Original paper

Central sensitisation as a mechanism of chronic pain in women with endometriosis – a systematic review of the literature

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Abstract

Introduction: Endometriosis in women is a chronic inflammatory disease in which pain is the dominant and often the most debilitating clinical symptom. The severity of pain often does not correlate with the extent of anatomical changes, and in a significant proportion of patients it persists despite surgical and hormonal treatment.
Aim of the research: To analyse the available scientific evidence on central sensitisation as a mechanism of chronic pain in women with endometriosis, taking into account its relationship with neuropathic pain, nociceptive pain, myofascial pain, and quality of life after surgical treatment.
Material and methods: The systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines. In November and December 2025, electronic databases, including PubMed, Scopus, and Web of Science, were searched. The search strategy was based on the keywords "endometriosis", "central nervous system sensitisation", and "central sensitisation". Ultimately, 14 publications were included in the review.
Results: The findings clearly indicate a frequent occurrence of central sensitisation, reduced pain thresholds, and symptoms of neuropathic pain in women with endometriosis. They also suggest strong links between these mechanisms and pain intensity, sexual dysfunction, anxiety and depressive disorders, and poorer quality of life, including in women who have undergone surgical treatment.
Conclusions: Central sensitisation appears to be a major mechanism underlying chronic pain in women with endometriosis. Failure to take it into account in the therapeutic process may lead to the ineffectiveness of surgical and pharmacological treatment.

Introduction

Endometriosis is a chronic, oestrogen-dependent inflammatory disease characterised by the presence of endometrium-like tissue outside the uterine cavity. It affects approximately 10% of women of reproductive age [1–4]. This condition is associated with a wide range of pain symptoms, including painful menstruation, dyspareunia, painful bowel movements, and persistent chronic pelvic pain (CPP) [1, 3, 5]. Traditionally, the pathophysiology of pain in endometriosis has been viewed primarily as the result of nociceptive mechanisms resulting from local inflammation and irritation of receptors by disease foci [1, 5, 6]. An increasing number of studies indicate that multiple factors contribute significantly to the pathogenesis of pain in women with endometriosis [7]. An important clinical problem is that in approximately 40% to 50% of women, pain persists or recurs within 5 years of surgical removal of endometrial lesions, even after radical procedures such as hysterectomy [1, 7–9]. In recent years, a growing number of studies have indicated that central mechanisms, in particular central sensitisation, play an important role in the pathophysiology of pain associated with endometriosis, leading to the perpetuation of pain regardless of peripheral stimuli [10]. This suggests that the chronicity of pain involves mechanisms that extend beyond peripheral tissue damage alone, which has led to the recognition of the category of nociceptive pain [4, 5, 11, 12]. Central sensitisation is a process of neuroplasticity defined as persistent hyperexcitability of neurons in the central nervous system (CNS), which leads to the amplification and distortion of pain perception [1, 5, 10, 11, 13]. Persistent nociceptive stimulation induces presynaptic and postsynaptic changes that lower the threshold of neuronal excitability, manifesting clinically as allodynia (pain in response to a non-painful stimulus) and hyperalgesia (an exaggerated response to pain) [2, 5, 13, 14]. Despite the increasing volume of publications, the available data remain scattered, and their synthesis is necessary for a better understanding of pain mechanisms and their clinical implications [15].

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Aim of research

The aim of this systematic review is to analyse and synthesise the available scientific data on the role of central sensitisation as a mechanism underlying chronic pain in women with endometriosis. Consideration of these processes allows for a more complete understanding of the pathophysiology of pain and may form a basis for more individualised therapeutic management [2, 6, 15].

Materials and methods

The systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines [16]. In November and December 2025, electronic databases, including PubMed, Scopus, and Web of Science, were searched. The search terms included combinations of PubMed MeSH and text terms. The text terms were: "endometriosis", "central nervous system sensitisation", and "central sensitisation". The "AND" operator was used in the review, representing the intersection of sets (i.e. their common part). Other criteria for inclusion of publications in the analysis are presented in Table 1.

The titles and abstracts of publications identified in the search process were preliminarily selected to exclude works that did not fall within the scope of the review. Subsequently, the full texts of potentially eligible articles were independently assessed by the authors of this paper for compliance with the inclusion criteria. During the full-text analysis stage, the CART criteria (completeness, accuracy, relevance, and timeliness) were applied to enable exclusion of studies that referred to the analysed phenomenon only to a limited extent.

