

Dysmenorrhea and Related Disorders

¹Dr Ayesha, ²Dr Anees Fatima, ³Dr Maira Sahar

^{1,2}MBBS, Khawaja Muhammad Safdar Medical College, Sialkot.

³MBBS, Nawaz Sharif Medical College, Gujrat.

Corresponding Author: Dr Ayesha, MBBS, Khawaja Muhammad Safdar Medical College, Sialkot.

Type of Publication: Original Research Paper

Conflicts of Interest: Nil

Abstract

Dysmenorrhea is common secondary to many gynecological issues, but in women it has represented as a primary disease. Hyper secretion of prostaglandins and an increased uterine contractility is linked with the pain of dysmenorrhea. Primary dysmenorrhea has been linked with young girls very commonly with good prognosis even though it lowers the quality of life. Whereas the secondary dysmenorrhea is associated with endometriosis and adenomyosis which may represents major symptoms. The diagnosis is based upon the clinical history and physical examination done by ultrasound which is very helpful to exclude secondary causes. The best treatment regime includes non-steroidal anti-inflammatory drugs alone or combined with progestins.

Introduction

The presence of distressing cramps of uterine origin that happen during menstruation and represents one of the most common causes of pelvic pain and menstrual disorder is known as dysmenorrhea [1]. The International Association for the Study of Pain has defined the pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage”. In particular, chronic pelvic pain is located in the pelvic area and lasts for 6 months or longer [2].

Dysmenorrhea is one of the major issue than any other gynecological complain: it is the main cause of gynecological morbidity in women of reproductive age regardless of age, nationality, and economic status [3]. Such effects expand far away from individual women to society which results annually in an important loss of productivity. Therefore, World Health Organization has stated that dysmenorrhea is the most important cause of chronic pelvic pain [4]. The estimated prevalence of dysmenorrhea is high, although it varies widely, ranging from 45 to 93% of women of reproductive age and the highest rates are reported in adolescents. Since it has been accepted as a normal situation of the menstrual cycle and women usually do not report and go for primary medical care [5]. But there are some women have unbearable pain, severe enough to take leave from office or school due to this pain for 1-3 days. Indeed, dysmenorrhea has a high impact on women’s lives, resulting in a restriction of daily activities a lower academic performance in adolescents and poor quality of sleep, and has negative effects on mood, causing anxiety and depression [6]. Dysmenorrhea is classified as primary dysmenorrhea (menstrual pain without organic disease) or secondary dysmenorrhea according to pathophysiology [7]. The cause of primary dysmenorrhea is not well established. However, the responsible cause has been identified on the hyper-

production of uterine prostaglandins, particularly of PGF₂a and PGF₂, thus resulting in increased uterine tone and high-amplitude contractions [8]. Women with dysmenorrhea have higher levels of prostaglandins, which are highest during the first two days of menses. Prostaglandin production is controlled by progesterone: when progesterone levels drop, immediately prior to menstruation, prostaglandin levels increase [9]. If the exposure of endometrium to luteal phase is crucial for the increased production of progesterone, dysmenorrhea occurs only with ovulatory cycles.

However, multiple other factors may play a role in the perception and the severity of pain, which does not depend only on endocrine factors [10]. The recurrent menstrual pain is associated with central sensitization, which is associated with structural and functional modification of the central nervous system [11]. Dysmenorrhea could lead to long-term consequences and may be increasing women's susceptibility to others chronic pain conditions later in life, it is mandatory to treat menstrual pain in order to limit the noxious input into the central nervous system. In young women major causes of secondary dysmenorrhea are endometriosis and adenomyosis.

Endometriosis is characterized by the presence of endometrial tissue (glands and stroma) outside the uterine cavity and is the most common cause of secondary dysmenorrhea [12].

Pain symptoms can have negative impact in physical and psychological well-being of women with endometriosis. Most of the pains persuade elevated sympathetic nervous system activity and this is considered a stressor, inducing changes in neuromediators, neuroendocrine, and hormonal secretions [13]. Women suffering from endometriosis wait before getting the right diagnosis, a great deal of effort has been made in recent years to try to find signs and symptoms that would help in making an earlier diagnosis. The early spotting of these symptoms could help reduce

the delay necessary for diagnosis and enable the use of less invasive procedures [14].

