower women to take an active role in managing their symptoms, and may offer benefit as part of a multimodal approach. However, meaningful change may be challenging, and appropriate support, through primary care, specialist nurses, lifestyle practitioners or social prescribers, is often needed. Such discussions should be approached with sensitivity to avoid invalidating the patient's experience and to promote meaningful engagement. *[Evidence level 4 (extrapolated from generalised chronic pain)]*

4.6.1. Diet

Many individuals with CPP explore dietary modification as a self-management strategy. In a large international survey of 2,388 people with confirmed endometriosis, 84% reported making dietary changes and 59% used supplements; of these, 67% and 43% respectively perceived an improvement in pain (208). However, high-quality evidence supporting specific dietary interventions remains limited. A 2021 systematic review of 43 studies found that whole-food (i.e. non-supplement-based) dietary interventions were associated with reduced chronic pain (primarily in conditions such as rheumatoid arthritis, fibromyalgia, and osteoarthritis) (209). A meta-analysis of 25 groups showed a significant reduction in pain scores when analysed by combined dietary groups (standardised MD -0.44; 95% CI -0.63– -0.24). While no single diet emerged as superior, commonalities such as improved diet quality, increased nutrient density, anti-inflammatory effects, and weight loss may underlie the observed benefits. Specifically, anti-inflammatory diets have been associated with improvements in both pain and quality-of-life outcomes, potentially mediated through reduced systemic inflammation and oxidative stress (210). Where gastrointestinal symptoms are prominent, it is important to consider the possibility of overlapping IBS (See Section 6.2.1). Dietary strategies such as a low FODMAP diet may be appropriate in this subgroup, ideally with the support of a dietitian (211). Restrictive or exclusion diets should be approached cautiously, as they may carry

risks including nutritional deficiencies or disordered eating patterns, particularly if undertaken without professional support.

4.6.2. Physical activity

Physical inactivity is recognised as a risk factor for the development of chronic pain. A longitudinal cohort study found that inactivity was associated with an increased risk of developing chronic pain within one year (adjusted OR 1.8; 95% CI 1.1–3.0), particularly when combined with non-restorative sleep or stress (212). Animal models provide mechanistic support, showing that regular physical activity prevents chronic muscle pain by reducing central sensitisation via downregulation of NMDA receptor activation in the CNS (199). A 2017 Cochrane review found low-quality evidence that physical activity and exercise may lead to small-to-moderate improvements in pain severity and physical function in adults with chronic pain, with inconsistent effects on quality of life and psychological outcomes (213). Exercise is often limited in women with CPP due to fear of flaring pain, and any recommendation should be tailored. Gentle or non-weight-bearing forms of activity such as yoga, pilates or swimming may be more suitable. Physiotherapy input may be needed to guide safe engagement especially for those with limited mobility or joint and connective tissue disorders.

4.6.3. Weight

There is no clear evidence that overweight or obesity is directly associated with the severity of CPP, and the impact of weight loss on pain outcomes remains uncertain. In a study of 91 women with CPP, pain scores did not differ significantly between normal, overweight, and obese groups (214). However, obesity can exacerbate conditions often associated with CPP, such as lower back pain and sleep disorders (215, 216). Furthermore, obesity presents several challenges in CPP care, including difficulties with physical examination and imaging, increased anaesthetic and surgical risk, and limitations in pharmacologic treatment options. Hormonal therapies such as combined oral contraceptives may carry elevated thromboembolic risk, and surgical risks may outweigh potential benefits. While endometriosis is more commonly associated with a lower BMI, other conditions such as adenomyosis and myofascial pain may be more prevalent in individuals with obesity (217). Patients should be supported with weight management if desired, using non-judgmental and inclusive approaches.

4.6.4. Sleep

Poor sleep quality and reduced sleep duration commonly co-exist in individuals with chronic pain including CPP; and the prevalence of sleep disorders such as insomnia are highly prevalent (218-220). The relationship between pain and sleep is bidirectional: pain can disrupt sleep, while disturbed sleep lowers pain thresholds and heightens spontaneous pain (221). As such, identifying and addressing poor sleep through educational interventions such as sleep hygiene advice, may be beneficial (See NICE practical guidance on good sleep hygiene (222) (223).

4.6.5. Smoking

Cigarette smoking is associated with an increased risk of chronic musculoskeletal pain. A meta-analysis of 32 studies involving over 296,000 participants found that current smokers had a significantly higher risk of developing chronic musculoskeletal pain compared to non-smokers (OR

1.23; 95% CI 1.09−1.40); notably, past or ever smoking was not significantly associated with increased risk (224). Individuals with chronic pain tend to have higher smoking prevalence than the general population (225), and emerging evidence suggests smoking may contribute to central sensitisation and neuroinflammation (226, 227). Additionally, smoking status may prevent use of oestrogen containing hormonal therapies due to elevated thromboembolic risk. Smoking cessation should be encouraged as part of CPP management, and patients should be signposted to local stop-smoking services for support.

4.7. Management of pain flares/exacerbations of chronic pelvic pain

Recommendation	Evidence quality	Strength	Rationale for the recommendation
Clinicians should ensure that acute flares of CPP are managed by balancing excluding acute pelvic pathology while also considering other contributing factors and known pain triggers.	**4**	**GPP**	**CPP flares are multifactorial and poorly understood. Patients with known CPP are at risk of bias during acute assessments.**
Clinicians should work with patients to identify flare triggers and develop tailored strategies to manage or mitigate pain flares, as part of a long-term management plan that includes relevant stakeholders such as primary care, emergency departments, and gynaecology services.	**4**	**GPP**	**Evidence suggests flare triggers are heterogeneous and patient-specific. A personalised, collaborative approach may support self-management, reduce acute service use, and improve patient outcomes in this chronic condition with episodic exacerbations.**
Opioids should not be used routinely in the management of CPP flares. If required for acute episodes, a plan for short-term use and dose reduction should be made and communicated with the patient and GP prior to discharge.	**4**	**GPP**	**There is no evidence for long-term opioid benefit in CPP, as stated by NICE NG193. Risks include dependence and tolerance.**

Pain flares are a common feature in women with CPP and contribute to symptom burden and reduced quality of life. The underlying mechanisms driving flare episodes is poorly understood. In the Translational Research in Pelvic Pain (TRiPP) cohort (n=100), 76% of women with CPP reported experiencing symptom flares (228). The majority (68.5%) identified at least one trigger such as stress, menstruation, sexual activity or arousal, and dietary factors. In additional cross-sectional analysis of 769 women within the TRiPP cohort, the most frequently reported exacerbating factors were stress, a full bladder or urination, and exercise (19). Qualitative data similarly describe flares as disruptive to daily functioning and wellbeing (229). *[Evidence level 2++]*

Flares often result in repeat presentations to acute care. The All-Party Parliamentary Group on Endometriosis Inquiry Report (2020) found that 53% of 9965 respondents had attended an emergency department prior to receiving a diagnosis of endometriosis, with 27% attending three or more times (230). Women presenting with acute CPP often have normal investigations (147), although analysis of acute CPP presentations at a Scottish tertiary centre found 35% of women had a positive urine culture, underscoring the importance of not overlooking concurrent pathology such as urinary tract infection, ovarian torsion or pelvic inflammatory disease. Clinicians should work with patients to identify flare triggers and develop personalised flare-management plans across primary care, emergency care, and gynaecology services, an approach supported by evidence that personalised care planning can improve outcomes in in long-term conditions (231). *[Evidence level 4]*

There is no clear evidence supporting the routine use of opioids in the management of CPP, and NICE (NG193) does not recommend opioids for primary chronic pain (177). In acute presentations, opioids may be necessary and should not be withheld if clinically appropriate, but use should be restricted to the lowest effective dose, used for the shortest duration necessary, and accompanied by a clear plan for reduction and cessation, involving both the patient and their general practitioner (232). *[Evidence level 4]*

5. Summary

Chronic pelvic pain (CPP) is a complex, multifactorial condition that significantly impacts quality of life. CPP is best understood through a biopsychosocial framework: contributing factors include gynaecological conditions such as endometriosis and adenomyosis, visceral pain syndromes, altered nervous system function, musculoskeletal dysfunction, and psychological factors like anxiety, depression, and trauma. The diagnosis is clinical and should focus on identification of contributing factors through history, examination, and selective use of investigations including imaging and, where appropriate, diagnostic laparoscopy. Effective management relies on a personalised, multimodal approach. Pharmacological treatments, such as combined hormonal contraceptives and progestogens for women with cyclical pain or non-hormonal options such as NSAIDs and neuromodulators may be considered. Pain education and psychological therapies may empower women and support coping. Physiotherapy has demonstrated clinically meaningful benefit in pain reduction. Care may require input from gynaecology, pain medicine, physiotherapy, psychology, and primary care, and should be individualised in response to individuals' values and goals, recognising these may change across the life course.

6. Recommendations for future research

- Investigate differences in prevalence, presentation, diagnostic pathways, access to care, and outcomes of CPP across population groups, including by ethnicity, socioeconomic status, geography, and other markers of health inequality.
- Understand the aetiology, best treatment and prevention of acute-on-chronic pain flares in patients with CPP.
- Investigate the contribution of the gut microbiome to CPP.
- Develop and validate diagnostic tools to stratify CPP into clinically meaningful subtypes.
- Understand the optimal management of adolescent CPP and the potential of this to reduce long term impact.

