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Review Article

A Review on Dysmenorrhea

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ABSTRACT

Dysmenorrhea is the most common gynaecologic complaint among adolescent and young adult females. Dysmenorrhea in adolescents and young adults is usually primary (functional), and is associated with normal ovulatory cycles and with no pelvic pathology. In approximately 10% of adolescents and young adults with severe dysmenorrhea symptoms, pelvic abnormalities such as endometriosis or uterine anomalies may be found. Potent prostaglandins and potent leukotrienes play an important role in generating dysmenorrhea symptoms. Nonsteroidal anti-inflammatory drugs (NSAID) are the most common pharmacologic treatment for dysmenorrhea. Adolescents and young adults with symptoms that do not respond to treatment with NSAIDs for 3 menstrual periods should be offered combined oestrogen/progestin oral contraceptive pills for 3 menstrual cycles. Adolescents and young adults with dysmenorrhea who do not respond to this treatment should be evaluated for secondary causes of dysmenorrhea. The care provider's role is to explain about pathophysiology of dysmenorrhea to every adolescent and young adult female, address any concern that the patient has about her menstrual period, and review effective treatment options for dysmenorrhea with the patient.

INTRODUCTION

Painful menstruation, or dysmenorrhea, is the most prevalent gynaecological disorder affecting women. The reported prevalence ranges from 17% to 90%, which is a significant variation. While some women have very little pain during their periods, others have severe functional limitations.[1] It only manifests during ovulatory cycles, therefore the first sign typically arises six months following menarche. Usually, the pain lasts eight to seventy-two hours. Most intense during the first and second days of the menstrual cycle because heightened prostaglandin release throughout this time frame. What's The symptoms are consistent from one menstrual cycle to one more. It is linked

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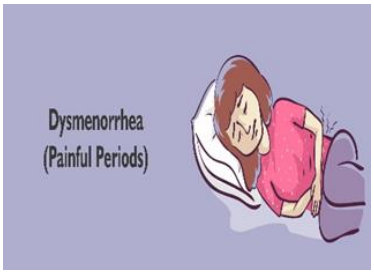
to low blood sugar, diarrhoea, vomiting, and headaches, exhaustion, sleeplessness, dizziness, back pain, and seldom, as well as syncope and fever.[2]Up to 15% of women who suffer with dysmenorrhea have symptoms that are severe enough to keep them from going to work, school, and other events.[3] This issue may be lessened by flexible scheduling or the option to work from home, but even for women who do not miss work or school due to menstrual-related symptoms, performance is adversely affected by the decreased attention and productivity brought on by such symptoms.[3] Dysmenorrhea, which literally translates to "difficult menstrual flow," is a Greek phrase. This is a typical adolescent menstruation problem. The symptoms of dysmenorrhea during this stage of life include painful periods that typically begin two to three years following menarche, when ovulation begins. A woman's frequent absences from job, school, or other activities are often caused by this. It may worsen overall wellbeing and life quality.[4] The evaluation and diagnosis of women with primary dysmenorrhea does not require specialization in women's health or pelvic pain. In many cases, a diagnosis of primary dysmenorrhea can be reached by obtaining a detailed medical, psychosocial, and gynecologic (including menstrual and sexual) history.[7] Uterine hypercontractility, increased peripheral nerve hypersensitivity, and decreased uterine blood flow cause discomfort. The uterus is made to contract erratically and repeatedly, which raises basal tone and active pressure. When omitting side effects including headache, indigestion, and drowsiness, the failure rate of non-steroidal anti-inflammatory medications, which are currently mostly used to treat primary dysmenorrhea, can reach 20% to 25%. Thus, it is essential to look for an alternate therapy to help ladies who are experiencing menstrual discomfort.[8] Although primary dysmenorrhea has long been regarded as an epiphenomenon, it has gained more attention recently due to the identification of prostaglandins' crucial involvement in myometrial hypercontractility and arterial vasoconstriction. Additionally, it has been shown that some non-steroidal anti-inflammatory medications (NSAIDs) called propionates are surprisingly effective in treating this teenage illness. Teenage care providers have a crucial responsibility to assess and treat patients with dysmenorrhea, as well as to educate teenage females about the symptoms related with menstruation.[9]



Epidemiology

Primary dysmenorrhea is the most common gynaecological disease in menstruating women. Its prevalence is more significant during the second and third decades of life and decreases with advancing age, unlike secondary dysmenorrhea.[2] Its prevalence is underestimated and difficult to determine because only a small number of women seek medical treatment. Contributing to this are the different definitions of dysmenorrhea that exist and the lack of standard methods for defining its severity. According to a systematic review by the World Health Organization in 2006, the prevalence of dysmenorrhea in menstruating women is between 17 and 81%. Severe dysmenorrhea was identified in only 12 to 14% of cases.[2]

