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COMBINED MAGNETOTHERAPY AND ELECTROPHORESIS IN THE MANAGEMENT OF CHRONIC PELVIC PAIN IN WOMEN WITH CHRONIC ENDOMETRITIS: A RANDOMIZED CONTROLLED STUDY

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Abstract Background. Chronic pelvic pain (CPP) is a common reason for gynaecological visits in women of reproductive age, and chronic endometritis (CE) is one of its inflammatory causes. Antibacterial and anti-inflammatory drugs often leave residual pain.

Aim. To evaluate whether adding magnetotherapy and electrophoresis to standard medical treatment improves pain, inflammatory and hormonal markers, clinical symptoms and quality of life in women with CE-associated CPP.

Methods. Eighty women aged 18–40 years with CE and pelvic pain lasting at least six months were treated at the multidisciplinary clinic of Samarkand State Medical University in 2024–2025. They were randomized 1:1 to standard therapy (doxycycline, metronidazole, ibuprofen, drotaverine and vitamin E; n = 40) or to the same therapy plus pulsed magnetotherapy (10–12 sessions) and electrophoresis with calcium chloride, lidocaine and magnesium sulfate (8–12 sessions; n = 40). Pain was rated on a visual analogue scale (VAS). ESR, C-reactive protein (CRP), sex and stress hormones, prostaglandins E_2 and $F_{2\alpha}$, symptom frequency and SF-36 scores were assessed before treatment and 1 and 3 months after it.

Results. Three months after treatment, the mean VAS score fell from 7.85 ± 1.12 to 2.35 ± 1.05 in the main group (−70.1%) and from 7.92 ± 1.08 to 5.80 ± 1.41 in the comparison group (−26.8%; $p < 0.001$; Cohen's $d = 2.89$). CRP decreased by 67.8% vs 31.8% and ESR by 62.5% vs 24.0%. In the main group, progesterone rose by 69.7% and estradiol by 43.1%, whereas cortisol, prolactin, PGE_2 and $PGF_{2\alpha}$ fell by 33.3%, 32.5%, 47.7% and 45.8%. Dysmenorrhoea persisted in 22.5% vs 60.0% of women and dyspareunia in 15.0% vs 47.5% ($p < 0.001$). All eight SF-36 domains improved more after combined treatment ($p < 0.001$). No adverse events occurred.

Conclusion. In women with CE-associated CPP, magnetotherapy and electrophoresis added to standard therapy relieved pain faster and to a greater extent, reduced inflammatory activity and prostaglandin levels, and improved quality of life. A five-stage management algorithm based on these findings is proposed.

Keywords: chronic endometritis; chronic pelvic pain; magnetotherapy; electrophoresis; physiotherapy; prostaglandins; quality of life; SF-36.

Introduction

Chronic pelvic pain (CPP) is usually defined as pain perceived to arise from the pelvic organs or structures that persists for more than six months and has cognitive, behavioural, sexual or emotional consequences (American College of Obstetricians and Gynecologists [ACOG], 2020). Published prevalence estimates in women range from 5.7% to 26.6% (ACOG, 2020), and the World Health Organization review by Latthe et al. (2006) showed how strongly the figure depends on the definition used and the population studied. Population surveys give a sense of the scale. In a United States survey covering 17,927 households, about one woman in seven reported CPP (Mathias et al., 1996). A New Zealand study found a three-month prevalence of 25.4%, and almost half of the affected women (47.7%) had no diagnosis (Grace & Zondervan, 2004). In Australia, 21.5% of 1,983 women reported non-cyclic pelvic pain and 71.7% reported dysmenorrhoea (Pitts et al., 2008).

Pain intensity is only part of the burden. Women with CPP have poorer health-related quality of life, lose working days and use more health care; in the United States, outpatient visits for CPP were estimated to cost US$881.5 million a year (Mathias et al., 1996). Because the pain often has gynaecological, urological, gastrointestinal, musculoskeletal and psychological contributors, many women see several specialists before a working diagnosis is reached (Lamvu et al., 2021; Speer et al., 2016).

Chronic endometritis (CE) is a persistent inflammation of the endometrium marked by plasma-cell infiltration of the stroma. It is often oligosymptomatic, and a firm diagnosis requires histology, preferably with CD138 immunohistochemistry, or hysteroscopy (Kitaya et al., 2016; Puente et al., 2020). The organisms most often implicated are common bacteria such as streptococci, Escherichia coli and Enterococcus faecalis, together with mycoplasmas and ureaplasmas; a 14-day course of doxycycline is the usual first-line treatment, and metronidazole-containing combinations are used as alternatives (Kitaya et al., 2016). Most recent work on CE concerns infertility, repeated implantation failure and pregnancy loss, conditions in which antibiotic treatment has been associated with better reproductive outcomes (Cicinelli et al., 2015; Kitaya et al., 2018). Its role in pain has received less attention, although ACOG (2020) lists chronic pelvic inflammatory disease and chronic endometritis among the gynaecological conditions associated with CPP. Inflammatory mediators sensitise pelvic nociceptors, and prostaglandins E_2 and $F_{2\alpha}$ are the main drivers of uterine hypercontractility and menstrual pain (Iacovides et al., 2015). Long-standing nociceptive input can alter central pain processing and the stress response, and changes in the hypothalamic–pituitary–adrenal (HPA) axis have been described in women with CPP (Heim et al., 1998).

Antibiotics and non-steroidal anti-inflammatory drugs act on the infectious and inflammatory component, yet pain frequently outlasts the infection, and the evidence for non-surgical treatment of CPP remains limited and heterogeneous (Cheong et al., 2014). Physical methods have been

studied mainly in chronic pelvic inflammatory disease. In a small randomized trial, short-wave diathermy reduced pain and pus-cell counts more than analgesics did (Lamina et al., 2011), and in a two-group study of 80 women its addition to medical treatment improved the results (Morsi & Nofal, 2020). Low-intensity pulsed magnetic fields at 1–100 Hz relieve musculoskeletal pain with few side effects (Paolucci et al., 2020), while electrophoresis (iontophoresis) uses a weak direct current to carry ionised drugs through the skin into the underlying tissues (Wang et al., 2022). Both methods are non-invasive and can be delivered on an outpatient basis. We found few controlled studies of their combined use in CE-associated CPP that assessed pain together with inflammatory markers, sex and stress hormones, prostaglandins and quality of life.

The aim of this study was to evaluate the effect of adding magnetotherapy and electrophoresis to standard medical treatment on pain, inflammatory and hormonal markers, clinical symptoms and quality of life in women of reproductive age with CPP associated with CE, and to propose a management algorithm based on the results.

Materials and methods

Study design and participants. This prospective randomized controlled study was carried out at the gynaecology department and outpatient clinic of the multidisciplinary clinic of Samarkand State Medical University in 2024–2025. Eighty women aged 18–40 years with CE and pelvic pain were enrolled consecutively as they presented.

Inclusion criteria were reproductive age, pelvic pain lasting at least six months, chronic inflammatory disease of the pelvic organs with a diagnosis of CE (alone or combined with salpingo-oophoritis or endocervicitis), and written informed consent. We excluded women with severe extragenital disease, malignancy or endometriosis, those whose pelvic pain had lasted less than six months, those with allergic reactions to the drugs or procedures used, and those who withdrew consent. CE was diagnosed on the basis of the history, gynaecological examination, laboratory and microbiological findings and transvaginal ultrasound.

