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ORIGINAL RESEARCH

Chronic Pelvic Pain Syndrome in Women: Clinical Covariates and Comorbidity Patterns

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ABSTRACT

***Introduction*:** Chronic pelvic pain syndrome (CPPS) in women is a debilitating condition with a high prevalence (5–25%), yet its etiology remains unclear. This prospective observational study aimed to identify clinical and medical history covariates associated with CPPS to elucidate potential pathophysiological mechanisms.

***Methods*:** A total of 225 women were evaluated in a gynecological pain clinic in Germany, including 41 patients with CPPS (≥ 6 months of lower abdominal pain) and 184 control patients undergoing routine gynecological screening. Exclusion criteria included pregnancy, pelvic malignancy, acute pelvic inflammation, and abnormal uterine bleeding. Covariates were assessed through structured clinical history and physical examination.

***Results*:** Significant associations with CPPS were observed for prior pelvic surgery (72% vs. 45%, $p = 0.003$), bowel constipation (37% vs. 11%, $p = 0.002$), history of endometriosis (33% vs. 10%, $p = 0.043$), and prior trauma (27% vs. 11%, $p = 0.013$). In contrast, there were no significant differences in rates of depression ($p = 0.376$), use of psychopharmaceuticals ($p = 0.757$), pelvic

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floor abnormalities ($p=0.503$), uterine retroversion ($p=0.330$), or pelvic congestion (p=0.455). Dysmenorrhea (59% vs. 42%) and vulvar pain (31% vs. 8%) were more frequent in the CPPS group, though not statistically significant. No differences were found in delivery mode, use of intrauterine devices, analgesics, hormonal replacement therapy, and other medications, or comorbidities such as diabetes, thyroid disease, hypertension, other pain diseases, or musculoskeletal disorders.

Conclusions: CPPS was not associated with several commonly suspected cofactors, including psychosomatic factors, pelvic congestion, or pelvic floor dysfunction. The findings suggest the existence of two subgroups of CPPS, the endometriosis-associated type and the neurovegetative type, associated with prior pelvic surgery, constipation, and trauma. This concept allows for the development of new targeted therapeutic strategies to successfully treat CPPS.

Keywords: Adnexal tenderness; Cofactors; Gynecological pain; Lower abdominal pain; Pelvic congestion; Pelvic floor hypertension; Uterovaginal plexus; Pelvipathy

Key Summary Points

Why carry out this study?

Chronic pelvic pain syndrome (CPPS) is a burden for many women, has a high prevalence, and poses a significant financial strain on healthcare systems.

What did the study ask?/What was the hypothesis of the study?

This prospective observational study investigated the covariates of patients with CPPS compared to controls to detect potential risk factors and possible pathophysiological mechanisms of the disease.

We found a high correlation of CPPS with bowel constipation, a history of endometriosis, or previous gynecological surgery, whereas the rate of risk factors previously suspected, such as depression, pelvic hypertension, or pelvic congestion, was not elevated.

What has been learned from the study?

According to our findings, the chronic pelvic pain syndrome has two subgroups, an endometriosis-related type and a predominantly neurovegetative type linked with other functional disorders.

The absence of muscular hypertension or pelvic congestion suggests that musculoskeletal and angiological factors do not contribute to the development of CPPS.

INTRODUCTION

Unresolved Complexities of CPPS

The chronic pelvic pain syndrome (CPPS) in women is defined as lower abdominal pain without an obvious origin for more than 6 months [1–4]. With a prevalence ranging from 5 to 25% across different populations [5], CPPS represents a significant clinical burden that imposes substantial healthcare costs and profoundly impacts quality of life [6, 7]. The condition affects women across all age groups, often leading to repeated medical consultations, extensive diagnostic workups, and therapeutic interventions with variable success rates [8, 9].

The concept of neurovegetative dysregulation, historically termed "parametropathia spastica", proposes that elevated sympathetic tone and autonomic dysfunction underlie the condition [10–13]. Additionally, vascular theories have gained attention, with some researchers proposing pelvic congestion syndrome as a primary mechanism, attributing symptoms to venous insufficiency and parauterine varicosities [4, 14–18]. Pelvic floor hypertension was also suspected to cause CPPS [19–22]. However, empirical support for these proposed mechanisms has been inconsistent across studies, with

weak associations reported for most putative risk factors, leaving the etiology of CPPS largely unresolved.

The heterogeneous nature of CPPS presentation suggests the condition may encompass multiple distinct pathophysiological subtypes rather than representing a single disease entity [23]. Previous investigations have yielded conflicting results regarding potential risk factors and associated comorbidities, including psychological factors [1, 3, 24–27], anatomical variations, hormonal influences, and inflammatory processes. This inconsistency highlights the critical need for systematic covariate analysis to identify clinically meaningful subgroups and establish evidence-based diagnostic and therapeutic approaches.

Furthermore, the substantial overlap between CPPS and other chronic pain conditions, including endometriosis, irritable bowel syndrome [28], and vulvodynia, suggests shared pathophysiological mechanisms that remain incompletely characterized. Understanding these relationships is essential for developing targeted therapeutic strategies and avoiding ineffective treatments that may perpetuate patient suffering and healthcare resource utilization.

Aim of the Study

In this study, we defined chronic pelvic pain (CPP) as a symptom and chronic pelvic pain syndrome (CPPS) as a clinical diagnosis characterized by persistent pelvic pain without an identifiable organic cause. We hypothesized that systematic identification of clinical, demographic, and medical history covariates would elucidate distinct pathophysiological mechanisms underlying this debilitating condition.

Our primary aim was to identify potential risk factors and pathophysiological associations through a comprehensive comparison of clinical covariates between women with CPPS and asymptomatic controls, with the goal of both enhancing diagnostic strategies and therapeutic interventions.

METHODS

Patients

Between July 2018 and December 2020, a total of 225 consecutive non-pregnant female patients aged 18 years and older were examined at an obstetrics/gynecology and chronic pain clinic in Karlsruhe, Germany. All bimanual gynecological examinations were conducted by the same experienced gynecologist to ensure consistency. The study cohort included two distinct groups: 41 patients presenting with chronic pelvic pain (CPPS group) and 184 asymptomatic individuals undergoing routine preventive examinations (control group).

Inclusion Criteria

Inclusion criteria were admittance for preventive Pap smear or other preventive reasons without complaints, such as control of contraceptives, or patients admitted for lower abdominal pain lasting ≥ 6 months. The data evaluation was conducted independently of treatment and did not influence medical procedures.

Exclusion Criteria

Patients were excluded from data evaluation for the following reasons: Pregnancy, chronic neurological diseases with loss of neuronal sensitivity (e.g. multiple sclerosis), systemic inflammatory disease, acute complaints ≤ 6 weeks, such as bleeding, acute pain disorders such as acute abdominal pain, acute vaginitis, or cystitis, for chemotherapy ≤ 3 months, malignant pelvic tumors, and ≤ 3 months following lower abdominal surgery, and the impossibility of a full gynecological examination (as in upper vaginal stenosis). Patients with menopause, genital atrophy, or vulvodynia were not excluded, altogether resulting in $N = 225$ patients, 184 without and 41 with chronic lower abdominal pain.