- Investigate precision medicine approaches to target treatment according to pain mechanisms and patient phenotype.
- Evaluate the effectiveness of structured pain and self-management education interventions in improving outcomes for women with CPP.
- Assess the role of lifestyle interventions, including diet, physical activity and sleep, in the management of CPP.
- Determine optimal patient reported outcomes in patients with CPP, not only symptom reduction but also quality of life measures.
- Evaluate the societal impact of CPP, including individual impact on career, family life, relationships and social life.

7. Auditable topics

- Availability of a dedicated CPP service within the service
    - A dedicated CPP service or clinic should be available in 100% of tertiary care gynaecology services.
    - A lead clinician for CPP care should be identified in 100% of secondary and tertiary gynaecology services.
- Impact of a dedicated CPP clinic within the service
    - Following implementation of a dedicated CPP service, services should aim to demonstrate a ≥10% reduction in emergency department attendances or unscheduled admissions for CPP flares among service users within 12 months.
- Inclusion of core multidisciplinary team members in CPP care
    - A core MDT (including gynaecology, pain medicine, physiotherapy, psychology, specialist nursing) should be available in ≥80% of tertiary CPP service structures.
    - Access to pelvic physiotherapy should be available in 100% of tertiary CPP service structures
    - Access to pain psychology should be available in 100% of tertiary CPP service structures
- Provision of pain education
    - 100% of women with CPP should have access to pain education Accessibility of a structured pain management programme for CPP patients
    - All women with CPP offered a structured pain management programme, this should be available within 6 months of referral within ≥90% of cases.
- Training of obstetrics and gynaecology speciality trainees
    - Services should aim for 100% of ST3+ trainees to have access to formal teaching or supervised clinical exposure in CPP management during their training.
    - The proportion of obstetrics and gynaecology trainees who report feeling adequately trained in pain science and the management of chronic pelvic pain should be ≥80%.

8. Useful links and support groups

- Pelvic Pain Support Network – https://www.pelvicpain.org.uk/
- Endometriosis UK – https://www.endometriosis-uk.org/
- International Painful Bladder Foundation (IPBF) – https://www.painful-bladder.org/
- Cystitis and Overactive Bladder Foundation (COB) – https://www.cobfoundation.org/

- IBS Network – https://www.theibsnetwork.org/
- Pain Concern – https://painconcern.org.uk/
- Live Well with Pain – https://livewellwithpain.co.uk/
- Sexual Assault Referral Centre (SARC)
    - England: https://www.nhs.uk/live-well/sexual-health/help-after-rape-and-sexual-assault/
    - Scotland: https://www.nhsinform.scot/turn-to-sarcs/
    - Wales: https://executive.nhs.wales/networks/programmes/wsas/access-a-sarc/
    - Northern Ireland: https://www.nidirect.gov.uk/articles/rowan-sexual-assault-referral-centre-sarc

Funding information

All those involved in the development of the Green-top Guidelines, including the Guidelines Committee, Guidelines Committee co-chairs, guideline developers, peer reviewers and other reviewers, are unpaid volunteers and receive no direct funding for their work in producing the guideline. The exception to this are the RCOG staff involved who are salaried employees of the College and Guidelines Committee members who receive reimbursement for expenses for attending Guidelines Committee meetings. Please see more information on travel expense rules on the RCOG website.

Conflict of interest statement

UP and RK have no conflict of interest to disclose. SCM, KV and AWH are listed as co-inventors on a patent application (UK Patent App No. 2217921.2, International Patent App No. PCT/GB2023/053076) relating to the use of the genetic test to determine the efficacy of gabapentin for chronic pelvic pain treatment. AWH is co-inventor of a UK patent for endometriosis markers and methods of diagnosing endometriosis (submitted; TEC1104533 - PG450519GB). AWH's institution has received honoraria for consultancy regarding endometriosis diagnosis and management from Gedeon Richter, Roche Diagnostics, Gesynta and Joii. AWH has received lecture fees (on endometriosis diagnosis and management) from Theramex and Gedeon Richter. AWH's institution has received grant funding from Roche Diagnostics. KV declares research funding from UKRI, NIHR, NIH US, and honoraria for consultancy and talks and associated travel expenses paid to her institution from Gedeon Richter, Gesynta, Reckitts and Eli Lilly.

1598

9. References

1. Latthe P, Latthe M, Say L, Gülmezoglu M, Khan KS. WHO systematic review of prevalence of chronic pelvic pain: a neglected reproductive health morbidity. BMC Public Health. 2006;6:177.
2. Graeve C, Gao G, Stephenson V, Helland R, Jeffery AD. Impact of chronic pelvic pain on quality of life in diverse young adults. Arch Gynecol Obstet. 2024;310(6):3147-56.
3. Mathias SD, Kuppermann M, Liberman RF, Lipschutz RC, Steege JF. Chronic pelvic pain: prevalence, health-related quality of life, and economic correlates. Obstet Gynecol. 1996;87(3):321-7.
4. Ahangari A. Prevalence of chronic pelvic pain among women: an updated review. Pain Physician. 2014;17(2):E141-7.
5. Zondervan KT, Yudkin PL, Vessey MP, Jenkinson CP, Dawes MG, Barlow DH, et al. The community prevalence of chronic pelvic pain in women and associated illness behaviour. Br J Gen Pract. 2001;51(468):541-7.
6. Ayorinde AA, Bhattacharya S, Druce KL, Jones GT, Macfarlane GJ. Chronic pelvic pain in women of reproductive and post-reproductive age: a population-based study. Eur J Pain. 2017;21(3):445-55.
7. Vincent K, Evans E. An update on the management of chronic pelvic pain in women. Anaesthesia. 2021;76 Suppl 4:96-107.
8. Huang G, Le AL, Goddard Y, James D, Thavorn K, Payne M, et al. A Systematic Review of the Cost of Chronic Pelvic Pain in Women. J Obstet Gynaecol Can. 2022;44(3):286-93.e3.
9. The L. ICD-11. Lancet. 2019;393(10188):2275.
10. Leow HW, Szubert W, Horne AW. 45% of UK gynaecologists think chronic pelvic pain is managed badly. Eur J Obstet Gynecol Reprod Biol. 2018;224:200-2.
11. Tewhaiti-Smith J, Semprini A, Bush D, Anderson A, Eathorne A, Johnson N, et al. An Aotearoa New Zealand survey of the impact and diagnostic delay for endometriosis and chronic pelvic pain. Sci Rep. 2022;12(1):4425.
12. As-Sanie S, Mackenzie SC, Morrison L, Schrepf A, Zondervan KT, Horne AW, et al. Endometriosis: A Review. Jama. 2025;334(1):64-78.
13. Terminology: International Association for the Study of Pain; 2025 [cited 2025 14th July]. Available from: https://www.iasp-pain.org/resources/terminology/.
14. Aziz Q, Giamberardino MA, Barke A, Korwisi B, Baranowski AP, Wesselmann U, et al. The IASP classification of chronic pain for ICD-11: chronic secondary visceral pain. Pain. 2019;160(1):69-76.
15. Zondervan KT, Yudkin PL, Vessey MP, Jenkinson CP, Dawes MG, Barlow DH, et al. Chronic pelvic pain in the community--symptoms, investigations, and diagnoses. Am J Obstet Gynecol. 2001;184(6):1149-55.
16. Schrepf A, Phan V, Clemens JQ, Maixner W, Hanauer D, Williams DA. ICD-10 Codes for the Study of Chronic Overlapping Pain Conditions in Administrative Databases. J Pain. 2020;21(1-2):59-70.
17. Lamvu G, Villegas-Echeverri JD, Allaire C, As-Sanie S, Carrillo J, Khalil S, et al. Developing the FIGO-IPPS "R U MOVVING SOMe" classification system for female chronic pelvic pain. Int J Gynaecol Obstet. 2025;171(2):550-65.
18. Yosef A, Allaire C, Williams C, Ahmed AG, Al-Hussaini T, Abdellah MS, et al. Multifactorial contributors to the severity of chronic pelvic pain in women. Am J Obstet Gynecol. 2016;215(6):760.e1-.e14.
19. Demetriou L, Krassowski M, Abreu Mendes P, Garbutt K, Vitonis AF, Wilkins E, et al. Clinical profiling of specific diagnostic subgroups of women with chronic pelvic pain. Front Reprod Health. 2023;5:1140857.
20. Ghiasi M, Kulkarni MT, Missmer SA. Is Endometriosis More Common and More Severe Than It Was 30 Years Ago? J Minim Invasive Gynecol. 2020;27(2):452-61.