Participants were allocated 1:1 to the main group (n = 40) or the comparison group (n = 40) with Random Allocation Software 2.0, using blocks of four. Blinding was partial: the physician who assessed outcomes and the statistician did not know the group assignment.

The study followed the principles of the Declaration of Helsinki. The protocol was approved by the Ethics Committee of Samarkand State Medical University, and each woman gave written informed consent after the aims, methods, possible risks and benefits had been explained to her.

Treatment. Both groups received the same standard drug therapy: doxycycline 100 mg orally twice daily and metronidazole 500 mg twice daily for 14 days, ibuprofen 400 mg two or three times daily after meals for 5–7 days, drotaverine 40 mg three times daily and vitamin E 400 IU once daily. The doxycycline–metronidazole combination is part of the outpatient regimen recommended for pelvic inflammatory disease (Workowski et al., 2021).

From day 7 the main group also received a combined physiotherapy course. Magnetotherapy was delivered with a BTL-5000 device in pulsed mode (induction 5–50 mT, frequency 10–100 Hz) through inductors placed over the suprapubic region, for 15 minutes per session, 10–12 sessions given daily or every other day. Electrophoresis was applied to the suprapubic and lumbosacral

regions with 2% calcium chloride, 0.5% lidocaine and 10% magnesium sulfate solutions used in alternation, at a current of 5–10 mA for 10–15 minutes, 8–12 sessions; a one-day break between sessions was allowed. Parameters were adjusted to each patient's clinical state. Before the course we checked for contraindications: malignancy, coagulation disorders, decompensated heart failure, fever, early pregnancy, skin lesions in the electrode area and intolerance of the drugs used.

Assessments. The clinical assessment covered menstrual, reproductive and gynaecological history, previous treatment, general and bimanual examination. Laboratory tests included a complete blood count with erythrocyte sedimentation rate (ESR), urinalysis, serum biochemistry (total protein, glucose, ALT, AST, creatinine, urea) and microscopy and culture of vaginal and cervical discharge. Serum C-reactive protein (CRP) was measured in fasting morning samples by immunoturbidimetry; values up to 5 mg/L were taken as normal. Estradiol (E_2), progesterone (P_4), follicle-stimulating hormone (FSH), luteinising hormone (LH), prolactin and cortisol were measured by enzyme-linked immunosorbent assay (ELISA) on a Mindray MR-96A analyser with XEMA reagent kits, and prostaglandins E_2 and $F_{2\alpha}$ by ELISA with RD&S kits. Blood for hormonal tests was drawn in the morning on days 21–23 of the menstrual cycle. Material for microscopy was taken before bimanual examination with a sterile swab from the posterior vaginal fornix and, after the cervix had been cleaned, with a sterile brush inserted 1.0–1.5 cm into the cervical canal; Gram-stained smears gave a first orientation to the likely pathogens. Transvaginal ultrasound with Doppler was performed on a Mindray DC-4000 scanner with 3.5–5 MHz convex and transvaginal probes on days 3–6 of the cycle. Colposcopy was carried out with an EDAN C6A HD colposcope on days 5–10 of the cycle, using 3% acetic acid and Lugol's solution to assess the transformation zone, vascular pattern and signs of inflammation. Neurologists and proctologists examined the patients when needed to exclude non-gynaecological sources of pain.

Pain intensity was rated on a 10-cm visual analogue scale (VAS): the patient marked her pain on a horizontal line running from "no pain" (0) to "worst pain" (10), and the distance from the left end in centimetres gave the score in points, with 1–3 points regarded as mild, 4–6 as moderate and 7–10 as severe pain (Hawker et al., 2011). Quality of life was measured with the 36-item SF-36 questionnaire, which covers eight domains – physical functioning, role limitations due to physical problems, bodily pain, general health, vitality, social functioning, role limitations due to emotional problems and mental health – each scored from 0 to 100, higher scores indicating better health (Ware & Sherbourne, 1992). We recorded dysmenorrhoea, dyspareunia, lower abdominal discomfort, menstrual irregularity and general weakness. Assessments were made before treatment and 1 and 3 months after its completion; hormones, prostaglandins and SF-36 scores were compared before treatment and at three months. Overall response was graded by the degree of symptom regression as complete (regression of more than 90% or remission), partial (50–90%), weak (20–50%) or absent (less than 20% or no change).

Statistical analysis. Data were analysed with SPSS Statistics 26 and Statistica; Microsoft Excel was used for data entry and charts. Normality was checked with the Shapiro–Wilk test. Continuous variables are given as mean ± standard deviation (M ± SD). Within-group changes were tested with the paired t-test and between-group differences with the independent t-test;

repeated-measures ANOVA was used for outcomes measured at three time points. The Mann–Whitney and Wilcoxon tests were applied to non-normally distributed data, and categorical variables were compared with the χ^2 test. Associations between VAS scores and clinical and laboratory variables were assessed with Pearson's correlation coefficient (r). Effect size was expressed as Cohen's d (Cohen, 1988). Differences were considered significant at $p < 0.05$.

Results

Baseline characteristics. All 80 women completed the treatment course and follow-up. The groups did not differ in age, body mass index, duration of disease and pain, or obstetric history (Table 1).

Table 1. Baseline characteristics of the study groups (M ± SD)

Characteristic	Main group (n = 40)	Comparison group (n = 40)	p
Age, years	32.4 ± 4.8	31.9 ± 5.1	0.672
Body mass index, kg/m^2	24.8 ± 3.2	25.1 ± 3.4	0.745
Duration of disease, years	2.7 ± 1.3	2.8 ± 1.4	0.813
Duration of pain, months	14.6 ± 5.2	15.1 ± 4.9	0.691
Caesarean sections, number	1.4 ± 0.7	1.5 ± 0.8	0.624
Abortions, number	0.8 ± 0.6	0.9 ± 0.7	0.582
Births, number	1.7 ± 0.9	1.8 ± 1.0	0.671

Women aged 31–35 years formed the largest age band (30.0%), followed by those aged 26–30 years (27.5%), 36–40 years (26.2%) and 18–25 years (16.3%); the age structure was the same in both groups ($p > 0.05$). More than half of the women lived in towns (57.5%), 61.2% were employed and 38.8% had higher education. Women from rural areas had lived with pain longer before their first visit to a gynaecologist (1.8 vs 1.3 years on average).

Cyclic pain related to menstruation was the most frequent pattern (42.5%); constant pain was reported by 37.5% of women and dyspareunia by 21.3%. Moderate pain predominated on the VAS (45.0%), and almost a third of the women (31.3%) rated their pain as severe. Psychoemotional stress (60.0%), a history of chronic genital infection (51.3%) and previous caesarean section (45.0%) were the commonest anamnestic factors (Table 2).

