Research paper

Lifestyle-Based Interventions for Menstrual Pain in Obese Young Women: A Narrative Review of Physiological Mechanisms and Rehabilitation Outcomes

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ABSTRACT

Background: Primary dysmenorrhea—painful menstrual cramping without underlying pelvic disease—frequently affects young women. While its traditional pathology stems from heightened prostaglandin release, uterine hypercontractility, and localized ischemia (restricted blood flow), obesity can significantly compound these symptoms. In young, obese females, painful menstruation is often exacerbated by systemic low-grade inflammation, disrupted estrogen metabolism, insulin resistance, compromised pelvic blood flow, sedentary habits, and lumbopelvic mechanical issues.

Aims: This narrative review maps out the complex intersections among obesity, microbiome-driven inflammation, physical inactivity, lumbopelvic instability, and primary dysmenorrhea. Ultimately, it evaluates how holistic lifestyle rehabilitation can serve as a comprehensive management strategy for this specific population.

Methods: A descriptive and narrative synthesis was conducted by gathering literature on the complex intersections among obesity, microbiome-driven inflammation, physical inactivity, lumbopelvic instability, and primary dysmenorrhea and the effect of lifestyle modification, diet and exercise on obese females with primary dysmenorrhea. Systematic searches were deployed across electronic databases—including PubMed, Scopus, Web of Science, Cochrane Library, ClinicalTrials.gov, ScienceDirect, PEDro, SAGE Journals, and Google Scholar—using targeted terms such as primary dysmenorrhea, obesity, lifestyle modification, microbiome diet, aerobic exercise, core stability, and lumbar spine.

Results: The reviewed evidence indicates that lifestyle modification may target multiple interacting pathways involved in primary dysmenorrhea. Aerobic exercise may reduce menstrual pain through improved pelvic circulation, activation of endogenous analgesic mechanisms, reduction of inflammatory mediators, and improvement of metabolic regulation. Core stabilization exercises may contribute to pain reduction by enhancing lumbopelvic stability, postural control, lumbar mobility, and neuromuscular coordination. Microbiome-supportive dietary strategies may reduce gut dysbiosis, improve intestinal barrier integrity, increase short-chain fatty acid production, modulate estrogen metabolism, and attenuate systemic inflammation. Together, these mechanisms suggest that combining exercise-based rehabilitation with weight management and microbiome-targeted dietary modification may provide broader benefits than symptom-focused management alone.

Conclusion: Lifestyle-based interventions represent a safe, non-pharmacological, and multidimensional approach for managing primary dysmenorrhea in obese young females. Integrating aerobic exercise, core stabilization, weight management, and microbiome-supportive dietary strategies may improve pain, metabolic health, lumbopelvic function, and quality of life. Further well-designed randomized controlled trials are warranted to standardize intervention protocols, evaluate long-term outcomes, and clarify the mechanistic links among gut microbiota, adiposity, inflammation, posture, and menstrual pain.

Keywords: primary dysmenorrhea; obesity; lifestyle modification; microbiome diet; aerobic exercise; core stability; lumbar spine; quality of life.

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1. Introduction

Primary dysmenorrhea (PD) refers to painful menstrual episodes occurring in the absence of identifiable pelvic disease. The onset of discomfort typically coincides with or immediately precedes the start of menstrual bleeding, with symptoms lasting anywhere from 12 to 72 hours. This condition is generally characterized by sharp, cramping sensations localized in the lower abdomen, which frequently radiate outward to the lower back, pelvic floor, buttocks, or upper thighs. Beyond localized pain, individuals often experience systemic distress, including gastrointestinal disturbances (nausea, vomiting, and diarrhea), systemic fatigue, headaches, diaphoresis (excessive sweating), emotional fluctuations, and a distinct decline in daily operational capacity [1,2].

While PD does not pose a threat to mortality, its profound clinical significance lies in its capacity to disrupt academic focus, occupational productivity, physical endurance, sleep patterns, social interactions, and psychological health, ultimately diminishing overall quality of life. Despite these extensive disruptions, a significant portion of young women tend to normalize their menstrual discomfort or manage it independently via localized heat therapy, bed rest, or over-the-counter analgesics. This widespread reliance on self-treatment frequently delays necessary clinical evaluations and prevents access to holistic, long-term care [3,4].

