

The Prevalence of Dysmenorrhea among Women

Sarah Abdulaziz Fallatah¹, Afnan Mohmmmed Mulla Ebrahim Khan², Hannin Mohammed Al Reqeï³, Zahra Ahmed Alalshaikh⁴, Jabir Mohammed A Alnabhani⁵, Mashail Ali Alomari⁶, Ruzanah Abdulaziz Almarzugi⁵, Abdulaziz Faisal M Khyat⁷, Umklthom Mohmmad A Masmali⁸, Noha Mohammed I Hawsawi⁹, Manal Mohamed A Khayat¹⁰, Basma Hamed M Alhawiti¹⁰

1- King Abdulaziz Hospital, 2- Since and Technology University, Sana'a, 3- Maternity and Children Hospital in Buraydah, 4- Soochow University, 5- King Abduaziz University, 6- King Khalid University, 7- University of Jeddah, 8- Jazan University, 9- Maternity Children Hospital in Makkah, 10- Cairo University, Egypt

ABSTRACT

Menstrual disorders and abnormal uterine bleeding are common worries of young women. Complaints comprise menses that are: too painful (dysmenorrhea), prolonged and heavy (menorrhagia, or excessive uterine bleeding), or absent or arise irregularly (amenorrhea or oligoamenorrhea). In providing optimal reproductive care, the medical provider should be capable of distinguishing between normal developmental patterns or symptoms necessitating education and reassurance from pathologic conditions needing early evaluation and management. This article discusses the normal menstrual patterns seen in adolescent females and provides treatment and management approach to primary and secondary dysmenorrhea.

Keywords: Menorrhagia, Excessive uterine bleeding, Dysmenorrhea, Menstrual problems.

INTRODUCTION

Dysmenorrhea is defined as difficult menstrual flow or painful menstruation. It is one of the most common gynecologic complaints in young women who present to clinicians^[1]. Optimal treatment of these symptoms rely on an understanding of the underlying causes. Dysmenorrhea can be divided into 2 broad categories: primary (spasmodic) and secondary (congestive)^[2]. Primary dysmenorrhea is defined as menstrual pain that is not allied with macroscopic pelvic pathology (ie, occurs in the absence of pelvic disease). It normally occurs in the first few years after menarche^[3] and influences as many as 50% of post pubertal females^[4]. Secondary dysmenorrhea is defined as menstrual pain resulting from anatomic or macroscopic pelvic pathology^[3, 5], as is seen in women with endometriosis or chronic pelvic inflammatory disease. It is most regularly perceived in women aged 30-45 years.

Table 1. Differential diagnosis of primary and secondary dysmenorrhea

| | Primary dysmenorrhea | Secondary dysmenorrhea |
|----------------------------------|----------------------------|-------------------------------------|
| Onset | Within 3 yr after menarche | More than 5 yr after menarche |
| Age | 15–25 yr old | Over 30 yr old |
| Aging | Gradually improve | Become worse |
| Postpartum | Improve | No change |
| Time | Menstruation | Menstruation or other time if worse |
| Findings of internal examination | Normal | Endometriosis, fibroma, etc. |
| Marriage | Improve | No change |
| Duration | 4–48 h | 1–5 d |

The following risk factors are associated with more severe episodes of dysmenorrhea^[6]:

- Smoking
- Earlier age at menarche
- Positive family history
- Long menstrual periods
- Heavy menstrual flow

Some (not all) studies have found obesity and alcohol intake to be linked with dysmenorrhea^[7, 8]. Physical activity and the length of the menstrual cycle do not appear to be related with increased menstrual pain. Even though dysmenorrhea is not life-threatening, it can be debilitating and psychologically taxing for many women. Some choose to self-medicate at home and never pursue medical consideration for their pain. Dysmenorrhea is accountable for substantial absenteeism from work, and it is the most common cause for school absence amid youths^[9].

