

## COMMENTARY

# Endometriosis—The scapegoat for pelvic pain?

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## Abstract

Cyclic and noncyclic chronic pelvic pain (CPP) represent a major problem for both affected patients and healthcare providers due to its effects on physical and mental health. In times of social media and digitalization, awareness of endometriosis as a leading cause of CPP is increasing. However, a close look at the current literature does not support endometriosis as the predominant and most common cause of pelvic pain syndromes and associated morbidities. Consequently, other or additional factors may be overlooked, complex pain mechanisms simplified, and symptoms misunderstood, resulting in less optimal treatment concepts. This commentary underlines the necessity to evaluate patients with CPP and suspected endometriosis in a multifactorial and holistic context to provide a better framework of patient care.

## KEYWORDS

attitudes, chronic pelvic pain, endometriosis, hyperalgesia, practice

## 1 | INTRODUCTION

The prevalence of endometriosis ranges between 1% up to 19% in the premenopausal population depending on the study population, diagnostic method, and source of healthcare insurance data investigated.<sup>1,2</sup> Within this, the condition is a well-known cause for chronic pelvic pain (CPP) and symptoms such as dysmenorrhea, dyspareunia, defecation pain, and impairment of gastrointestinal

function.<sup>3,4</sup> CPP is defined as cyclic or noncyclic pelvic pain lasting for at least 6 months with or without dysmenorrhea, dyspareunia, dysuria, and cyclic defecation pain.<sup>5,6</sup>

Endometriosis is found in about 30%<sup>7</sup> of patients with CPP with a range of prevalence rates between 15% and 70%.<sup>7,8</sup> However, CPP is a multifactorial condition, with contributing factors that include complex pain mechanisms (such as central sensitization), as well as psychological, behavioral, and social influences.<sup>9</sup> Therefore, while

**Abbreviations:** CPP, chronic pelvic pain; DE, deep endometriosis.

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endometriosis is commonly associated with CPP, its role as a certain cause should be interpreted with caution.

Beyond its psychosocial burden, endometriosis and CPP have a major economic impact, with annual total costs per patient exceeding 9500 Euros.<sup>10</sup> Recent awareness campaigns and social media activism have significantly increased public awareness of endometriosis as a cause of pelvic pain and subfertility.<sup>11</sup> However, given the complexity of pain mechanisms and the numerous factors that contribute to CPP—many of which may be unrelated to tissue injury,<sup>12</sup> it is worth critically reviewing the true relationship between pelvic pain and endometriosis. While tailored treatment for endometriosis in symptomatic patients is crucial, there is a risk of oversimplifying CPP by neglecting the complex interplay of factors beyond endometriosis. In this commentary, we review the literature to highlight the problem inherent in attributing CPP predominantly to endometriosis—the possible scapegoat for pelvic pain.

## 2 | EXTENT OF ENDOMETRIOSIS AND PAIN

There is conflicting scientific evidence on the correlation of the extent of endometriosis and the severity of pain symptoms. Whether endometriosis necessarily causes pain is highly questionable since a significant proportion of women with extensive endometriosis are asymptomatic—with endometriosis being accidentally diagnosed in up to 50% of women undergoing surgery for other reasons.<sup>13</sup> Correlation studies tried to evaluate a possible association between the extent, that is, number, size, location, and infiltration depth of endometriotic lesions and the location, type, and severity of pain. However, most of these studies lack control groups. Furthermore, the studies investigating this topic are heterogenous, which is partly caused by the multitude of different classification systems used to quantify and stage endometriosis. In addition, disease extent when historically measured by the rASRM classification system does not take into account deep endometriosis (DE) and adenomyosis, which are well-accepted causes of pain.<sup>14</sup> Studies which have tried to correlate superficial, ovarian, and/or DE or rASRM stage with dysmenorrhea scores reported only minor or no direct correlation.<sup>15,16</sup> Although Montanari et al. observed that the number of affected #Enzian compartments (A, B, C, and FA, which correspond to vagina/ torus uterinus, uterosacral ligaments/ parametrium, rectum and uterus) was significantly associated with the severity of dysmenorrhea, the correlation coefficient was weak ( $r=0.256$ ).<sup>17</sup> Similarly, a correlation between the extent of bowel endometriosis and degree of defecation pain is lacking—Pashkunova et al.<sup>18</sup> prospectively evaluated #Enzian compartment C (rectum) grade and anatomical height and intensity of dyschezia in 162 women undergoing surgery for symptomatic colorectal DE without observing any direct correlation between these variables.