Physical Examination

Physical examination was performed according to standard operating procedures of the clinic involving inspection of vulva, vagina, and uterine cervix, vaginal smear for bedside microbiome assessment, pH, and hormonal cell analysis, bimanual palpation of the pelvic organs, and transvaginal ultrasound. For pelvic floor assessment, functional classification based on palpation of the muscles of the pelvic floor during contraction (modified 5-digit Oxford pelvic score) [29, 30] was performed using a five-digit scale: 0 = no innervation possible, 1 = weak innervation, 2 = muscle strength below average, 3 = normal average muscle innervation, and 4 = muscle power above normal average. Vaginal tension was assessed on a three-digit scale: 0 = no tension (vaginal laxity), 1 = normal tension, 2 = elevated, rigid vaginal tension [8]. Cervical motion tenderness was examined in all patients using the three-digit scale described in [28]. "Pelvic congestion" was defined as adnexal varicosis of the respective side of the uterus when two or more parauterine venous vessels of ≥ 5 mm diameter were found in a transvaginal ultrasound examination.

Medical History

All participants underwent a comprehensive clinical assessment to exclude organic pathology or severe psychiatric conditions as potential causes of chronic pelvic pain. Data on body weight, height, body mass index (BMI), and age were obtained either from patient records or measured during the initial clinical examination. Additional information—such as current medication use, comorbidities, smoking status, parity, number of cesarean sections, history of miscarriage, and exposure to psychological or physical trauma was collected through structured patient interviews as part of the standard patient care of this clinic. All patients had the possibility to not answer questions. Patients gave their informed consent about the pseudonymized data evaluation for all data, including psychological issues.

A trauma history was recorded if the patient reported having experienced a severe psychological or physical event that had a lasting impact on her life. Due to the real-world observational nature of the study, standardized questionnaires for trauma or depression were not employed. "Depression" was identified in accordance with ICD-10 criteria (F32.9) and recorded if the patient reported being under psychiatric care or using psychopharmaceuticals at the time of evaluation.

For this study, "gynecological surgery" was defined as any prior cervical conization, laparoscopy, hysterectomy, curettage, adnexal surgery, or similar procedures. "Lower abdominal non-gynecological surgery" included appendectomy, hernia repair, and diagnostic or therapeutic laparoscopy for non-gynecologic indications. A positive history of endometriosis was recorded only if the diagnosis had been laparoscopically confirmed. "Dysmenorrhea" was defined as a history of painful menstrual periods lasting at least 6 months at any point in the patient's lifetime.

Long-term medication use was noted if a drug such as oral contraceptives, hormone replacement therapy, intrauterine hormonal devices, analgesics, thyroxine or other medication had been taken continuously for at least 3 months. Nicotine use was classified as abuse if the patient smoked the equivalent of one or more packs of cigarettes per week. "Dental disease" was defined as the presence of one or more acutely infected teeth or evidence of apical osteitis on radiographic imaging following root canal treatment.

Statistical Analysis

All analyses were done using statistical software R version ≥ 4.4.0. (R-Foundation for Statistical Computing, Vienna, Austria). Demographic variables of the patients were described as frequencies and percentages for categorical variables, and as means and standard deviations or median and range for continuous variables. Differences between subgroups were evaluated using chi-squared tests or Fisher's exact tests, as appropriate, for categorical variables, while *t* tests were performed for evaluating continuous variables. Furthermore, to compensate for possible bias caused by the age variable, age-adjusted *p* values were calculated post hoc by matching

the age ranges of the two cohorts and excluding patients who did not match the age range.

Due to the exploratory character of the study, no missing data was imputed. p values given here have a descriptive meaning and were not adjusted for multiplicity. p values < 0.05 were labeled as statistically significant.

Ethical Approval

This study was performed in accordance with the Helsinki Declaration of 1964 and its later amendments. All patients gave their informed consent for participating in the study, pseudonymized data evaluation, and publication. The structured interviews to collect the patients' medical history were administered as part of our standard procedures in patient's care. The study was approved by the ethical committee of the University Hospital Heidelberg (approval number 487/2011).

RESULTS

Patients

In this prospective observational study, 225 patients at first examination were included in the evaluation; 184 of them visited a doctor for a preventive annual Pap smear or other preventive reasons, such as contraceptive controls, whereas 41 patients sought medical advice for chronic pelvic pain (CPP) for ≥ 6 months. The patients' flow chart is shown in Fig. 1. Patients' average age was 57.6 ± 14.4 years; average body mass index (BMI) was 23.2 ± 4.0 kg/m^2.

Biometric Data and Gynecological History

Patients in the control group were older ($p = 0.001$) and had a 1.1 kg/m^2 higher BMI on average ($p = 0.046$) (Table 1). To estimate the possible bias due to this age difference, we additionally calculated the differences in an age-adjusted model. This second set of p values is depicted in all tables in a column named "p value age-adjusted". In this calculation, the BMI difference of the two groups was correlated with age but not an independent covariate ($p_{\text{adjusted}} = 0.236$).

The average duration of chronic pelvic pain on admission in the CPP group was 111 months or 9.3 years (median 66 months), with a range of 6.4–364 months (0.53–30.3 years).

The rate of known endometriosis was significantly higher in the CPP group (33.3% vs. 10.7%; $p = 0.043$). The rate of patients who had one or several gynecological surgeries was significantly higher in CPP patients than in the control group (73% vs. 46%, $p < 0.001$), see Table 1 and Fig. 2. In contrast, the rate and duration of dysmenorrhea were not significantly different between the two groups.

For the covariates: history of endometriosis, previous gynecological surgery, previous conscious trauma, and bowel constipation the difference to controls also remained significant after adjusting for age difference (Table 1).

Gynecological Examination

Upon gynecological examination, we did not find statistically significant differences in almost all variables between CPPS and control patients, see Table 2 and Fig. 3. We found a difference in vaginal microbiome, with more *lactobacilli* than in control patients ($p = 0.015$), and in the vaginal pH, which was lower in CPPS patients ($p < 0.001$). However, in the age-adjusted calculation model, none of these differences reached a significance level of $p < 0.05$ (Table 2). Pelvic congestion, described as ultrasound adnexal varicosis, was not elevated in CPPS patients ($p = 0.242$ and 0.756, Table 2). Patients in the CPPS group had an even lower rate of pelvic congestion than the control patients, as shown by the lower incidence of adnexal varicosis on both parauterine sides. Paracervical tenderness on gynecological examination was significantly higher in the CPPS group (data not shown, see our previous publication on paracervical tenderness [29]).

Pre-existing Diseases

Patients suffering from CPP complained far more frequently of bowel constipation than the controls (36 vs. 11%, $p = 0.008$), see Table 3.