Risk factors

Risk factors for primary and secondary dysmenorrhea include the following:

Table 2. Risk factors for primary and secondary dysmenorrhea

| primary dysmenorrhea | secondary dysmenorrhea |
|------------------------------------|-----------------------------|
| Early age at menarche (< 12 years) | Leiomyomata (fibroids) |
| Nulliparity | PID |
| Heavy or prolonged menstrual flow | Tubo-ovarian abscess |
| Smoking | Ovarian torsion |
| Positive family history | Endometriosis |
| Obesity | Pelvic inflammatory disease |

In the following sections, the more common causes of secondary dysmenorrhea are briefly summarized.

- **Tubo-ovarian abscess**

Tubo-ovarian abscess is a loculated infection within the fallopian tubes or ovaries, usually occurring as a sequela of PID. It is frequently polymicrobial. Most regularly, patients give fever and step by step declining pelvic torment and delicacy; queasiness, spewing, and vaginal draining or release might be available also. Examination may evoke delicacy on cervical movement and in the adnexal territory. A pelvic mass might be available, however it is frequently hard to palpate^[10].

Tubo-ovarian abscesses can be distinguished on pelvic ultrasonography or stomach CT as a complex cystic structure in the pelvis, with or without loculations^[11]. Patients are often admitted for intravenous (IV) antibiotic therapy covering *Neisseria gonorrhoeae*, anaerobes, *Chlamydia*, and gram-negative organisms. If medical therapy fails or if peritoneal signs are found on examination, surgical drainage is specified. Infertility is almost always a complication of tubo-ovarian abscess^[10]. The most feared complication, however, is rupture, which can lead to septic shock and death; this is a true surgical emergency^[12].

- **Ovarian torsion**

Ovarian torsion includes twisting of the adnexal structures, which prompts ischemia and eventually rot if the procedure isn't turned around in time. In a non-pregnant woman, it is quite often caused by a variation from the norm in the ovary, for example, a blister or a tumor. Torsion can happen in pregnancy without an essential adnexal irregularity, and in one expansive arrangement, 20% of the patients found to have torsion were pregnant^[13]. Patients regularly give extreme, irregular, colicky, one-sided pelvic or bring down stomach torment, much of the time related with sickness and heaving. The analysis is frequently postponed on the grounds that the introduction of ovarian torsion can look like those of other infection elements, for example, a ruptured appendix or renal colic^[12].

Due to these resemblances and the consequent potential for diagnostic improbability, CT is often performed before any other imaging modality. It is important to be familiar with the typical CT findings for torsion: ovarian enlargement exceeding 5 cm with a corkscrew appearance of the ipsilateral fallopian tube^[11].

A sonogram will usually show a large ovarian mass or cyst, but ultrasonographic evidence of torsion is difficult to obtain, because the

appearance changes depending on the length of time elapsed^[12]. In the event that there is an abnormal state of doubt for ovarian torsion, a gynecologic discussion ought to be acquired early. Laparoscopy isn't just analytic yet additionally remedial and conceivably fruitfulness sparing. These patients are altogether conceded.

- **Ovarian cyst rupture or hemorrhage**

A hemorrhagic ovarian cyst comes from an ovarian follicle in the absence of ovulation; consequently, these cysts are exclusively found in menstruating females.

Patients often present with the acute onset of pelvic or abdominal pain, along with nausea and vomiting. Examination may reveal an adnexal mass, but almost all patients with ruptured ovarian cysts have some level of adnexal tenderness. Signs of peritoneal irritation may be apparent as well. Although CT and ultrasonography can be used to visualize hemoperitoneum and the cyst^[14], laparoscopy is required for the definitive diagnosis^[10].

