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DYSMENORRHOEA AND *UDAVARTINI YONIVYAPAD*: NEUROINFLAMMATION, *APANA VATA*, AND INTEGRATIVE PAIN MANAGEMENT

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ABSTRACT

Dysmenorrhoea is often normalized as an unavoidable part of menstruation, even when pain repeatedly interrupts education, work, sleep, mobility, and emotional well-being. Contemporary evidence explains primary dysmenorrhoea mainly through progesterone withdrawal, increased endometrial prostaglandin production, uterine hypercontractility, vasoconstriction, and transient ischaemia. The emerging picture is broader: inflammatory cytokines, oxidative stress, peripheral nociceptor activation, altered descending pain modulation, and central sensitization may help explain why pain severity differs markedly between individuals and why recurrent menstrual pain may persist beyond a purely uterine event. In Ayurveda, painful and difficult menstruation with relief after the establishment of menstrual flow is described under *Udavartini Yonivyapad*. Its pathogenesis centres on aggravated *Vata*, especially disturbed or upward-moving *Apana Vata*, resulting in *Rajahkrichhrata* and *Yoni Shoola*. Although *Apana Vata* cannot be equated directly with a single neuroendocrine pathway, it provides a functional framework for understanding disordered pelvic movement, pain, and elimination. This narrative review examines dysmenorrhoea through biomedical and Ayurvedic perspectives and proposes a safe, layered model of integrative pain management. Evidence-based pharmacological care, appropriate screening for secondary causes, heat, physical activity,

yoga, sleep and stress regulation may be combined with individualized Ayurvedic measures under qualified supervision. Published Ayurvedic trials are encouraging but are generally small and methodologically limited. Integration should therefore complement—not delay—diagnosis or established treatment. Future research should use rigorous randomized designs, standardized outcomes, adverse-event reporting, and mechanistic biomarkers to determine whether Ayurvedic interventions influence inflammation, uterine contractility, pain sensitization, or functional recovery.

KEYWORDS: dysmenorrhoea; *Udavartini Yonivyapad*; *Apana Vata*; neuroinflammation; menstrual pain; central sensitization; integrative medicine; Ayurveda.

INTRODUCTION

For many women and adolescents, menstruation is not simply a monthly biological event. It can arrive with cramping pain, nausea, fatigue, diarrhoea, headache, backache, disturbed sleep, anxiety about the next episode, and the practical burden of missing classes or work. A recent systematic review covering 70 countries estimated that dysmenorrhoea affects approximately seven in ten menstruating individuals worldwide, although prevalence varies according to population, definition, and measurement method.[1] Earlier epidemiological work similarly reported wide prevalence ranges and identified younger age, heavy or irregular bleeding, family history, smoking, and psychosocial factors among commonly associated variables.[2] Dysmenorrhoea also has a measurable effect on quality of life, academic participation, concentration, and social functioning.[3] The World Health Organization now treats menstrual health as a health and human-rights issue, emphasizing that severe or disruptive menstrual pain should not be dismissed as normal.[4]

Modern gynaecology distinguishes primary dysmenorrhoea—painful menstruation without demonstrable pelvic pathology—from secondary dysmenorrhoea caused by conditions such as endometriosis, adenomyosis, leiomyoma, pelvic inflammatory disease, congenital outflow obstruction, or an intrauterine pathology.[5] The distinction is clinically essential, but it does not mean that primary dysmenorrhoea is “only functional” or insignificant. Increasing evidence shows that repeated menstrual pain involves an interaction between uterine biochemistry, inflammatory signalling, the peripheral nervous system, central pain processing, behaviour, sleep, and emotional context.

Ayurveda describes a clinically comparable pattern under *Udavartini Yonivyapad*, a *Vata*-predominant disorder in which disturbed *Apana Vata* causes painful or difficult expulsion of menstrual blood. The comparison is useful because both frameworks recognize disturbed uterine function and pain during menstrual flow. However, they arise from different knowledge systems. The purpose of integration is therefore not to force equivalence, but to build a clinically responsible dialogue that improves assessment, symptom relief, self-care, and timely referral.

AIM AND OBJECTIVES

This review aims to examine the relationship between primary dysmenorrhoea and *Udavartini Yonivyapad*, with particular attention to neuroinflammation, *Apana Vata*, and integrative pain management. Its objectives are to.