Types

On the basis of pathophysiology, dysmenorrhea is classified as primary dysmenorrhea (menstrual pain without organic disease) or secondary dysmenorrhea (menstrual pain associated with underlying pelvic pathology) .[5] Primary dysmenorrhea is menstrual pain that does not have

an underlying cause, while secondary dysmenorrhea is pain brought on by diseases such interstitial cystitis, endometriosis, pelvic inflammatory disease, and leiomyomas. Secondary dysmenorrhea is treated by addressing the underlying pelvic disease or illness. Most ovulatory women who experience painful menses have primary dysmenorrhea. [3]

-Menstrual pain that is linked to regular ovulatory cycles, free of pelvic disease, and with a definite physiological cause is known as primary dysmenorrhea (PD). Young adults and adolescents are more likely to experience it.[6]

- Menstrual pain linked to a specific illness (endometriosis, fibroids, adenomyosis, pelvic adhesions, endometrial polyps, pelvic inflammatory disease) or the use of an intrauterine contraceptive device is known as secondary dysmenorrhea.[6]

Primary Dysmenorrhoea	Secondary Dysmenorrhoea
Onset shortly after menarche	Onset can occur at any time after menarche (typically, after 25 years of age)
Lower pelvic or abdominal pain is usually associated with onset of menstrual flow and lasts 8-72 hours	Women may complain of change of time of pain onset during menstrual cycle or in intensity of pain
Back and thigh pain, headache, diarrhoea, nausea and vomiting may be present	Other gynaecological symptoms (such as dyspareunia, menorrhagia) may be present
No abnormal findings on examination	Pelvic abnormality on physical examination

Differential diagnosis of primary and secondary dysmenorrhoea

Pathophysiology Of Primary Dysmenorrhea

A regular ovulatory cycle, no pelvic pathology, and a distinct physiological etiology are characteristics of the majority of primary (or functional) dysmenorrhea in teens and young adults.[10] The phospholipids in the cell membranes accumulate fatty acids following ovulation. Omega-6 fatty acids predominate in cell wall phospholipids due to the western diet's increased consumption of these fatty acids.15 A series of prostaglandins (PG) and leukotrienes (LT) are produced in the uterus along with these omega-6 fatty acids, especially arachidonic acid, following the start of progesterone withdrawal before to menstruation. Along with cramping, the inflammatory response—which is mediated by these PG and LT—also causes systemic symptoms as headaches, bloating, nausea, and vomiting. The cyclooxygenase (COX) metabolite of arachidonic acid, prostaglandin F2a, in particular, produces strong vasoconstriction and myometrial contractions, which results in ischemia and discomfort.[11] Nine classes of prostaglandins are thought to be involved in the pathophysiology of primary dysmenorrhea. These include decreased uterine norepinephrine in the third trimester of pregnancy, neuronal degeneration in the uterus following a term delivery, and decreased endometrial prostaglandin release following a term delivery.[12]

Pathophysiology Of Sececonday Dysmenorrhea

In over 10% of adolescents and young adults with dysmenorrhea, secondary dysmenorrhea—painful menstruation linked to pelvic abnormalities—occurs. Metrorrhagia, dyspareunia, mid-cycle discomfort, and persistent pelvic pain are more likely to be linked to secondary dysmenorrhea.[10]

Endometriosis. Endometriosis is the most common cause of secondary dysmenorrhea in adolescents and young adults. It is defined as the presence and growth of uterine glands and stroma outside the uterine cavity. The condition known as endometriosis is dependent on estrogen. According to immunohistochemical investigations, the epithelial and stromal cells of endometriotic tissues and the peritoneum exhibit elevated expression of aromatase and estrogen receptors. [13,14] As a result, whereas aromatase activity is undetectable in healthy endometrium, it is abnormally expressed in endometriosis, which causes an increase in local estrogen production. Despite the absence of ovarian steroids during menstruation, this development of steroidogenic ability may allow the ectopic endometrial tissues to persist. Furthermore, the development and spread of these ectopic endometrial implants may be impacted by abnormal production of cytokines such interleukin-1 and 9 necrosis factor-alpha.[15] According to immunohistochemical research, endometriotic lesions have increased COX-2 expression.[16] And the rise in estrogen is probably the primary cause of this increase in COX-2.[17] Increased COX activity leads to the synthesis of PG, including PG E2, which strongly stimulates the expression and activity of aromatase in endometriotic stromal cells.[18] Deficient expression of 17beta-hydroxysteroid dehydrogenase (17b-HSD) type 2 hinders the conversion of estradiol to estrone, which is another anomaly that raises oestrogen levels in endometriosis.[19] One possible explanation for this 17b-HSD type 2 deficit is that progesterone's activity is flawed, failing to stimulate this enzyme in endometriotic tissue. Accordingly, the positive feedback loop in endometriosis is comprised of a high local level of estrogen that triggers COX-2 transcription and PG E2 production, which in turn leads to further aromatase expression and activity and an additional rise in estrogen (Fig. 1). A strong inflammatory response and pelvic discomfort are caused by the buildup of PG and estrogen. There are several factors that influence how severe endometriosis pain is. These consist of the tissue's stretching or scarring, the lesion's location, and the depth of invasion. Women who have deep implants in particular typically experience more intense pain and more active illness.[20] Nevertheless, the degree of endometriosis is not necessarily predicted by the existence of symptoms.[21]