Table 2. Clinical characteristics of pelvic pain and anamnestic factors, n (%)

Characteristic	Main group (n = 40)	Comparison group (n = 40)	Total (n = 80)
Type of pain			
Constant pain	17 (42.5)	13 (32.5)	30 (37.5)
Cyclic pain related to menstruation	15 (37.5)	19 (47.5)	34 (42.5)
Pain during intercourse (dyspareunia)	10 (25.0)	7 (17.5)	17 (21.3)
Pain intensity on the VAS			
Mild (1–3 points)	8 (20.0)	11 (27.5)	19 (23.8)
Moderate (4–6 points)	19 (47.5)	17 (42.5)	36 (45.0)
Severe (7–10 points)	13 (32.5)	12 (30.0)	25 (31.3)
Anamnestic factors			
Stress and psychoemotional strain	27 (67.5)	21 (52.5)	48 (60.0)
History of chronic genital infection	23 (57.5)	18 (45.0)	41 (51.3)
Previous caesarean section	20 (50.0)	16 (40.0)	36 (45.0)
History of abortion	16 (40.0)	13 (32.5)	29 (36.3)
Early onset of sexual activity	12 (30.0)	8 (20.0)	20 (25.0)
Three or more births	9 (22.5)	7 (17.5)	16 (20.0)

Pain affected most areas of daily life (Table 3): emotional state was impaired in 81.3% of women, physical activity was limited in 78.8%, capacity for work was reduced in 73.8%, sleep was disturbed in 67.5%, sexual life suffered in 66.3% and social activity was restricted in 56.3%. Women rarely sought help early. Only 5.0% had seen a doctor within three months of pain onset, whereas 68.75% had lived with pain for more than a year before the first visit (1–2 years in 30.0%, 2–3 years in 25.0% and more than 3 years in 13.75%). Chronic cervicitis (38.8%), anaemia (30.0%), cervical ectropion (27.5%), cystic changes of the ovaries (23.8%) and autonomic (vegetative-vascular) dystonia (21.2%) were the most frequent comorbidities.

Table 3. Impact of pelvic pain on daily life, duration of pain before the first visit and comorbid conditions, n (%)

Indicator	Main group (n = 40)	Comparison group (n = 40)	Total (n = 80)
Areas of daily life affected by pain			
Emotional state	35 (87.5)	30 (75.0)	65 (81.3)
Physical activity	34 (85.0)	29 (72.5)	63 (78.8)
Capacity for work	31 (77.5)	28 (70.0)	59 (73.8)
Sleep	29 (72.5)	25 (62.5)	54 (67.5)
Sexual life	30 (75.0)	23 (57.5)	53 (66.3)
Social activity	25 (62.5)	20 (50.0)	45 (56.3)
Duration of pain before the first visit to a gynaecologist			
1–3 months	3 (7.5)	1 (2.5)	4 (5.0)
4–6 months	4 (10.0)	3 (7.5)	7 (8.75)
7–12 months	8 (20.0)	6 (15.0)	14 (17.5)
1–2 years	11 (27.5)	13 (32.5)	24 (30.0)
2–3 years	9 (22.5)	11 (27.5)	20 (25.0)
More than 3 years	5 (12.5)	6 (15.0)	11 (13.75)
Comorbid conditions			
Chronic cervicitis	18 (45.0)	13 (32.5)	31 (38.8)
Anaemia	14 (35.0)	10 (25.0)	24 (30.0)
Cervical ectropion	13 (32.5)	9 (22.5)	22 (27.5)
Cystic changes of the ovaries	11 (27.5)	8 (20.0)	19 (23.8)
Autonomic (vegetative-vascular) dystonia	10 (25.0)	7 (17.5)	17 (21.2)
Endometrial polyp	8 (20.0)	5 (12.5)	13 (16.2)
Chronic cystitis	7 (17.5)	5 (12.5)	12 (15.0)
Thyroid dysfunction	6 (15.0)	4 (10.0)	10 (12.5)

Correlation analysis. Before treatment, VAS scores correlated positively with stress level, serum cortisol and duration of disease and negatively with estradiol, progesterone and the quality-of-life score in both groups (Table 4). The inverse correlation with quality of life was the strongest ($r = -0.764$ in the main group and -0.691 in the comparison group; $p < 0.001$), and age showed only a weak association with pain intensity.

Table 4. Correlation between VAS pain scores and clinical and laboratory variables before treatment (Pearson's r)

Variable	Main group (n = 40)		Comparison group (n = 40)	
	r	p	r	p

Age	0.312	0.049	0.251	0.041
Duration of disease	0.561	< 0.001	0.473	< 0.001
Serum cortisol	0.654	< 0.001	0.582	< 0.001
Serum estradiol	−0.418	0.003	−0.366	0.008
Serum progesterone	−0.486	< 0.001	−0.401	0.004
Quality-of-life score	−0.764	< 0.001	−0.691	< 0.001
Stress level	0.689	< 0.001	0.612	< 0.001

Inflammatory markers. Both regimens lowered ESR and CRP, but the decrease was far greater in the main group (Table 5; Figure 1). Three months after treatment, CRP had fallen by 67.8% in the main group and by 31.8% in the comparison group. As a result, mean CRP in the main group was within the reference range (≤ 5 mg/L), whereas in the comparison group it remained above it. ESR decreased by 62.5% and 24.0%, respectively.

Table 5. Inflammatory markers before and 3 months after treatment (M ± SD)

Marker	Group	Before treatment	After 3 months	Change (Δ)	Fall, %	p
ESR, mm/h	Main	27.85 ± 4.92	10.45 ± 3.21	17.40 ± 4.12	62.5	< 0.001
	Comparison	28.15 ± 5.08	21.40 ± 4.75	6.75 ± 3.92	24.0	0.012
CRP, mg/L	Main	11.95 ± 2.87	3.85 ± 1.64	8.10 ± 2.45	67.8	< 0.001
	Comparison	12.10 ± 3.05	8.25 ± 2.31	3.85 ± 2.18	31.8	0.008

Note. p refers to the within-group change (paired t-test).

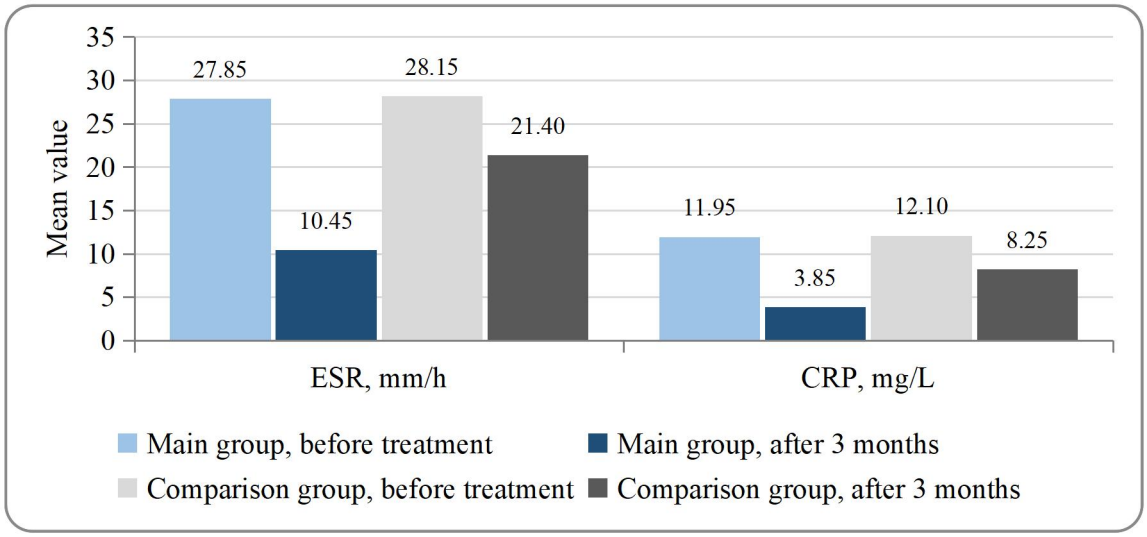


Figure 1. ESR and CRP before and 3 months after treatment

Hormones and prostaglandins. Baseline hormonal profiles were almost identical in the two groups (all p > 0.7), including FSH (7.85 ± 2.14 vs 7.92 ± 2.08 mIU/mL) and LH (6.42 ± 1.87 vs 6.51 ± 1.92 mIU/mL). After combined treatment, luteal-phase progesterone rose by 69.7% and

estradiol by 43.1%, while prolactin, cortisol, PGE_2 and $PGF_{2\alpha}$ decreased by 32.5–47.7%; effect sizes were large (d = 1.87–2.42). In the comparison group the changes were three to four times smaller (8.9–16.2%), and the rise in progesterone was not significant (p = 0.062) (Table 6).