1. summarize the contemporary pathophysiology of dysmenorrhoea, including inflammatory and central pain mechanisms;
2. describe the classical Ayurvedic understanding of *Udavartini Yonivyapad* and the role of *Apana Vata*;
3. identify clinically meaningful areas of convergence and important limits of comparison;
4. present a safe, evidence-informed, stepped approach to integrative management; and
5. identify research priorities for evaluating Ayurvedic interventions in menstrual pain.

MATERIALS AND METHODS

A focused narrative review was undertaken using PubMed, PubMed Central, the Cochrane Library, World Health Organization resources, American College of Obstetricians and Gynecologists guidance, and the European Society of Human Reproduction and Embryology guideline. Literature relating to primary dysmenorrhoea, prostaglandins, cytokines, oxidative stress, neuroimaging, central sensitization, exercise, heat, yoga, transcutaneous electrical nerve stimulation, hormonal treatment, and selected complementary interventions was considered. Classical descriptions were examined in the *Charaka Samhita*, *Sushruta Samhita*, *Ashtanga Hridaya*, and *Madhava Nidana*. Published clinical reports on Ayurvedic management of *Udavartini Yonivyapad* or *Kashtartava* were included to assess the present evidence base. The review is interpretive rather than systematic; consequently, it does not claim exhaustive retrieval or formal certainty grading.

Dysmenorrhoea: From Uterine Contraction to a Pain-System Disorder

Prostaglandins, hypercontractility, and ischaemia

The most established mechanism of primary dysmenorrhoea begins near the end of an ovulatory cycle. Withdrawal of progesterone promotes endometrial breakdown and the release of arachidonic acid, which is converted through cyclooxygenase pathways into prostaglandins. Increased prostaglandin F2 alpha and prostaglandin E2 contribute to high-amplitude uterine contractions, elevated resting tone, vasoconstriction, reduced uterine perfusion, and ischaemic pain.[5,6] Systemic spillover of prostaglandins helps explain nausea, vomiting, diarrhoea, headache, fatigue, and dizziness. This mechanism also explains why non-steroidal anti-inflammatory drugs (NSAIDs), which inhibit cyclooxygenase activity, are effective for many patients.

The intensity of pain, however, cannot always be predicted from uterine activity alone. Some individuals have severe disability despite apparently normal pelvic findings, while others respond incompletely to prostaglandin inhibition. This clinical variability has encouraged investigation of inflammation, oxidative stress, pain amplification, and psychosocial modulation.

Inflammatory signalling and neuroinflammation

Menstruation is physiologically inflammatory: tissue breakdown, leukocyte activity, repair, and angiogenesis are coordinated through cytokines, chemokines, prostaglandins, and matrix-remodelling pathways. In primary dysmenorrhoea, this response may become excessive or poorly regulated. A case-control study demonstrated increased expression of pro-inflammatory cytokine genes, including *IL1B*, *TNF*, *IL6*, and *IL8*, with reduced expression of selected anti-inflammatory signals across the menstrual cycle.[7] Reviews have further described the potential participation of tumour necrosis factor-alpha, interleukins, C-reactive protein, and prostaglandin–cytokine interactions in pain generation.[8]

The term neuroinflammation in this context should be used carefully. It does not imply a proven inflammatory lesion in the brain of every patient with dysmenorrhoea. Rather, inflammatory mediators can sensitize peripheral uterine afferents, lower nociceptor thresholds, increase spinal transmission, and interact with central pain-modulating networks. Recurrent nociceptive input may then strengthen pain responses over time. Oxidative stress may add to this process through lipid peroxidation, altered endothelial function, and

amplification of inflammatory signalling, although the available biomarker literature remains limited and heterogeneous.[9]

Peripheral and central sensitization

Pain is not a passive readout of uterine contraction. It is an experience produced by the nervous system after integrating sensory input with attention, expectation, previous pain, sleep, mood, and social context. Experimental work has found altered pain thresholds and inhibitory pain processing in young women with primary dysmenorrhea.[10] More recent research suggests that women with a longer history of dysmenorrhoea may show generalized hyperalgesia, enlarged pain distribution, and facilitated central pain mechanisms across the menstrual cycle.[11] Functional neuroimaging has reported altered connectivity involving the periaqueductal grey and other networks associated with descending pain modulation, salience, and sensory integration.[12]

These findings do not mean that menstrual pain is "psychological." They show that repeated pelvic pain can influence how the nervous system processes later pain. This distinction matters in practice. A person with severe dysmenorrhoea needs validation, adequate analgesia, and evaluation for secondary disease—not reassurance that normal imaging means nothing is wrong.