Recent clinical insights highlight obesity as a critical, adjustable risk factor that can exacerbate both the incidence and intensity of PD. Heightened adipose tissue levels have been shown to alter ovarian steroid metabolism, accelerate the expression of pro-inflammatory cytokines, elevate prostaglandin synthesis, compromise insulin sensitivity, impair vascular elasticity, and reduce pelvic perfusion (blood flow), thereby heightening overall pain perception. Consequently, managing menstrual pain in obese patients requires moving away from the conventional, isolated use of prostaglandin-inhibiting analgesics. Instead, effective treatment must address the patient's broader metabolic, musculoskeletal, nutritional, and behavioral profiles [5,6].

Multidimensional lifestyle modification provides a comprehensive therapeutic blueprint by integrating structured physical rehabilitation, targeted dietary adjustments, weight stabilization, stress management, sleep optimization, and behavioral compliance strategies. For obese individuals suffering from PD, these combined interventions work synergistically to alleviate uterine pain, optimize body composition, correct lumbopelvic structural mechanics, elevate mechanical pain thresholds, and restore overall well-being. This narrative review explores the underlying biological mechanisms and clinical justification for deploying integrated lifestyle interventions within this patient cohort.

2. Methods:

A descriptive and narrative synthesis was conducted by gathering literature on the complex intersections among obesity, microbiome-driven inflammation, physical inactivity, lumbopelvic instability, and primary dysmenorrhea and the effect of life style modification, diet and exercise on obese females with primary dysmenorrhea.

A literature search was performed using Medical Subject Headings (MeSH) and related keywords, including "primary dysmenorrhea," "obesity," "lifestyle modification," "microbiome diet," "gut microbiome," "aerobic exercise," "core stability," "lumbopelvic dysfunction," and "lumbar spine." The searches were conducted across PubMed, Scopus, Web of Science, Cochrane Library, ClinicalTrials.gov, ScienceDirect, PEDro, SAGE Journals, and Google Scholar.

The review process spanned two months, from **October 2024 to November 2024**, and focused on articles published up to 2024, including only studies written in English. Additionally, references from the selected studies were screened to identify further relevant articles. To provide a comprehensive understanding of the topic, studies addressing menstrual physiology, obesity-related inflammation, gut microbiome modulation, aerobic exercise, core stabilization, postural changes, lumbopelvic dysfunction, and rehabilitation outcomes were included. The selected literature was descriptively analyzed and narratively summarized.

3. Menstrual Physiology and Pathophysiology of Primary Dysmenorrhea

3.1 Menstrual and ovarian cycle regulation

The menstrual cycle is regulated by the hypothalamic–pituitary–ovarian axis, which controls follicular development, ovulation, and cyclic estrogen and progesterone production. Estrogen promotes endometrial growth, while progesterone maintains the secretory endometrium after ovulation. If pregnancy does not occur, the decline in these hormones triggers menstruation [7,8].

Menstruation involves endometrial shedding, spiral artery vasoconstriction, inflammatory mediator release, and tissue repair. Although this is a normal physiological process, excessive prostaglandin and inflammatory activity may increase uterine contractions, reduce blood flow, and contribute to primary dysmenorrhea [9].

3.2 Prostaglandins, ischemia, and pain generation

Primary dysmenorrhea is primarily driven by a drop in progesterone, which triggers the endometrium to overproduce prostaglandins F2 alpha and E2. These

chemicals cause violent uterine contractions, elevated internal pressure, and severe blood vessel constriction. The resulting lack of oxygenated blood flow (ischemia) leads to a buildup of anaerobic metabolites that irritate local nerve endings, causing severe pelvic cramping [10,11].