As a result of the initial database search, 229 records were identified, of which 226 publications were retained for further analysis after removing duplicates (n = 3). During the screening of titles and abstracts, 143 articles were excluded. Subsequently, a full-text analysis of the remaining works was conducted, resulting in the exclusion of 69 publications that did not meet the inclusion criteria. Ultimately, 14 publications were included in the review. The individual stages of the electronic database search are presented in Figure 1. Publications excluded at the full-text analysis stage did not meet the predefined inclusion criteria.

Results

The eligible publications included three systematic reviews, two prospective studies, three cohort studies, and six cross-sectional studies. The studies covered a total of over 2000 women with endometriosis.

The publications reviewed focused on the following areas:

1) Prevalence and identification of central sensitisation [1, 2, 4–8, 10, 14];
2) Evidence from quantitative sensory testing (QST) [8, 11–13];
3) Central sensitisation and the severity of endometriosis [7, 14];
4) Co-occurrence with myofascial dysfunction and neuropathic pain [1–5, 12];
5) Impact on treatment outcomes and quality of life [1, 5, 6, 9–11, 14];
6) Biological and imaging mechanisms [8, 12] (Table 2) [17–20].

Discussion

Reports in the literature indicate a paradigm shift in the understanding of the mechanism of pain associated with endometriosis. This phenomenon is no longer viewed solely as a consequence of local disease processes but has become a complex condition associated with abnormal pain processing, in which central sensitisation (CS) plays a key role [5, 13].

One of the most consistent research findings is the lack of correlation between the severity of endometriosis (according to the rASRM and ENZIAN scales) and the intensity of pain and the degree of sensitisation of the nervous system.

Studies have shown that patients with mild forms of the disease may exhibit significantly higher levels of central sensitisation than those with deep infiltrat-

Table 1. Criteria for inclusion of articles in the analysis and criteria for exclusion from the analysis

Inclusion criteria	Exclusion criteria
• Language of publication: English and/or Polish • Year of publication: 2015–2025 • Type of publication: original research (RCT, cohort, case-control, cross-sectional), systematic review, meta-analysis • Full access • Compliance with the accepted combination of keywords	• Publication language: other than English and/or Polish • Year of publication: before 2015 • Publication type other than: original studies (RCT, cohort, case-control, cross-sectional), systematic review, meta-analysis • Animal studies • No full access to the article • No compliance with the accepted combination of keywords • No separate group of patients with endometriosis

RTC – randomised controlled trial.

ing endometriosis. This suggests that it is not the extent of endometrial changes but the way signals are processed by the CNS that determines the occurrence of pain in these women. This phenomenon supports the theory of nociceptive pain, in which there is persistent hyperactivity of neurons in the dorsal horns of the spinal cord and higher brain centres. From a practical perspective, this means that women with a "mild" form of the disease in anatomical terms may present with very severe pain symptoms and a high degree of central sensitisation, while a group of women with deep infiltrating endometriosis may experience less pain or no pain at all [2, 6, 10, 11, 20–22]. Analysis of the available data calls into question the validity of surgical treatment as the only and sufficient method of pain management in patients with endometriosis [1, 4]. Numerous studies have shown that high scores on the Central Sensitisation Inventory (CSI) questionnaire in the preoperative period are a significant indicator of persistent pain after surgery [1, 4, 15, 23]. Surgical removal of peripheral pain generators in the form of endometrial foci reduces peripheral nociception but does not lead to the normalisation of malfunctioning central pain processing mechanisms [4]. In addition, in patients with multiple comorbidities within the spectrum of central sensitivity syndromes (CSSs), high CSI values often persist despite effective resection of endometrial lesions [9, 10, 23]. This phenomenon may partly explain the high rate of pain recurrence and reoperation, reaching up to 50% within 5 years after surgery [1]. The Central Sensitisation Inventory is therefore becoming an essential tool for pain phenotyping and for providing realistic counselling of patients regarding the expected outcomes of surgical treatment [1, 4, 15, 24].