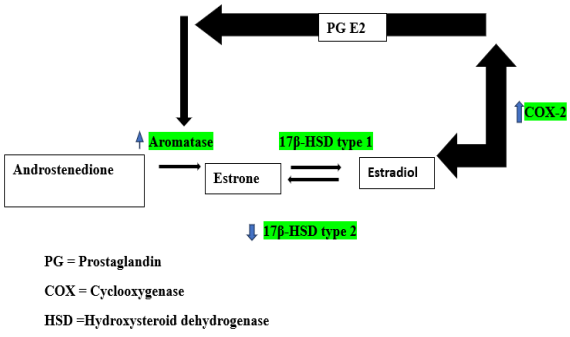


Fig. 1. Pathophysiology of Endometriosis.

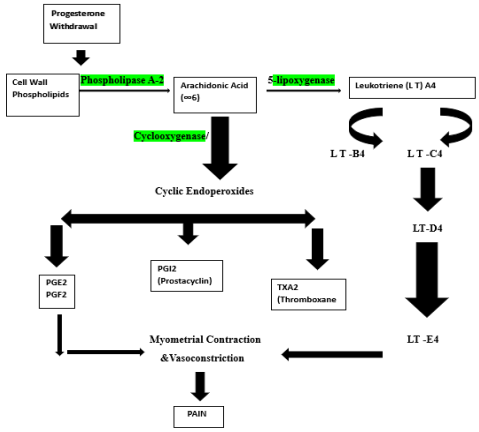


Fig. 2. Pathophysiology of Dysmenorrhea.

Dysmenorrhea Symptoms and Risk Factor

Many teenagers have other menstruation-related symptoms, such headaches and vomiting, even though lower abdomen cramps are the most typical dysmenorrhea symptom. Symptoms usually appear a few hours before or after the initiation of menstrual flow, or they accompany it and remain for the first 24 to 48 hours.[22] Increased menstrual flow length and volume, as well as an early menarche, are positively correlated with the severity of dysmenorrhea symptoms. In two trials, the severity of dysmenorrhea was associated with low fish consumption. [21,22] Premenstrual symptoms are less common in teenage females and are frequently relieved by proper treatment of dysmenorrhea. These symptoms are more prevalent beginning in the third decade of life.

Symptoms of Dysmenorrhea

- Cramps
- Nausea
- Vomiting
- Loss of appetite
- Headaches
- Weakness
- Dizziness
- Diarrhoea
- Facial blemishes
- Abdominal pain
- Sleeplessness
- Irritability

Diagnosis

It is crucial to rule out pelvic illness while evaluating menstruation discomfort, which should involve a thorough clinical history and physical examination. Menstrual history (age at menarche, amount of flow, regularity and duration of cycles, time between menarche and onset of dysmenorrhea), pain characterization (type, location, irradiation, associated symptoms, chronology), treatments used, sexual history, family history of dysmenorrhea, and system review (gastrointestinal, genitourinary, musculoskeletal, and psychosocial) should all be included in the clinical history.[24] Pain that starts just before or at the beginning of bleeding and lasts for up to 72 hours is described by patients as cramping and varying in intensity.[25] Located in the suprapubic area, the discomfort may spread to the back, upper thigh, or both. When menstruation begins, pain often peaks 24 to 36 hours later and lasts for a few days at most.[26]

Treatment

In addition to improving function, such as fewer days missed from work, school, or extracurricular activities, the ultimate objective of treating dysmenorrhea is to decrease reported pain and related systemic symptoms.