- **Uterine leiomyoma**

Uterine leiomyomata are benign tumors of the uterine musculature that are a common cause of dysmenorrhea as they enlarge when stimulated by estrogen. They are up to 9 times more common in black women than in white women^[15]. As well as pain with menses, patients may present with menorrhagia, abdominal distention, or pressure. Pelvic examination may reveal a uterine mass or irregularity. Ultrasonography is often used for determining size and location of fibroids, though computed tomography (CT) is used if ultrasonographic information is limited^[10, 11]. Unless patients are symptomatic from profound anemia, these patients can be safely discharged with appropriate gynecologic follow-up. Potential complications are anemia and infertility.

Physical Examination

A complete physical examination ought to be implemented. For youngsters who have never been sexually active, a careful abdominal examination is suitable. In older adolescents or those known to be sexually active, a pelvic examination is crucial for excluding uterine irregularities, cul-de-sac tenderness, or suggestive nodularities. This examination contains the following^[10]:

- Inspection of the vaginal vault for discharge, blood, or foreign bodies
- Inspection of the external genitalia for rashes, swelling, or discoloration

- Inspection of the cervix for the above, plus any masses or signs of infection.
- Bimanual examination to assess cervical motion tenderness, uterine or adnexal tenderness, or any masses in the pelvis.

Women with primary dysmenorrhea regularly have normal outcomes on pelvic examination. Lower abdominal or uterine tenderness might be current. Cervical stenosis may contribute to retrograde flow. Women with secondary dysmenorrhea might have pelvic pathology, however ordinary discoveries don't reject the condition. Ladies with endometriosis who give auxiliary dysmenorrhea have physical discoveries around 40% of the time^[16, 17]. A substantial uterine mass may be obtainable. Cervical movement delicacy may be prominent. There could be adnexal delicacy or a substantial mass. Vaginal or cervical release could be perceived. Obvious vaginal pathology (e.g., mucosal tears, masses, or prolapse) could be unmistakable. Pelvic ultrasonography ought to be considered in ladies who are associated with having optional dysmenorrhea. Consideration ought to likewise be paid to the stomach examination and back-flank examinations to discount pelvic torment as an introduction of gastrointestinal (GI) pathology and upper genitourinary (GU) pathology, respectively.

Treatment & Management of Dysmenorrhea

Many women never seek medical attention for dysmenorrhea. Self-medication with analgesics and nonsteroidal anti-inflammatory drugs (NSAIDs) and direct application of heat are common effective strategies. When a patient is seen in the emergency department (ED), evaluation should begin with the ABCs (Airway, Breathing, Circulation) and should consider serious diagnoses such as hemorrhagic shock and sepsis. A patient whose history and clinical presentation clearly suggest primary dysmenorrhea may be treated symptomatically and provided with appropriate follow-up. A patient whose presentation is less clear or whose vital signs or physical findings are abnormal deserves a more thorough workup, comprising full laboratory studies, pelvic ultrasonography, and potentially an obstetrics/gynecology consultation^[18].

Treatment of dysmenorrhea is aimed at providing symptomatic relief as well as inhibiting the underlying processes that cause symptoms. Grading dysmenorrhea according to the severity of pain and the degree of limitation of daily activity might help guide to the treatment strategy. Medications used may include NSAIDs and opioid analgesics, as well as oral contraceptives (OCs). In

addition to pain relief, mainstays of treatment include reassurance and education. Other therapies have been proposed, but most are not well studied. In patients with refractory symptoms, a multidisciplinary method might be specified. Patients with pelvic pain do not routinely need consultation with a gynecologist in the ED, though they ought to be directed to follow up on an outpatient basis. Exceptions comprise assured infectious entities (e.g., abscesses), in addition to endometriosis. Patients with both primary and secondary dysmenorrhea ought to be provided with suitable gynecologic follow-up. If they do not have consistent medical care, an appointment with a primary medical doctor is correspondingly specified.

