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Role Of Homoeopathy In The Management Of Dysmenorrhoea: A Review

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Abstract:
Dysmenorrhoea is one of the most prevalent gynecological complaints among women of reproductive age and is a leading cause of absenteeism from school and work. It is characterized by painful menstruation severe enough to interfere with daily activities and is classified into primary and secondary types based on etiology. Primary dysmenorrhoea occurs in the absence of pelvic pathology and is mainly attributed to increased prostaglandin secretion, leading to uterine hypercontractility and ischemic pain. Secondary dysmenorrhoea is associated with underlying pelvic disorders such as endometriosis, adenomyosis, fibroids, and pelvic inflammatory disease. This article reviews the epidemiology, etiopathogenesis, and pathophysiology of dysmenorrhoea, highlighting key biochemical mechanisms involved in pain generation. Furthermore, a homoeopathic approach to the management of dysmenorrhoea is discussed, emphasizing commonly indicated remedies based on characteristic symptomatology. The repertorial perspective is also presented to aid individualized remedy selection. A holistic homoeopathic approach offers a promising, patient-centered strategy for effective management of dysmenorrhoea.

Keywords:
Dysmenorrhoea, Primary dysmenorrhoea, Secondary dysmenorrhoea, Menstrual pain, Homoeopathy, Therapeutics, Repertory

Introduction:

Dysmenorrhoea is derived from Greek words meaning “painful monthly bleeding”.[(1)] Dysmenorrhoea is one of the most common gynecological complaints among women of reproductive age and is a frequent cause of absenteeism from school and work. It is defined as painful menstruation severe enough to interfere with normal daily activities.[(2)] Dysmenorrhoea is a frequent concern in menstruating individuals throughout the reproductive period. It can be associated with substantial emotional, psychological, and functional health consequences.[(5)]
Based on etiology and clinical characteristics, dysmenorrhoea is classified into primary and secondary types.[(2)] Primary dysmenorrhoea refers to recurrent lower abdominal pain occurring during the menstrual cycle in the absence of any underlying pelvic pathology or associated disease.[(3)] Primary dysmenorrhoea typically develops within two years of menarche, coinciding with the establishment of ovulatory cycles. It is most commonly diagnosed in adolescents and young adults. The pain is cyclic, beginning within a few hours after the onset of menses and generally subsiding within 72 hours. It is usually midline pelvic in location and may radiate to the lumbar region or upper legs.[(4)] The pain is often crampy and episodic, with a consistent pattern in each

menstrual cycle. Associated symptoms may include nausea, vomiting, headache, dizziness, fatigue, and sleep disturbances.[6] Conversely, secondary dysmenorrhoea occurs in association with identifiable or suspected pelvic pathology. [2]

Etiology:

Since the 1960s, multiple theories have been proposed to explain the etiology of dysmenorrhoea, including psychological, biochemical, and anatomical mechanisms. The anatomical theory emphasizes abnormal uterine position and variations in cervical shape or length. However, evidence from several studies indicates that the biochemical theory has the strongest scientific support.[6]

Several risk factors have been associated with dysmenorrhoea, including age (commonly up to 30 years), smoking, attempts to lose weight, abnormal body mass index, depression or anxiety, longer menstrual cycles, early age at menarche, nulliparity, history of sexual assault, previous cesarean section with incomplete uterine scar healing (uterine niche), prolonged and heavy menstrual flow, positive family history of dysmenorrhoea, and disruption of social networks. [7][8]

(i) Primary Dysmenorrhoea: Prostaglandins (PGs) are considered the primary mediators of dysmenorrhoeal[14]. Increased concentrations of PGs have been identified in the menstrual fluid and endometrial tissue of women affected by dysmenorrhoeaV. Endometrial shedding is initiated by declining hormone levels during the menstrual cycle. At the onset of menstruation, endometrial cells release PGs, which induce uterine contractions. The severity of cramping correlates with the quantity of PGs released. These uterine contractions lead to tissue hypoxia and ischemia, resulting in pain and, in some cases, associated symptoms such as nausea and diarrhea[4].

(i) Secondary Dysmenorrhoea: Secondary dysmenorrhoea refers to menstrual pain resulting from an underlying disease, disorder, or structural abnormality located within or outside the uterus[2]. It may occur at any time following menarche and can present as a new symptom in women in their 30s or 40s. The pain intensity varies and may be accompanied by additional symptoms such as dyspareunia, menorrhagia, intermenstrual bleeding, and postcoital bleeding. Common causes of secondary dysmenorrhoea include endometriosis, large cesarean scar niche, fibroids, adenomyosis, endometrial polyps, interstitial cystitis, pelvic inflammatory disease, and possible use of an intrauterine contraceptive system. [14]

Epidemiology:

Dysmenorrhoea is among the most prevalent gynecological complaints in menstruating individuals, irrespective of age or race, and is a common cause of pelvic pain. Its prevalence in the reproductive age group ranges from 16% to 91%, while severe dysmenorrhoea is reported in approximately 2% to 29% of affected individuals.[8]

Pathophysiology:

The pathophysiology of primary dysmenorrhoea remains incompletely understood; however, it is primarily attributed to hypersecretion of prostaglandins (PGs) from the uterine inner lining. PGs induce pain by increasing uterine contractions and intrauterine pressure. Reduced uterine perfusion, ischemia, hypoxia, and accumulation of anaerobic metabolic byproducts may also contribute to pain generation[9]. During menstruation, decreased levels of progesterone and estradiol are associated with increased activity of collagenases, inflammatory cytokines, and matrix metalloproteinases in the endometrium. Subsequent endometrial breakdown releases phospholipids, which are converted into arachidonic acid[10]. Arachidonic acid is further metabolized via cyclooxygenase into prostacyclins, PGs, and thromboxane-2a [11].