Table 6. Hormones and prostaglandins before and 3 months after treatment (M ± SD)

Indicator	Group	Before treatment	After 3 months	Change, %	p	d
Estradiol, pmol/L	Main	218.4 ± 42.7	312.6 ± 38.9	+43.1	< 0.001	2.42
	Comparison	215.9 ± 39.8	238.4 ± 41.2	+10.4	0.048	–
Progesterone, nmol/L	Main	8.75 ± 3.12	14.85 ± 3.67	+69.7	< 0.001	1.98
	Comparison	8.92 ± 3.25	10.15 ± 3.18	+13.8	0.062	–
Prolactin, mIU/L	Main	428.6 ± 87.4	289.4 ± 64.2	−32.5	< 0.001	1.87
	Comparison	431.2 ± 91.5	392.7 ± 84.3	−8.9	0.041	–
Cortisol, nmol/L	Main	512.3 ± 94.6	341.7 ± 72.5	−33.3	< 0.001	2.05
	Comparison	518.7 ± 88.2	467.9 ± 82.6	−9.8	0.039	–
PGE_2, pg/mL	Main	245.7 ± 51.3	128.4 ± 37.9	−47.7	< 0.001	2.31
	Comparison	249.4 ± 48.9	208.6 ± 45.2	−16.2	0.028	–
$PGF_{2\alpha}$, pg/mL	Main	312.8 ± 64.2	169.5 ± 48.7	−45.8	< 0.001	2.18
	Comparison	308.5 ± 59.7	267.3 ± 55.8	−13.3	0.034	–

Note. p refers to the within-group change; d is Cohen's effect size. Baseline differences between the groups were not significant for any indicator (p = 0.765–0.912).

Pain and clinical symptoms. Pain decreased faster and further with combined therapy (Figure 2). One month after treatment, the mean VAS score in the main group was 4.15 ± 1.34 points, a 47.1% reduction, compared with 6.45 ± 1.27 points in the comparison group. At three months the scores were 2.35 ± 1.05 and 5.80 ± 1.41 points, reductions of 70.1% and 26.8% (p < 0.001; d = 2.89). The gap between the groups widened with time.

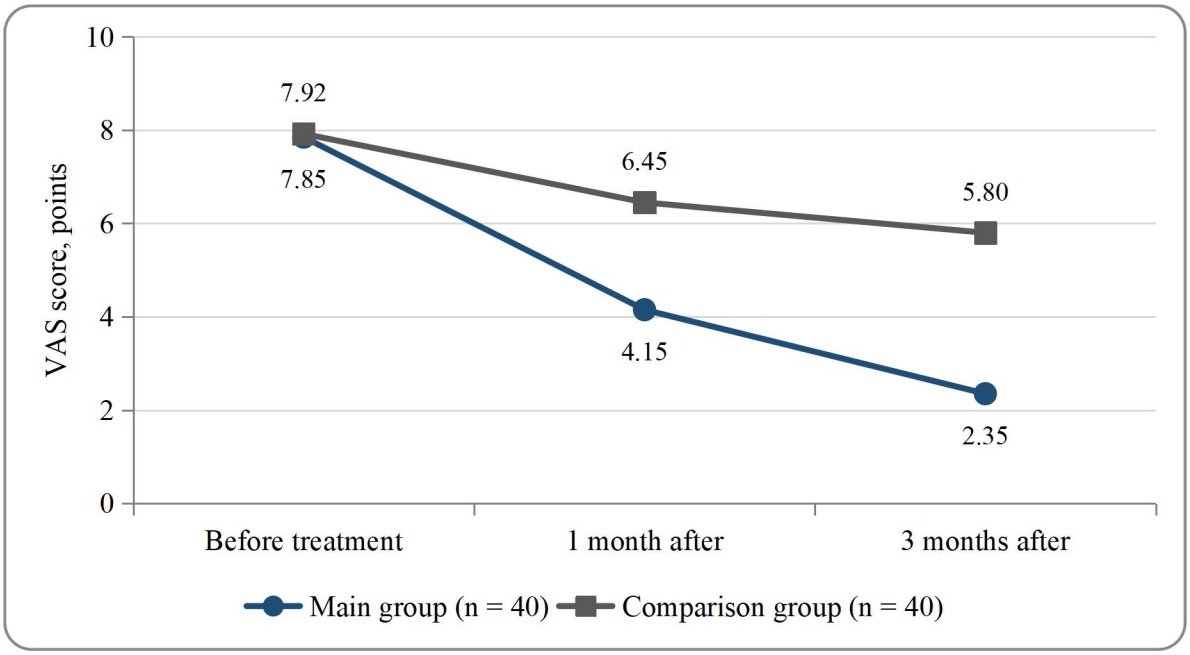


Figure 2. Mean VAS pain scores before and after treatment, points

Before treatment the frequency of each symptom was similar in the two groups ($p > 0.7$). Three months later, dysmenorrhoea persisted in 22.5% of women in the main group and 60.0% in the comparison group, dyspareunia in 15.0% and 47.5%, lower abdominal discomfort in 20.0% and 65.0%, menstrual irregularity in 10.0% and 37.5%, and general weakness in 17.5% and 55.0% (all $p < 0.001$; Table 7).

Table 7. Frequency of clinical symptoms before and 3 months after treatment, n (%)

Symptom	Main group (n = 40)		Comparison group (n = 40)		p between groups	
	Before	After	Before	After	Before	After
Dysmenorrhoea	34 (85.0)	9 (22.5)	33 (82.5)	24 (60.0)	0.756	< 0.001
Dyspareunia	28 (70.0)	6 (15.0)	27 (67.5)	19 (47.5)	0.812	< 0.001
Lower abdominal discomfort	36 (90.0)	8 (20.0)	35 (87.5)	26 (65.0)	0.724	< 0.001
Menstrual irregularity	22 (55.0)	4 (10.0)	23 (57.5)	15 (37.5)	0.821	< 0.001
General weakness	31 (77.5)	7 (17.5)	30 (75.0)	22 (55.0)	0.789	< 0.001

In relative terms, symptom frequency fell by 73.5–81.8% in the main group and by 25.7–34.8% in the comparison group (Figure 3). Menstrual irregularity (81.8%) and dyspareunia (78.6%) showed the largest relative decline after combined treatment.