The literature also points to a very important link between central sensitisation and myofascial pain syndrome (MPS) [15, 24]. The presence of trigger points in the pelvic floor and abdominal wall muscles increases the likelihood of central sensitisation by more than ninefold [15]. This is evidence of a viscerosomatic convergence process, whereby pain originating from internal organs is "transferred" to tissues and muscles, thus creating a vicious circle of muscle tension and pain [5, 18, 24]. Similarly, pain during sexual intercourse – both deep dyspareunia and pain during orgasm – shows a strong correlation with high CSI scores and levator ani muscle tenderness, indicating a central and nociceptive component to these complaints [3, 14].

In addition, 36–44.1% of patients exhibit a neuropathic pain component (DN4 ≥ 4), which is associated with greater severity of symptoms and significant deterioration in quality of life [6, 11]. These data confirm the multi-mechanistic nature of pain in endometriosis and the need for its comprehensive assessment [23].

The relationship between central sensitisation and the mental state of patients is bidirectional [10, 17].

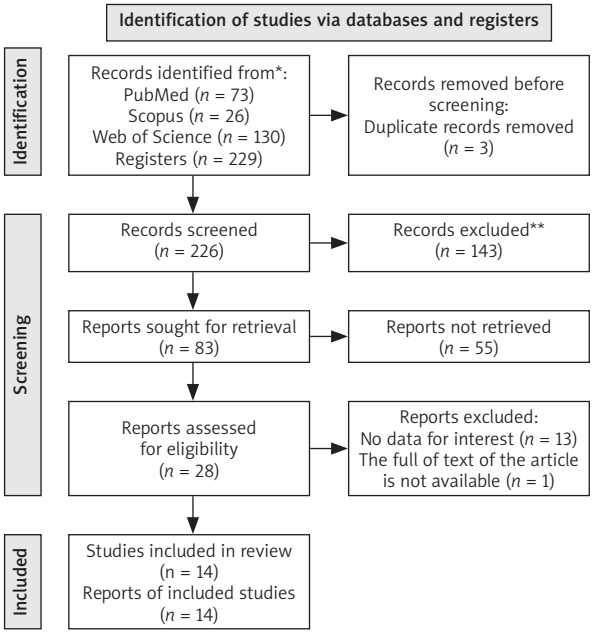

Figure 1. PRISMA 2020 flow diagram for new systematic reviews that included searches of databases and registers

Neuroimaging (fMRI) and biomarker analyses provide objective evidence of central sensitisation, such as reduced grey matter volume in the thalamus and elevated levels of neuroplasticity mediators (BDNF, TNF-α, nitric oxide).

Psychological factors such as pain catastrophising, anxiety, and depression are not solely a consequence of the disease but also play an active role in modulating pain processing, exacerbating sensitisation mechanisms. Patients with features of central sensitisation demonstrate a significantly higher severity of these factors [4, 6, 10, 15]. In addition, patients with a history of sexual abuse are more prone to developing central sensitisation, suggesting that previous trauma may "sensitise" the nervous system to future nociceptive stimuli [15].

The traditional therapeutic approach, based solely on hormonal and surgical treatment, is insufficient for patients with a dominant mechanism of central sensitisation [2, 6, 15]. Effective therapy should be based on an integrated, interprofessional therapeutic strategy.

Conclusions

Central sensitisation appears to be a major mechanism underlying chronic pain in women diagnosed with endometriosis. Therefore, it is very important to thoroughly understand the nature of this phenomenon and the mechanisms behind it. The routine use of tools such as the Central Sensitisation Inventory (CSI) or the Neuropathic Pain Questionnaire (DN4) in the midwife's and/or gynaecologist's practice may contribute to the early identification of patients who require treatment targeted at pain-inducing mecha-