21. Vercellini P, Fedele L, Aimi G, Pietropaolo G, Consonni D, Crosignani PG. Association between endometriosis stage, lesion type, patient characteristics and severity of pelvic pain symptoms: a multivariate analysis of over 1000 patients. Hum Reprod. 2007;22(1):266-71.
22. Carey ET, Martin CE, Siedhoff MT, Bair ED, As-Sanie S. Biopsychosocial correlates of persistent postsurgical pain in women with endometriosis. Int J Gynaecol Obstet. 2014;124(2):169-73.
23. Khan KN, Kitajima M, Fujishita A, Hiraki K, Matsumoto A, Nakashima M, et al. Pelvic pain in women with ovarian endometrioma is mostly associated with coexisting peritoneal lesions. Hum Reprod. 2013;28(1):109-18.
24. Chapron C, Santulli P, de Ziegler D, Noel JC, Anaf V, Streuli I, et al. Ovarian endometrioma: severe pelvic pain is associated with deeply infiltrating endometriosis. Hum Reprod. 2012;27(3):702-11.
25. Coxon L, Demetriou L, Vincent K. Current developments in endometriosis-associated pain. Cell Rep Med. 2024;5(10):101769.
26. Struble J, Reid S, Bedaiwy MA. Adenomyosis: A Clinical Review of a Challenging Gynecologic Condition. J Minim Invasive Gynecol. 2016;23(2):164-85.
27. Rahmioglu N, Mortlock S, Ghiasi M, Møller PL, Stefansdottir L, Galarneau G, et al. The genetic basis of endometriosis and comorbidity with other pain and inflammatory conditions. Nat Genet. 2023;55(3):423-36.
28. Upson K, Missmer SA. Epidemiology of Adenomyosis. Semin Reprod Med. 2020;38(2-03):89-107.
29. Dason ES, Maxim M, Sanders A, Papillon-Smith J, Ng D, Chan C, et al. Guideline No. 437: Diagnosis and Management of Adenomyosis. J Obstet Gynaecol Can. 2023;45(6):417-29.e1.
30. Naftalin J, Bean E, Saridogan E, Barton-Smith P, Arora R, Jurkovic D. Imaging in gynecological disease (21): clinical and ultrasound characteristics of accessory cavitated uterine malformations. Ultrasound Obstet Gynecol. 2021;57(5):821-8.
31. Pinzauti S, Lazzeri L, Tosti C, Centini G, Orlandini C, Luisi S, et al. Transvaginal sonographic features of diffuse adenomyosis in 18-30-year-old nulligravid women without endometriosis: association with symptoms. Ultrasound Obstet Gynecol. 2015;46(6):730-6.
32. Vannuccini S, Meleca C, Toscano F, Mertino P, Pampaloni F, Fambrini M, et al. Adenomyosis diagnosis among adolescents and young women with dysmenorrhoea and heavy menstrual bleeding. Reprod Biomed Online. 2024;48(5):103768.
33. Kresch AJ, Seifer DB, Sachs LB, Barrese I. Laparoscopy in 100 women with chronic pelvic pain. Obstet Gynecol. 1984;64(5):672-4.
34. Rapkin AJ. Adhesions and pelvic pain: a retrospective study. Obstet Gynecol. 1986;68(1):13-5.
35. Howard FM, El-Minawi AM, Sanchez RA. Conscious pain mapping by laparoscopy in women with chronic pelvic pain. Obstet Gynecol. 2000;96(6):934-9.
36. Cheong Y, Saran M, Hounslow JW, Reading IC. Are pelvic adhesions associated with pain, physical, emotional and functional characteristics of women presenting with chronic pelvic pain? A cluster analysis. BMC Womens Health. 2018;18(1):11.
37. van den Beukel BA, de Ree R, van Leuven S, Bakkum EA, Strik C, van Goor H, et al. Surgical treatment of adhesion-related chronic abdominal and pelvic pain after gynaecological and general surgery: a systematic review and meta-analysis. Hum Reprod Update. 2017;23(3):276-88.
38. Swank DJ, Swank-Bordewijk SC, Hop WC, van Erp WF, Janssen IM, Bonjer HJ, et al. Laparoscopic adhesiolysis in patients with chronic abdominal pain: a blinded randomised controlled multi-centre trial. Lancet. 2003;361(9365):1247-51.
39. Molegraaf MJ, Torensma B, Lange CP, Lange JF, Jeekel J, Swank DJ. Twelve-year outcomes of laparoscopic adhesiolysis in patients with chronic abdominal pain: A randomized clinical trial. Surgery. 2017;161(2):415-21.
40. Cheong YC, Reading I, Bailey S, Sadek K, Ledger W, Li TC. Should women with chronic pelvic pain have adhesiolysis? BMC Womens Health. 2014;14(1):36.

41. Peters AA, Trimbos-Kemper GC, Admiraal C, Trimbos JB, Hermans J. A randomized clinical trial on the benefit of adhesiolysis in patients with intraperitoneal adhesions and chronic pelvic pain. Br J Obstet Gynaecol. 1992;99(1):59-62.
42. Hardy I, Rousseau S. Captive uterus syndrome: An unrecognized complication of cesarean sections? Med Hypotheses. 2019;122:98-102.
43. Champaneria R, Shah L, Moss J, Gupta JK, Birch J, Middleton LJ, et al. The relationship between pelvic vein incompetence and chronic pelvic pain in women: systematic reviews of diagnosis and treatment effectiveness. Health Technol Assess. 2016;20(5):1-108.
44. Loos MJ, Scheltinga MR, Mulders LG, Roumen RM. The Pfannenstiel incision as a source of chronic pain. Obstet Gynecol. 2008;111(4):839-46.
45. Possover M, Schneider T, Henle KP. Laparoscopic therapy for endometriosis and vascular entrapment of sacral plexus. Fertil Steril. 2011;95(2):756-8.
46. Dietrich JE, Millar DM, Quint EH. Obstructive reproductive tract anomalies. J Pediatr Adolesc Gynecol. 2014;27(6):396-402.
47. Breech LL, Laufer MR. Müllerian anomalies. Obstet Gynecol Clin North Am. 2009;36(1):47-68.
48. Setty T, Naftalin J, Jurkovic D. Accessory cavitated uterine malformations (ACUMs): an unfamiliar cause of dysmenorrhoea. The Obstetrician & Gynaecologist. 2022;24(1):40-9.
49. Timmerman S, Stubbe L, Van den Bosch T, Van Schoubroeck D, Tellum T, Froyman W. Accessory cavitated uterine malformation (ACUM): A scoping review. Acta Obstet Gynecol Scand. 2024;103(6):1036-45.
50. Lowe S, Vincent S, Sumida M, Dietrich J. 9. Abdominopelvic Pain and Coexisting Pain Disorders in Patients with Mayer-Rokitansky-Kuster-Hauser Syndrome. Journal of Pediatric and Adolescent Gynecology. 2025;38(2):233-4.
51. Marsh CA, Will MA, Smorgick N, Quint EH, Hussain H, Smith YR. Uterine remnants and pelvic pain in females with Mayer-Rokitansky-Küster-Hauser syndrome. J Pediatr Adolesc Gynecol. 2013;26(3):199-202.
52. Gaikaiwari RU, Prinsloo C, Grover SR, Wright I, Drever N. Experiences of Pelvic and Generalized Persistent Pain Syndromes in MRKH: A Scoping Review. J Pediatr Adolesc Gynecol. 2024;37(5):477-94.
53. McEvoy M, McElvaney R, Glover R. Understanding vaginismus: a biopsychosocial perspective. Sexual and Relationship Therapy. 2024;39(3):680-701.
54. Bornstein J, Goldstein AT, Stockdale CK, Bergeron S, Pukall C, Zolnoun D, et al. 2015 ISSVD, ISSWSH, and IPPS Consensus Terminology and Classification of Persistent Vulvar Pain and Vulvodynia. J Sex Med. 2016;13(4):607-12.
55. Reed BD, Harlow SD, Sen A, Legocki LJ, Edwards RM, Arato N, et al. Prevalence and demographic characteristics of vulvodynia in a population-based sample. Am J Obstet Gynecol. 2012;206(2):170.e1-9.
56. Edwards SK, Lewis F, Fernando I, Haddon L, Grover D. 2024 British Association for Sexual Health and HIV (BASHH) UK national guideline on the management of vulval conditions. Int J STD AIDS. 2025;36(5):346-71.
57. Williams RE, Hartmann KE, Sandler RS, Miller WC, Steege JF. Prevalence and characteristics of irritable bowel syndrome among women with chronic pelvic pain. Obstet Gynecol. 2004;104(3):452-8.
58. Tirlapur SA, Kuhrt K, Chaliha C, Ball E, Meads C, Khan KS. The 'evil twin syndrome' in chronic pelvic pain: a systematic review of prevalence studies of bladder pain syndrome and endometriosis. Int J Surg. 2013;11(3):233-7.
59. Leuenberger J, Kohl Schwartz AS, Geraedts K, Haeberlin F, Eberhard M, von Orellie S, et al. Living with endometriosis: Comorbid pain disorders, characteristics of pain and relevance for daily life. Eur J Pain. 2022;26(5):1021-38.
60. Giamberardino MA, Costantini R, Affaitati G, Fabrizio A, Lapenna D, Tafuri E, et al. Viscero-visceral hyperalgesia: characterization in different clinical models. Pain. 2010;151(2):307-22.