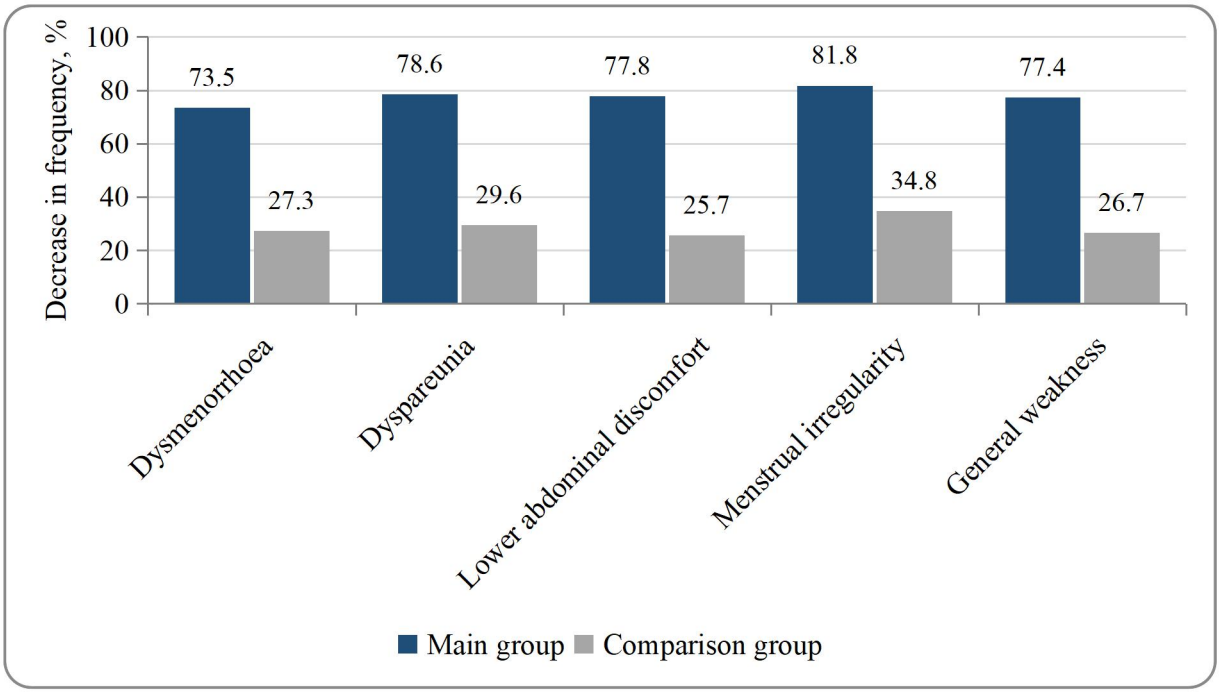


Figure 3. Relative decrease in symptom frequency 3 months after treatment, %

Quality of life. Baseline SF-36 scores were low and similar in both groups. At three months all eight domains had improved more in the main group ($p < 0.001$ for each; Table 8; Figure 4). The bodily pain score changed most, rising from 32.1 ± 10.6 to 76.4 ± 11.2 points (+138%), against +65.7% in the comparison group. Role limitations due to physical problems improved by 78.4% vs 36.2%, and role limitations due to emotional problems by 70.5% vs 32.0%; vitality rose by 78.5% and mental health by 58.1% after combined treatment.

Table 8. SF-36 scores before and 3 months after treatment, points (M ± SD)

Domain	Main group (n = 40)		Comparison group (n = 40)		p
	Before	After	Before	After	
Physical functioning (PF)	48.2 ± 12.4	78.5 ± 9.8	47.9 ± 13.1	62.3 ± 11.5	< 0.001
Role–physical (RP)	42.5 ± 15.2	75.8 ± 12.4	43.1 ± 14.8	58.7 ± 13.2	< 0.001
Bodily pain (BP)	32.1 ± 10.6	76.4 ± 11.2	31.8 ± 11.3	52.9 ± 13.5	< 0.001
General health (GH)	45.3 ± 11.8	72.6 ± 10.4	46.1 ± 12.2	58.4 ± 11.9	< 0.001
Vitality (VT)	38.6 ± 13.5	68.9 ± 11.7	39.2 ± 12.8	52.4 ± 13.6	< 0.001
Social functioning (SF)	52.4 ± 14.2	79.3 ± 10.6	51.8 ± 13.9	63.5 ± 12.8	< 0.001
Role–emotional (RE)	44.7 ± 15.8	76.2 ± 12.3	45.3 ± 14.7	59.8 ± 14.1	< 0.001
Mental health (MH)	47.5 ± 13.2	75.1 ± 10.9	48.1 ± 12.7	61.7 ± 13.4	< 0.001

Note. p refers to the difference between the groups after treatment.

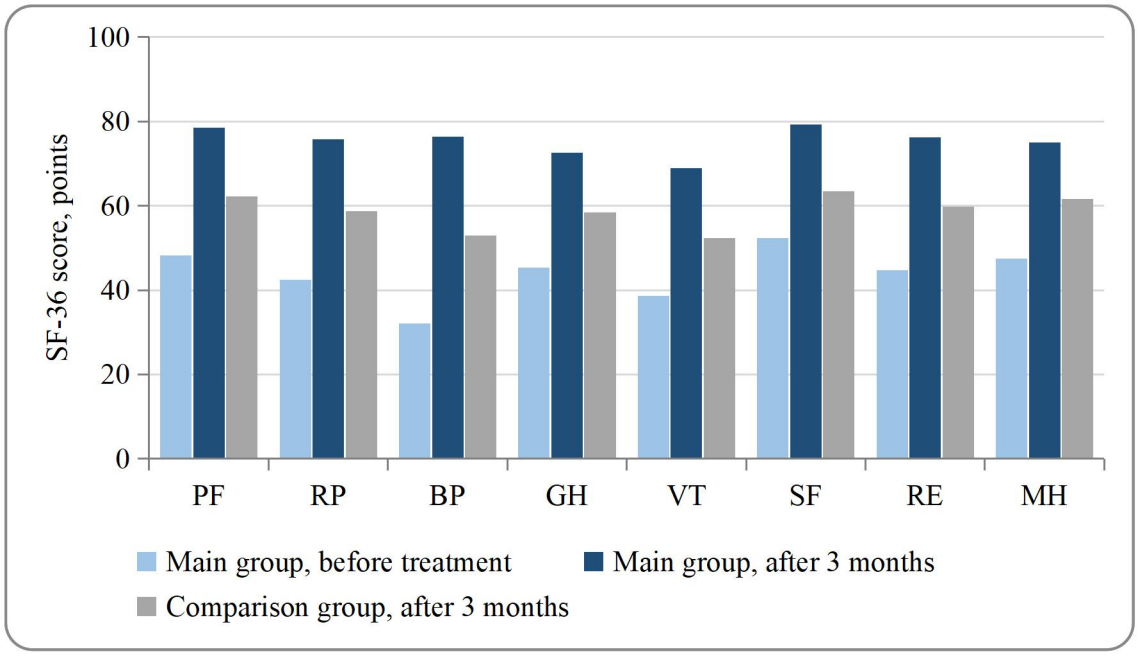


Figure 4. SF-36 profile before and 3 months after treatment, points

Note. PF – physical functioning; RP – role–physical; BP – bodily pain; GH – general health; VT – vitality; SF – social functioning; RE – role–emotional; MH – mental health. Baseline scores of the comparison group were close to those of the main group (Table 8).