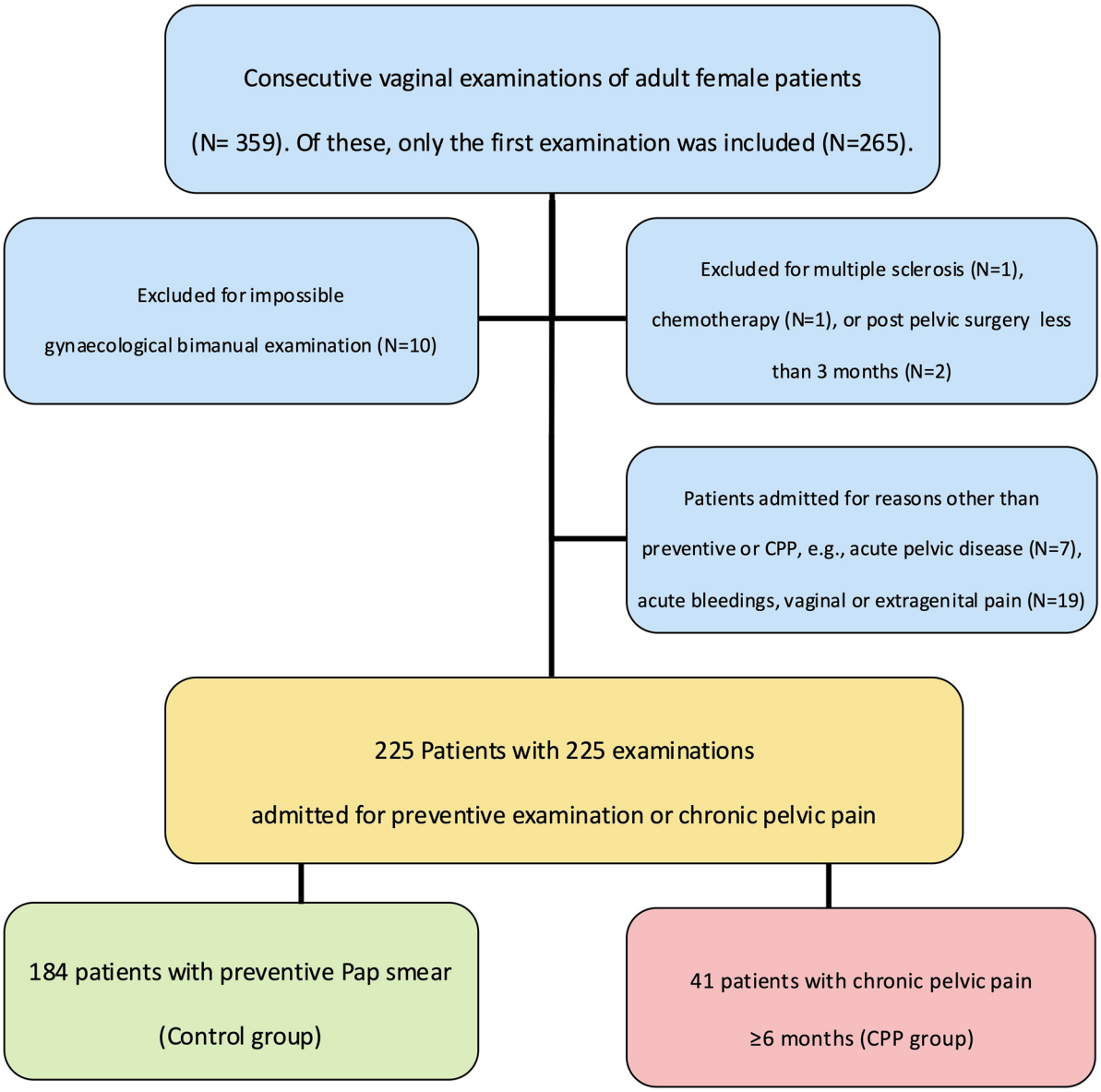

Fig. 1 Patient selection according to the STARD criteria (Standards for Reporting of Diagnostic Accuracy) [25]. All patients were consecutively recruited and registered between July 2018 and December 2020. *Yellow*: all patients included in the data evaluation; *green*: controls, and *red*: patients suffering from chronic pelvic pain (CPP)

Bowel constipation with a threefold higher rate was a remarkable and unexpected covariate of CPPS (37% vs. 11%, $p=0.002$), which also remained constant in the age-adjusted calculation ($p=0.004$). We found a higher incidence of vulvodynia in CPPS patients, defined as vulvar pain of ≥ 6 months (31% vs. 8%, $p=0.011$). However, in the age-adjusted calculation, this difference did not reach statistical significance.

The rate of other diseases, such as thyroid diseases, hypertension, diabetes, musculoskeletal disorders, and different pain disorders (e.g., headache, coccygodynia, and fibromyalgia) were not different between the two groups (Table 3). None of the patients suffered from diabetic neuropathy. In contrast to depression ($p=0.376$), trauma history was significantly higher in the CPPS group. The distribution of

Table 1 Biometric factors and gynecological history of patients with chronic pelvic pain (CPP compared to patients with preventive examination as asymptomatic controls

	CPPS ($N=41$)	Controls ($N=184$)	All ($N=225$)	p value, all	p value, age-adjusted
Biometric factors					
Age (years)	49.6 ± 12.9	59.4 ± 14.1	57.6 ± 14.4	< **0.001****	0.118
BMI (kg/m^2)	22.1 ± 3.8	23.5 ± 4.0	23.2 ± 4.0	**0.046***	0.236
Gynecological history					
Parity > 0	29/41 (70.7%)	113/180 (62.8%)	142/221 (64.3%)	0.372	0.322
Cesarean section (yes)	10/41 (24.4%)	34/177 (19.2%)	44/218 (20.2%)	0.517	0.902
Vaginal surgical delivery	4/37 (10.8%)	6/130 (4.6%)	10/167 (6.0%)	0.235	0.165
History of dysmenorrhea	19/32 (59.4%)	48/114 (42.1%)	67/146 (45.9%)	0.090	0.497
Dysmenorrhea, duration (years)	13.0 ± 8.7	13.9 ± 11.2	13.6 ± 10.4	0.745	0.860
Known endometriosis	5/15 (33.3%)	8/75 (10.7%)	13/90 (14.4%)	**0.043***	**0.024***
History of any gynecological surgery	29/40 (72.5%)	81/177 (45.8%)	110/217 (50.7%)	**0.003****	**0.009****
Status post hysterectomy	8/41 (19.5%)	18/184 (9.8%)	26/225 (11.6%)	0.102	0.107
Non-gynecological lower abdominal surgery	10/39 (25.6%)	47/172 (27.3%)	57/211 (27.0%)	0.999	0.651

abdomin. abdominal, *age adjusted* the p values yielded after adjusting for age of the control group to the CPPS patient group, see M&M section, *BMI* body mass index, *CPPS* patients with chronic pelvic pain > 6 months, *gyn* gynecological, *HRT* hormonal replacement therapy, *hx* medical history, *N* number of individuals, *tx* therapy

Differences to the total number of 184 and 41 are due to missing values

*$p < 0.05$, **$p < 0.01$

all other concomitant diseases was similar for both groups (Table 3).

Medication

None of the medications taken by the patients was significantly different between CPPS patients and controls (Table 4). In detail, neither the frequent use of analgesics, nor the usage of psychopharmaceutic drugs was more frequent in CPPS patients. The rate of vaginal estriol (E3) therapy was higher in CPPS patients (22% vs. 11%). After age adjustment, this difference became significant ($p=0.029$). Nicotine abuse (≥ 20 cigarettes per week) was evenly distributed between the two groups ($p=0.810$) and, therefore, does not exhibit an obvious impact on CPPS.