However, individual pain levels vary based on broader inflammatory and neurological factors. Elevated cytokines, oxidative stress, and vasopressin further intensify these contractions, while recurrent monthly pain can induce central sensitization, rewiring the nervous system to amplify pain signals. Over time, this chronic discomfort forces physical adaptations, leading to protective muscle guarding, altered movement patterns, and heightened sensitivity across the entire lumbopelvic region[11].

4. Obesity as a Modifying Factor in Primary Dysmenorrhea (Fig.1)

4.1 Adipose tissue inflammation and prostaglandin activity

Obesity promotes chronic low-grade inflammation through enlarged adipocytes and immune cell infiltration. This increases pro-inflammatory cytokines such as TNF-α and IL-6, which may stimulate COX activity and prostaglandin production. As prostaglandins increase uterine contractions and pain, obesity-related inflammation may worsen primary dysmenorrhea [5,12].

Inflammation may also lower pain thresholds by sensitizing nociceptors. Therefore, anti-inflammatory lifestyle strategies, including exercise, weight control, and dietary modification, may be clinically useful [5].

4.2 Hormonal imbalance and insulin resistance

Adipose tissue functions as an active endocrine organ, using aromatase activity to convert androgens into estrogens. In obese individuals, this expanded fat mass can trigger relative estrogen dominance and disrupt the hypothalamic-pituitary-ovarian axis. This excess estrogen fuels endometrial proliferation, leading to greater prostaglandin release during menstruation [5,6].

This hormonal imbalance is often worsened by visceral adiposity-driven insulin resistance. The resulting hyperinsulinemia lowers sex hormone-binding globulin, increasing free androgens and disrupting ovarian steroidogenesis. Together, metabolic inflammation and elevated insulin levels intensify menstrual symptoms and prolong pain perception, supporting the use of insulin-sensitizing strategies like exercise, weight loss, and high-fiber diets [6,13].

4.3 Central obesity, pelvic blood flow, and symptom severity

Central adiposity, typically measured via waist circumference or waist-to-hip ratio, is more closely linked to metabolic and vascular dysfunction than body mass index (BMI) alone. Excess visceral fat impairs endothelial function and reduces tissue perfusion, altering pelvic circulation. During menstruation, this restricted pelvic blood flow intensifies uterine ischemia, directly worsening pain severity [13,14].

Consequently, monitoring both BMI and waist-to-hip ratio is clinically valuable for obese females with primary dysmenorrhea. Reductions in these metrics indicate more than just weight loss; they reflect a decrease in systemic inflammatory and vascular risks, which directly contributes to relieving menstrual pain.