In adolescence dysmenorrhea is considered a risk factor for endometriosis; other menstrual characteristics such as cycle length and menstrual bleeding duration and quantity are not related to the development of endometriosis. Parameters that may predict a later finding of deep infiltrating endometriosis are prolonged use of oral contraceptives (OCs) for treating primary dysmenorrhea, absenteeism from school during menstruation, and a positive family history of dysmenorrhea [15].

Adolescents are more prevalent in the condition suffering from endometriosis with chronic pelvic pain resistant to treatment with OC pills and non-steroidal anti-inflammatory drugs (NSAIDs) and in girls with dysmenorrhea. So that severe dysmenorrhea that does not respond to medical therapy warrants further investigation such as by laparoscopy [16]. Adenomyosis is defined as the presence of endometrial glands and stroma within the myometrium and is associated with dysmenorrhea and abnormal uterine bleeding (AUB). Adenomyosis is one of the most common causes of AUB [17]. The diagnosis is usually confirmed through transvaginal ultrasonography and magnetic resonance imaging. Thorough specific USG criteria by bidimensional and tridimensional transvaginal ultrasound (morphological uterus sonographic assessment), the detection of adenomyosis features by imaging is accepted and the association with menstrual pain, heavy menstrual bleeding, and infertility may facilitate the diagnosis of adenomyosis [18]. A 34% incidence of adenomyosis ultrasonographic features is found in young nulligravid women 18 to 30 years of age and is associated with dysmenorrhea [19].

Heavy menstrual bleeding and longer menstrual bleeding duration type risk factors are often associated with dysmenorrhea [20]. Childbearing is a very influential factor for the decrease of dysmenorrhea. Increasing age is

inversely proportion to dysmenorrhea, although a longitudinal study found that the proportion of women with moderate to severe dysmenorrhea remained constant with increasing age [21]. The early onset of pain is associated with more severe pain, and a family history of dysmenorrhea is associated with a significantly higher prevalence of dysmenorrhea [22].

Diagnosis

A focused history and physical examination are usually sufficient for making a diagnosis of primary dysmenorrhea [23]. After menarche the onset of primary dysmenorrhea is usually 6 to 12 months. The typical pain is sharp and intermittent, is located in the suprapubic area, and develops within hours of the start of menstruation and peaks with maximum blood flow [24].

Mostly the physical examination is completely normal, and the menstrual pain may be linked with systemic symptoms, such as nausea, vomiting, diarrhea, fatigue, fever, headache, and insomnia [25]. There is no indication for routine use of ultrasound in the evaluation of primary dysmenorrhea, although ultrasound is very useful in excluding the secondary causes of dysmenorrhea, such as endometriosis and adenomyosis (Figure 1). Dysmenorrhea that occurs any time after menarche, that is associated with other gynecological symptoms such as dyspareunia, heavy menstrual bleeding, AUB, and infertility, and that does not respond to treatment with NSAIDs might be hinting towards for secondary dysmenorrhea [26]. The analysis of menstrual bleeding abnormalities associated with dysmenorrhea might be helpful for the diagnosis of adenomyosis (Figure 1).

Treatment

The aim of the treatment for primary dysmenorrhea is pain relief. NSAIDs are usually immediate therapy for dysmenorrhea and should be tried for at least three menstrual periods [27]. If NSAIDs alone are not sufficient, OCs can be combined with it. NSAIDs are

drugs that act by blocking prostaglandin production through the inhibition of cyclooxygenase, an enzyme responsible for formation of prostaglandins. Common NSAIDs (aspirin, naproxen, and ibuprofen) are very effective in relieving period pain [28]. They make the menstrual cramps less severe and can prevent other symptoms such as nausea and diarrhea. NSAIDs minimize moderate to severe pain in women with primary dysmenorrhea [29]. With the widespread availability of NSAIDs, the management of dysmenorrhea is mainly self-care. Oral contraceptives Contraceptive hormones act by suppressing ovulation and causing no endometrial proliferation. OCs bring almost immediate relief from symptoms associated with menstruation: heavy periods, painful periods, and irregular bleeding. In addition, OCs often are used as therapeutic drugs for women with symptomatic menorrhagia or endometriosis. For the reduction of associated menstrual disorders and the improvement in women's pain relief regular use of OC can be considered to treat primary dysmenorrhea . However, limited evidence supports the use of OCs as a standard treatment.

The choice between the use of combined OCs and oral progesterone should be guided by the patient's pain relief, the toleration of possible adverse effects especially linked to the frequency of breakthrough bleeding and weight gain, and the patient's basal risk of venous thromboembolism.