DISCUSSION

Principal Findings: Covariates of Chronic Pelvic Pain Syndrome

This prospective analysis identified several clinical covariates significantly associated with CPPS, including prior gynecological surgery (72% vs. 45%, $p=0.003$), confirmed history of endometriosis (33% vs. 11%, $p=0.043$), and bowel constipation (37% vs. 11%, $p=0.002$). These findings suggest distinct pathophysiological mechanisms

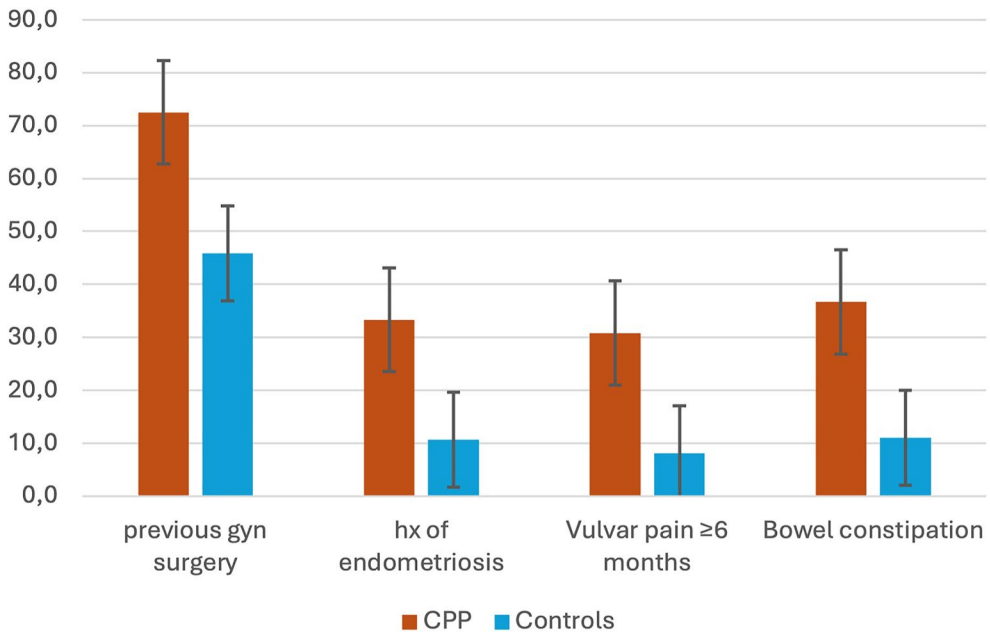

Fig. 2 Covariates of the chronic pelvic pain syndrome (CPPS): Examination findings with a significant difference between patients with chronic pelvic pain (CPP, *red*) and control patients (*blue*): Constipation, number of gynecological surgeries, and a history of endometriosis. Vulvar pain was found in 30% of the CPPS vs. 8% in the control patients, but this difference was in part due to the age of the patients. *gyn* gynecological, *hx* history, * = significance on a $p < 0.05$ level. *y-axis*: percentage of the individuals

defining different CPPS phenotypes. Several commonly proposed etiological factors showed no association with CPPS in our cohort, thus challenging prevailing assumptions about disease mechanisms.

Evidence Questioning Established Theories

Pelvic Floor Dysfunction Hypothesis

Our data provides compelling evidence against pelvic floor muscle hypertension as a primary mechanism in CPPS, contradicting extensive literature and supporting myofascial theories [19–22]. The myofascial pelvic pain syndrome concept, as described by Kotarinos [21] and Spitznagle and Robinson [22], proposes that pelvic floor muscle dysfunction is central to CPPS pathophysiology. However, our pelvic floor assessment using the validated Oxford scale [30] showed no differences between CPPS and control patients ($p = 0.503$), and vaginal tension measurements were similarly unremarkable.

This finding is significant because it challenges a substantial body of literature that has influenced current treatment approaches. While Proulx et al. [31] found increased lumbopelvic muscle stiffness in CPPS patients, they questioned whether muscular abnormalities represent causative factors or secondary consequences of chronic pain, supporting our findings that pelvic floor dysfunction may not be a primary cause.

Pelvic Congestion

Furthermore, our data does not support the theory of a pelvic congestion syndrome [4, 14–18]. This theory, first proposed by Beard et al. [16] and subsequently supported by studies from Borghi and Dell'Atti [17] and others, attributes chronic pelvic pain to pelvic venous congestion and varicosities. However, our data showed adnexal varicosis was actually lower in CPPS

Table 2 Gynecological examination findings in two groups of patients: chronic pelvic pain (CPP) and asymptomatic controls

	CPPS ($N = 41$)	Controls ($N = 184$)	All ($N = 225$)	p value, all	p value, age-adjusted
Gynecological examination					
Vaginal pH (average value)	4.61 ± 0.94	5.3 ± 1.3	5.2 ± 1.3	**< 0.001****	0.082
Vulvar pain	8/27 (30.8%)	5/62 (8.1%)	13 (14.8%)	**0.011***	0.148
Vaginal microbiome: presence of lactobacilli	24/39 (61.5%)	79/180 (43.9%)	103/219 (47.0%)	**0.015***	0.106
Adnexal varicosis right side (1)	0/22 (0.0%)	10/116 (8.6%)	10/138 (7.3%)	0.242	0.142
Vaginal tension on the left side (scale 0–2)	0.667	0.195	0.264	0.318	0.125
Uterine retroversion	2/32 (6.3%)	19/143 (13.3%)	21/175 (12.0%)	0.330	0.120
Vaginal tension on the right side (scale 0–2)	0.571	0.203	0.257	0.455	0.285
Pelvic floor quality (scale 0–4)	2.65 ± 0.66	2.56 ± 0.61	2.57 ± 0.62	0.503	0.831
Uterine fibroids > 25 ml	0/41 (0.0%)	5/184 (2.7%)	5/225 (2.2%)	0.588	0.798
Adnexal varicosis left side (1)	1/21 (4.7%)	10/115 (8.7%)	11/136 (8.1%)	0.756	0.508
Ovarian cysts > 25 ml	0/41 (0.0%)	3/184 (1.6%)	3/225 (1.3%)	0.999	0.999

Gynecological findings are ordered along with their probability to correlate with CPPS according to their p values

Age adjusted the p values yielded after adjusting for age of the control group to the CPPS patient group, see M&M section, *CPPS* patients with chronic pelvic pain > 6 months, *N* number of individuals, *pH* pondus hydrogenii, acidity of the vagina. (1) for definition, see Material section. (2) vaginal tension on a 3-digit scale, see Material section. Differences to the total number of 184 and 41 are due to missing values. *$p < 0.05$, **$p < 0.01$

patients than in controls (0–7% vs. 10–12%). This finding challenges the rationale for interventional procedures targeting pelvic veins and suggests that pelvic congestion may represent incidental, age-dependent venous insufficiency rather than a pain-generating mechanism.

Factors Without Obvious Correlation with CPPS

Vaginal Microbiome

The vaginal microbiome and vaginal pH were more dominated by the presence of *lactobacilli* than in the control group. However, in the age-adjusted model calculation, both factors were no more significant and therefore, could not be attributed to CPPS. We conclude that vaginal microbiome and pH are more likely correlated to age rather than covariates of the disease. All other gynecological findings were similar in the CPPS group compared to controls.