61. Nijs J, George SZ, Clauw DJ, Fernández-de-Las-Peñas C, Kosek E, Ickmans K, et al. Central sensitisation in chronic pain conditions: latest discoveries and their potential for precision medicine. Lancet Rheumatol. 2021;3(5):e383-e92.
62. Costantini R, Affaitati G, Wesselmann U, Czakanski P, Giamberardino MA. Visceral pain as a triggering factor for fibromyalgia symptoms in comorbid patients. Pain. 2017;158(10):1925-37.
63. Schmulson MJ, Drossman DA. What Is New in Rome IV. J Neurogastroenterol Motil. 2017;23(2):151-63.
64. Foundation R. Rome IV Criteria 2026 [cited 2026 20th Jan]. Available from: https://theromefoundation.org/rome-iv/rome-iv-criteria/.
65. van de Merwe JP, Nordling J, Bouchelouche P, Bouchelouche K, Cervigni M, Daha LK, et al. Diagnostic criteria, classification, and nomenclature for painful bladder syndrome/interstitial cystitis: an ESSIC proposal. Eur Urol. 2008;53(1):60-7.
66. Krieger JN, Stephens AJ, Landis JR, Clemens JQ, Kreder K, Lai HH, et al. Relationship between chronic nonurological associated somatic syndromes and symptom severity in urological chronic pelvic pain syndromes: baseline evaluation of the MAPP study. J Urol. 2015;193(4):1254-62.
67. Lai HH, Jemielita T, Sutcliffe S, Bradley CS, Naliboff B, Williams DA, et al. Characterization of Whole Body Pain in Urological Chronic Pelvic Pain Syndrome at Baseline: A MAPP Research Network Study. J Urol. 2017;198(3):622-31.
68. Schrepf AD, Mawla I, Naliboff BD, Gallop B, Moldwin RM, Tu F, et al. Neurobiology and long-term impact of bladder-filling pain in humans: a Multidisciplinary Approach to the Study of Chronic Pelvic Pain (MAPP) research network study. Pain. 2023;164(10):2343-51.
69. Clemens JQ, Mullins C, Ackerman AL, Bavendam T, van Bokhoven A, Ellingson BM, et al. Urologic chronic pelvic pain syndrome: insights from the MAPP Research Network. Nat Rev Urol. 2019;16(3):187-200.
70. Wolff DT, Tranchina S, Schrepf A, Christopher Doiron R, Chai TC, Walker SJ. Interstitial Cystitis/Bladder Pain Syndrome Patient Phenotyping. Neurourol Urodyn. 2025.
71. Iacovides S, Avidon I, Baker FC. What we know about primary dysmenorrhea today: a critical review. Hum Reprod Update. 2015;21(6):762-78.
72. Armour M, Parry K, Manohar N, Holmes K, Ferfolja T, Curry C, et al. The Prevalence and Academic Impact of Dysmenorrhea in 21,573 Young Women: A Systematic Review and Meta-Analysis. J Womens Health (Larchmt). 2019;28(8):1161-71.
73. Latthe PM, Champaneria R. Dysmenorrhoea. BMJ Clin Evid. 2014;2014.
74. Jarrell J, Arendt-Nielsen L. Allodynia and Dysmenorrhea. J Obstet Gynaecol Can. 2016;38(3):270-4.
75. Hardi G, Evans S, Craigie M. A possible link between dysmenorrhoea and the development of chronic pelvic pain. Aust N Z J Obstet Gynaecol. 2014;54(6):593-6.
76. Reid-McCann R, Poli-Neto OB, Stein K, Dixon S, Cox E, Coxon L, et al. Longitudinal association between dysmenorrhoea in adolescence and chronic pain in adulthood: a UK population-based study. Lancet Child Adolesc Health. 2025;9(11):766-75.
77. Li R, Li B, Kreher DA, Benjamin AR, Gubbels A, Smith SM. Association between dysmenorrhea and chronic pain: a systematic review and meta-analysis of population-based studies. Am J Obstet Gynecol. 2020;223(3):350-71.
78. Li R, Kreher DA, Jusko TA, Chapman BP, Bonham AD, Seplaki CL. Prospective Association between Dysmenorrhea and Chronic Pain Development in Community-Dwelling Women. J Pain. 2021;22(9):1084-96.
79. Vincent K, Warnaby C, Stagg CJ, Moore J, Kennedy S, Tracey I. Dysmenorrhoea is associated with central changes in otherwise healthy women. Pain. 2011;152(9):1966-75.
80. de Arruda GT, Driusso P, Rodrigues JC, de Godoy AG, Degani A, Danna-Dos-Santos A, et al. Are menstrual symptoms associated with central sensitization inventory? A cross-sectional study. Eur J Pain. 2022;26(8):1759-67.

81. Coxon L, Wiech K, Vincent K. Is There a Neuropathic-Like Component to Endometriosis-Associated Pain? Results From a Large Cohort Questionnaire Study. Front Pain Res (Lausanne). 2021;2:743812.
82. Fitzcharles MA, Cohen SP, Clauw DJ, Littlejohn G, Usui C, Häuser W. Nociplastic pain: towards an understanding of prevalent pain conditions. Lancet. 2021;397(10289):2098-110.
83. Schrepf A, Gallop R, Naliboff B, Harte SE, Afari N, Lai HH, et al. Clinical Phenotyping for Pain Mechanisms in Urologic Chronic Pelvic Pain Syndromes: A MAPP Research Network Study. J Pain. 2022;23(9):1594-603.
84. Till SR, Schrepf A, Clauw DJ, Harte SE, Williams DA, As-Sanie S. Association Between Nociplastic Pain and Pain Severity and Impact in Women With Chronic Pelvic Pain. J Pain. 2023;24(8):1406-14.
85. Coxon L, Vollert J, Perro D, Lunde CE, Ferreira-Gomes J, Charrua A, et al. Comprehensive quantitative sensory testing shows altered sensory function in women with chronic pelvic pain: results from the Translational Research in Pelvic Pain (TRiPP) Study. Pain. 2023;164(11):2528-39.
86. Beales D, Asinelli R, Klokset M, O'Kane L, Urstad T, Wise E, et al. Association between pelvic pain bothersomeness and pain sensitivity: A community-based cross-sectional study of young adult females in the Raine Study. Bjog. 2022;129(12):1981-91.
87. Quaghebeur J, Wyndaele JJ, De Wachter S. Pain areas and mechanosensitivity in patients with chronic pelvic pain syndrome: a controlled clinical investigation. Scand J Urol. 2017;51(5):414-9.
88. Fuentes-Márquez P, Valenza MC, Cabrera-Martos I, Ríos-Sánchez A, Ocón-Hernández O. Trigger Points, Pressure Pain Hyperalgesia, and Mechanosensitivity of Neural Tissue in Women with Chronic Pelvic Pain. Pain Med. 2019;20(1):5-13.
89. Kaya S, Hermans L, Willems T, Roussel N, Meeus M. Central sensitization in urogynecological chronic pelvic pain: a systematic literature review. Pain Physician. 2013;16(4):291-308.
90. Brawn J, Morotti M, Zondervan KT, Becker CM, Vincent K. Central changes associated with chronic pelvic pain and endometriosis. Hum Reprod Update. 2014;20(5):737-47.
91. Orr NL, Wahl KJ, Lisonek M, Joannou A, Noga H, Albert A, et al. Central sensitization inventory in endometriosis. Pain. 2022;163(2):e234-e45.
92. Ryan A, Healey M, Cheng C, Dior U, Reddington C. Central sensitisation in pelvic pain: A cohort study. Aust N Z J Obstet Gynaecol. 2022;62(6):868-74.
93. Chelimsky G, Simpson P, McCabe N, Zhang L, Chelimsky T. Autonomic Testing in Women with Chronic Pelvic Pain. J Urol. 2016;196(2):429-34.
94. Chelimsky GG, Yang S, Sanses T, Tatsuoka C, Buffington CAT, Janata J, et al. Autonomic neurophysiologic implications of disorders comorbid with bladder pain syndrome vs myofascial pelvic pain. Neurourol Urodyn. 2019;38(5):1370-7.
95. Foster DC, Dworkin RH, Wood RW. Effects of intradermal foot and forearm capsaicin injections in normal and vulvodynia-afflicted women. Pain. 2005;117(1-2):128-36.
96. Hao M, Liu X, Rong P, Li S, Guo SW. Reduced vagal tone in women with endometriosis and auricular vagus nerve stimulation as a potential therapeutic approach. Sci Rep. 2021;11(1):1345.
97. Kornum DS, Terkelsen AJ, Bertoli D, Klinge MW, Høyer KL, Kufaishi HHA, et al. Assessment of Gastrointestinal Autonomic Dysfunction: Present and Future Perspectives. J Clin Med. 2021;10(7).
98. Gyang A, Hartman M, Lamvu G. Musculoskeletal causes of chronic pelvic pain: what a gynecologist should know. Obstet Gynecol. 2013;121(3):645-50.
99. Abu-Alnadi N, Frame B, Moore KJ, Carey ET. Myofascial Pain in Hysterectomy Patients. J Minim Invasive Gynecol. 2021;28(12):2067-72.
100. Mieritz RM, Thorhauge K, Forman A, Mieritz HB, Hartvigsen J, Christensen HW. Musculoskeletal Dysfunctions in Patients With Chronic Pelvic Pain: A Preliminary Descriptive Survey. J Manipulative Physiol Ther. 2016;39(9):616-22.
101. Fitzgerald CM, Neville CE, Mallinson T, Badillo SA, Hynes CK, Tu FF. Pelvic floor muscle examination in female chronic pelvic pain. J Reprod Med. 2011;56(3-4):117-22.