Progestins Hormonal progestins-only treatment produces a benefit on menstrual pain, causing endometrial atrophy and inhibiting ovulation. Several long-acting reversible progestin contraceptives have been found to be effective treatments for primary dysmenorrhea [30].

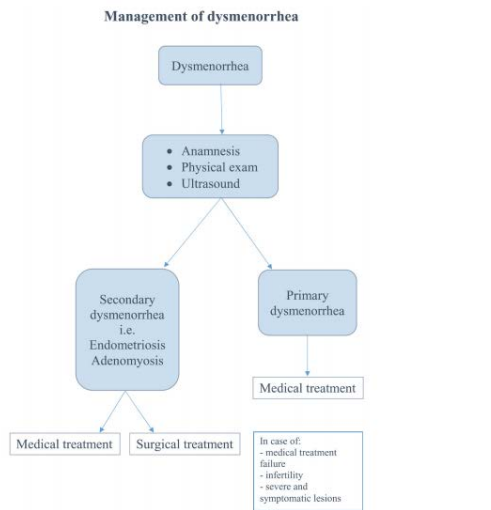


Figure 1. Flowchart for the management of patients with dysmenorrhea. Flowchart for the management of patients with dysmenorrhea.

References

- Hanoch Kumar K, Elavarasi P: Definition of pain and classification of pain disorders. *Journal of Advanced Clinical & Research Insight*. 2016; 3: 87–90. [Publisher Full Text](#)
- ACOG Committee on Practice Bulletins--Gynecology: ACOG Practice Bulletin No. 51. Chronic pelvic pain. *Obstet Gynecol*. 2004; 103(3): 589–605. [PubMed Abstract](#)
- Patel V, Tanksale V, Sahasrabhojane M, et al.: The burden and determinants of dysmenorrhoea: a population-based survey of 2262 women in Goa, India. *BJOG*. 2006; 113(4): 453–63. [PubMed Abstract](#) | [Publisher Full Text](#)
- Harlow SD, Campbell OM: Epidemiology of menstrual disorders in developing countries: a systematic review. *BJOG*. 2004; 111(1): 6–16. [PubMed Abstract](#) | [Publisher Full Text](#)
- Weissman AM, Hartz AJ, Hansen MD, et al.: The natural history of primary dysmenorrhoea: a longitudinal study. *BJOG*. 2004; 111(4): 345–52. [PubMed Abstract](#) | [Publisher Full Text](#)
- Wong LP, Khoo EM: Dysmenorrhea in a multiethnic population of adolescent Asian girls. *Int J Gynaecol Obstet*. 2010; 108(2): 139–42. [PubMed Abstract](#) | [Publisher Full Text](#)
- De Sanctis V, Soliman A, Bernasconi S, et al.: Primary Dysmenorrhea in Adolescents: Prevalence, Impact and Recent Knowledge. *Pediatr Endocrinol Rev*. 2015; 13(2): 512–20. [PubMed Abstract](#)
- Thomas SL, Ellertson C: Nuisance or natural and healthy: should monthly menstruation be optional for women? *Lancet*. 2000; 355(9207): 922–4. [PubMed Abstract](#) | [Publisher Full Text](#)
- Eryilmaz G, Ozdemir F, Pasinlioglu T: Dysmenorrhea prevalence among adolescents in eastern Turkey: its effects on school performance and relationships with family and friends. *J Pediatr Adolesc Gynecol*. 2010; 23(5): 267–72. [PubMed Abstract](#) | [Publisher Full Text](#)
- Latthe P, Latthe M, Say L, et al.: WHO systematic review of prevalence of chronic pelvic pain: a neglected reproductive health morbidity. *BMC Public Health*. 2006; 6: 177. [PubMed Abstract](#) | [Publisher Full Text](#) | [Free Full Text](#)
- Parker MA, Sneddon AE, Arbon P: The menstrual disorder of teenagers (MDOT) study: determining typical menstrual patterns and menstrual disturbance in a large population-based study of Australian teenagers. *BJOG*. 2010; 117(2): 185–92. [PubMed Abstract](#) | [Publisher Full Text](#)
- Lindh I, Ellström AA, Milsom I: The effect of combined oral contraceptives and age on dysmenorrhoea: an epidemiological study. *Hum Reprod*. 2012; 27(3): References F1000 recommended Page 5 of 7 F1000Research 2017, 6(F1000 Faculty Rev):1645 Last updated: 05 SEP 2017 676–82. [PubMed Abstract](#) | [Publisher Full Text](#)
- Wong CL, Farquhar C, Roberts H, et al.: Oral contraceptive pill for primary dysmenorrhoea. *Cochrane Database Syst Rev*. 2009; (4): CD002120. [PubMed Abstract](#) | [Publisher Full Text](#)