Uterine Abnormalities or Genital Tumors

Uterine retroversion does not seem to play a role in CPPS, as it was assumed before [32]. Furthermore, the rate of ovarian cysts and fibroids > 25 ml volume was not statistically different between CPPS and control patients. Thus,

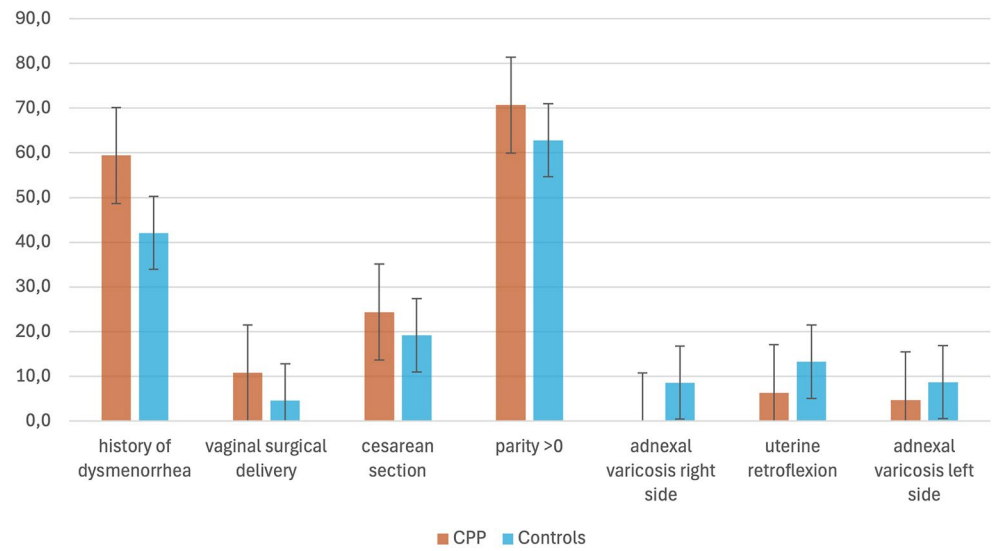

Fig. 3 Non-significant covariates of chronic pelvic pain syndrome (CPPS). A trend towards a higher rate of dysmenorrhea, vaginal surgical delivery, parity, and the number of cesarean sections was seen in CPPS patients (*red*), however, the difference to the control group (*blue*) was not significant. Gynecological findings of pelvic congestion (measured from ultrasound adnexal varicosis), and uterine retroversion were not found in the CPPS group, but even more frequently in the control group. *y-axis*: percentage of the individuals

CPPS does not seem to be induced by or correlated with these morphological findings.

Depression

Contrary to influential studies of several working groups [24, 25, 27, 33] who reported high rates of depression and psychological trauma in CPPS, we only found a weak but not significant difference in depression rates between the groups (12.2% vs. 8.2%, $p=0.376$). This questions the widespread assumption that psychological factors are the primary drivers of CPPS. However, due to methodological constraints, the results should be treated with caution.

Diabetes, Thyroid Disease, and Neuropathies

In our investigation, the rate of diabetes or hyper- or hypothyroidism was similar in both groups. CPPS seems to be independent from these diseases. The rate of other neuropathic diseases was not increased, and there was no patient with diabetic neuropathy, both of which argue against a systemic neuropathic factor in the pathogenesis of CPPS.

Dental and Other Diseases

There is a weak but not significant correlation with a lower dental health status in CPPS patients (5% vs. 0.5%). This putative correlation needs further investigation. Dental diseases are correlated with gynecological disorders such as endometriosis [34] and may also be associated with CPPS.

Among all other co-existing diseases known in the patients, none of them showed any correlation with CPPS. Furthermore, even smoking did not seem to play a role in the pathogenesis of CPPS.

Medication and CPP

Hormones

There was no correlation of CPPS with estradiol medication, oral contraceptives, or the use levonorgestrel intrauterine devices. Only the rate of vaginal usage of estriol (E3) was higher in the CPPS group, independent of age, which

Table 3 Non-gynecological diseases in two groups of patients: chronic pelvic pain (CPP) and asymptomatic controls

	CPPS ($N = 41$)	Controls ($N = 184$)	All ($N = 225$)	p value, all	p value, age adjusted
Diseases					
Bowel constipation	11/30 (36.7%)	15/136 (11.0%)	26/166 (15.7%)	**0.002****	**0.004****
Trauma history	11/41 (26.8%)	20/184 (10.8%)	31/225 (13.8%)	**0.012***	**0.005****
Dental diseases	2/41 (4.9%)	1/184 (0.5%)	3/225 (1.3%)	0.086	0.350
Fibromyalgia	1/31 (3.2%)	0/158 (0%)	1/225 (0.5%)	0.182	0.503
Neuralgia (other locations)	1/41 (2.4%)	0/184 (0.0%)	1/225 (0.4%)	0.182	0.503
Arthrosis	6/41 (14.6%)	15/184 (8.2%)	21/225 (9.3%)	0.232	0.083
Depression	5/41 (12.2%)	15/184 (8.2%)	20/225 (8.9%)	0.376	0.350
Other gastrointestinal disorders	1/41 /2.4%)	12/184 (6.5%)	13/225 (5.8%)	0.471	0.655
Hypertension	6/41 (14.6%)	37/184 (20.1%)	43/225 (19.1%)	0.514	0.739
Diabetes	0/41 (0%)	5/184 (2.7%)	5/225 (2.2%)	0.588	0.588
Thyroid disease	7/41 (17.0%)	27/184 (14.7%)	34/225 (15.1%)	0.638	0.598
Coccygodynia	0/41 (0%)	2/184 (1.1%)	2/225 (0.9%)	0.999	0.798
Allergy	2/41 (4.9%)	12/184 (6.5%)	14/225 (6.2%)	0.999	0.903
Headache, migraine	1/41 (2.4%)	6/184 (3.3%)	7/225 (3.1%)	0.999	0.999
Diabetic neuropathy	0%	0%	0%	–	–

Diseases are ordered along with their probability to correlate with CPPS according to their p values, independent from the location

Age adjusted the p values yielded after adjusting the age of the control group to the CPPS patient group, see M&M section, *CPP* patients with chronic pelvic pain > 6 months, *N* number of individuals

Differences to the total numbers of 184 and 41 are due to missing values

*$p < 0.05$, **$p < 0.01$

may be rather due to a therapeutic application in the context of the therapy than a covariate of the disease. This assumption is supported by the fact that in the age-adjusted calculation, the difference in vaginal E3 application reached a significance level of $p < 0.05$. Taking all these into account, we conclude that CPPS is not influenced by hormonal factors.

Analgesics

Do analgetic drugs influence the CPPS? The use of analgesics or corticoids in the CPPS group was approximately identical to that in the control group. We had expected a higher rate due to the persistent complaints of the patients. Our findings argue against a potential concealing effect of analgesics on chronic pelvic pain.

Other Drugs. We found no correlation with the intake of any of the other medications and conclude that none of them are covariates of the disease.