However, there is a general consensus that endometriosis patients with higher rates of severe deep dyspareunia and cyclic defecation pain appear to exhibit a higher frequency of DE affecting

### Key Message

Endometriosis is a predominant but not the only cause for chronic pelvic pain. An overestimation of the role of endometriosis and the underestimation of other causes including pain sensitization are common. Different perspectives, fresh approaches and understanding of pain mechanisms are necessary to improve patient care.

the vagina, uterosacral ligaments, and rectovaginal area.<sup>15,18,19</sup> To assess the true association of all types of endometriosis (including superficial) with clinical symptoms is difficult, if not impossible. This would require performing diagnostic laparoscopies on a large sample of women representative of the general premenopausal population, which would be unethical.

## 3 | CYCLIC AND NONCYCLIC PAIN AND ENDOMETRIOSIS

To evaluate a causal relation of endometriosis with cyclic pain symptoms would necessitate cohorts representing the general premenopausal population with and without endometriosis that are large enough to allow for the analysis of confounding factors. However, the study cohorts investigated to date do not reflect the general population or are small. In addition, the majority of studies performed so far on this subject analyzed cohorts of women scheduled for surgery due to pelvic pain, thereby introducing a selection bias. Nevertheless, there are some studies that investigate less selected, broader populations trying to ascertain the correlation between endometriosis and cyclic pain. Chaggar et al.<sup>20</sup> analyzed whether patients attending a general gynecological outpatient clinic showing signs of ovarian or DE on ultrasound were more likely to experience dyspareunia and pelvic pain compared to those without the disease. On multivariable analysis, cyclic defecation pain was the only symptom that was significantly more prevalent among 146 women with endometriosis compared to 368 without (47.9% vs. 28.3%;  $p<0.001$ ). A nonsignificant association was observed for dysmenorrhea and the presence of endometriosis when compared to women with other pathologies (87.3% vs. 79.9%;  $p=0.09$ ). A particular strength of the work by Chaggar et al. was that they also adjusted for the presence of adenomyosis, which has a significant symptom overlap with endometriosis.

Although this is in line with two other works on the prevalence of endometriosis in women with dysmenorrhea,<sup>16,21</sup> other studies have not been able to support this observation. A Cochrane review reported an uncertain benefit of removal of endometriosis lesions on pain.<sup>22</sup> This finding can be interpreted in two ways: either endometriosis was not the primary cause of the pain, or while endometriosis may contribute to pain, the underlying mechanisms of chronic pain are independent of the mere presence of endometriotic lesions.

It is important to note that the general prevalence of cyclic pain symptoms is surprisingly high. A WHO systematic review<sup>23</sup> on the prevalence of CPP reports rates of dysmenorrhea in 106 studies including 125 249 women ranging between 2% and 97% with 12% of women reporting severe dysmenorrhea. When looking at the most plausible prevalence rates of endometriosis in the general population<sup>1</sup> we will realize the absurdity of automatically connecting cyclic pain symptoms with endometriosis. However, the initiation of pain treatment and pain education may still be beneficial for patients with cyclic pelvic pain, especially for those with central sensitization symptoms, irrespective of pathognomonic signs of endometriosis on MRI or sonography.

Noncyclic pelvic pain has been linked with the presence of endometriosis by a multitude of studies. However, given that noncyclic pelvic pain most often originates from nongynecological sources, does this reflect a tendency to use endometriosis as a convenient scapegoat for pelvic pain?