Pharmacologic Treatment

Treatment of primary dysmenorrhea is directed at providing relief from the cramping pelvic pain and accompanying symptoms (e.g., vomiting, nausea, headache, diarrhea, and flushing) that usually accompany or immediately precede the onset of menstrual flow. The pelvic pain may be distressing and infrequently radiates to the back and thighs, regularly requiring rapid intervention. To date, pharmacotherapy has been the most solid and viable treatment for diminishing dysmenorrhea. Since the agony comes about because of uterine vasoconstriction, anoxia, and withdrawals intervened by prostaglandins, symptomatic alleviation can frequently be acquired by utilizing specialists that restrain prostaglandin amalgamation and have mitigating and pain relieving properties. NSAIDs and blend OCs are the most regularly utilized helpful modalities for the administration of essential dysmenorrhea. These operators have diverse systems of activity and can be utilized adjunctively in head strong cases. Absence of reaction to NSAIDs and OCs (or a combination thereof) can improve the probability of a secondary reason for dysmenorrhea. Management of optional dysmenorrhea comprises revision of the basic natural reason.

Particular measures (therapeutic or surgical) might be required to treat pelvic pathologic conditions (e.g., endometriosis) and to improve the related dysmenorrhea. Occasional utilization of pain relieving operators as adjunctive treatment might be advantageous.

- **Nonsteroidal anti-inflammatory drugs**

NSAIDs are the most shared management for both primary and secondary dysmenorrhea. They reduce menstrual pain by reducing intrauterine pressure and lowering prostaglandin F_{2α} (PGF_{2α}) levels in menstrual fluid^[19]. NSAIDs that inhibit

type I prostaglandin synthetase and suppress production of cyclic endoperoxides (e.g., indole acetic acids, cyclooxygenase [COX]-2-selective agents, propionic acids, and fenamates) alleviate symptoms by lessening endometrial and menstrual fluid prostaglandin concentrations. On the off chance that taken sufficiently early and in adequate amount, NSAIDs are to a great degree fruitful in mitigating menstrual agony. Since they are utilized for brief periods in generally solid young ladies, they are for the most part all around endured and free of genuine danger. Gastrointestinal (GI) disturb is the most well-known unfavorable impact related with NSAIDs, and patients getting these medicines ought to be observed for more genuine unfriendly impacts, including GI draining and renal brokenness. Patients ought to likewise be observed for potential pharmacokinetic and pharmacodynamic mediate connections and conceivable consequences for platelet conglomeration. NSAIDs are contraindicated in patients with renal deficiency, peptic ulcer infection, gastritis, draining diatheses, or headache medicine touchiness. These specialists must be utilized all the time (as-required utilize isn't satisfactory in many patients) for a few days. To keep away from accidental introduction to these specialists amid early pregnancy, NSAIDs ought to be begun at the beginning of menstrual dying.

While some NSAIDs (especially the fenamates) have been touted as being particularly effective for dysmenorrhea, scientific data to support such claims are sparse and generally weak^[20]. Additionally, well-designed prospective comparative studies have not been accomplished. The NSAIDs precisely approved by the US Food and Drug Administration (FDA) for management of dysmenorrhea are as follows:

- Ibuprofen
- Naproxen
- Diclofenac
- Ketoprofen
- Meclofenamate
- Mefenamic acid

Aspirin may not be as effective as these NSAIDs, and acetaminophen may be a useful adjunct for alleviating only mild menstrual cramping pain^[20, 21]. NSAIDs that accomplish top serum focuses inside 30-a hour and have a quicker beginning of activity (e.g., ibuprofen, naproxen, and meclofenamate) might be favored. In any case, singular patient reaction shifts, and patients may need to attempt a few specialists previously discovering one that works. A few NSAIDs (e.g., indomethacin) ought to be stayed away from, in light of the fact that they have a higher frequency

of adverse impacts. COX-2 particular inhibitors have likewise demonstrated powerful in alleviating menstrual torment. Their selectivity decreases the GI indications caused by hindrance of the COX-1 receptor. In spite of some preparatory information proposing viability in patients with essential dysmenorrhea, COX-2 inhibitors have not been verifiably better than ordinary NSAIDs^[22]. Notwithstanding, these operators might be utilized as a part of patients who can't endure different NSAIDs or in whom these specialists are contraindicated. COX-2-inferred prostanoids regardless have all the earmarks of being engaged with the pathophysiology of essential dysmenorrhea^[23].