102. Loving S, Thomsen T, Jaszczak P, Nordling J. Pelvic floor muscle dysfunctions are prevalent in female chronic pelvic pain: a cross-sectional population-based study. Eur J Pain. 2014;18(9):1259-70.
103. Proulx L, Brizzolara K, Thompson M, Wang-Price S, Rodriguez P, Koppenhaver S. Women with Chronic Pelvic Pain Demonstrate Increased Lumbopelvic Muscle Stiffness Compared to Asymptomatic Controls. J Womens Health (Larchmt). 2023;32(2):239-47.
104. Stratton P, Khachikyan I, Sinaii N, Ortiz R, Shah J. Association of chronic pelvic pain and endometriosis with signs of sensitization and myofascial pain. Obstet Gynecol. 2015;125(3):719-28.
105. Prendergast SA, Weiss JM. Screening for musculoskeletal causes of pelvic pain. Clin Obstet Gynecol. 2003;46(4):773-82.
106. Sanses TV, Chelimsky G, McCabe NP, Zolnoun D, Janata J, Elston R, et al. The Pelvis and Beyond: Musculoskeletal Tender Points in Women With Chronic Pelvic Pain. Clin J Pain. 2016;32(8):659-65.
107. Gupta P, Gallop R, Spitznagle T, Lai H, Tu F, Krieger JN, et al. Is Pelvic Floor Muscle Tenderness a Distinct Urologic Chronic Pelvic Pain Syndrome Phenotype? Findings from the Multidisciplinary Approach to the Study of Chronic Pelvic Pain Research Network Symptom Pattern Study. J Urol. 2022;208(2):341-9.
108. Till SR, As-Sanie S, Schrepf A. Psychology of Chronic Pelvic Pain: Prevalence, Neurobiological Vulnerabilities, and Treatment. Clin Obstet Gynecol. 2019;62(1):22-36.
109. Yao C, Zhang Y, Lu P, Xiao B, Sun P, Tao J, et al. Exploring the bidirectional relationship between pain and mental disorders: a comprehensive Mendelian randomization study. J Headache Pain. 2023;24(1):82.
110. Bryant C, Cockburn R, Plante AF, Chia A. The psychological profile of women presenting to a multidisciplinary clinic for chronic pelvic pain: high levels of psychological dysfunction and implications for practice. J Pain Res. 2016;9:1049-56.
111. Quartana PJ, Campbell CM, Edwards RR. Pain catastrophizing: a critical review. Expert Rev Neurother. 2009;9(5):745-58.
112. Martin CE, Johnson E, Wechter ME, Leserman J, Zolnoun DA. Catastrophizing: a predictor of persistent pain among women with endometriosis at 1 year. Hum Reprod. 2011;26(11):3078-84.
113. McPeak AE, Allaire C, Williams C, Albert A, Lisonkova S, Yong PJ. Pain Catastrophizing and Pain Health-Related Quality-of-Life in Endometriosis. Clin J Pain. 2018;34(4):349-56.
114. Bourdon M, Antoine V, Combes U, Maitrot-Mantelet L, Marcellin L, Maignien C, et al. Severe pelvic pain is associated with sexual abuse experienced during childhood and/or adolescence irrespective of the presence of endometriosis. Hum Reprod. 2023;38(8):1499-508.
115. As-Sanie S, Clevenger LA, Geisser ME, Williams DA, Roth RS. History of abuse and its relationship to pain experience and depression in women with chronic pelvic pain. Am J Obstet Gynecol. 2014;210(4):317.e1-.e8.
116. Krantz TE, Andrews N, Petersen TR, Dunivan GC, Montoya M, Swanson N, et al. Adverse Childhood Experiences Among Gynecology Patients With Chronic Pelvic Pain. Obstet Gynecol. 2019;134(5):1087-95.
117. Lamvu G, Carrillo J, Ouyang C, Rapkin A. Chronic Pelvic Pain in Women: A Review. Jama. 2021;325(23):2381-91.
118. Adler H, Lewis M, Ng CHM, Brooks C, Leonardi M, Mikocka-Walus A, et al. Social Media, Endometriosis, and Evidence-Based Information: An Analysis of Instagram Content. Healthcare (Basel). 2024;12(1).
119. Shiplo SN, Gholiof M, Sarin N, Leonardi M. Endometriosis Influencers on Instagram: Who are They and What are They Posting? J Minim Invasive Gynecol. 2025.
120. Rosenberger DC, Pogatzki-Zahn EM. Chronic post-surgical pain - update on incidence, risk factors and preventive treatment options. BJA Educ. 2022;22(5):190-6.
121. Sousa Shimamura LK, Bettiol H, Moura da Silva AA, Nogueira AA, Barbieri MA, Rosa ESJC, et al. Incidence of chronic pelvic pain after childbirth and its causal association with C-section. Pain. 2025.

122. Kainu JP, Sarvela J, Tiippana E, Halmesmäki E, Korttila KT. Persistent pain after caesarean section and vaginal birth: a cohort study. Int J Obstet Anesth. 2010;19(1):4-9.
123. Li WY, Liabsuetrakul T, Stray-Pedersen B, Li YJ, Guo LJ, Qin WZ. The effects of mode of delivery and time since birth on chronic pelvic pain and health-related quality of life. Int J Gynaecol Obstet. 2014;124(2):139-42.
124. Helden E, Reijnders-Boerboom G, Ki A-W, Langenhuijsen H, d'Ancona F, Keijzer C, et al. Chronic postsurgical pain after laparoscopic surgery: a systematic review2024.
125. Transvaginal mesh repair of anterior or posterior vaginal wall prolapse: Interventional procedures guidance. : National Institute of Health and Care Excellence; 2017 [Available from: https://www.nice.org.uk/guidance/ipg599/chapter/5-Safety.
126. Glazener CM, Breeman S, Elders A, Hemming C, Cooper KG, Freeman RM, et al. Mesh, graft, or standard repair for women having primary transvaginal anterior or posterior compartment prolapse surgery: two parallel-group, multicentre, randomised, controlled trials (PROSPECT). Lancet. 2017;389(10067):381-92.
127. Arjona Berral JE, Rodríguez Jiménez B, Velasco Sánchez E, Povedano Cañizares B, Monserrat Jordan J, Lorente Gonzalez J, et al. Essure® and chronic pelvic pain: a population-based cohort. J Obstet Gynaecol. 2014;34(8):712-3.
128. Yunker AC, Ritch JM, Robinson EF, Golish CT. Incidence and risk factors for chronic pelvic pain after hysteroscopic sterilization. J Minim Invasive Gynecol. 2015;22(3):390-4.
129. Ross WT, Snyder B, Stuckey H, Ross IR, McCall-Hosenfeld J, Harkins GJ, et al. Gynaecological care of women with chronic pelvic pain: Patient perspectives and care preferences. Bjog. 2023;130(5):476-84.
130. Price J, Farmer G, Harris J, Hope T, Kennedy S, Mayou R. Attitudes of women with chronic pelvic pain to the gynaecological consultation: a qualitative study. Bjog. 2006;113(4):446-52.
131. Wygant JN, McGuire LJ, Bush NM, Burnett TL, Green IC, Breitkopf DM. What makes a chronic pelvic pain patient satisfied? J Psychosom Obstet Gynaecol. 2019;40(3):239-42.
132. Working definition of trauma-informed practice: GOV.UK; 2022 [Available from: https://www.gov.uk/government/publications/working-definition-of-trauma-informed-practice/working-definition-of-trauma-informed-practice.
133. O'Leary MP, Sant GR, Fowler FJ, Jr., Whitmore KE, Spolarich-Kroll J. The interstitial cystitis symptom index and problem index. Urology. 1997;49(5A Suppl):58-63.
134. Edgley K, Horne AW, Saunders PTK, Tsanas A. Symptom tracking in endometriosis using digital technologies: Knowns, unknowns, and future prospects. Cell Rep Med. 2023;4(9):101192.
135. Becker CM, Bokor A, Heikinheimo O, Horne A, Jansen F, Kiesel L, et al. ESHRE guideline: endometriosis. Hum Reprod Open. 2022;2022(2):hoac009.
136. Quaghebeur J, Wyndaele JJ. Comparison of questionnaires used for the evaluation of patients with chronic pelvic pain. Neurourol Urodyn. 2013;32(8):1074-9.
137. Droz J, Howard FM. Use of the Short-Form McGill Pain Questionnaire as a diagnostic tool in women with chronic pelvic pain. J Minim Invasive Gynecol. 2011;18(2):211-7.
138. Lin T, Allaire C, As-Sanie S, Stratton P, Vincent K, Adamson GD, et al. World Endometriosis Research Foundation Endometriosis Phenome and Biobanking Harmonization Project: V. Physical examination standards in endometriosis research. Fertil Steril. 2024;122(2):304-15.
139. Thompson HD, Tang S, Jarrell JF. Temporal Summation in Chronic Pelvic Pain. J Obstet Gynaecol Can. 2020;42(5):556-60.
140. Kielstra SC, Reezigt RR, Coppieters MW, de Vries R, Arendt-Nielsen L, Petersen KK, et al. A myriad of methods to determine temporal summation of pain in people with musculoskeletal pain and healthy participants: a scoping review. Pain Rep. 2024;9(5):e1176.
141. Iraola E, Menard JP, Chariot P. Experience of Pelvic Examination and Uptake of Gynecological Care Following Domestic or Sexual Violence: a Systematic Review. Trauma Violence Abuse. 2024;25(5):4030-44.