Ayurvedic Understanding of *Udavartini Yonivyapad*

Classical description

The *Charaka Samhita* describes *Udavartini* among the twenty *Yonivyapad*. Aggravated *Vata*, moving in an abnormal or upward direction, produces painful menstruation; relief is characteristically obtained after the menstrual blood is discharged.[13] The *Sushruta Samhita* similarly describes painful, difficult menstrual flow in a *Vata*-dominant condition.[14] Vagbhata discusses the disorder within the diseases of the female reproductive tract, retaining the central relationship between abnormal movement of *Vata* and pain.[15] The *Madhava Nidana* also recognizes painful menstruation associated with disturbed *Vata*.[16]

The clinical resemblance to primary dysmenorrhoea is strongest when the patient has cyclical lower-abdominal pain beginning shortly before or with menstruation, difficult or initially scanty flow, and relief after flow becomes established, without identifiable pelvic pathology. Nevertheless, *Udavartini Yonivyapad* should not be treated as a universal label for all painful menstruation. Endometriosis, adenomyosis, infection, fibroids, obstructive anomalies,

gastrointestinal disease, urinary pathology, and pregnancy-related emergencies require appropriate biomedical evaluation.

Role of *Apana Vata*

Apana Vata governs downward movement and physiological expulsion from the pelvic region, including defecation, urination, menstruation, and childbirth. In *Udavartini*, the normal downward direction (*Anuloma Gati*) is disturbed, producing *Pratiloma Gati* or obstructed movement. This disturbance is expressed through *Rajahkrichhrata* (painful or difficult menstruation), *Yoni Shoola* (pelvic or genital pain), abdominal cramping, backache, and associated disturbances of bowel or bladder function.

From an Ayurvedic standpoint, pain is strongly associated with *Vata*. *Ruksha* (dry), *Sheeta* (cold), *Laghu* (light), *Chala* (mobile), and *Khara* (rough) qualities may be aggravated by irregular meals, inadequate nourishment, sleep disruption, excessive exertion, chronic stress, suppression of natural urges, and exposure to cold. These factors are not diagnostic biomarkers, but they provide a practical history of routines that may worsen pain, constipation, sleep, and stress reactivity.

Samprapti

A clinically useful *Samprapti* can be summarized as follows: *Vata*-provoking diet and behaviour, suppression of natural urges, stress, or local pelvic factors aggravate *Vata*; *Apana Vata* loses its normal direction; movement of *Artava* becomes difficult or obstructed; uterine and pelvic pain develops; and relief occurs when the flow is properly established. Depending on the individual, *Pitta* may contribute burning, irritability, heavy flow, or inflammatory features, while *Kapha* or *Ama* may contribute heaviness, congestion, sluggish digestion, or a sense of obstruction. This individualized assessment is one advantage of the Ayurvedic framework, provided it does not replace investigation for secondary pathology.

Neuroinflammation and *Apana Vata*: A Responsible Integrative Interpretation

The biomedical and Ayurvedic explanations operate at different levels. Prostaglandins, cytokines, nociceptors, uterine perfusion, and pain-network connectivity are measurable biological processes. *Apana Vata* is a functional Ayurvedic construct describing coordinated downward movement, pelvic elimination, and reproductive physiology. It would therefore be scientifically inappropriate to state that *Apana Vata* "is" a particular nerve, neurotransmitter, cytokine, or autonomic pathway.

The two perspectives can nevertheless be placed in dialogue. Uterine hypercontractility, vasoconstriction, ischaemia, bowel disturbance, pelvic muscle guarding, and nociceptive amplification together produce disturbed pelvic function and painful flow. Ayurveda organizes this pattern as abnormal *Vata Gati*. Neuroinflammation may help explain why the pain becomes intense, spreads beyond the lower abdomen, or remains disproportionate to observable pelvic pathology. *Apana Vata* offers a whole-person clinical model that also attends to bowel regularity, sleep, food timing, stress, movement, and the subjective experience of flow.

This comparison is best regarded as hypothesis-generating. It can guide research questions—for example, whether a *Vata-Anulomana* intervention changes pain scores, rescue analgesic use, uterine artery Doppler indices, prostaglandin metabolites, inflammatory cytokines, heart-rate variability, pelvic-floor tone, or quantitative sensory testing. Until such studies are performed, claims of mechanistic equivalence should be avoided.