The prostaglandin derivatives PGF-2α and PGE2 enhance uterine tone and produce high-amplitude uterine contractions. Expression of cyclooxygenase-2 (COX-2) is markedly elevated during menstruation, providing the rationale for NSAID use in treatment[10]. Vasopressin has also been implicated in primary dysmenorrhoea,

as it increases uterine contractility and may induce ischemic pain through vasoconstriction.[12] Elevated levels of leukotrienes C4 and D4 have been observed in patients with dysmenorrhoea and are associated with increased uterine contractions[9]. Uterine contractility is greatest during the first two days of menstruation, corresponding to the period when dysmenorrhoea is most frequent and severe.[13]

Endometriosis and adenomyosis are the leading causes of secondary dysmenorrhoea in premenopausal women.[13]

Homoeopathic Approach:

(i) Belladonna

- Menstrual discharge bright red, feeling very hot like hot sealing-wax. Metrorrhagia of clear red blood, with a discharge of fetid clots; with violent pain in the small of the back and bearing-down. Menstrual blood of bright colour, or of a bad smell [17]
- Cutting pain from hip to hip. Menses and lochia very offensive and hot[16].
- Sensitive forcing downwards, as if all the viscera would protrude at genitals[16].
- Dryness and heat of vagina[16].
- Dragging around loins. Pain in sacrum[16].

(ii) Cactus Grandiflorus

- Menses early, dark, pitch-like; cease on lying down, with heart symptoms[16].
- Very painful menstruation; extorting loud cries. [17]
- Constriction in uterine region and ovaries[16].
- Dysmenorrhoea; pulsating pain in uterus and ovaries[16].

(iii) Calcerea Phosphorica

- Menses too early, excessive, and bright in girls[16].
- If late, blood is dark; sometimes, first bright, then dark, with violent backache[16].
- Labour-like pains[17]

(iv) Chamomilla

- Menstrual colic, before the catamenia[17].
- Pressure towards the uterus, as if from the pains of child-birth[17].
- Metrorrhagia, with discharge of deep-red blood, and of clots, accompanied by labour pains[17].
- Discharge of blood between the regular catamenia[17].
- Burning pains and smarting in the vagina[17].

(v) Cimicifuga Racemosa

- Menses profuse, dark, coagulated, offensive with backache, nervousness; always irregular[16].
- Pain immediately before menses[16].
- Ovarian neuralgia. Pain in ovarian region; shoots upward and down anterior surface of thighs[16].
- Pain across pelvis, from hip to hip[16].
- After-pains, with great sensitiveness and intolerance to pain[16].

(vi) Kalium Carbonicum

- Menses early, profuse or too late, pale and scanty, with soreness about genitals; pains from back pass down through gluteal muscles, with cutting in abdomen[16].
- Pain through left labium, extending through abdomen to chest[16].
- Delayed menses in young girls, with chest symptoms or ascites[16].
- Difficult, first menses[16].
- Complaints after parturition.
- Uterine haemorrhage; constant oozing after copious flow, with violent backache, relieved by sitting and pressure[16].

(vii) Psorinum

- Menses too late; and scanty.
- Dysmenorrhoea; near climaxis.

Repertorial View:

As Per Kent Repertory-

Female Genitalia- Menses- painful, dysmenorrhœa

Abrot., *acon.*, agar., alet., alum., *am-c.*, anan., ant-c., apis., arg-n., ars-i., *ars.*, asar., bar-c., bar-m., **Bell.**, *berb.*, *bor.*, bov., brom., bry., **Cact.**, **Calc-p.**, *calc.*, canth., carb-an., carb-s., carb-v., *caul.*, *caust.*, **Cham.**, chin-a., chin., *cic.*, **Cimic.**, cinnb., *cocc.*, *coff.*, coll., *coloc.*, *con.*, *croc.*, crot-c., cupr., *cycl.*, *dios.*, *dulc.*, euphr., ferr-ar., ferr-i., ferr-p., ferr., *gels.*, goss., *graph.*, grat., ham., *helon.*, hyos., hyper., *ign.*, iod., ip., *kali-ar.*, kali-bi., **Kali-c.**, *kali-i.*, kali-n., *kali-p.*, *kali-s.*, kalm., kreos., *lac-c.*, *lach.*, *lap-a.*, laur., led., *lil-t.*, lob., *lyc.*, mag-c., mag-m., mag-s., mang., *med.*, *meli.*, *merc.*, *merl.*, mosch., mur-ac., murx., *nat-c.*, nat-m., nat-p., nat-s., nicc., nit-ac., nux-m., *nux-v.*, ol-an., petr., ph-ac., *phos.*, phyt., *plat.*, plb., podo., **Psor.**, *puls.*, *rhus-t.*, *sabin.*, sang., sars., *sec.*, *senec.*, *sep.*, sil., spong., stram., sul-ac., *sulph.*, ther., thuj., *tub.*, *verat.*, vib., *xan*[18].

Conclusion:

Dysmenorrhoea remains a significant health concern affecting the physical, emotional, and social well-being of menstruating individuals. Understanding its multifactorial etiology and underlying pathophysiological mechanisms is essential for effective management. While conventional treatment primarily focuses on symptomatic relief using NSAIDs and hormonal therapy, homoeopathy offers an individualized, holistic approach addressing the root cause and overall constitution of the patient. Remedies such as Belladonna, Cactus grandiflorus, Cimicifuga racemosa, and others, when prescribed based on totality of symptoms, can provide significant relief. The repertorial approach further supports accurate remedy selection. Thus, homoeopathy plays a valuable role in the comprehensive management of dysmenorrhoea, improving quality of life and reducing dependency on conventional analgesics.

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