142. Gynaecologists. L-BNaCSobotRCoOa. Female Genital Mutilationand its Management. Green-top Guideline No. 53. July 2015.
143. Tellum T, Nygaard S, Lieng M. Noninvasive Diagnosis of Adenomyosis: A Structured Review and Meta-analysis of Diagnostic Accuracy in Imaging. J Minim Invasive Gynecol. 2020;27(2):408-18.e3.
144. Nisenblat V, Bossuyt PM, Farquhar C, Johnson N, Hull ML. Imaging modalities for the non-invasive diagnosis of endometriosis. Cochrane Database Syst Rev. 2016;2(2):Cd009591.
145. Young SW, Jha P, Chamié L, Rodgers S, Kho RM, Horrow MM, et al. Society of Radiologists in Ultrasound Consensus on Routine Pelvic US for Endometriosis. Radiology. 2024;311(1):e232191.
146. Singh SS, Allaire C, Al-Nourhji O, Bougie O, Bridge-Cook P, Duigenan S, et al. Guideline No. 449: Diagnosis and Impact of Endometriosis - A Canadian Guideline. J Obstet Gynaecol Can. 2024;46(5):102450.
147. Endometriosis: diagnosis and management (NG73): National Institute for Heath and Care Excellence; 2024 [Available from: https://www.nice.org.uk/guidance/ng73.
148. Leonardi M, Uzuner C, Mestdagh W, Lu C, Guerriero S, Zajicek M, et al. Diagnostic accuracy of transvaginal ultrasound for detection of endometriosis using International Deep Endometriosis Analysis (IDEA) approach: prospective international pilot study. Ultrasound Obstet Gynecol. 2022;60(3):404-13.
149. Gerner-Rasmussen J, Donatsky AM, Bjerrum F. The role of non-invasive imaging techniques in detecting intra-abdominal adhesions: a systematic review. Langenbecks Arch Surg. 2019;404(6):653-61.
150. Ahmed TM, Coco A, Vaught AJ, Gomez EN. MR imaging for preoperative characterization of pelvic adhesions: role in diagnosis and surgical planning. Abdom Radiol (NY). 2025;50(2):966-78.
151. Llarena NC, Shah AB, Milad MP. Bowel injury in gynecologic laparoscopy: a systematic review. Obstet Gynecol. 2015;125(6):1407-17.
152. NHS Diagnostic laparoscopy consent form: NHS England; 2022 [available via the Getting It Right First Time (GIRFT) workspace on the FutureNHS platform]. Available from: https://future.nhs.uk/.
153. Daniels J, Gray R, Hills RK, Latthe P, Buckley L, Gupta J, et al. Laparoscopic uterosacral nerve ablation for alleviating chronic pelvic pain: a randomized controlled trial. Jama. 2009;302(9):955-61.
154. Howard FM. The role of laparoscopy in chronic pelvic pain: promise and pitfalls. Obstet Gynecol Surv. 1993;48(6):357-87.
155. Tempest N, Efstathiou E, Petros Z, Hapangama DK. Laparoscopic Outcomes after Normal Clinical and Ultrasound Findings in Young Women with Chronic Pelvic Pain: A Cross-Sectional Study. J Clin Med. 2020;9(8).
156. Hebbar S, Chawla C. Role of laparoscopy in evaluation of chronic pelvic pain. J Minim Access Surg. 2005;1(3):116-20.
157. Newham AP, van der Spuy ZM, Nugent F. Laparoscopic findings in women with chronic pelvic pain. S Afr Med J. 1996;86(9 Suppl):1200-3.
158. Sharma D, Dahiya K, Duhan N, Bansal R. Diagnostic laparoscopy in chronic pelvic pain. Arch Gynecol Obstet. 2011;283(2):295-7.
159. Brichant G, Denef M, Tebache L, Poismans G, Pinzauti S, Dechenne V, et al. Chronic pelvic pain and the role of exploratory laparoscopy as diagnostic and therapeutic tool: a retrospective observational study. Gynecological Surgery. 2018;15(1):13.
160. Kuan KK, Neilson AR, Horne AW, Whitaker LH. Comparing inpatient management of chronic pelvic pain flares before and after the COVID-19 pandemic. Reprod Fertil. 2023;4(2).
161. Gibbons T, Rahmioglu N, Zondervan KT, Becker CM. Crimson clues: advancing endometriosis detection and management with novel blood biomarkers. Fertil Steril. 2024;121(2):145-63.
162. Werneburg GT, Moldwin R, Lowell Parsons C, Shivam Priyadarshi M, Sinha S, Quentin Clemens J. Interstitial Cystitis/Bladder Pain Syndrome (IC/BPS) Diagnosis: Current Limitations and a Pragmatic Clinical Diagnostic Definition. Neurourol Urodyn. 2025.

163. Allaire C, Williams C, Bodmer-Roy S, Zhu S, Arion K, Ambacher K, et al. Chronic pelvic pain in an interdisciplinary setting: 1-year prospective cohort. Am J Obstet Gynecol. 2018;218(1):114.e1-.e12.
164. Miller-Matero LR, Saulino C, Clark S, Bugenski M, Eshelman A, Eisenstein D. When treating the pain is not enough: a multidisciplinary approach for chronic pelvic pain. Arch Womens Ment Health. 2016;19(2):349-54.
165. Twiddy H, Lane N, Chawla R, Johnson S, Bradshaw A, Aleem S, et al. The development and delivery of a female chronic pelvic pain management programme: a specialised interdisciplinary approach. Br J Pain. 2015;9(4):233-40.
166. Mardon AK, Leake HB, Szeto K, Moseley GL, Chalmers KJ. Recommendations for patient education in the management of persistent pelvic pain: a systematic review of clinical practice guidelines. Pain. 2024;165(6):1207-16.
167. Mardon AK, Chalmers KJ, Heathcote LC, Curtis LA, Freedman L, Malani R, et al. "I wish I knew then what I know now" - pain science education concepts important for female persistent pelvic pain: a reflexive thematic analysis. Pain. 2024;165(9):1990-2001.
168. Mardon AK, Leake HB, Wilson MV, Karran EL, Parker R, Malani R, et al. Pain science education concepts for pelvic pain: an e-Delphi of expert clinicians. Front Pain Res (Lausanne). 2025;6:1498996.
169. Cheong YC, Smotra G, Williams A. Non-surgical interventions for the management of chronic pelvic pain. Cochrane Database of Systematic Reviews. 2014(3).
170. Priya K, Rajaram S, Goel N. Comparison of combined hormonal vaginal ring and low dose combined oral hormonal pill for the treatment of idiopathic chronic pelvic pain: a randomised trial. Eur J Obstet Gynecol Reprod Biol. 2016;207:141-6.
171. Etrusco A, Agrifoglio V, D'Amato A, Chiantera V, Laganà AS, Haydamous J, et al. Efficacy and safety of hormone therapies for treating adenomyosis-associated pelvic pain: a systematic review and network meta-analysis of randomized controlled trials. Front Endocrinol (Lausanne). 2025;16:1571727.
172. Till SR, Nakamura R, Schrepf A, As-Sanie S. Approach to Diagnosis and Management of Chronic Pelvic Pain in Women: Incorporating Chronic Overlapping Pain Conditions in Assessment and Management. Obstet Gynecol Clin North Am. 2022;49(2):219-39.
173. UK Medical Eligibility Criteria for Contraceptive Use (UKMEC) April 2016 (Amended September 2019): Faculty of Sexual and Reproductive Healthcare; 2019 [
174. Society BM. Migraine and HRT 2022 [cited 2026 20th Jan]. Available from: https://thebms.org.uk/wp-content/uploads/2022/12/06-BMS-TfC-Migraine-and-HRT-NOV2022-A.pdf?utm.
175. Lethaby A, Ayeleke RO, Roberts H. Local oestrogen for vaginal atrophy in postmenopausal women. Cochrane Database Syst Rev. 2016;2016(8):Cd001500.
176. Marjoribanks J, Ayeleke RO, Farquhar C, Proctor M. Nonsteroidal anti-inflammatory drugs for dysmenorrhoea. Cochrane Database Syst Rev. 2015;2015(7):Cd001751.
177. Chronic pain (primary and secondary) in over 16s: assessment of all chronic pain and management of chronic primary pain (NG193): National Institute of Health and Care Excellence; 2021 [Available from: https://www.nice.org.uk/guidance/ng193.
178. Horne AW, Vincent K, Hewitt CA, Middleton LJ, Koscielniak M, Szubert W, et al. Gabapentin for chronic pelvic pain in women (GaPP2): a multicentre, randomised, double-blind, placebo-controlled trial. Lancet. 2020;396(10255):909-17.
179. Neuropathic pain in adults: pharmacological management in non-specialist settings (CG173) National Institute of Health and Care Excellence2020 [Available from: https://www.nice.org.uk/guidance/cg173.
180. Soliman N, Moisset X, Ferraro MC, de Andrade DC, Baron R, Belton J, et al. Pharmacotherapy and non-invasive neuromodulation for neuropathic pain: a systematic review and meta-analysis. Lancet Neurol. 2025;24(5):413-28.