Table 4 Medication usage (analgesics and drugs taken for other reasons) in two groups of patients: chronic pelvic pain (CPP) and asymptomatic controls

Medication	CPPS ($N = 41$)	Controls ($N = 184$)	All ($N = 225$)	p value, all	p value, age-adjusted
Statins	0/41 (0%)	15/184 (8.2%)	15/225 (6.7%)	0.079	0.344
Vaginal estriol (E3) therapy	8/35 (22.9%)	19/167 (11.4%)	27/202(13.4%)	0.097	**0.029***
Oral contraceptives	4/40 (10.0%)	10/184 (5.4%)	14/224 (6.3%)	0.284	0.438
Anticoagulants	1/30 (3.3%)	17/150 (11.3%)	18/180 (10.0%)	0.240	0.999
Anti-hypertensive drugs	3/41 (7.3%)	26/184 (14.1%)	29/225 (12.9%)	0.309	0.930
Intrauterine device	6/40 (15.0%)	18/182 (9.9%)	24/222 (10.8%)	0.397	0.865
Corticoids	0/41 (0%)	5/184 (2.7%)	5/225 (2.2%)	0.588	0.798
Antacids (pantoprazole, etc.)	0/41 (0%)	5/184 (2.7%)	5/225 (2.2%)	0.588	0.999
Analgesics	1/41 (2.4%)	9/184 (4.9%)	10/225 (4.4%)	0.694	0.999
Psychopharmaceuticals	4/41 (9.8%)	15/184 (8.2%)	19/225 (8.4%)	0.757	0.999
Nicotine abuse (> 1 pack per week)	4/40 (10.0%)	19/155 (12.3%)	23/195 (11.8%)	0.810	0.486
Hormonal replacement treatment	6/37 (16.2%)	37/178 (20.8%)	43/179 (20.0%)	0.939	0.999
Beta blockers	5/41 (12.2%)	23/184 (12.5%)	28/225 (12.4%)	0.999	0.243
Thyroxine	7/41 (17.1%)	32/184 (17.4%)	39/225 (17.3%)	0.999	0.491

Drugs are ordered along with their probability to correlate with CPPS according to their p values, independent of their indication. Nicotine usage is listed here as a "drug"

Age-adjusted the p values yielded after adjusting the age of the control group to the CPPS patient group, see M&M section, *CPP* patients with chronic pelvic pain > 6 months, *N* number of individuals

Differences to the total numbers of 184 and 41 are due to missing values

*$p < 0.05$

Evidence for Different Pathophysiological Mechanisms of CPPS

Concomitant Vulvodynia

The rate of vulvar pain of ≥ 6 months (vulvodynia) in CPPS patients was about 3.5 times higher than in the control group. Vulvodynia was initially thought to be correlated with CPPS in the past [26]. Our data support this view (30.8% vs. 8.1%). However, after the age-adjusted calculation, this correlation was no longer significant for CPPS. Although vulvodynia seems to have a different risk profile, as demonstrated by its covariates [35], some common characteristics with CPPS may exist.

Endometriosis and Dysmenorrhea

Our confirmation of significantly higher endometriosis rates in CPPS patients supports inflammatory theories of pain generation and aligns with extensive literature linking endometriosis to chronic pelvic pain [36, 37]. The threefold higher rate in our CPPS cohort is consistent with Zondervan et al.'s [37] population studies showing 10% prevalence in reproductive-age women. Interestingly, the lack of difference in

dysmenorrhea duration between groups suggests that residual inflammatory processes, rather than active endometriotic symptoms, may drive persistent pain. This may explain why some patients continue to experience pain despite apparently successful endometriosis treatment.

Trauma History

Our trauma history findings (27% vs. 11%, $p=0.013$) partially support psychological theories [24, 25, 27, 33], suggesting that trauma may contribute to CPPS development in a subset of patients. This finding was independent of age and is in line with former research assuming a post-traumatic covariate in CPPS [24, 25, 27, 33]. However, in this analysis of real-world data, our data relied on self-reported information gathered as a part of a standard anamnesis without conducting a validated trauma questionnaire. Although the information was collected in the same way in both groups, the rate of unknown trauma may have been different. The findings indicate that post-traumatic factors may be relevant for some patients but are not universal CPPS characteristics.

Surgical Trauma and Neurogenic Sensitization

The significantly higher rate of prior gynecological surgery in CPPS patients providing strong support for neuropathic pain theories [38]. Kronenberg et al. [38] demonstrated that nerve injury following pelvic surgery can lead to chronic pain through peripheral and central sensitization. Our finding that 72% of CPPS patients had a history of gynecological surgery, compared to 45% of controls, suggests that iatrogenic nerve irritation may be an underrecognized CPPS mechanism.

The specificity for gynecological versus general abdominal surgery (no significant difference in appendectomy rates) supports the view of an anatomically specific neural pathway involvement. This finding has important implications for surgical decision-making in CPPS patients and suggests that additional pelvic procedures should be approached with caution.

Neurovegetative Dysfunction and Visceral Hypersensitivity

The remarkable association with bowel constipation (37% vs. 11%, $p=0.002$) provides strong support for autonomic dysfunction theories and reasserts the relevance of the historical concept of "parametropathia spastica" [10–13, 39, 40]. This finding aligns with modern understanding of visceral hypersensitivity in chronic pain conditions [28, 39, 41].

The magnitude of this association was unexpected and suggests that autonomic nervous system dysfunction may be a unifying mechanism in non-endometriosis CPPS. This connects CPPS to broader concepts of functional disorders and visceral pain syndromes [39], indicating that gastrointestinal symptoms deserve greater attention in CPPS evaluation.

Clinical Implications and Proposed Phenotypes

Based on our covariate analysis, we propose two distinct CPPS subtypes with different therapeutic implications:

Endometriosis-Associated CPPS

This subtype, representing approximately one-third of our CPPS patients, is characterized by a confirmed endometriosis history. The absence of active dysmenorrhea in many patients suggests that residual inflammatory processes drive pain persistence, requiring targeted anti-inflammatory or neuromodulatory approaches beyond standard hormonal and surgical treatments.

Neurovegetative CPPS

This subtype is characterized by constipation, gyn-surgical history, and absence of endometriosis, suggesting primary autonomic dysfunction with visceral hypersensitivity [39, 40]. These patients may benefit from treatments

targeting autonomic function [28, 38, 42] rather than treating structural abnormalities.

Research Implications

Our findings challenge current diagnostic and treatment paradigms for CPPS. The lack of association with pelvic floor dysfunction and vascular congestion questions the utility of extensive assessments in routine CPPS evaluation. Instead, comprehensive surgical history and gastrointestinal symptom assessment may be diagnostically more relevant.

The surgical trauma association raises important questions about repeat interventions in CPPS patients. Future research should examine whether surgical approaches perpetuate symptoms through continued neural injury. Additionally, the proposed phenotypic classification requires validation in larger cohorts with standardized assessment tools.

Limitations and Strengths

Monocenter Design and Small Group Size

We observed a limited number of patients in a single clinic. However, this prospective, descriptive data reveals first insights into possible pathophysiological mechanisms of CPPS. The consistency provided by a single experienced gynecologist strengthens methodological clarity, but at the same time introduces a lack of objectivity. Furthermore, the number of patients with CPPS was significantly lower than the number of control patients. This was caused by the fact that CPPS patients receive intensive therapy with several appointments in the respective period, whereas preventive patients usually come only once a year. These limitations will be overcome by future multicenter studies based on the findings of this pilot study.

Observational Design

This study is observational in nature, and the findings should be interpreted as exploratory and hypothesis-generating rather than confirmatory.

Age and BMI Difference

CPPS patients were significantly younger and had a lower BMI than the patients in the control group. We therefore re-calculated all covariates in an age-adjusted approach. The main covariates of CPPS—endometriosis, bowel constipation, and the number of previous gynecological surgeries—remained as significant covariates with this calculation as well, whereas BMI, vaginal pH, and the vaginal microbiome were revealed to be age-dependent and are therefore not found to be significant covariates of CPPS. Furthermore, some other factors could not be attributed to CPPS. There were differences regarding some other potential factors between the two groups in both the whole cohort as well as in the age-adopted model, but these differences were not on a significant level. Thus, even if there were an age difference between the two groups, it seemed to have no influence on the main results of this investigation.