• Other analgesic agents

In an emergency setting, patients who do not respond to NSAIDs may require treatment with narcotics for pain control. Patients whose symptoms are not relieved by NSAIDs are very likely to have an underlying pelvic condition (e.g., endometriosis). In a study comparing montelukast, a leukotriene-receptor antagonist, with placebo in patients with dysmenorrhea, montelukast was effective in reducing pain^[24]. Clinicians might deliberate this agent as an alternative to hormonal therapy or in lieu of NSAIDs. Simple analgesics, for example, aspirin and acetaminophen, may similarly be valuable, particularly when NSAIDs are contraindicated.

• Oral contraceptives

Oral contraceptives, which block monthly ovulation and might reduce menstrual flow, might correspondingly discharge symptoms. An update of a Cochrane review showed some indication of symptomatic benefit in patients with primary dysmenorrhea, however, no precise preparation presented superiority over any other^[25]. In some patients, oral contraceptives can avoid dysmenorrhea completely, although these agents are not approved by the FDA for this suggestion.

Oral contraceptives might be a fitting decision for patients who don't wish to imagine. Blend oral contraceptives stifle the hypothalamic-pituitary-ovarian hub, in this way hindering ovulation and counteracting prostaglandin generation in the late luteal stage. Utilization of oral contraceptives in a way that decreases the quantity of menstrual cycles might be valuable for few patients^[26].

Combination oral contraceptives, the levonorgestrel intrauterine device, and depot medroxyprogesterone acetate^[27] provide effective pain relief and are allied with decreased menstrual flow. It might be essential to add an NSAID to the

oral contraceptive, particularly throughout the first few cycles after beginning of the oral contraceptive. The ethinyl estradiol dose should generally be less than 50 µg; a monophasic oral contraceptive containing 30 µg is a rational choice.

To date, studies comparing the efficacy of various oral contraceptive formulations in the treatment of dysmenorrhea have not been implemented. In an investigation of women with essential dysmenorrhea, Petraglia *et al* (28) found that estradiol valerate in addition to dienogest and ethinyl estradiol in addition to levonorgestrel were equivalently compelling in diminishing dysmenorrheic torment. Each of the medications was taken orally by more than 200 ladies day by day for three 28-day cycles, with the quantity of days of torment and the level of agony being assessed. In view of the patients' self-appraisals, the specialists established that agony was lessened by the two medications by around a similar number of days (by 4.6 days for estradiol valerate in addition to dienogest, by 4.2 days for ethinyl estradiol in addition to levonorgestrel).

• Dietary and Other Therapies

Other therapies for dysmenorrhea have been proposed, but most are not well studied. A low-fat vegetarian diet, pyridoxine, magnesium, and vitamin E are examples^[29]. Furthermore, acupuncture, acupressure, numerous herbal medicines and dietary supplements, transdermal nitroglycerin, calcium-channel blockers, beta-adrenergic agonists, antileukotrienes, transcutaneous electrical nerve stimulation (TENS) units, and massage therapy and isometric exercise^[30] have been suggested for therapeutic use in this setting. Topical application of continuous low-level heat may be beneficial for some patients. Interruption of nerve pathways has been performed, but data are limited^[31].

PREVENTION

Various measures have been used to manage dysmenorrhea in the outpatient setting, including the following:

- Lifestyle modification seems to be helpful.
- Smoking cessation should be encouraged, in that smoking may be a risk factor for dysmenorrhea^[32]
- Exercise has been shown to alleviate symptoms of dysmenorrhea, though the mechanism is not well understood^[32].