Clinical Assessment and the Need to Exclude Secondary Dysmenorrhoea

Assessment should establish the age at onset, relation of pain to menarche, cycle regularity, timing and duration of pain, bleeding volume, pain location and radiation, gastrointestinal and urinary symptoms, sexual history when appropriate, medication response, family history, functional impairment, and possibility of pregnancy. Pain intensity can be recorded on a numerical rating scale or visual analogue scale, but functional outcomes—school or work absence, sleep disturbance, physical activity, and rescue medication—are equally important.

Features that should increase suspicion of secondary dysmenorrhoea include pain beginning immediately after menarche or later after previously painless cycles, progressive worsening, persistent non-cyclical pelvic pain, dyspareunia, dyschezia, dysuria, infertility, abnormal uterine bleeding, fever, vaginal discharge, a pelvic mass, poor response to correctly used NSAIDs or hormonal therapy, and a family history of endometriosis.[17]

Current guidance increasingly supports a clinical and imaging-based approach to suspected endometriosis rather than waiting for diagnostic surgery in every case. The 2026 ACOG guideline recommends clinical evaluation and appropriate imaging, with transvaginal ultrasonography generally used first when suitable and magnetic resonance imaging reserved for selected cases.[18] The ESHRE guideline similarly emphasizes symptom recognition, imaging, shared decision-making, and individualized treatment.[19] Severe pain should

therefore trigger thoughtful evaluation, not simply stronger analgesics or repeated empirical Ayurvedic treatment.

Integrative Pain Management

An integrative plan should be layered. It begins with diagnosis and reliable pain relief, then adds low-risk self-management and individualized Ayurvedic measures. The aim is not to make the patient choose between systems but to combine compatible interventions while avoiding duplication, interactions, invasive procedures without indication, or delayed referral.

Education and shared decision-making

The first therapeutic act is to acknowledge that disabling menstrual pain is not trivial. Patients should understand the likely mechanisms, available options, expected time to benefit, adverse effects, warning signs, and the difference between primary and secondary dysmenorrhoea. A menstrual diary recording pain, bleeding, gastrointestinal symptoms, sleep, medicine use, and functional limitation often reveals patterns and improves follow-up decisions.

Evidence-based pharmacological treatment

NSAIDs remain first-line treatment for most patients with primary dysmenorrhoea. They are most effective when started shortly before expected menstruation or at the earliest onset of bleeding or pain and then taken on a regular schedule for the first one to three days, according to the selected medicine and the patient's clinical profile. A Cochrane review found NSAIDs more effective than placebo, but gastrointestinal, renal, allergic, and other adverse effects must be considered.[20] The choice and dose should follow clinical guidance, especially in patients with asthma, kidney disease, peptic ulcer disease, anticoagulant use, or other contraindications.

Hormonal therapy reduces endometrial proliferation and menstrual prostaglandin production and is appropriate for many patients who also desire contraception or have inadequate relief from NSAIDs. Combined oral contraceptive pills have demonstrated pain reduction compared with placebo, although irregular bleeding, headache, nausea, contraindications, and individual preferences require discussion.[21] Progestin-only methods and other menstrual-suppression options may also be considered under gynaecological supervision.

Heat, physical activity, and TENS

Local heat is simple, accessible, and often provides rapid comfort through muscle relaxation, improved local blood flow, and altered nociceptive input. Reviews of heat and transcutaneous electrical nerve stimulation (TENS) suggest benefit, although trial quality and protocols vary.[22] A more recent systematic review also supports heat therapy as an effective low-risk option, while noting heterogeneity across trials.[23] Care is required to prevent burns, especially with prolonged use or reduced skin sensation.

Regular physical activity may reduce menstrual pain through improved circulation, endogenous pain modulation, mood regulation, and reduced stress. The Cochrane review found potentially meaningful benefit, but the certainty of evidence was low to very low because of small and heterogeneous studies.[24] A 2024 network meta-analysis reported improvement across several exercise types after sustained practice, reinforcing the importance of regularity rather than relying only on exercise during acute pain.[25]

***Yoga*, breathing, and nervous-system regulation**

Yoga is especially relevant to an integrative model because it combines movement, breathing, attention, and relaxation. Meta-analytic evidence suggests that yoga may reduce menstrual pain, but the number and quality of trials remain limited.[26] Randomized studies in young women have reported reductions in menstrual cramps and distress and improvements in physical fitness and quality of life after structured programmes.[27,28] Gentle, individualized practice may include relaxed diaphragmatic breathing, pelvic and spinal mobility, and restorative postures. Forceful practice during severe pain, heavy bleeding, dizziness, or suspected secondary pathology should be avoided. Claims that a specific posture "corrects" the uterus or directly normalizes hormones are not adequately established.