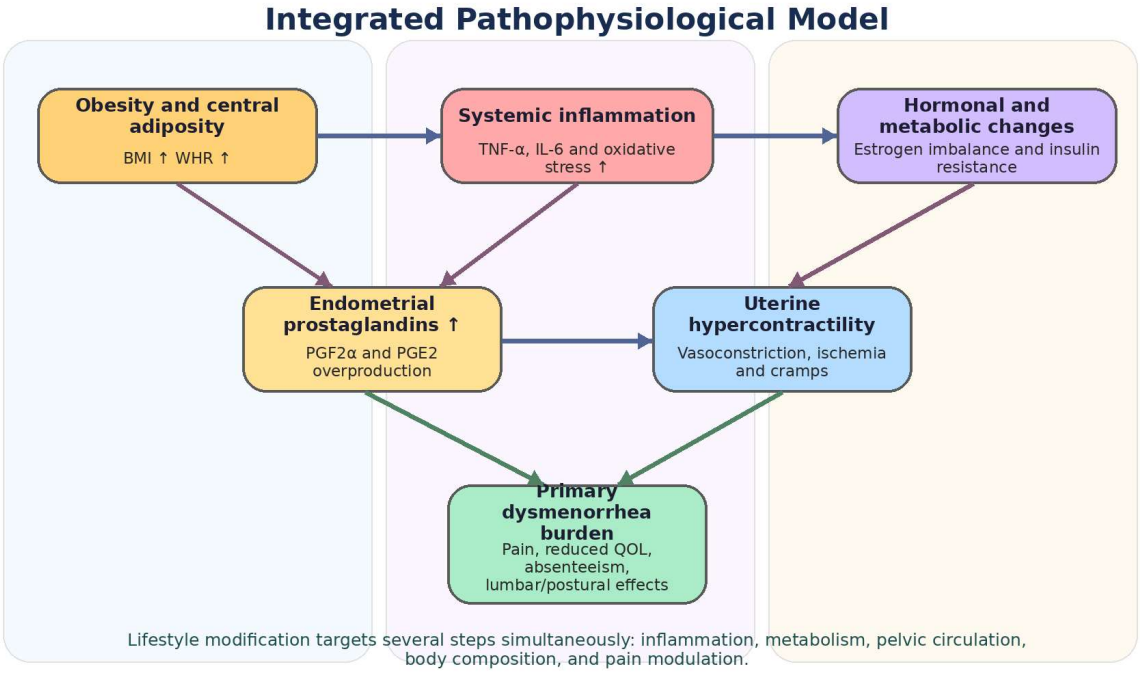

Fig. 1: Integrated pathophysiological model linking obesity, inflammation, hormonal-metabolic changes, prostaglandin activity, uterine ischemia, and primary dysmenorrhea burden.

5. Physical Activity and Exercise-Based Management

5.1 Aerobic exercise and endogenous pain modulation

Regular physical activity is inversely associated with the severity of dysmenorrhea. Aerobic exercise mitigates menstrual pain through multiple physiological pathways, including enhancing pelvic circulation, reducing uterine ischemia, and boosting local tissue oxygenation. Additionally, cardiovascular training stimulates the release of endogenous opioids like endorphins, optimizes autonomic nervous system balance, and curbs stress-induced pain amplification [15-17].

Aerobic conditioning also drives positive systemic adaptations by improving body composition, lipid metabolism, insulin sensitivity, vascular health, and systemic inflammatory profiles. These systemic benefits are especially critical for obese females, whose underlying metabolic dysfunction can amplify menstrual pain. Practical modalities like brisk walking, cycling, swimming, jogging, and structured aerobic classes can be seamlessly integrated into rehabilitation programs, with intensity progressed based on patient tolerance and phase-specific menstrual symptoms [17].

5.2 Core stability exercises and lumbopelvic control

Core stabilization targets the deep and superficial musculature supporting the trunk and pelvis, including the transversus abdominis, rectus abdominis, internal and external obliques, multifidus, diaphragm, pelvic floor, and hip stabilizers. Standard clinical interventions utilize exercises such as pelvic bridging, the cat-and-camel stretch, planks, curl-ups, and single- or double-leg abdominal presses to restore lumbopelvic stability, neuromuscular coordination, spinal alignment, and dynamic movement control [18,19].

In individuals with primary dysmenorrhea, recurrent pelvic and lower back pain frequently triggers protective muscle guarding and disrupts the normal activation patterns of deep stabilizing muscles. Core stabilization exercises alleviate mechanical strain on the lumbar spine and pelvis, improve localized blood flow, and reinforce descending pain-inhibitory pathways via enhanced proprioceptive and motor feedback. When combined with aerobic training, core exercises address both the systemic metabolic and local mechanical contributors to painful menstruation [19].

6. Postural and Lumbar Spine Considerations

Primary dysmenorrhea is frequently associated with lumbopelvic discomfort and may be accompanied by postural adaptations such as increased lumbar lordosis, anterior pelvic tilt, altered pelvic alignment, and changes in thoracic posture. These changes may occur as protective responses to recurrent pain or may contribute mechanically to symptom persistence [20,21].

Biomechanically, altered pelvic alignment can increase tension in pelvic ligaments, affect uterine position, limit pelvic lymphatic drainage, and reduce local blood flow. Lumbar hyperlordosis and reduced lumbar mobility may increase mechanical loading in the lower back and pelvis, intensifying pain during menstruation. Pain-related anxiety and muscle tension may further reinforce antalgic posture and impaired postural control [20-22].