A Cochrane review of 5 randomized controlled trials showed that certain behavioral interventions

may be effective at treating primary and secondary dysmenorrhea^[33].

CONCLUSION

Dysmenorrhea is found to be highly prevalent among college going girls. Studies suggest family history, bleeding duration and presence of clots as significant risk factors for dysmenorrhea in these girls. Majority of girls were suffering from pre-menstrual symptoms indicating the magnitude of problem and thus, need an appropriate intervention through a change in lifestyle.

The study was done according to the ethical board of King Abdulaziz university.

REFERENCES

1. **Jamieson DJ, Steege JF(1996):** The prevalence of dysmenorrhea, dyspareunia, pelvic pain, and irritable bowel syndrome in primary care practices. *Obstet Gynecol.*, 87(1):55-8.
2. **Dawood MY(1985):** Dysmenorrhea. *J Reprod Med.* , 30(3):154-67.
3. **Koltz MM(1992):** Dysmenorrhea, endometriosis and pelvic pain. Lemeke DP, Pattison J, Marshall LA, Cowley DS, eds. *Primary Care of Women*. Norwalk Conn: Appleton & Lange.
4. **Dawood MY(1988):** Nonsteroidal anti-inflammatory drugs and changing attitudes toward dysmenorrhea. *Am J Med.*,84(5A):23-9.
5. **Dawood MY(1990):** Dysmenorrhea. *ClinObstet Gynecol.*,33(1):168-78.
6. **Harlow SD, Park M(1996):** A longitudinal study of risk factors for the occurrence, duration and severity of menstrual cramps in a cohort of college women. *Br J ObstetGynaecol.*,103(11):1134-42.
7. **Parazzini F, Tozzi L, Mezzopane R, Luchini L, Marchini M, Fedele L(1994):** Cigarette smoking, alcohol consumption, and risk of primary dysmenorrhea. *Epidemiology*,5(4):469-72.
8. **Sundell G, Milsom I, Andersch B(1990):** Factors influencing the prevalence and severity of dysmenorrhoea in young women. *Br J ObstetGynaecol.*,97(7):588-94.
9. **Durain D(2004):** Primary dysmenorrhea: assessment and management update. *J Midwifery Womens Health*, 49(6):520-8.
10. **Baines PA, Allen GM(2001):** Pelvic pain and menstrual related illnesses. *Emerg Med Clin North Am.*,19(3):763-80.
11. **Kalish GM, Patel MD, Gunn ML, Dubinsky TJ(2007):** Computed tomographic and magnetic resonance features of gynecologic abnormalities in women presenting with acute or chronic abdominal pain. *Ultrasound Q.* , 23(3):167-75.
12. **Kamaya A, Shin L, Chen B, Desser TS(2008):** Emergency gynecologic imaging. *Semin Ultrasound CT MR.*,29(5):353-68.
13. **Houry D, Abbott JT(200):** Ovarian torsion: a fifteen-year review. *Ann Emerg Med.*, 38(2):156-9.