Table 2. Thematic analysis of articles included in the systematic review

Author [year]	Title	Type of study	Population (*n*)	Project/research tools	Main mechanisms assessed	Key findings
Dolci *et al.*; 2025 [6]	Neuropathic-like pain affects pain perception in patients with deep endometriosis: an observational study	Cohort study, retrospective	*N* = 149 (DE + CPP)	NRS, DN4, QDSA, EQ-VAS, PCS, HAD, abridged Beck questionnaire	Neuropathic-like pain	Pain in endometriosis is not exclusively nociceptive (resulting from tissue damage). In more than one third of patients, neuropathic pain is a significant component. The presence of neuropathic pain, higher intensity of sensations and catastrophising are consistent with central and peripheral sensitisation mechanisms.
Quintas-Marques; 2025 [17]	Multidimensional Evaluation of Myofascial Pelvic Pain and Other Comorbidities in Endometriosis Patients	Cross-sectional study	*N* = 175 (MPP = 84, non-MPP = 91)	NRS, SF-36, HADS, CSI	Central sensitisation	Patients with endometriosis and MPP present with higher pain intensity, poorer quality of life and a greater number of comorbidities and symptoms (including anxiety and depression). This pattern of symptoms is consistent with the mechanism of central sensitisation.
Ding *et al.*; 2024 [3]	Pain with orgasm in endometriosis: potential etiologic factors and clinical correlates,	Prospective study	*N* = 358	CSI, GAD-7, PHQ-9, PCS, FSDS-R	Central sensitisation, sexual pain	Pelvic pain exacerbated by orgasm may occur in endometriosis, with potential aetiological factors including pelvic floor myalgia and underlying central sensitisation.
Bouko-Levy *et al.*; 2024 [11]	Prevalence of neuropathic pain in patients with symptomatic endometriosis: Assessment using the DN4 score.	Cohort study	*N* = 236	EPH-5, DN4, PPSC, VIRAGE	Central sensitisation, neuropathic pain	In the cohort of patients with endometriosis who completed the PPSC (Pelvic and Perineal Pain Central Sensitisation) questionnaire, 39.4% of women showed signs of central sensitisation (score ≥ 5). The study showed a strong association between sensitisation and neuropathic pain.
Gentles *et al.*; 2024 [5]	Nociplastic Pain in Endometriosis: A Scoping Review	Systematic review (scoping review)	30 articles	Literature analysis	Nociplastic pain	Patients with endometriosis who experience CS symptoms and pain with a significant nociceptive component are more likely to have more severe pelvic pain symptoms, regardless of the type of endometriosis or previous surgery. What is more, CS is clinically significant because patients with its indicators (e.g. higher CSI scores) have a poorer quality of life and higher scores for chronic pelvic pain, deep dyspareunia, dyschezia, and back pain after surgical treatment of endometriosis.

Table 2. cont.

Author [year]	Title	Type of study	Population (*n*)	Project/research tools	Main mechanisms assessed	Key findings
Tucker *et al.*; 2023 [4]	Pelvic pain comorbidities associated with quality of life after endometriosis surgery.	Prospective longitudinal study	*N* = 444	EHP 30, GAD-7, PCS, PHQ-9	Central sensitisation and surgical treatment outcomes	Central sensitisation is the main mechanism of pain maintenance after endometriosis surgery. Comorbid pain syndromes strongly indicate CS. Patients with CS may not experience significant improvement after surgical treatment of endometriosis.
Quintas-Marquès *et al.*; 2023 [10]	Central sensitisation in patients with deep endometriosis.	Cross-sectional study	*N* = 235 (patients with DE = 169; healthy patients = 66)	CSI, SF-36, HADS	Central sensitisation	Patients with deep endometriosis have significantly higher CSI scores, poorer quality of life, and a higher risk of depression and anxiety.
Centera *et al.*; 2023 [13]	Central Sensitisation in Vulvodynia and Endometriosis: What Have We Been Overlooking So Far?	Systematic review, Meta-analysis	10 articles	Literature analysis	Central sensitisation	Central sensitisation may play a key role in both endometriosis and vulvodynia. There is ample evidence that the pain experienced by these patients is not solely related to peripheral changes but is sustained by processes in the central nervous system.
Orr *et al.*; 2023 [1]	Association of Central Sensitisation Inventory Scores with Pain Outcomes After Endometriosis Surgery.	Cohort study, longitudinal	*N* = 239	CSI; NRS; GAD-7; PHQ-9; PCS	Central sensitisation and surgical treatment outcomes	The results indicate that patients with higher severity of CS-related symptoms have worse pain outcomes after surgery, which is consistent with the hypothesis that surgical treatment of peripheral pain generators (endometriosis lesions) may not directly treat central pain generators (CS).
Orr *et al.*; 2020 [14]	Phenotyping Sexual Pain in Endometriosis Using the Central Sensitisation Inventory.	Cross-sectional (prospective) study	*N* = 163	GAD-7; PHQ-9; NRS; CSI	Central sensitisation, sexual pain;	A subgroup of women with endometriosis who experience severe pain during intercourse and bladder/pelvic floor tenderness show higher levels of central sensitisation. This suggests that their pain may not be solely due to endometrial changes (peripheral factors) but to the amplification of pain signals by the nervous system.
Grundström *et al.*; 2019 [19]	Reduced pain thresholds and signs of sensitisation in women with persistent pelvic pain and suspected endometriosis	Cross-sectional (observational) study	*N* = 92 (patients with PPP = 37; healthy patients = 55)	Quantitative sensory tests (heat, cold, pressure pain); SF-36, HADS	Central sensitisation	Extensive changes in pain thresholds in women with PPP indicate the presence of central sensitisation, i.e. the amplification of pain perception by the central nervous system. The results support the theory that it is the state of persistent pain itself, rather than the presence of visible endometrial changes, that is the main factor driving the development of central sensitisation.

