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An Overview and Management of Painful Menstrual Disorder (Dysmenorrhea): A Narrative Literature Review

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ABSTRACT

Primary dysmenorrhea is menstrual pain associated with the release of prostaglandins in the ovulatory cycle, but not with the pelvic disease. Approximately 90% of all women experience dysmenorrhea, 15% of whom are unable to last 1 to 3 days because of the severity of the pain. This literature review aimed to describe an overview and management of dysmenorrhea. Primary dysmenorrhea usually begins with the onset of ovulation cycles, with the highest prevalence in adolescence. In contrast, secondary dysmenorrhea is associated with pelvic pathology (i.e., ovarian cysts, adenomyosis, endometriosis) that manifests in later reproductive years and can occur at any point in the menstrual cycle. Administration of nonsteroidal anti-inflammatory drugs (NSAIDs) is the treatment of choice because these drugs reduce the activity of the cyclooxygenase (COX) enzyme and, thus, the production of prostaglandins. NSAIDs work in the majority of women with primary dysmenorrhea and are most effective when started at the first sign of bleeding or cramping.

1. Introduction

Dysmenorrhea is a condition characterized by menstrual pain that occurs during a woman's menstrual cycle. This pain usually occurs in the lower abdomen and can be felt as a cramping, dull, or shooting pain. Dysmenorrhea can occur in women of any age but is most common in young women during the first years of menstruation. This condition can be divided into two types, namely primary dysmenorrhea and secondary dysmenorrhea. Primary dysmenorrhea occurs due to hormonal imbalance and usually begins in adolescence or early adulthood. Meanwhile, secondary dysmenorrhea occurs as a result of an underlying medical condition, such as endometriosis, uterine fibroids, or pelvic infection.¹⁻³

The incidence or incidence of dysmenorrhea is quite high, especially in women who are young and have just experienced menstruation. According to some studies, around 45-95% of women experience dysmenorrhea at some point in their life. Primary dysmenorrhea is more common than secondary dysmenorrhea. In women with primary dysmenorrhea, about 60-80% of them experience symptoms of dysmenorrhea in each menstrual cycle. Meanwhile, in women with secondary dysmenorrhea, the incidence depends on the underlying cause. For example, in women with endometriosis, the incidence of secondary dysmenorrhea can be as high as 90%.⁴⁻⁶

Primary dysmenorrhea versus secondary dysmenorrhea

Primary dysmenorrhea is menstrual pain associated with the release of prostaglandins in the ovulatory cycle, but not with the pelvic disease. Approximately 90% of all women experience dysmenorrhea, 15% of whom are unable to last 1 to 3 days because of the severity of the pain. Primary dysmenorrhea usually begins with the onset of ovulation cycles, with the highest prevalence in adolescence. In contrast, secondary dysmenorrhea is associated with pelvic pathology (i.e., ovarian cysts, adenomyosis, endometriosis) that manifests in later reproductive years and can occur at any point in the menstrual cycle.^{7,8}

Pathophysiology of dysmenorrhea

Primary dysmenorrhea is associated with excessive production of endometrial prostaglandins, mainly released during the first 48 hours of menstruation when symptoms are most intense. Women with painful periods produced 10 times the amount of prostaglandin F (PGF₂), a potent myometrial stimulant and vasoconstrictor, compared to asymptomatic women. Increased levels of prostaglandins (especially PGF₂ and PGE₂) cause uterine hypercontractility, decreased blood flow to the uterus, and increased nerve hypersensitivity, resulting in pain. Women with dysmenorrhea may have upregulated cyclooxygenase (COX) enzyme activity, which contributes to increased prostaglandin synthesis. Furthermore, leukotriene production increases, which further contributes to an increase in pain levels. Women are more likely to report primary dysmenorrhea if they are younger than 30 years; have not yet given birth; have a history of pelvic inflammatory disease, sexual assault, syndrome premenstrual, or sterilization; is a heavy users of tobacco or alcohol; have a family history of dysmenorrhea; or have a body mass index (BMI) of less than 20. Women with severe primary dysmenorrhea appear to have increased pain sensitivity; Emerging research shows changes in their pain modulation system, placing them at higher risk for functional pain disorders (e.g., fibromyalgia) later in life. Women who are anovulatory due to oral contraceptive use rarely experience primary dysmenorrhea. Secondary dysmenorrhea occurs due to disorders such as

endometriosis (most common cause), endometritis (infection), adenomyosis, pelvic inflammatory disease, obstructive uterine or vaginal disorders, uterine fibroids, polyps, tumors, ovarian cysts, pelvic congestion syndrome, or non-hormonal contraceptives (IUDs).⁹⁻¹²

Clinical symptoms of dysmenorrhea

The main symptom of dysmenorrhea is pelvic pain associated with the onset of menstruation. The pain often radiates to the groin and may be accompanied by backache, anorexia, vomiting, diarrhea, syncope, insomnia, and headache. The latter symptoms are due to the entry of prostaglandins and prostaglandin metabolites into systemic circulation. Usually, the discomfort associated with primary dysmenorrhea begins shortly before the onset of menstruation and lasts for the first 1 to 3 days of menstrual flow.¹³⁻¹⁵