Assessment of lumbar mobility and lordotic angle can therefore provide clinically useful information. In the thesis intervention, lumbar mobility was assessed by the modified Schober test, and lumbar lordotic curve was assessed using a flexible ruler. These measures reflect the musculoskeletal dimension of PD and support the integration of exercise therapy within menstrual pain management [22].

7. Gut Microbiome, Diet, and Menstrual Pain

7.1 Diet-microbiome interaction and inflammatory regulation

The gut microbiome contributes to metabolic control, immune regulation, gut barrier integrity, and inflammation. Diet is one of the main factors shaping microbial balance. High-fiber, plant-based diets increase the production of short-chain fatty acids, including butyrate, acetate, and propionate, which support gut barrier function, reduce endotoxemia, regulate cytokines, and improve metabolic health. In contrast, diets rich in refined sugar, saturated fat, and ultra-processed foods may promote dysbiosis, intestinal permeability, and chronic low-grade inflammation. Since both obesity and primary dysmenorrhea are associated with inflammation, microbiome-supportive dietary strategies may help reduce menstrual pain and improve overall health [23–25].

7.2 Estrobolome and reproductive implications

The gut microbiome may influence estrogen metabolism through the estrobolome, a group of microbial genes involved in estrogen processing. When dysbiosis occurs, estrogen recycling may be disturbed, leading to changes in circulating estrogen levels. These changes may affect endometrial growth, prostaglandin release, and menstrual symptoms. Although this mechanism is still being studied, it suggests a possible link between diet, gut microbiota, hormonal regulation, and primary dysmenorrhea [26,27].

7.3 Microbiome diet as a lifestyle component

A microbiome-supportive diet focuses on reducing processed foods, added sugars, artificial additives, and excessive saturated fat, while increasing prebiotic fibers, vegetables, fruits, legumes, whole grains, fermented foods, and lean protein sources [23–25].

Clinically, this diet should be viewed as part of a wider lifestyle program rather than as a stand-alone cure. Nutritional advice should be individualized, culturally appropriate, nutritionally balanced, and realistic for long-term use, especially for young women with limited resources or restrictive eating habits [28,29].

8. Lifestyle Modification as an Integrated Therapeutic Strategy

Lifestyle modification is a structured behavioral approach designed to improve health outcomes through sustainable changes in daily habits. In PD, relevant components include aerobic exercise, core stabilization, anti-inflammatory and microbiome-supportive dietary

patterns, weight management, sleep regulation, stress reduction, and patient education as shown in **Fig.2** [28,29].