14. Subasinghe AK, Happo L, Jayasinghe YL, et al.: Prevalence and severity of dysmenorrhoea, and management options reported by young Australian women. *Aust Fam Physician*. 2016; 45(11): 829–34. PubMed Abstract
15. Zannoni L, Giorgi M, Spagnolo E, et al.: Dysmenorrhea, absenteeism from school, and symptoms suspicious for endometriosis in adolescents. *J Pediatr Adolesc Gynecol*. 2014; 27(5): 258–65. PubMed Abstract | Publisher Full Text
16. Ortiz MI, Rangel-Flores E, Carrillo-Alarcón LC, et al.: Prevalence and impact of primary dysmenorrhea among Mexican high school students. *Int J Gynaecol Obstet*. 2009; 107(3): 240–3. PubMed Abstract | Publisher Full Text
17. Chantler I, Mitchell D, Fuller A: Actigraphy quantifies reduced voluntary physical activity in women with primary dysmenorrhea. *J Pain*. 2009; 10(1): 38–46. PubMed Abstract | Publisher Full Text
18. Banikarim C, Chacko MR, Kelder SH: Prevalence and impact of dysmenorrhea on Hispanic female adolescents. *Arch Pediatr Adolesc Med*. 2000; 154(12): 1226–9.
19. Hailemeskel S, Demissie A, Assefa N: Primary dysmenorrhea magnitude, associated risk factors, and its effect on academic performance: evidence from female university students in Ethiopia. *Int J Womens Health*. 2016; 8: 489–96. PubMed Abstract | Publisher Full Text | Free Full Text | F1000 Recommendation
20. Unsal A, Ayranci U, Tozun M, et al.: Prevalence of dysmenorrhea and its effect on quality of life among a group of female university students. *Ups J Med Sci*. 2010; 115(2): 138–45. PubMed Abstract | Publisher Full Text | Free Full Text
21. Baker FC, Driver HS, Rogers GG, et al.: High nocturnal body temperatures and disturbed sleep in women with primary dysmenorrhea. *Am J Physiol*. 1999; 277(6 Pt 1): E1013–21. PubMed Abstract
22. Dorn LD, Negriff S, Huang B, et al.: Menstrual symptoms in adolescent girls: association with smoking, depressive symptoms, and anxiety. *J Adolesc Health*. 2009; 44(3): 237–43. PubMed Abstract | Publisher Full Text | Free Full Text
23. Proctor M, Farquhar C: Diagnosis and management of dysmenorrhoea. *BMJ*. 2006; 332(7550): 1134–8. PubMed Abstract | Publisher Full Text | Free Full Text | F1000 Recommendation
24. Iacovides S, Avidon I, Baker FC: What we know about primary dysmenorrhea today: a critical review. *Hum Reprod Update*. 2015; 21(6): 762–78. PubMed Abstract | Publisher Full Text | F1000 Recommendation
25. Dawood MY: Primary dysmenorrhea: advances in pathogenesis and management. *Obstet Gynecol*. 2006; 108(2): 428–41. PubMed Abstract | Publisher Full Text
26. Lefebvre G, Pinsonneault O, Antao V, et al.: Primary dysmenorrhea consensus guideline. *J Obstet Gynaecol Can*. 2005; 27(12): 1117–46. PubMed Abstract | Publisher Full Text
27. Brawn J, Morotti M, Zondervan KT, et al.: Central changes associated with chronic pelvic pain and endometriosis. *Hum Reprod Update*. 2014; 20(5): 737–47. PubMed Abstract | Publisher Full Text | Free Full Text | F1000 Recommendation
28. Bulun SE: Endometriosis. *N Engl J Med*. 2009; 360(3): 268–79. PubMed Abstract | Publisher Full Text
29. Morotti M, Vincent K, Brawn J, et al.: Peripheral changes in endometriosis-associated pain. *Hum Reprod Update*. 2014; 20(5): 717–36. PubMed Abstract | Publisher Full Text | Free Full Text | F1000 Recommendation

29. Arruda MS, Petta CA, Abrão MS, et al.: Time elapsed from onset of symptoms to diagnosis of endometriosis in a cohort study of Brazilian women. *Hum Reprod.* 2003; 18(4): 756–9.