Management of dysmenorrhea

Primary dysmenorrhea can be distinguished from secondary dysmenorrhea by careful history and pelvic examination. Administration of non-steroidal anti-inflammatory drugs (NSAIDs, e.g., ibuprofen) is the treatment of choice because these drugs reduce COX enzyme activity and, thus, prostaglandin production. NSAIDs work in the majority of women with primary dysmenorrhea and are most effective when started at the first sign of bleeding or cramping. In women who desire contraception or do not respond to NSAIDs, dysmenorrhea can be reduced by hormonal contraceptives, such as the combined estrogen-progestin pill or progestin-only intrauterine device (IUD). Hormonal contraceptives stop ovulation and create an atrophic endometrium, thereby reducing prostaglandin synthesis and myometrial contractility. Other treatment approaches with some evidence of effectiveness in pain relief include transdermal nitroglycerin patches to reduce uterine contractility, local heat application, acupuncture, transcutaneous electrical nerve stimulation (TENS), increased physical activity, stress reduction through meditation, extra sleep, and such supplements as thiamine, vitamin E, and herbal remedies.¹⁶⁻²⁰

2. Conclusion

Primary dysmenorrhea occurs due to hormonal imbalance and usually begins in adolescence or early adulthood. Meanwhile, secondary dysmenorrhea occurs as a result of an underlying medical condition, such as endometriosis, uterine fibroids, or pelvic infection.

3. References

1. Vlachou E, Owens DA, Lavdaniti M, Kalemikerakis J, Evagelou E, Margari N, et al. Prevalence, wellbeing, and symptoms of dysmenorrhea among university nursing students in Greece. *Diseases*. 2019; 7(1).
2. Burnett M, Lemyre M. No. 345-primary dysmenorrhea consensus guideline. *J Obstet Gynaecol Can*. 2017; 39(7): 585-95.
3. French L. Dysmenorrhea. *Am Fam Physician*. 2005; 71(2): 285-91.
4. Chauhan M, Kala J. Relation between dysmenorrhea and body mass index in adolescents with rural versus urban variation. *J Obstet Gynaecol India*. 2012; 62(4): 442-5.
5. Zebitay AG, Verit FF, Sakar MN, Keskin S, Cetin O, Ulusoy AI. Importance of cervical length in dysmenorrhoea aetiology. *J Obstet Gynaecol*. 2016; 36(4): 540-3.
6. Dawood MY. Current concepts in the etiology and treatment of primary dysmenorrhea. *Acta Obstet Gynecol Scand Suppl*. 1986; 138: 7-10.
7. Ju H, Jones M, Mishra G. The prevalence and risk factors of dysmenorrhea. *Epidemiol Rev*. 2014; 36: 104-13.
8. Proctor M, Farquhar C. Diagnosis and management of dysmenorrhoea. *BMJ*. 2006; 332(7550): 1134-8.
9. Lundström V, Gréen K, Svanborg K. Endogenous prostaglandins in dysmenorrhea and the effect of prostaglandin synthetase inhibitors (PGSI) on uterine contractility. *Acta Obstet Gynecol Scand Suppl*. 1979; 87: 51-6.
10. Coco AS. Primary dysmenorrhea. *Am Fam Physician*. 1999; 60(2): 489-96.
11. Ylikorkala O, Dawood MY. New concepts in dysmenorrhea. *Am J Obstet Gynecol*. 1978; 130(7): 833-47.
12. Mrugacz G, Grygoruk C, Sieczyński P, Grusza M, Bołkun I, Pietrewicz P. Etiopathogenesis of dysmenorrhea. *Med Wieku Rozwoj*. 2013; 17(1): 85-9.
13. Agarwal AK, Agarwal A. A study of dysmenorrhea during menstruation in adolescent girls. *Indian J Community Med*. 2010; 35(1): 159-64.
14. Alsaleem MA. Dysmenorrhea, associated symptoms, and management among students at King Khalid University, Saudi Arabia: An exploratory study. *J Family Med Prim Care*. 2018; 7(4): 769-74.
15. Zannoni L, Giorgi M, Spagnolo E, Montanari G, Villa G, Seracchioli R. Dysmenorrhea, absenteeism from school, and symptoms suspicious for endometriosis in adolescents. *J Pediatr Adolesc Gynecol*. 2014; 27(5): 258-65.
16. Word RA, Kamm KE, Casey ML. Contractile effects of prostaglandins, oxytocin, and endothelin-1 in human myometrium in vitro: refractoriness of myometrial tissue of pregnant women to prostaglandins E2 and F2 alpha. *J Clin Endocrinol Metab*. 1992; 75(4): 1027-32.
17. Ekström P, Akerlund M, Forsling M, Kindahl H, Laudanski T, Mrugacz G. Stimulation of vasopressin release in women with primary dysmenorrhoea and after oral contraceptive treatment--effect on uterine contractility. *Br J Obstet Gynaecol*. 1992; 99(8): 680-4.
18. Bernardi M, Lazzeri L, Perelli F, Reis FM, Petraglia F. Dysmenorrhea and related disorders. *F1000Res*. 2017; 6: 1645.
19. Falcone T, Flyckt R. Clinical management of endometriosis. *Obstet Gynecol*. 2018; 131(3): 557-71.
20. Agarwal SK, Chapron C, Giudice LC, Laufer MR, Leyland N, Missmer SA, et al. Clinical diagnosis of endometriosis: a call to action. *Am J Obstet Gynecol*. 2019; 220(4): 354.