Pelvic Floor and Vaginal Wall Assessment

Pelvic floor assessment was done by a routine screening with palpation of the muscle contraction according to the five-digit modified Oxford scale [29, 30]. This may have induced a certain bias in pelvic floor assessment. However, the examination is well established and has a high inter-rater reproducibility [43], so the bias most likely remained low. Furthermore, the examination was performed by a single, well-experienced gynecologist. We found no difference in the results of CPPS and control patients ($p = 0.503$), with that we can assume that pelvic floor hypo- or hypertension is no relevant covariate of CPPS.

Also, the examination of the vaginal wall tension was done by palpation, not by measurement. Due to this, we cannot exclude bias caused by the palpation technique. Nevertheless, the results in both scales were far from a significant range, which supports the view that

there is no relevant influence of the pelvic floor quality on CPPS.

Missing Data

The database concerning some potential cofactors, such as trauma and fibromyalgia, was weak in our investigation. Due to the observational nature of the study, we were not able to use structured questionnaires in daily routines. Therefore, an underestimation or selection bias cannot be excluded but seems unlikely as the rate of missing data was similar in both groups for all covariates examined.

No Detailed Data on Psychological and Post-traumatic Factors

Some researchers have assumed that psychological factors play an important role in CPPS [1, 3, 24–27, 33]. In a clinic with all-day patient care, a psychological inventory questionnaire was not given out to the patients. Using structured questionnaires for study reasons only would have violated the character of an observational study. However, as the patient's self-reports on prevalence and diagnosis of depression or traumatic experiences were taken similarly by all patients in both groups, a comparison was possible but is limited by a possible under-assessment of these factors.

Conclusions from our data concerning depression and trauma experience as possible cofactors of CPPS should be drawn with caution. Further studies on this important factor should be planned to distinguish between depression as a cause or as a sequela of CPPS.

Due to the design of the study, a possible under-assessment of psychological factors, such as depression and trauma history, may have occurred.

Hidden Endometriosis

Information on the presence or absence of proven endometriosis conducted using laparoscopy was available in only approximately 45% of the patients. However, the positive correlation with a known history of endometriosis suggests endometriosis to be a strong cofactor of CPPS. In contrast, one of the most important covariates of endometriosis, dysmenorrhea, was not different in either group.

Given that endometriosis is a significant potential cause of CPPS, and it affects 10% of women within reproductive ages [37], its detection in a preventive care setting, such as during a Pap smear examination, is crucial. Due to the design of our study, we cannot rule out undiagnosed cases of endometriosis. The even distribution of dysmenorrhea in both groups suggests that active endometriosis is not a main denominator of CPPS. This question should be examined in further studies focusing on patients with CPPS and a proven endometriosis.

Strengths of the Study

The study is based on a prospective observational design. Therefore, there were no dropouts, which could have caused a data selection bias. We examined a relatively large population of 225 individuals, including a control group of 184 individuals. Our data serve as a foundation for further research and randomized studies on risk factors, covariates, and possible therapeutic approaches to the CPPS, which can be performed based on this study.

CONCLUSIONS

This exploratory study identified several covariates significantly associated with CPPS, including prior gynecological surgery, confirmed history of endometriosis, and bowel constipation. These associations suggest that CPPS may encompass distinct phenotypic subtypes with different underlying mechanisms. In some patients, residual endometriotic processes may be associated with pain persistence, while in others, patterns consistent with autonomic dysfunction may be observed.

Our findings generate hypotheses for two potential CPPS subgroups: an endometriosis-associated phenotype and a neurovegetative phenotype characterized by gastrointestinal dysfunction and surgical history. While these observations require validation in larger

controlled studies, they can already provide a novel conceptual framework for understanding CPPS heterogeneity.

We recommend a comprehensive endometriosis evaluation in patients presenting with CPPS. Additionally, in the case where endometriosis is absent, assessment of gastrointestinal symptoms and autonomic function may be clinically relevant. The proposed phenotypic classification warrants further investigation in prospective multicenter studies to confirm these preliminary observations and guide the development of different targeted therapeutic approaches.

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Data Availability. The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Conflict of Interest. Stefan Weinschenk is a member of the scientific board of the German Society of Acupuncture and Neural Therapy (DGfAN). All other authors (Nura Fitnat Topbas Selcuki, Thomas Strowitzki, Axel Gerhardt, Oliver Zivanovic, and Manuel Feisst) declare no conflicts of interest.

Ethical Approval. This study was performed in accordance with the Helsinki Declaration of 1964 and its later amendments. All patients gave their informed consent for participating in the study, pseudonymized data evaluation, and publication. The structured interviews to collect the patients' medical history were administered as part of our standard procedures in patient's care. The study was approved by the ethical committee of the University Hospital Heidelberg (approval number 487/2011).

REFERENCES

1. Speer LM, Mushkbar S, Erbele T. Chronic pelvic pain in women. Am Fam Phys. 2016;93(5):380–7. https://www.ncbi.nlm.nih.gov/pubmed/26926975.

2. Lamvu G, Carrillo J, Ouyang C, Rapkin A. Chronic pelvic pain in women: a review. JAMA. 2021;325(23):2381–91. https://doi.org/10.1001/jama.2021.2631.

3. Meisenheimer ES, Carnevale AM. Chronic pelvic pain in women: evaluation and treatment. Am Fam Phys. 2025;111(3):218–229

4. Balabuszek K, Toborek M, Pietura R. Comprehensive overview of the venous disorder known as pelvic congestion syndrome. Ann Med. 2022;54(1):22–36. https://doi.org/10.1080/07853890.2021.2014556.

5. Ahangari A. Prevalence of chronic pelvic pain among women: an updated review. Pain Phys. 2014;17(2):E141-7. https://www.ncbi.nlm.nih.gov/pubmed/24658485.

6. 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. https://doi.org/10.1016/0029-7844(95)00458-0.

7. Grace V, Zondervan K. Chronic pelvic pain in women in New Zealand: comparative well-being, comorbidity, and impact on work and other activities. Health Care Women Int. 2006;27(7):585–99. https://doi.org/10.1080/07399330600803725.

8. Zondervan KT, Yudkin PL, Vessey MP, et al. Chronic pelvic pain in the community–symptoms, investigations, and diagnoses. Am J Obstet Gynecol. 2001;184(6):1149–55. https://doi.org/10.1067/mob.2001.112904.

9. Engeler DS, Baranowski AP, Dinis-Oliveira P, et al. The 2013 EAU guidelines on chronic pelvic pain: is management of chronic pelvic pain a habit, a philosophy, or a science? 10 years of development. Eur Urol. 2013;64(3):431–9. https://doi.org/10.1016/j.eururo.2013.04.035.

10. Kamocsay D. [Ultrasonic Therapy in Spastic Parametropathy (Martius)]. Rehabilitation (Bonn) 1964;17:77–9. http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=14309202. (**in Ger**).