181. Zolnoun DA, Hartmann KE, Steege JF. Overnight 5% lidocaine ointment for treatment of vulvar vestibulitis. Obstet Gynecol. 2003;102(1):84-7.
182. Foster DC, Kotok MB, Huang LS, Watts A, Oakes D, Howard FM, et al. Oral desipramine and topical lidocaine for vulvodynia: a randomized controlled trial. Obstet Gynecol. 2010;116(3):583-93.
183. Jhang JF, Yu WR, Jiang YH, Kuo HC. Pathophysiology and potential multimodal therapeutic strategies for IC/BPS. Nat Rev Urol. 2025.
184. Wouters MM, Balemans D, Van Wanrooy S, Dooley J, Cibert-Goton V, Alpizar YA, et al. Histamine Receptor H1-Mediated Sensitization of TRPV1 Mediates Visceral Hypersensitivity and Symptoms in Patients With Irritable Bowel Syndrome. Gastroenterology. 2016;150(4):875-87.e9.
185. Grundy L, Caldwell A, Garcia Caraballo S, Erickson A, Schober G, Castro J, et al. Histamine induces peripheral and central hypersensitivity to bladder distension via the histamine H(1) receptor and TRPV1. Am J Physiol Renal Physiol. 2020;318(2):F298-f314.
186. Champaneria R, Daniels JP, Raza A, Pattison HM, Khan KS. Psychological therapies for chronic pelvic pain: systematic review of randomized controlled trials. Acta Obstet Gynecol Scand. 2012;91(3):281-6.
187. Johnson S, Bradshaw A, Bresnahan R, Evans E, Herron K, Hapangama DK. Biopsychosocial Approaches for the Management of Female Chronic Pelvic Pain: A Systematic Review. Bjog. 2025;132(3):266-77.
188. Starzec-Proserpio M, Frawley H, Bø K, Morin M. Effectiveness of nonpharmacological conservative therapies for chronic pelvic pain in women: a systematic review and meta-analysis. Am J Obstet Gynecol. 2025;232(1):42-71.
189. Ariza-Mateos MJ, Cabrera-Martos I, Ortiz-Rubio A, Torres-Sánchez I, Rodríguez-Torres J, Valenza MC. Effects of a Patient-Centered Graded Exposure Intervention Added to Manual Therapy for Women With Chronic Pelvic Pain: A Randomized Controlled Trial. Arch Phys Med Rehabil. 2019;100(1):9-16.
190. Ariza-Mateos MJ, Cabrera-Martos I, López-López L, Rodríguez-Torres J, Torres-Sánchez I, Valenza MC. Effects of a patient-centered program including the cumulative-complexity model in women with chronic pelvic pain: a randomized controlled trial. Maturitas. 2020;137:18-23.
191. Rodríguez-Torres J, López-López L, Cabrera-Martos I, Prados-Román E, Granados-Santiago M, Valenza MC. Effects of an Individualized Comprehensive Rehabilitation Program on Impaired Postural Control in Women With Chronic Pelvic Pain: A Randomized Controlled Trial. Arch Phys Med Rehabil. 2020;101(8):1304-12.
192. Nygaard AS, Rydningen MB, Stedenfeldt M, Wojniusz S, Larsen M, Lindsetmo RO, et al. Group-based multimodal physical therapy in women with chronic pelvic pain: A randomized controlled trial. Acta Obstet Gynecol Scand. 2020;99(10):1320-9.
193. Bafort C, Beebeejaun Y, Tomassetti C, Bosteels J, Duffy JM. Laparoscopic surgery for endometriosis. Cochrane Database Syst Rev. 2020;10(10):Cd011031.
194. Singh SS, Gude K, Perdeaux E, Gattrell WT, Becker CM. Surgical Outcomes in Patients With Endometriosis: A Systematic Review. J Obstet Gynaecol Can. 2020;42(7):881-8.e11.
195. Tummers F, Peltenburg SI, Metzemaekers J, Jansen FW, Blikkendaal MD. Evaluation of the effect of previous endometriosis surgery on clinical and surgical outcomes of subsequent endometriosis surgery. Arch Gynecol Obstet. 2023;308(5):1531-41.
196. Grundström H, Fredrikson M, Alehagen S, Berterö C, Kjølhede P. Incidence of self-reported pelvic pain and risk factors for pain 1 year after benign hysterectomy: A register study from the Swedish National Quality Registry for Gynecological Surgery. Acta Obstet Gynecol Scand. 2023;102(10):1359-70.
197. Hillis SD, Marchbanks PA, Peterson HB. The effectiveness of hysterectomy for chronic pelvic pain. Obstet Gynecol. 1995;86(6):941-5.
198. As-Sanie S, Till SR, Schrepf AD, Griffith KC, Tsodikov A, Missmer SA, et al. Incidence and predictors of persistent pelvic pain following hysterectomy in women with chronic pelvic pain. Am J Obstet Gynecol. 2021;225(5):568.e1-.e11.

199. Honigberg MC, Zekavat SM, Aragam K, Finneran P, Klarin D, Bhatt DL, et al. Association of Premature Natural and Surgical Menopause With Incident Cardiovascular Disease. Jama. 2019;322(24):2411-21.
200. Laughlin-Tommaso SK, Khan Z, Weaver AL, Smith CY, Rocca WA, Stewart EA. Cardiovascular and metabolic morbidity after hysterectomy with ovarian conservation: a cohort study. Menopause. 2018;25(5):483-92.
201. Mamlouk MD, vanSonnenberg E, Dehkharghani S. CT-guided nerve block for pudendal neuralgia: diagnostic and therapeutic implications. AJR Am J Roentgenol. 2014;203(1):196-200.
202. Malec-Milewska M, Horosz B, Kolęda I, Sękowska A, Kucia H, Kosson D, et al. Neurolytic block of ganglion of Walther for the management of chronic pelvic pain. Wideochir Inne Tech Maloinwazyjne. 2014;9(3):458-62.
203. Chung Y-C, Chen H-H, Yeh M-L. Acupoint stimulation intervention for people with primary dysmenorrhea: Systematic review and meta-analysis of randomized trials. Complementary Therapies in Medicine. 2012;20(5):353-63.
204. Jiang HR, Ni S, Li JL, Liu MM, Li J, Cui XJ, et al. Systematic review of randomized clinical trials of acupressure therapy for primary dysmenorrhea. Evid Based Complement Alternat Med. 2013;2013:169692.
205. Luo FY, Nasr-Esfahani M, Jarrell J, Robert M. Botulinum toxin injection for chronic pelvic pain: A systematic review. Acta Obstet Gynecol Scand. 2020;99(12):1595-602.
206. Spruijt MA, Klerkx WM, Kelder JC, Kluivers KB, Kerkhof MH. The efficacy of botulinum toxin a injections in pelvic floor muscles in chronic pelvic pain patients: a systematic review and meta-analysis. Int Urogynecol J. 2022;33(11):2951-61.
207. Nijs J, D'Hondt E, Clarys P, Deliens T, Polli A, Malfliet A, et al. Lifestyle and Chronic Pain across the Lifespan: An Inconvenient Truth? Pm r. 2020;12(4):410-9.
208. Hearn-Yeates F, Edgley K, Horne AW, O'Mahony SM, Saunders PTK. Dietary Modification and Supplement Use For Endometriosis Pain. JAMA Netw Open. 2025;8(3):e253152.
209. Field R, Pourkazemi F, Turton J, Rooney K. Dietary Interventions Are Beneficial for Patients with Chronic Pain: A Systematic Review with Meta-Analysis. Pain Med. 2021;22(3):694-714.
210. Kaushik AS, Strath LJ, Sorge RE. Dietary Interventions for Treatment of Chronic Pain: Oxidative Stress and Inflammation. Pain Ther. 2020;9(2):487-98.
211. Staudacher HM, Whelan K. The low FODMAP diet: recent advances in understanding its mechanisms and efficacy in IBS. Gut. 2017;66(8):1517-27.
212. Lindell M, Grimby-Ekman A. Stress, non-restorative sleep, and physical inactivity as risk factors for chronic pain in young adults: A cohort study. PLoS One. 2022;17(1):e0262601.
213. Geneen LJ, Moore RA, Clarke C, Martin D, Colvin LA, Smith BH. Physical activity and exercise for chronic pain in adults: an overview of Cochrane Reviews. Cochrane Database Syst Rev. 2017;4(4):Cd011279.
214. Gurian MB, Mitidieri AM, da Silva JB, da Silva AP, Pazin C, Poli-Neto OB, et al. Measurement of pain and anthropometric parameters in women with chronic pelvic pain. J Eval Clin Pract. 2015;21(1):21-7.
215. Shiri R, Karppinen J, Leino-Arjas P, Solovieva S, Viikari-Juntura E. The association between obesity and low back pain: a meta-analysis. Am J Epidemiol. 2010;171(2):135-54.
216. Chaput JP, McHill AW, Cox RC, Broussard JL, Dutil C, da Costa BGG, et al. The role of insufficient sleep and circadian misalignment in obesity. Nat Rev Endocrinol. 2023;19(2):82-97.
217. Tan IF, Horne A. Obesity and chronic pelvic pain. 2020. p. 281-91.
218. Mathias JL, Cant ML, Burke ALJ. Sleep disturbances and sleep disorders in adults living with chronic pain: a meta-analysis. Sleep Med. 2018;52:198-210.
219. Cosar E, Çakır Güngör A, Gencer M, Uysal A, Hacivelioğlu SO, Özkan A, et al. Sleep disturbance among women with chronic pelvic pain. Int J Gynaecol Obstet. 2014;126(3):232-4.

220. de Souza RJ, Villela NR, Brollo LCS, Oliveira MAP. Impact of chronic pelvic pain and painful bladder syndrome on the Pittsburgh Sleep Quality Index on women with deep endometriosis: a cross-sectional study. Int Urogynecol J. 2023;34(10):2487-93.
221. Haack M, Simpson N, Sethna N, Kaur S, Mullington J. Sleep deficiency and chronic pain: potential underlying mechanisms and clinical implications. Neuropsychopharmacology. 2020;45(1):205-16.
222. Scenario: Managing insomnia: National Institute for Health and Care Excellence; 2025 [Available from: https://cks.nice.org.uk/topics/insomnia/management/managing-insomnia/#good-sleep-hygiene.
223. Murawski B, Wade L, Plotnikoff RC, Lubans DR, Duncan MJ. A systematic review and meta-analysis of cognitive and behavioral interventions to improve sleep health in adults without sleep disorders. Sleep Med Rev. 2018;40:160-9.
224. Dai Y, Huang J, Hu Q, Huang L, Wu J, Hu J. Association of Cigarette Smoking with Risk of Chronic Musculoskeletal Pain: A Meta-Analysis. Pain Physician. 2021;24(8):495-506.
225. Orhurhu VJ, Pittelkow TP, Hooten WM. Prevalence of smoking in adults with chronic pain. Tob Induc Dis. 2015;13(1):17.
226. Liu Y, Li H, Wang J, Xue Q, Yang X, Kang Y, et al. Association of Cigarette Smoking With Cerebrospinal Fluid Biomarkers of Neurodegeneration, Neuroinflammation, and Oxidation. JAMA Netw Open. 2020;3(10):e2018777.
227. Chiba S, Yamada K, Kawai A, Hamaoka S, Ikemiya H, Hara A, et al. Association between smoking and central sensitization pain: a web-based cross-sectional study. J Anesth. 2024;38(2):198-205.
228. Coxon L, Lugt C, Horne AW, Evans E, Abreu-Mendes P, Arendt-Nielsen L, et al. Symptom flares in women with chronic pelvic pain: Questionnaire study within a cohort study (translational research in pelvic pain (TRiPP)). Bjog. 2024;131(13):1832-40.
229. Sutcliffe S, Bradley CS, Clemens JQ, James AS, Konkle KS, Kreder KJ, et al. Urological chronic pelvic pain syndrome flares and their impact: qualitative analysis in the MAPP network. Int Urogynecol J. 2015;26(7):1047-60.
230. Endometriosis in the UK: time for change. APPG on Endometriosis. Inquiry Report 2020 Endometriosis UK: All Party Parliamentary Group on Endometriosis; 2020 [Available from: https://www.endometriosis-uk.org/sites/default/files/files/Endometriosis%20APPG%20Report%20Oct%202020.pdf.
231. Coulter A, Entwistle VA, Eccles A, Ryan S, Shepperd S, Perera R. Personalised care planning for adults with chronic or long-term health conditions. Cochrane Database of Systematic Reviews. 2015(3).
232. Daniell HW. Opioid endocrinopathy in women consuming prescribed sustained-action opioids for control of nonmalignant pain. J Pain. 2008;9(1):28-36.