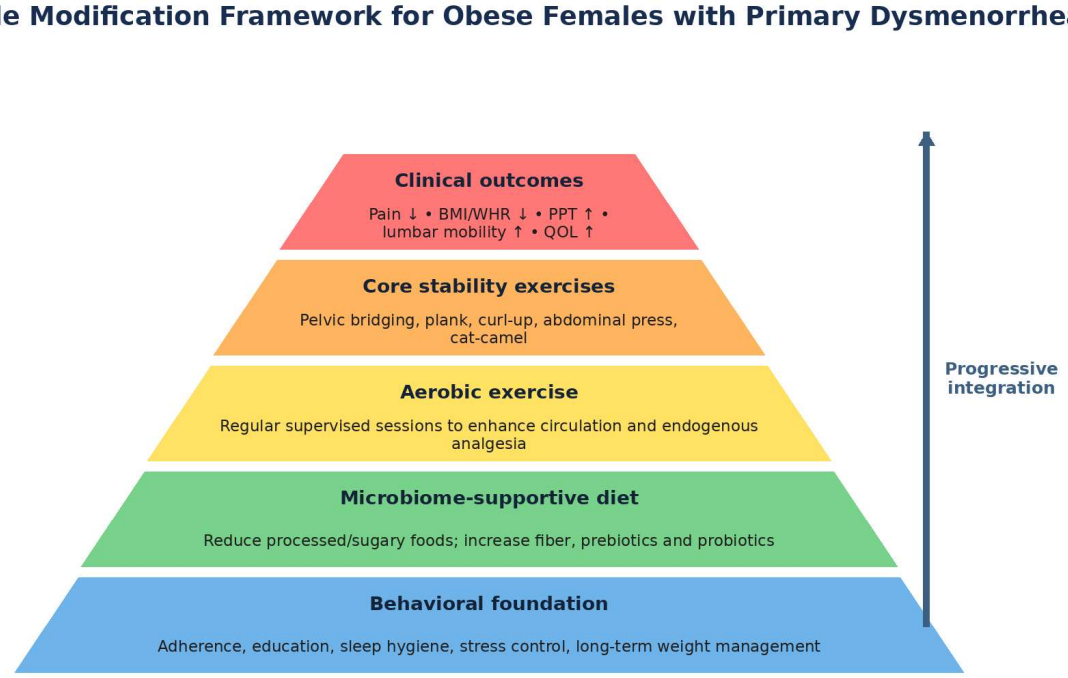

Fig. 2: lifestyle modification framework showing the progressive integration of behavioral foundations, microbiome-supportive diet, aerobic exercise, core stability, and clinical outcomes.

This approach is clinically attractive because it targets multiple interacting mechanisms rather than a single symptom pathway as shown in **Fig. 3**. Exercise can improve circulation, endogenous analgesia, mood, posture, and body composition. Diet can influence inflammation, microbiome composition, metabolic function, and hormonal regulation. Weight management can reduce adipose-derived cytokines and improve vascular and endocrine function. Education can improve self-efficacy and reduce normalization of severe menstrual pain.

Compared with pharmacological management, lifestyle modification has a favorable safety profile and may reduce dependence on repeated analgesic use. Nonsteroidal anti-inflammatory drugs and hormonal contraceptives remain effective options for many women, but they may be associated with gastrointestinal side effects, contraindications, hormonal concerns, or patient preference for non-pharmacological strategies. A combined model that includes medical screening and lifestyle intervention may therefore be most appropriate.

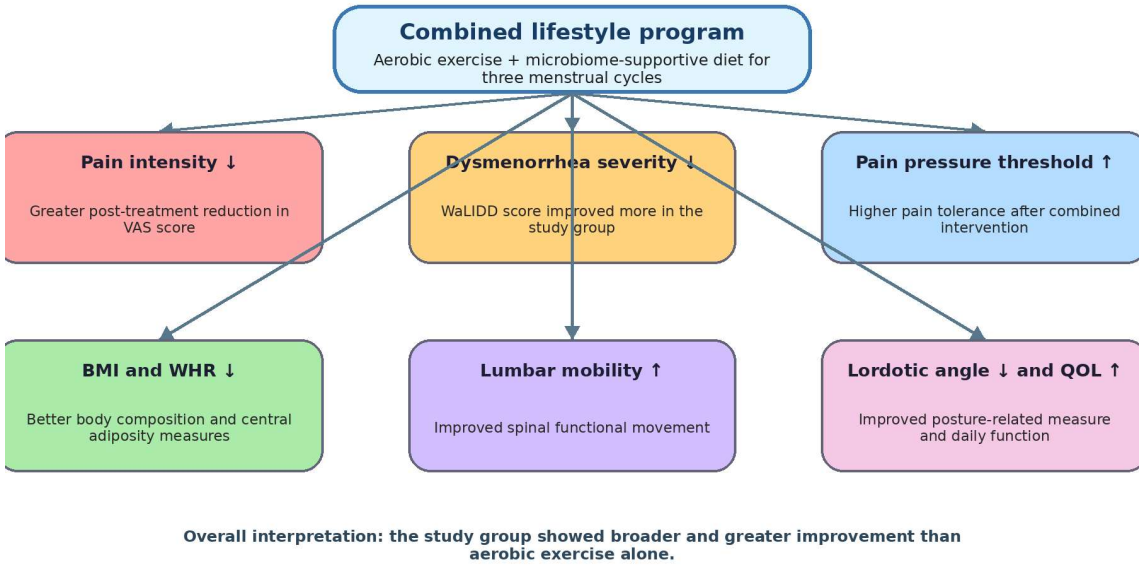

Fig. 3: Summary of thesis-based outcomes after three menstrual cycles, highlighting the greater improvements reported in the combined aerobic exercise and microbiome diet group.