Pharmacologic Treatment Options

Primary dysmenorrhea

Pharmacologic treatments aim to decrease the synthesis of prostaglandins and leukotrienes,

which cause menstruation discomfort and its related systemic consequences. Therefore, first-line treatments employ hormonal contraceptives and/or nonsteroidal anti-inflammatory medications (NSAIDs) to lower prostaglandins and leukotrienes**.**

Nonsteroidal anti-inflammatory drugs

NSAIDs are the most well-established first line of treatment for dysmenorrhea.[27] There are at least two potential ways that NSAIDs work: they reduce the amount of menstrual flow and suppress prostaglandin production to directly reduce pain. These are most likely both NSAID class effects. Celecoxib, a cyclo-oxygenase (COX)-2 inhibitor, is authorized to treat dysmenorrhea; nevertheless, it is not better than NSAIDs. There are very few head-to-head comparisons of various formulations, and when new medications have been compared to older ones, they have been shown to be comparable. The choice of a certain medication should be based on factors like price and ease of use. According to two meta-analyses of NSAID and acetaminophen (paracetamol) randomized controlled trials (RCTs), all of the NSAIDs under investigation—ibuprofen, naproxen, mefenamic acid, and aspirin [acetyl-salicylic acid]—were shown to be effective, and all of them were more effective than acetaminophen. It's possible that NSAIDs work best when taken before menstruation discomfort and flow start.[27]

Standard Use of Oral Contraceptives

Oral contraceptives (OCs) are a widely recognized off-label treatment for dysmenorrhea. Reduced prostaglandin release during menstruation is the suggested mechanism of action. Low levels of COX-2 and endometrial proliferative indicators are linked to OC use.[28] The usefulness of OCs in the treatment of dysmenorrhea is supported by a number of RCTs as well as a substantial amount of reliable observational data. [29,30] Monophasic formulations were shown to be more efficient than triphasic ones in an observational investigation of the severity of dysmenorrhea among users of various kinds of contraception. [31]

Injectable long-acting hormonal contraceptives

Medroxyprogesterone acetate (DMPA), an injectable contraceptive depot, is a long-acting, progestin-only, practical, and efficient form of birth control. It comes in two forms: the FDA-approved intramuscular formulation (Depo-Provera, 150 mg DMPA/1 ml) in 1992 and the FDA-approved subcutaneous formulation (Depo-sub-Provera 104, 104 mg DMPA/0.65 ml) in 2004. Both formulations are given every 12 weeks. Since a single intramuscular injection of DMPA can inhibit ovulation for up to seven or nine months,[32] it might be used to lessen the symptoms of dysmenorrhea. The subcutaneous version of DMPA suppresses ovulation for almost 13 weeks, although having a 30% lower total dosage than the intramuscular formulation.[33]

CONCLUSION

For a long time, teenage dysmenorrhea was thought to be a minor menarche issue. Physicians' lack of interest has been influenced by a number of factors, including the relative frequency, severity, intergenerational patterns of self-medication, and the minimizing views of other family members. [34,35] In 1981, Dawood, began to sensitize American physicians to the extent of the problem by attracting their attention to the social and economic repercussions of both school and work absenteeism because of dysmenorrhea. Since then, the demonstration of a hyperproduction of uterine prostaglandins (PGF2) has led to the development of a specific treatment [36] whose prototype, flurbiprofen (of the propionate group), has been found to have a remarkable efficacy in treating this adolescent syndrome. [37] Since dysmenorrhea is a pain condition, subjective reports are the primary method of evaluation. Therefore, we have defined it as the collection of symptoms that add up to

more than just pelvic pain, with a total score of about 5. In a cohort of 4,203 teenagers aged 14 to 18, the prevalence of dysmenorrhea was determined to be 21% using these criteria. Comparing this finding with those of other groups is challenging, partly due to the wide variations in the literature's definition of dysmenorrhea, which makes quantitative comparison extremely dubious. It is hardly surprising that the prevalence ranges (table 4) from 43 to 80% [38] – and up to 91% in one sample of American teens [39] – given the vast variances in definition! With so much conflicting evidence, it is uncertain how dysmenorrhea and ovulation are related. Some populations require the onset of ovulatory cycles in order to have dysmenorrhea. [36,40] In conclusion, teenage females who have dysmenorrhea have significant undertreated morbidity. It is unfortunate that only one-third of the teenagers who have dysmenorrhea on a regular basis take the straightforward, targeted, and effective drugs that are currently accessible to treat its symptoms [42–42]. This makes it abundantly evident that pediatricians, gynaecologists, and general practitioners must better educate their patients on the resources accessible to them.

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