Appendix 1: Explanation of grades and evidence levels

Classification of evidence levels

1++	High-quality meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a very low risk of bias
1+	Well-conducted meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a low risk of bias
1–	Meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a high risk of bias
2++	High-quality systematic reviews of case–control or cohort studies or high-quality case–control or cohort studies with a very low risk of confounding, bias or chance and a high probability that the relationship is causal
2+	Well-conducted case–control or cohort studies with a low risk of confounding, bias or chance and a moderate probability that the relationship is causal
2–	Case–control or cohort studies with a high risk of confounding, bias or chance and a significant risk that the relationship is not causal
3	Non-analytical studies, e.g. case reports, case series
4	Expert opinion

Grades of Recommendation

A At least one meta-analysis, systematic review or RCT rated as 1++, and directly applicable to the target population; or a systematic review of RCTs or a body of evidence consisting principally of studies rated as 1+, directly applicable to the target population and demonstrating overall consistency of results

B A body of evidence including studies rated as 2++ directly applicable to the target population, and demonstrating overall consistency of results; or
Extrapolated evidence from studies rated as 1++ or 1+

C A body of evidence including studies rated as 2+ directly applicable to the target population, and demonstrating overall consistency of results; or
Extrapolated evidence from studies rated as 2++

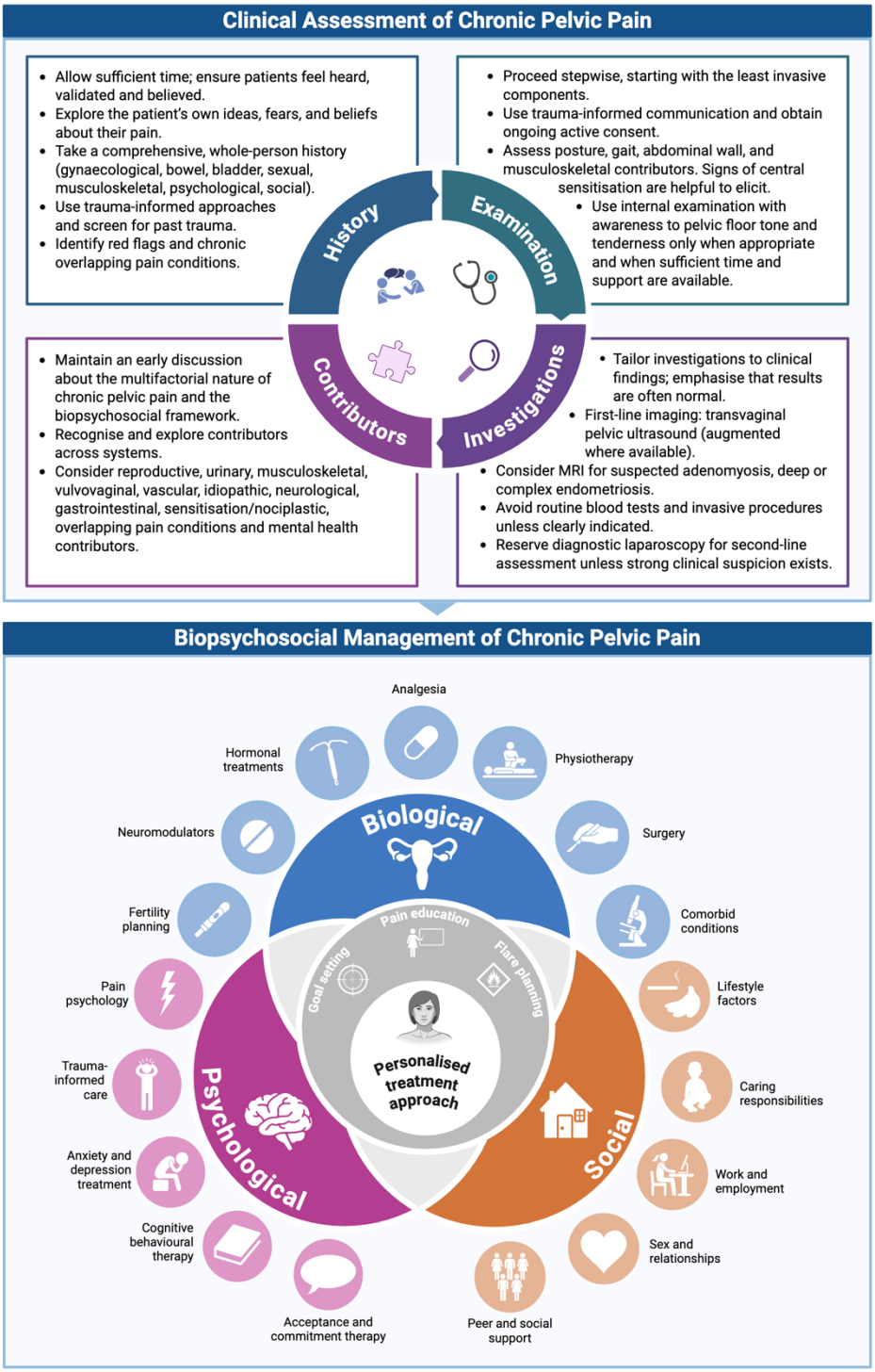
D Evidence level 3 or 4; or
Extrapolated evidence from studies rated as 2+

Good Practice Points

GPP Recommended best practice based on the clinical experience of the guideline development group.*

*on the occasion when the guideline development group find there is an important practical point that they wish to emphasise but for which there is not, nor is there likely to be any research evidence. This will typically be where some aspect of treatment is regarded as such sound clinical practice that nobody is likely to question it. These are marked in the guideline, and are indicated by GPP. It must be emphasised that these are NOT an alternative to evidence-based recommendations, and should only be used where there is no alternative means of highlighting the issue.

Appendix 2: Assessment and biopsychosocial management of chronic pelvic pain



The clinical assessment of chronic pelvic pain should be structured to identify the key contributors to an individual's symptoms, using targeted history, examination and investigations. This approach recognises that CPP is rarely driven by a single source and often reflects interactions across biological, psychological and social domains. Understanding each person's pain within this broader context provides the foundation for a personalised, biopsychosocial management plan that addresses not only potential pathology but also the cognitive, emotional, and social factors that shape the pain experience. Created in Biorender.

Appendix 3: Chronic Pelvic Pain Patient Questionnaire

(see attached pdf)

Chronic Pelvic Pain
Patient Questionnaire

Patient label or details:

Date: ___ / ___ / _____

This questionnaire is an opportunity to tell us about your symptoms and experiences. Your answers will help us understand how pelvic pain affects you and how we can best support you.

Section A: Pain History

We would like you to describe your pain as it is now, or as it has been over the last few weeks.

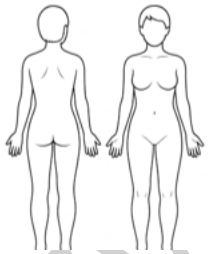
1. **How long have you had your pain?**
   - Q Less than 3 months
   - Q 3 months to 1 year
   - Q Over 1 year

2. **Can you remember the first time you experienced the pain that bothers you currently? If so, when was this and at that time, what did you think caused the pain?**

   _____
   _____
   _____
   _____

3. **Please mark the area of your pain(s) on the diagram to the right.**
4. **Does your pain radiate or spread anywhere?**
   - Q Yes
   - Q No

   If yes, please draw arrows where it radiates to.
5. **What is the character of your pain?**
   - Q Burning
   - Q Shooting



This guideline was produced on behalf of the Royal College of Obstetricians and Gynaecologists by: **Dr Scott C Mackenzie MBChB, Edinburgh*, Dr Una Pak MRCOG, Edinburgh*, Professor Katy Vincent FRCOG, Oxford, Professor Rajvinder Khasriya, MRCOG, London, Professor Andrew Horne FRCOG, Edinburgh. *joint first authors**

The peer reviewers were: **xxx**

Committee lead reviewers were: Dr A Campbell FRCOG, Edinburgh; Dr L Knight MRCOG, Devon.

The Chairs of the Guidelines Committee were: Dr A Campbell FRCOG, Edinburgh and Dr L Knight MRCOG, Devon.

The final version is the responsibility of the Guidelines Committee of the RCOG.

The guideline will be considered for update three years after publication, with an intermediate assessment of the need to update two years after publication.