14. Harada T , Momoeda M , Taketani Y , Aso T , Fukunaga M , Hagino H (2009): Dienogest is as effective as intranasal buserelin acetate for the relief of pain symptoms associated with endometriosis: a randomized, double-blind, multicenter, controlled rial. *Fertile Steril.*,91: 675-681
15. Proctor M, Farquhar C(2006): Diagnosis and management of dysmenorrhoea. *BMJ.*, 332(7550):1134-8.
16. Propst AM, Storti K, Barbieri RL(1998): Lateral cervical displacement is associated with endometriosis. *FertilSteril.*,70(3):568-70.
17. Barbieri RL, Propst AM(1999): Physical examination findings in women with endometriosis: uterosacral ligament abnormalities, lateral cervical displacement and cervical stenosis. *J Gynecol Tech.*,135:102.
18. Liu P, Yang J, Wang G, Liu Y, Liu X, Jin L *et al.*(2016): Altered regional cortical thickness and subcortical volume in women with primary dysmenorrhoea. *Eur J Pain*, 20 (4):512-20.
19. Dawood MY, Khan-Dawood FS(2007): Clinical efficacy and differential inhibition of menstrual fluid prostaglandin F2alpha in a randomized, double-blind, crossover treatment with placebo, acetaminophen, and ibuprofen in primary dysmenorrhea. *Am J Obstet Gynecol.*,196(1):35.e1-5.
20. Zhang WY, Li Wan Po A(1998): Efficacy of minor analgesics in primary dysmenorrhoea: a systematic review. *Br J ObstetGynaecol.*,105(7):780-9.
21. Rosenwaks Z, Jones GS, Henzl MR, Dubin NH, Ghodgaonkar RB, Hoffman S(1981): Naproxen sodium, aspirin, and placebo in primary dysmenorrhea. Reduction of pain and blood levels of prostaglandin F2-alpha metabolite. *Am J Obstet Gynecol.*, 140(5):592-8.
22. Daniels SE, Torri S, Desjardins PJ(2005): Valdecoxib for treatment of primary dysmenorrhea. A randomized, double-blind comparison with placebo and naproxen. *J Gen Intern Med.*,20(1):62-7.
23. Iacovides S, Avidon I, Baker FC(2015): What we know about primary dysmenorrhea today: a critical review. *Hum Reprod Update*, 21 (6):762-78.
24. Fujiwara H, Konno R, Netsu S *et al.*(2010): Efficacy of montelukast, a leukotriene receptor antagonist, for the treatment of dysmenorrhea: a prospective, double-blind, randomized, placebo-controlled study. *Eur J ObstetGynecolReprod Biol.*,148(2):195-8.
25. Wong CL, Farquhar C, Roberts H, Proctor M(2009): Oral contraceptive pill as treatment for primary dysmenorrhoea. *Cochrane Database Syst Rev.*,CD002120.
26. Sillem M, Schneidereit R, Heithecker R, Mueck AO(2003): Use of an oral contraceptive containing drospirenone in an extended regimen. *Eur J ContraceptReprod Health Care*, 8(3):162-9.
27. Cheewadhanaraks S, Choksuchat C, Dhanaworavibul K, Liabsuetrakul T(2012): Postoperative depot medroxyprogesterone acetate versus continuous oral contraceptive pills in the treatment of endometriosis-associated pain: a randomized comparative trial. *GynecolObstet Invest.*,74(2):151-6.
28. Petraglia F, Parke S, Serrani M *et al.*(2014): Estradiol valerate plus dienogest versus ethinylestradiol plus levonorgestrel for the treatment of primary dysmenorrhea. *Int J Gynaecol Obstet.*,125(3):270-4.
29. Ziaei S, Zakeri M, Kazemnejad A(2005): A randomised controlled trial of vitamin E in the treatment of primary dysmenorrhoea. *BJOG.*,112(4):466-9.
30. Azima S, Bakhshayesh HR, Kaviani M, Abbasnia K, Sayadi M(2015): Comparison of the effect of massage therapy and isometric exercises on primary dysmenorrhea: a randomized controlled clinical trial. *J PediatrAdolesc Gynecol.*,28 (6):486-91.
31. Latthe PM, Proctor ML, Farquhar CM, Johnson N, Khan KS(2007):Surgical interruption of pelvic nerve pathways in dysmenorrhea: a systematic review of effectiveness. *ActaObstetGynecol Scand.*,86(1):4-15.
32. Latthe P, Mignini L, Gray R, Hills R, Khan K(2006): Factors predisposing women to chronic pelvic pain: systematic review. *BMJ.*,332(7544):749-55.
33. Proctor ML, Murphy PA, Pattison HM, Suckling J, Farquhar CM(2007): Behavioural interventions for primary and secondary dysmenorrhoea. *Cochrane Database Syst Rev.*,18(3):CD002248.