Overall response and safety. A complete response was achieved in 68% of the main group and 18% of the comparison group, and a partial response in 22% and 30%, respectively. In total, a good or very good result was obtained in 90% of women after combined treatment and in 48% after drug therapy alone. Treatment was ineffective in 17% of the comparison group and in 2% of the main group (Figure 5).

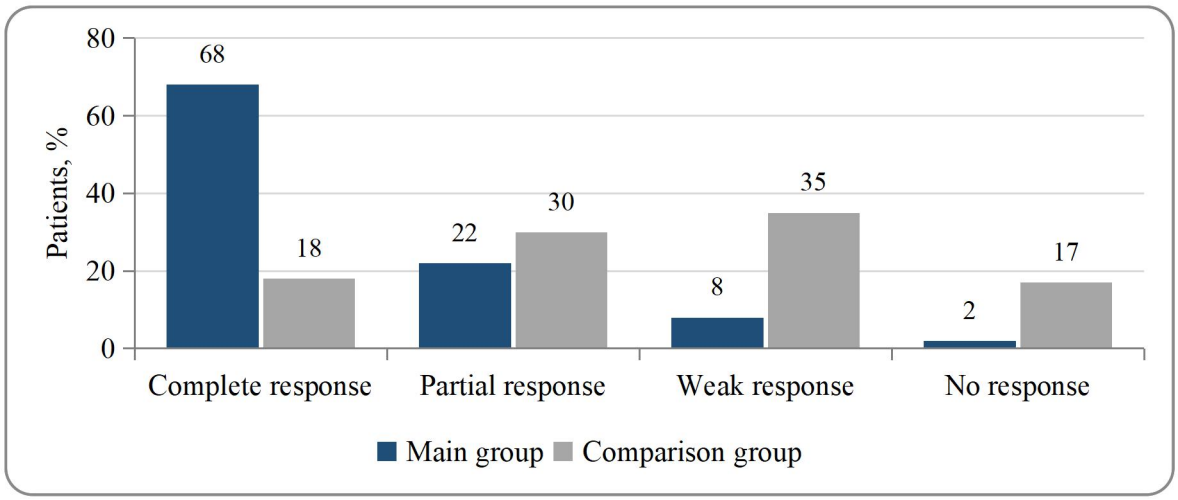


Figure 5. Overall treatment response in the study groups, % of patients

No adverse reactions to magnetotherapy or electrophoresis were recorded: there were no skin reactions, allergic reactions, changes in blood pressure, dizziness or nausea, and no patient

discontinued the course. During the three months of follow-up, 85% of women in the main group did not need additional analgesics.

Management algorithm. On the basis of these results and current recommendations we drew up a five-stage algorithm for the management of CPP in women with CE (Figure 6). The diagnostic stage (days 1–3) combines the history, examination, laboratory tests, ultrasound, colposcopy and VAS scoring with consultations of a neurologist, proctologist, urologist or psychiatrist when indicated. Etiotropic and pathogenetic drug therapy (days 1–14) is followed from day 7 by combined physiotherapy. Clinical and laboratory monitoring takes place 1 and 3 months after treatment, and treatment is regarded as effective when the VAS score falls by more than 50%, inflammatory markers return to normal and dysmenorrhoea and dyspareunia regress. Women are then reviewed every 6 months; a repeat physiotherapy course is advised after 3–6 months if symptoms persist or recur.

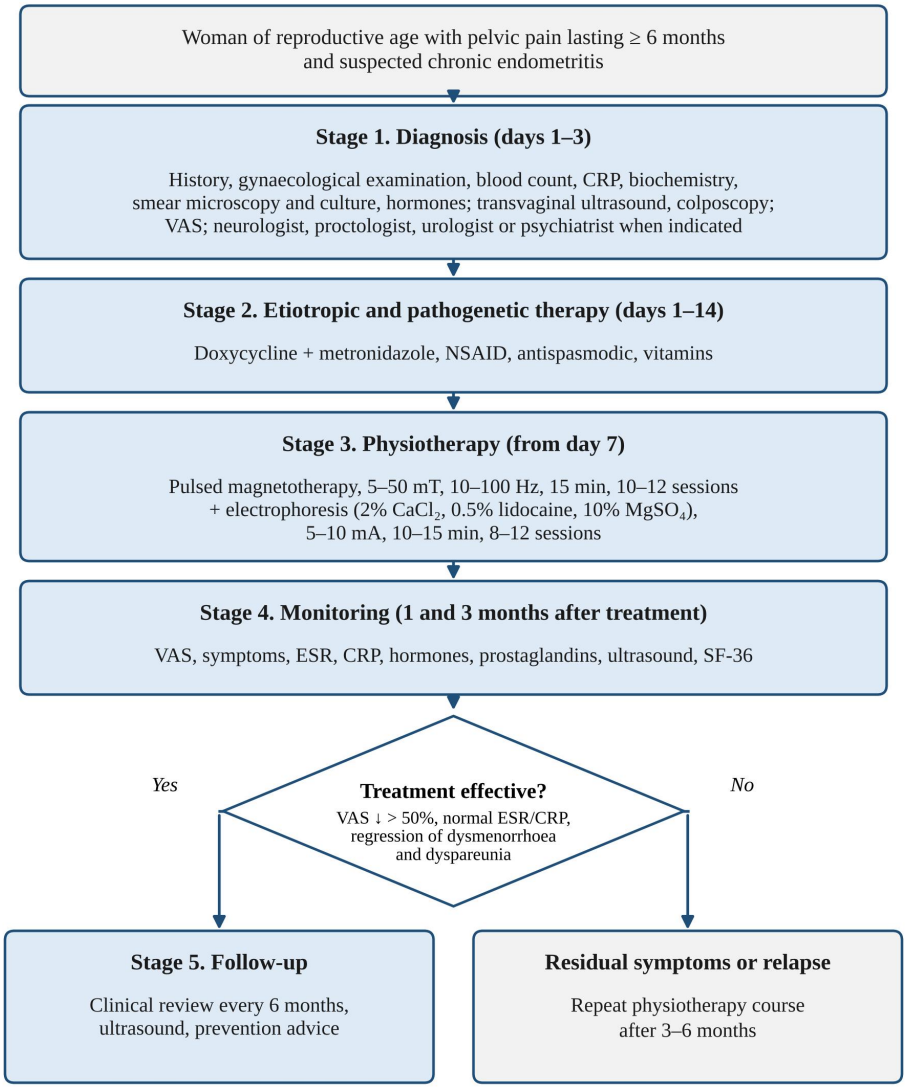


Figure 6. Algorithm for the management of chronic pelvic pain in women with chronic endometritis

Discussion

In this randomized study, adding pulsed magnetotherapy and electrophoresis to a standard 14-day antibacterial and anti-inflammatory regimen gave greater and faster pain relief than drug therapy alone in women with CE. The difference between the groups was already clear one month after treatment and grew by the third month. Inflammatory markers, prostaglandins, hormones, symptom frequency and SF-36 scores all moved in the same direction, which makes a chance finding unlikely.

The size of the effect is also clinically meaningful. On an 11-point numerical rating scale, a reduction in chronic pain of about two points or 30% corresponds to an improvement that patients regard as important (Farrar et al., 2001). The mean fall of 5.50 points (70.1%) in the main group was well above this threshold, whereas the fall of 2.12 points (26.8%) after drug therapy alone was close to it. In other words, standard treatment gave many women some relief, but combined treatment moved most of them from severe or moderate pain to mild pain.