11. Martius H. Spastische Parametropathie. Z Arztl Fortbildg. 1942;39:289–90.

12. Seese SA. [Neurotherapeutic aspect of parametropathy]. Zentralbl Gynakol. 1961;83:925–7.

13. Dietsch H. [Spastic Parametropathy and Its Treatment]. Med Welt 1963;43:2188–9. http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=14093064. (**in Ger**)

14. Koo S, Fan CM. Pelvic congestion syndrome and pelvic varicosities. Tech Vasc Interv Radiol. 2014;17(2):90–5. https://doi.org/10.1053/j.tvir.2014.02.005.

15. Basile A, Failla G, Gozzo C. Pelvic congestion syndrome. Semin Ultrasound CT MR. 2021;42(1):3–12. https://doi.org/10.1053/j.sult.2020.07.001.

16. Beard RW, Reginald PW, Wadsworth J. Clinical features of women with chronic lower abdominal pain and pelvic congestion. Br J Obstet Gynaecol 1988;95(2):153–61. http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=3349005. (**in Eng**).

17. Borghi C, Dell'Atti L. Pelvic congestion syndrome: the current state of the literature. Arch Gynecol Obstet. 2016;293(2):291–301. https://doi.org/10.1007/s00404-015-3895-7.

18. Herrera-Betancourt AL, Villegas-Echeverri JD, Lopez-Jaramillo JD, Lopez-Isanoa JD, Estrada-Alvarez JM. Sensitivity and specificity of clinical findings for the diagnosis of pelvic congestion syndrome in women with chronic pelvic pain. Phlebology. 2018;33(5):303–8. https://doi.org/10.1177/0268355517702057.

19. Dal Farra F, Aquino A, Tarantino AG, Origo D. Effectiveness of myofascial manual therapies in chronic pelvic pain syndrome: a systematic review and meta-analysis. Int Urogynecol J. 2022;33(11):2963–76. https://doi.org/10.1007/s00192-022-05173-x.

20. Bonder JH, Chi M, Rispoli L. Myofascial pelvic pain and related disorders. Phys Med Rehabil Clin N Am. 2017;28(3):501–15. https://doi.org/10.1016/j.pmr.2017.03.005.

21. Kotarinos R. Myofascial pelvic pain. Curr Pain Headache Rep. 2012;16(5):433–8. https://doi.org/10.1007/s11916-012-0277-8.

22. Spitznagle TM, Robinson CM. Myofascial pelvic pain. Obstet Gynecol Clin North Am. 2014;41(3):409–32. https://doi.org/10.1016/j.ogc.2014.04.003.

23. Nygaard I, Barber MD, Burgio KL, et al. Prevalence of symptomatic pelvic floor disorders in US

women. JAMA. 2008;300(11):1311–6. https://doi.org/10.1001/jama.300.11.1311.

24. Meltzer-Brody S, Leserman J, Zolnoun D, Steege J, Green E, Teich A. Trauma and posttraumatic stress disorder in women with chronic pelvic pain. Obstet Gynecol. 2007;109(4):902–8. https://doi.org/10.1097/01.AOG.0000258296.35538.88.

25. Leserman J, Zolnoun D, Meltzer-Brody S, Lamvu G, Steege JF. Identification of diagnostic subtypes of chronic pelvic pain and how subtypes differ in health status and trauma history. Am J Obstet Gynecol. 2006;195(2):554–60. https://doi.org/10.1016/j.ajog.2006.03.071. (**discussion 560-1**).

26. Reed BD, Haefner HK, Punch MR, Roth RS, Gorenflo DW, Gillespie BW. Psychosocial and sexual functioning in women with vulvodynia and chronic pelvic pain. A comparative evaluation. J Reprod Med 2000;45(8):624–32. https://www.ncbi.nlm.nih.gov/pubmed/10986680.

27. 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. https://doi.org/10.1097/GRF.0000000000000412.

28. Warren JW, Morozov V, Howard FM. Could chronic pelvic pain be a functional somatic syndrome? Am J Obstet Gynecol. 2011;205(3):199e1-205. https://doi.org/10.1016/j.ajog.2011.04.003.

29. Isherwood PJ, Rane A. Comparative assessment of pelvic floor strength using a perineometer and digital examination. BJOG. 2000;107(8):1007–11. https://doi.org/10.1111/j.1471-0528.2000.tb10404.x.

30. Ortiz OC, Nuñez FC. Dynamic assessment of pelvic floor function in women using the intravaginal device test. Int Urogynecol J. 1996;7(6):317–20.

31. 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. https://doi.org/10.1089/jwh.2022.0198.

32. Fauconnier A, Dubuisson JB, Foulot H, et al. Mobile uterine retroversion is associated with dyspareunia and dysmenorrhea in an unselected population of women. Eur J Obstet Gynecol Reprod Biol. 2006;127(2):252–6. https://doi.org/10.1016/j.ejogrb.2005.11.026.

33. 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. https://doi.org/10.2147/JPR.S115065.

34. Agneta MT, D'Albis G, Lorusso L, et al. Endometriosis-associated periodontal disease: a large cohort perspective study. Oral Dis. 2025. https://doi.org/10.1111/odi.70044.

35. Gerhardt A, Feisst M, Strowitzki T, Zivanovic O, Weinschenk S. Vulvodynia and chronic vulvar pain: influencing factors and long-term success after therapeutic local anesthesia (TLA). Pain Ther. 2025. https://doi.org/10.1007/s40122-025-00725-7.

36. Ortiz DD. Chronic pelvic pain in women. Am Fam Physician. 2008;77(11):1535–42.

37. Zondervan KT, Becker CM, Missmer SA. Endometriosis. N Engl J Med. 2020;382(13):1244–56. https://doi.org/10.1056/NEJMra1810764.

38. Kronenberg RM, Ludin SM, Fischer L. Severe case of chronic pelvic pain syndrome: recovery after injection of procaine into the vesicoprostatic plexus-case report and discussion of pathophysiology and mechanisms of action. Case Rep Urol. 2018;2018:9137215. https://doi.org/10.1155/2018/9137215.

39. Brumovsky PR, Gebhart GF. Visceral organ cross-sensitization - an integrated perspective. Auton Neurosci. 2010;153(1–2):106–15. https://doi.org/10.1016/j.autneu.2009.07.006.

40. Weinschenk S, Strowitzki T, Topbas Selcuki NF, Zivanovic O, Gerhardt A, Feisst M. Cervical tenderness (parametropathy) is a diagnostic tool for the chronic pelvic pain syndrome. Pain Ther. 2025. https://doi.org/10.1007/s40122-025-00760-4.

41. Wong MYW, Hebbard G, Gibson PR, Burgell RE. Chronic constipation and abdominal pain: independent or closely interrelated symptoms? J Gastroenterol Hepatol. 2020;35(8):1294–301. https://doi.org/10.1111/jgh.14970.

42. Kapural L, Narouze SN, Janicki TI, Mekhail N. Spinal cord stimulation is an effective treatment for the chronic intractable visceral pelvic pain. Pain Med. 2006;7(5):440–3. https://doi.org/10.1111/j.1526-4637.2006.00165.x.

43. Messelink B, Benson T, Berghmans B, et al. Standardization of terminology of pelvic floor muscle function and dysfunction: report from the pelvic floor clinical assessment group of the International Continence Society. Neurourol Urodyn. 2005;24(4):374–80. https://doi.org/10.1002/nau.20144.