While papers support a positive correlation, the large majority of the studies published to date fail to demonstrate a clear-cut causal relationship between endometriosis and noncyclic pain symptoms.<sup>24</sup> This is supported by Chaggar et al.<sup>20</sup> who did not detect any difference in rates of dyspareunia and noncyclic pelvic pain symptoms in patients with and without endometriosis attending a general outpatient clinic. However, a history of migraine and anxiety and depression was more common in women with dyspareunia,<sup>20</sup> indicating that the central sensitization mechanism might have been involved in increasing pain perception. As primary CPP syndromes and mechanisms of pain sensitization are by nature multifactorial and caused by complex neurobiological mechanisms, this should not be surprising.<sup>25</sup>

Moreover, research indicates that diminished quality of life in endometriosis patients is linked to a range of challenges including infertility, sexual dysfunction, mental health issues, chronic visceral pain, sleep disturbances, and fatigue.<sup>26,27</sup> While the presence of endometriosis contributes to pain, it may, on the other hand, represent just one phenotypical expression within a broader, more complex pain syndrome. Considering the multifactorial and complex nature of CPP syndromes and pain sensitization mechanisms, it is important to avoid attributing these symptoms exclusively to endometriosis.

Consequently, CPP should be seen in a comprehensive biopsychosocial context rather than being automatically linked to the possible presence of endometriosis. Furthermore, the current trend of establishing endometriosis treatment centers often results in facilities that function primarily as surgical hubs focused on advanced disease, which does not necessarily translate into a comprehensive service encompassing both diagnostics and non-surgical management with primary focus of improving patients' quality of life. As a result, many women with milder forms of endometriosis—but who still suffer with significant pain—find themselves without access to a high-quality, patient-centered, multidisciplinary care.

## 4 | ADENOMYOSIS AND PAIN

Adenomyosis shares most of the symptoms with endometriosis and is frequently found in women with endometriosis or vice versa.<sup>2</sup> As a result, adenomyosis represents an important confounder when investigating endometriosis-associated symptoms—yet this factor is often overlooked in many studies.<sup>28</sup> Traditionally, adenomyosis had been diagnosed via histopathology following hysterectomy, which limited the diagnosis to highly symptomatic patients and made it hard to study association with CPP. Although the development of noninvasive diagnosis of adenomyosis has been helpful in this respect, there are concerns about the diagnostic accuracy of imaging, with ambiguous sonographic signs sometimes used to diagnose adenomyosis. This is particularly problematic in adolescent or younger women, where overdiagnosis is a major concern and an adenomyosis prevalence up to 30% is described,<sup>29</sup> while definitive confirmation via histopathology is not feasible or appropriate. Consequently, while endometriosis has long been cited as the main explanation for CPP, emerging evidence that superficial endometriosis may not be the primary culprit has led clinicians to increasingly attribute pain to adenomyosis—potentially shifting the focus without resolving the underlying therapeutic challenges. This is apparent, since even in cases of clear preoperative signs of adenomyosis on pelvic imaging, hysterectomy not always relieves the pelvic pain, again indicating sensitization mechanisms that cannot be solved by surgery alone.

## 5 | CONCLUSION

While endometriosis is widely recognized as a significant contributor to pelvic pain it may, in many cases, merely play a contributory role or even be an incidental finding. Recognizing and understanding this complexity is essential for developing more effective and individualized treatment strategies.

A major challenge in the field is the entrenched perception among both patients and clinicians that endometriosis is always to blame for pelvic pain. Overcoming this construct is difficult and can leave patients feeling upset when the expected “culprit” is not found to be the sole cause of their symptoms. Complex pain mechanisms such as pain sensitization and their effects on pain-related morbidity seem to be underestimated in patients with endometriosis undergoing surgical or medical treatment and in women with other causes for CPP. To improve patient care, future efforts should focus on ways to identify, address, and rectify these problems. Addressing these gaps requires a concerted effort to develop integrated treatment models that consider the multifactorial nature of CPP in a multidisciplinary setting.

### AUTHOR CONTRIBUTIONS

Gernot Hudelist and Tina Tellum: Creation and design of the commentary. All authors: Literature search, writing and editing the manuscript equally, approval of the final version.

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