Diet and selected herbal evidence

Regular meals, adequate hydration, fibre, sleep-supportive routines, and correction of iron deficiency or other nutritional problems are more defensible than rigid "dysmenorrhoea diets." Ginger has been studied for primary dysmenorrhoea and may reduce pain, possibly through anti-inflammatory and cyclooxygenase-related effects. Systematic reviews report promising results but also emphasize small samples, heterogeneity, variable preparations, and risk of bias.[29,30] Ginger is not suitable for everyone; gastrointestinal intolerance, bleeding risk, concurrent anticoagulant or antiplatelet therapy, pregnancy status, and product quality should be considered.

Ayurvedic principles of management

Ayurvedic management is directed toward restoring the normal direction of *Apana Vata*, reducing pain, improving digestion and bowel regularity, and strengthening menstrual health across cycles. The major principles include.

6. *Nidana Parivarjana*: identifying and reducing aggravating factors such as irregular meals, fasting followed by overeating, sleep deprivation, excessive exertion, prolonged sitting, constipation, and suppression of natural urges.
7. *Vata Anulomana*: supporting normal downward movement, particularly when pain is associated with constipation, bloating, or difficult onset of flow.
8. *Snehana* and gentle *Swedana*: using appropriately selected unctuous and warm measures to counter excessive dryness, coldness, spasm, and stiffness.
9. *Shoola Prashamana*: individualized pain-relieving measures selected according to *Dosha*, digestive capacity, bleeding pattern, age, comorbidity, and concomitant medicines.
10. regulation of *Agni* and correction of clinically assessed *Ama* without aggressive restriction or purgation during acute pain; and
11. supervised *Basti* or other procedures only when clearly indicated, after excluding pregnancy, infection, structural disease, and contraindications.

Ayurvedic medicines should not be presented as universally safe because they are “natural.” Identity, purity, dose, duration, formulation, contaminants, herb–drug interactions, hepatic or renal disease, pregnancy, and bleeding risk require attention. Metallic or mineral preparations require particular quality assurance and qualified prescribing.

Evidence for Ayurvedic interventions

The direct clinical evidence remains preliminary. A small study of *Uttara Basti* with *Trivrit* and *Lasuna* oil reported improvement in primary dysmenorrhoea, but its sample size and study design limit generalizability.[31] A comparative study of *Dashamoola Taila Matra Basti* and *Tila Taila Matra Basti* reported symptomatic benefit, with more sustained improvement in the *Dashamoola Taila* group.[32] An open-label study of *Shoola Prashamana Dashemani Churna* also reported reduction in menstrual pain, but lacked a large randomized control group.[33] A recent review of published *Matra Basti* studies found promising results while identifying variation in formulations, schedules, comparators, and methodological quality.[34] Yoga has also been investigated specifically in *Udavartini Vyapad*, but the available report is preliminary.[35]

These studies justify further research, not definitive claims of superiority or cure. Invasive procedures such as *Uttara Basti* should never be converted into unsupervised self-care. They require specialist assessment, sterile technique, appropriate timing, informed consent, and monitoring.

A Practical Stepped Model

Clinical stage	Core actions	Integrative additions	Escalation criteria
Initial assessment	Confirm cyclical pattern; assess pain, bleeding, pregnancy possibility, medicines, comorbidities, and functional loss	Ayurvedic history of *Vata* aggravation, bowel pattern, sleep, diet timing, stress, and features of *Dosha* involvement	Any red flag, abnormal examination, acute severe pain, or pregnancy possibility
First-line care	Correctly timed NSAID when suitable; education; menstrual diary	Local heat, hydration, regular meals, gentle movement, sleep regulation, breathing practice	Inadequate relief after correctly used treatment across approximately three cycles
Persistent symptoms	Reassess adherence and diagnosis; consider hormonal therapy and pelvic imaging as indicated	Individualized, quality-assured Ayurvedic oral or external measures under supervision	Progressive pain, non-cyclical pain, dyspareunia, dyschezia, infertility, heavy or irregular bleeding
Refractory or complex pain	Gynaecology evaluation; assess endometriosis, adenomyosis, pelvic floor dysfunction, and chronic pain mechanisms	Multidisciplinary care involving Ayurveda physician, gynaecologist, physiotherapist, pain specialist, nutrition professional, or mental-health support as required	Functional decline, repeated emergency visits, suspected secondary disease, or significant central sensitization

This stepped model keeps the patient's immediate relief and safety at the centre. Integration is most useful when it reduces fragmentation: one coordinated plan, clear responsibilities, agreed outcome measures, and scheduled reassessment.