These results agree with earlier work on physical treatment of chronic pelvic inflammation. Lamina et al. (2011) found in a randomized trial that short-wave diathermy added to antibiotics reduced pain and inflammation more than analgesics, and Morsi and Nofal (2020), in a design similar to ours with 80 women in two groups, reported better results when diathermy was combined with medical treatment. Our physical approach was different. The magnetic field frequencies we used (10–100 Hz) lie within the low-frequency range that a systematic review found effective for musculoskeletal pain, although the same review noted that the biological mechanism of the analgesic effect is still unclear (Paolucci et al., 2020). Electrophoresis carries ionised drugs into the tissues under the electrodes by electromigration and electro-osmosis (Wang et al., 2022). In our protocol, lidocaine, magnesium and calcium ions were delivered over the suprapubic and lumbosacral regions, which may explain the early decline in pain and dyspareunia.

Several mechanisms have been proposed for the effect of low-frequency pulsed magnetic fields on inflamed tissue: changes in membrane polarisation and calcium ion flux, better capillary blood flow and a shift of macrophages towards an anti-inflammatory phenotype. These ideas come largely from experimental work, and we did not test them; the parallel fall in ESR and CRP is consistent with an anti-inflammatory action but does not prove it. Calcium chloride, lidocaine and magnesium sulfate introduced by electrophoresis may add local membrane-stabilising, analgesic and spasmolytic effects, and magnesium relaxes uterine smooth muscle, which could ease cyclic pain.

Prostaglandin levels deserve separate comment. PGE_2 and $PGF_{2\alpha}$ fell by almost half after combined treatment, in parallel with a 73.5% regression of dysmenorrhoea. Prostaglandins drive uterine hypercontractility and ischaemic menstrual pain (Iacovides et al., 2015), so lower synthesis offers a plausible link between local anti-inflammatory action and symptom relief. We cannot, however, separate the contribution of the magnetic field from that of electrophoresis, because both were applied together, nor exclude better local penetration of the antibiotics as a further factor.

Before treatment, pain intensity correlated with serum cortisol ($r = 0.654$) and stress level ($r = 0.689$), and after combined therapy cortisol and prolactin decreased by about a third. Heim et al.

(1998), by contrast, described low basal and stimulated cortisol in women with CPP, many of whom had a normal pelvis at laparoscopy. Our patients had an identifiable inflammatory cause, and the type and duration of pain may shape the HPA response differently. The correlations we observed were cross-sectional and do not show the direction of the relationship.

Our patients' clinical profile also deserves attention. Two-thirds of them had lived with pain for more than a year before seeing a gynaecologist, and women from rural areas waited longer. This fits population data: in Australia only about a third of women with pelvic pain had sought advice from a health professional (Pitts et al., 2008), and in New Zealand almost half of the women with CPP had no diagnosis (Grace & Zondervan, 2004). Population surveys also link CPP with depression and anxiety (Pitts et al., 2008). In our sample, psychoemotional stress was the most frequent anamnestic factor (60.0%) and impaired emotional state the most common consequence of pain (81.3%). Chronic cervicitis and anaemia were frequent comorbidities. A persistent cervical focus may sustain ascending infection and recurrence of endometritis, and anaemia may lower tolerance of pain, so both belong in the treatment plan rather than being recorded as incidental findings.

Luteal-phase progesterone rose by 69.7% and estradiol by 43.1% in the main group. CE has been linked with impaired endometrial receptivity, implantation failure and pregnancy loss, and antibiotic treatment of CE with better outcomes of in vitro fertilisation (Cicinelli et al., 2015; Kitaya et al., 2016). Whether the hormonal changes we observed bring reproductive benefit was not studied and needs separate follow-up.

Good tolerability and the lower need for analgesics are of practical value too. Eighty-five percent of women in the main group took no additional painkillers during follow-up, which matters for patients who would otherwise use non-steroidal anti-inflammatory drugs for months, with the gastrointestinal, renal and cardiovascular risks this carries.

SF-36 scores show that patients felt the benefit. The bodily pain domain changed most, but role limitations, vitality and mental health also improved, in keeping with the broad burden of CPP described in population studies (Grace & Zondervan, 2004; Mathias et al., 1996). Current guidance favours a stepwise, multidisciplinary approach to CPP that deals with all contributing conditions rather than with one organ (ACOG, 2020; Lamvu et al., 2021). The proposed algorithm follows this principle: it begins with a structured diagnostic stage that involves other specialists, combines etiotropic treatment with physiotherapy, and sets measurable criteria of success at 1 and 3 months.

Physical treatment of CPP is not new. ACOG (2020) supports physical therapy for the musculoskeletal and myofascial components of CPP, while a Cochrane review found the evidence for most non-surgical interventions to be of low quality (Cheong et al., 2014). Our data add a controlled comparison in a defined inflammatory subgroup, with laboratory end points alongside patient-reported ones. They do not show that physiotherapy can replace antibacterial treatment; in our protocol it was always added to it.

Several limitations should be noted. It was carried out in one centre with a modest sample, and follow-up ended at three months, so recurrence and reproductive outcomes are unknown. No sham physiotherapy was used, and patients knew their allocation, which may have influenced subjective

outcomes such as VAS and SF-36 scores; the outcome assessor and the statistician were blinded, and the laboratory markers changed in the same direction. Diagnostic criteria for CE are not standardised (Puente et al., 2020), and hormones were measured in one phase of the cycle only. Finally, the mechanisms discussed above were inferred, not measured directly. Future trials should include a sham procedure, separate arms for magnetotherapy and electrophoresis, histological confirmation of CE and follow-up long enough to capture relapses and pregnancies.

Conclusions

1. In women of reproductive age with CE, pelvic pain was mostly of moderate or severe intensity, had lasted more than a year before the first visit in 68.75% of cases and impaired emotional state (81.3%), physical activity (78.8%) and capacity for work (73.8%). Its intensity correlated with stress, cortisol and disease duration and inversely with quality of life.

2. Adding magnetotherapy and electrophoresis to standard therapy reduced VAS pain scores by 70.1% at three months, compared with 26.8% after standard therapy alone, and lowered CRP and ESR by 67.8% and 62.5%.

3. Combined treatment raised luteal-phase progesterone and estradiol, reduced cortisol, prolactin and prostaglandins E_2 and $F_{2\alpha}$, improved all eight SF-36 domains and produced a complete or partial response in 90% of patients without adverse events.

4. The proposed five-stage algorithm can be used in outpatient and inpatient gynaecological practice for women with CPP associated with CE.

Practical recommendations

1. Women of reproductive age with pelvic pain lasting more than six months should be examined for CE. The examination should include VAS scoring, assessment of quality of life and of comorbid conditions (chronic cervicitis, anaemia, thyroid and urinary disorders) and, when indicated, consultation with a neurologist, proctologist, urologist or psychiatrist.

2. In CE-associated CPP, magnetotherapy (pulsed mode, 5–50 mT, 10–100 Hz, 15 min, 10–12 sessions) and electrophoresis with 2% calcium chloride, 0.5% lidocaine and 10% magnesium sulfate (5–10 mA, 10–15 min, 8–12 sessions) can be added to antibacterial and anti-inflammatory therapy from day 7 once contraindications have been excluded.