9. Discussion

The available literature supports a multidimensional understanding of primary dysmenorrhea in obese females. Although menstrual pain is mainly driven by prostaglandin-mediated uterine hypercontractility and uterine ischemia, its severity may also be shaped by systemic inflammation, excess adiposity, hormonal imbalance, insulin resistance, physical inactivity, altered pain modulation, and lumbopelvic dysfunction. This may explain why approaches that target only one pathway often provide incomplete or temporary relief.

Current evidence suggests that lifestyle modification may be valuable because it addresses several contributors to pain at the same time. Aerobic exercise may improve pelvic circulation, enhance endogenous analgesic activity, support metabolic health, reduce inflammatory mediators, and improve psychological wellbeing. Core stabilization may help improve lumbopelvic control, spinal mobility, posture, and neuromuscular coordination. Dietary modification, particularly anti-inflammatory and microbiome-supportive dietary patterns, may reduce inflammatory load, improve gut-derived metabolites, support intestinal barrier function, and contribute to better hormonal and metabolic regulation.

Another important point in the available literature is that primary dysmenorrhea should not be evaluated by pain intensity alone. Functional limitation, pain sensitivity, body composition, lumbar mobility, postural alignment, and quality of life may provide a broader picture of the patient's condition and treatment response. Therefore, multidimensional outcome assessment is more consistent with contemporary rehabilitation practice, where menstrual pain is understood as a condition influenced by gynecological, musculoskeletal, metabolic, behavioral, and quality-of-life factors.

However, several clinical considerations remain. Lifestyle interventions require adherence, supervision, and individualized progression. Dietary programs should be nutritionally adequate, culturally acceptable, and should avoid unnecessary restrictions. Exercise prescriptions should consider menstrual cycle symptoms, baseline fitness, pain severity, and patient preference. In addition, more high-quality studies with long-term follow-up are needed to determine whether the benefits of lifestyle modification are maintained beyond the supervised intervention period.

11. Clinical Implications

Clinicians managing obese females with primary dysmenorrhea should use a comprehensive assessment approach. This should include menstrual history, pain characteristics, medication use, physical activity level, body mass index, waist-to-hip ratio, posture, lumbar mobility, pressure pain threshold, and quality of life. Screening for secondary dysmenorrhea and other red flags remains essential before starting conservative management.

Rehabilitation programs may benefit from integrating aerobic exercise, core stabilization, and individualized dietary counseling. Patient education is also important, as severe menstrual pain should not be dismissed as a normal part of menstruation. Women should be informed that modifiable lifestyle factors, including physical activity, weight management, diet, sleep, and stress, may influence symptom severity.

Interdisciplinary care may improve outcomes. Collaboration among physiotherapists, gynecologists, dietitians, and primary care providers can help ensure accurate diagnosis, safe intervention, better adherence, and more individualized care.

Outcome monitoring should include both symptom-based and functional measures. Pain intensity alone may not reflect meaningful changes in daily activity, pain sensitivity, posture, metabolic health, or quality of life.

Measures such as VAS, WaLIDD, body mass index, waist-to-hip ratio, modified Schober test, lordotic angle measurement, pressure pain threshold, and SF-36 may provide a practical multidimensional assessment framework.

12. Conclusion

Primary dysmenorrhea in obese females is influenced by several interacting reproductive, inflammatory, metabolic, vascular, neuromuscular, and psychosocial factors. Obesity may worsen menstrual pain through adipose-derived inflammation, hormonal imbalance, insulin resistance, central adiposity, impaired pelvic circulation, and altered pain sensitivity. Therefore, lifestyle modification is a clinically reasonable approach because it can address multiple contributing mechanisms at the same time.

Current evidence suggests that aerobic exercise, core stabilization, microbiome-supportive dietary modification, and weight management may help reduce pain intensity and dysmenorrhea severity while improving pressure pain threshold, body composition, lumbar mobility, postural alignment, and quality of life. However, further high-quality randomized controlled trials are needed to standardize intervention

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