Table 2. cont.

Author [year]	Title	Type of study	Population (*n*)	Project/research tools	Main mechanisms assessed	Key findings
Ping *et al.*; 2019 [20]	Research on central sensitisation of endometriosis-associated pain: a systematic review of the literature.	Systematic review	15 articles	Literature analysis	Central sensitisation – pain mechanisms	Pain associated with endometriosis is currently considered to be a type of neuropathic pain or neurogenic inflammation. Persistent pain after surgical removal of endometrial lesions is often the result of CS – persistent pain signals from tissues "teach" neurons in the spinal cord to be overly reactive, which sustains pain despite the absence of the original stimulus.
van Aken *et al.* 2018 [2]	Experimental pain tolerance is decreased and independent of clinical pain intensity in patients with endometriosis	Cross-sectional study	*N* = 82 (patients with endometriosis = 35, control group = 38)	QST pain tolerance tests	Central sensitisation	Significantly reduced pain tolerance was observed in patients with endometriosis, regardless of clinical pain intensity or American Society for Reproductive Medicine stage, compared to healthy controls.
Stratton *et al.*; 2015 [15]	Association of chronic pelvic pain and endometriosis with signs of sensitisation and myofascial pain.	Cross-sectional (prospective) study	*N* = 49	Pinch and roll technique; Wartenberg pinwheel; Pressure algometer; VAS; EHP-30;	Central and peripheral sensitisation, myofascial pain	Signs of sensitisation (measured as regional allodynia and hyperalgesia) were found in 83% of women with current, confirmed endometriosis and in 82% of women with pain without current endometriosis. For comparison, in healthy volunteers, this rate was only 15%. Women with any history of endometriosis (even if the lesions had been previously removed) showed the highest rate of sensitisation – as high as 87%. This suggests that pain may be sustained by changes in the nervous system and not just by current foci of the disease.

DE – deep endometriosis, CS – central sensitisation, CCP – chronic pelvic pain, NRS – numerical rating scale, DN4 – Neuropathic Pain Questionnaire in 4 parts, QDSA – Saint-Antoine Pain Questionnaire, EqVAS - Visual Analogue Scale in the EQ-5D, PCS – Pain Catastrophizing Scale, HAD – Hospital Anxiety and Depression, MPP – Myofascial Pelvic Pain, SF-36 – Short-Form Health Survey 36, HADS – Hospital Anxiety and Depression Scale, CSI – Central Sensitization Inventory, GAD-7 – Generalized Anxiety Disorder 7, PHQ-9 – Patient Health Questionnaire 9, FSDS-R – Female Sexual Distress Scale-Revised, EPH-5 – Endometriosis Health Profile-5, PPSC – Pelvic and Perineal Pain Central Sensitization, VIRAGE – Violence and Gender Relations, EHP 30 – Endometriosis Health Profile-30, PPP – Persistent Pelvic Pain, QST – quantitative sensory testing, VAS – Visual Analogue Scale.

nisms. Furthermore, the implementation of such procedures may contribute to the rational and optimised planning of further therapeutic management and high-quality care for this group of women.

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Ethical approval

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Conflict of interest

The authors declare no conflict of interest.

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