3. The response should be checked 1 and 3 months after the course by VAS scores, symptom frequency, ESR and CRP. Patients should then be reviewed every 6 months, and a repeat physiotherapy course is reasonable after 3–6 months if symptoms persist or recur.

4. Patients should be advised on regular moderate physical activity, sleep and stress reduction, timely treatment of genital infections, avoidance of abortion and appropriate contraception.

References

1. American College of Obstetricians and Gynecologists. (2020). Chronic pelvic pain: ACOG Practice Bulletin, Number 218. Obstetrics & Gynecology, 135(3), e98–e109. https://doi.org/10.1097/AOG.0000000000003716

2. Cheong, Y. C., Smotra, G., & Williams, A. C. de C. (2014). Non-surgical interventions for the management of chronic pelvic pain. Cochrane Database of Systematic Reviews, (3), Article CD008797. https://doi.org/10.1002/14651858.CD008797.pub2
3. Cicinelli, E., Matteo, M., Tinelli, R., Lepera, A., Alfonso, R., Indraccolo, U., Marrocchella, S., Greco, P., & Resta, L. (2015). Prevalence of chronic endometritis in repeated unexplained implantation failure and the IVF success rate after antibiotic therapy. Human Reproduction, 30(2), 323–330. https://doi.org/10.1093/humrep/deu292
4. Cohen, J. (1988). Statistical power analysis for the behavioral sciences (2nd ed.). Lawrence Erlbaum Associates.
5. Farrar, J. T., Young, J. P., Jr., LaMoreaux, L., Werth, J. L., & Poole, M. R. (2001). Clinical importance of changes in chronic pain intensity measured on an 11-point numerical pain rating scale. Pain, 94(2), 149–158. https://doi.org/10.1016/S0304-3959(01)00349-9
6. Grace, V. M., & Zondervan, K. T. (2004). Chronic pelvic pain in New Zealand: Prevalence, pain severity, diagnoses and use of the health services. Australian and New Zealand Journal of Public Health, 28(4), 369–375. https://doi.org/10.1111/j.1467-842X.2004.tb00446.x
7. Hawker, G. A., Mian, S., Kendzerska, T., & French, M. (2011). Measures of adult pain: Visual Analog Scale for Pain (VAS Pain), Numeric Rating Scale for Pain (NRS Pain), McGill Pain Questionnaire (MPQ), Short-Form McGill Pain Questionnaire (SF-MPQ), Chronic Pain Grade Scale (CPGS), Short Form-36 Bodily Pain Scale (SF-36 BPS), and Measure of Intermittent and Constant Osteoarthritis Pain (ICOAP). Arthritis Care & Research, 63(S11), S240–S252. https://doi.org/10.1002/acr.20543
8. Heim, C., Ehlert, U., Hanker, J. P., & Hellhammer, D. H. (1998). Abuse-related posttraumatic stress disorder and alterations of the hypothalamic-pituitary-adrenal axis in women with chronic pelvic pain. Psychosomatic Medicine, 60(3), 309–318. https://doi.org/10.1097/00006842-199805000-00017
9. Iacovides, S., Avidon, I., & Baker, F. C. (2015). What we know about primary dysmenorrhea today: A critical review. Human Reproduction Update, 21(6), 762–778. https://doi.org/10.1093/humupd/dmv039
10. Kitaya, K., Matsubayashi, H., Yamaguchi, K., Nishiyama, R., Takaya, Y., Ishikawa, T., Yasuo, T., & Yamada, H. (2016). Chronic endometritis: Potential cause of infertility and obstetric and neonatal complications. American Journal of Reproductive Immunology, 75(1), 13–22. https://doi.org/10.1111/aji.12438
11. Kitaya, K., Takeuchi, T., Mizuta, S., Matsubayashi, H., & Ishikawa, T. (2018). Endometritis: New time, new concepts. Fertility and Sterility, 110(3), 344–350. https://doi.org/10.1016/j.fertnstert.2018.04.012
12. Lamina, S., Hanif, S., & Gagarawa, Y. S. (2011). Short wave diathermy in the symptomatic management of chronic pelvic inflammatory disease pain: A randomized controlled trial. Physiotherapy Research International, 16(1), 50–56. https://doi.org/10.1002/pri.473
13. Lamvu, G., Carrillo, J., Ouyang, C., & Rapkin, A. (2021). Chronic pelvic pain in women: A review. JAMA, 325(23), 2381–2391. https://doi.org/10.1001/jama.2021.2631

14. Latthe, P., Latthe, M., Say, L., Gülmezoglu, M., & Khan, K. S. (2006). WHO systematic review of prevalence of chronic pelvic pain: A neglected reproductive health morbidity. BMC Public Health, 6, Article 177. https://doi.org/10.1186/1471-2458-6-177
15. Mathias, S. D., Kuppermann, M., Liberman, R. F., Lipschutz, R. C., & Steege, J. F. (1996). Chronic pelvic pain: Prevalence, health-related quality of life, and economic correlates. Obstetrics & Gynecology, 87(3), 321–327. https://doi.org/10.1016/0029-7844(95)00458-0
16. Morsi, D. I., & Nofal, A. M. (2020). Physiotherapy is a new line in management of chronic pelvic inflammatory diseases. The Egyptian Journal of Hospital Medicine, 81(5), 2054–2056. https://doi.org/10.21608/ejhm.2020.124873
17. Paolucci, T., Pezzi, L., Centra, A. M., Giannandrea, N., Bellomo, R. G., & Saggini, R. (2020). Electromagnetic field therapy: A rehabilitative perspective in the management of musculoskeletal pain – A systematic review. Journal of Pain Research, 13, 1385–1400. https://doi.org/10.2147/JPR.S231778
18. Pitts, M. K., Ferris, J. A., Smith, A. M. A., Shelley, J. M., & Richters, J. (2008). Prevalence and correlates of three types of pelvic pain in a nationally representative sample of Australian women. Medical Journal of Australia, 189(3), 138–143. https://doi.org/10.5694/j.1326-5377.2008.tb01945.x
19. Puente, E., Alonso, L., Laganà, A. S., Ghezzi, F., Casarin, J., & Carugno, J. (2020). Chronic endometritis: Old problem, novel insights and future challenges. International Journal of Fertility & Sterility, 13(4), 250–256. https://doi.org/10.22074/ijfs.2020.5779
20. Speer, L. M., Mushkbar, S., & Erbele, T. (2016). Chronic pelvic pain in women. American Family Physician, 93(5), 380–387.
21. Wang, Y., Zeng, L., Song, W., & Liu, J. (2022). Influencing factors and drug application of iontophoresis in transdermal drug delivery: An overview of recent progress. Drug Delivery and Translational Research, 12(1), 15–26. https://doi.org/10.1007/s13346-021-00898-6
22. Ware, J. E., Jr., & Sherbourne, C. D. (1992). The MOS 36-item short-form health survey (SF-36): I. Conceptual framework and item selection. Medical Care, 30(6), 473–483.
23. Workowski, K. A., Bachmann, L. H., Chan, P. A., Johnston, C. M., Muzny, C. A., Park, I., Reno, H., Zenilman, J. M., & Bolan, G. A. (2021). Sexually transmitted infections treatment guidelines, 2021. MMWR Recommendations and Reports, 70(4), 1–187. https://doi.org/10.15585/mmwr.rr7004a1