DISCUSSION

The central argument of this review is that dysmenorrhoea is simultaneously a uterine, inflammatory, neurological, and lived pain experience. Prostaglandin excess remains the most actionable biomedical mechanism, but cytokine signalling, oxidative stress, peripheral sensitization, and altered central pain modulation help explain incomplete treatment response and progressive disability. The Ayurvedic description of *Udavartini Yonivyapad* adds a functional account centred on disturbed *Apana Vata*, painful expulsion, and relief after

menstrual flow. Its clinical strength lies in attending to movement, elimination, warmth, nourishment, sleep, stress, and individual variability.

The risk of integrative writing is oversimplification. Prostaglandin excess is not synonymous with *Vata Prakopa*, and central sensitization is not proof of *Apana Vata Dushti*. A more defensible approach is to identify overlapping clinical phenomena while retaining conceptual boundaries. This makes integration testable. For example, an intervention intended to promote *Vata Anulomana* can be evaluated against pain intensity, pain interference, rescue medicine, uterine blood-flow measures, bowel symptoms, cytokines, heart-rate variability, or quantitative sensory testing.

Another risk is diagnostic delay. Endometriosis affects an estimated 10% of reproductive-age women and commonly presents with severe dysmenorrhoea, chronic pelvic pain, dyspareunia, bowel or urinary pain, fatigue, or infertility.[36] A patient whose pain is worsening, atypical, or resistant to first-line care should not receive repeated empirical treatment without reassessment. Integrative practice becomes clinically credible when it identifies referral needs early and continues supportive care alongside appropriate investigation.

The available Ayurvedic studies are valuable as early clinical signals, but most are limited by small samples, open-label designs, poorly described randomization, non-standard outcomes, short follow-up, and incomplete safety reporting. Future studies should compare well-defined adjunctive Ayurvedic care plus standard management against standard management alone. They should register protocols prospectively, use concealed allocation, blinded outcome assessment where feasible, validated pain and quality-of-life instruments, adequate follow-up, rescue-medication recording, and transparent adverse-event reporting.

Research Priorities

Future research should prioritize.

1. consensus diagnostic criteria distinguishing primary dysmenorrhoea, *Udavartini Yonivyapad*, and secondary pelvic disease;
2. development of a reproducible Ayurvedic phenotyping tool for *Apana Vata Dushti*;
3. adequately powered randomized controlled trials with standard-care comparators;
4. core outcomes including pain intensity, pain interference, school or work absence, sleep, quality of life, bleeding, rescue medication, and recurrence;

5. mechanistic substudies examining prostaglandins, cytokines, oxidative stress, uterine perfusion, autonomic function, pelvic-floor activity, and sensory testing;
6. pharmacovigilance, product standardization, contaminant testing, and herb–drug interaction monitoring;
7. pragmatic trials of low-risk combinations such as heat, yoga, sleep regulation, dietary regularity, and individualized oral Ayurvedic therapy; and
8. longer follow-up to determine whether integrative care changes pain trajectories rather than only reducing pain during one cycle.

CONCLUSION

Dysmenorrhoea should not be normalized when it repeatedly disrupts daily life. Contemporary science describes a pathway beginning with endometrial prostaglandins and uterine ischaemia but extending into cytokine signalling, peripheral sensitization, and central pain modulation. Ayurveda describes a comparable clinical pattern as *Udavartini Yonivyapad*, in which disturbed *Apana Vata* produces painful or difficult menstruation and relief after the flow is established. These perspectives can enrich one another when their conceptual differences are respected.

A responsible integrative approach begins with exclusion of secondary disease and access to effective pain relief. It then combines education, correctly used pharmacological treatment, heat, regular physical activity, yoga or breathing practices, sleep and bowel regulation, and individualized Ayurvedic care under qualified supervision. Existing Ayurvedic studies are promising but insufficient for claims of cure or equivalence to established treatment. The next step is not broader assertion, but better research—clinically relevant, mechanistically informed, methodologically rigorous, and centred on the patient's ability to live, study, work, and menstruate without disabling pain.

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