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PRIMARY DYSMENORRHOEA – A CRITICAL REVIEW

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Abstract

Dysmenorrhoea literally means painful menstruation. A more realistic and practical definition includes cases of painful menstruation of sufficient magnitude so as to incapacitate day-to-day activities. It means cramping pain accompanying menstruation. It is mainly of two types – Primary, Secondary. Primary Dysmenorrhoea one that is not associated with any identifiable pelvic pathology. It is now clear that the pathogenesis of pain is attributed to a biochemical derangement. It affects more than 50% postpubescent women in the age group of 18-25 years with ovulatory cycles.

Key words: Primary Dysmenorrhoea, painful menstruation

Introduction

Dysmenorrhoea literally means painful menstruation. A more realistic and practical definition includes cases of painful menstruation of sufficient magnitude so as to incapacitate day-to-day activities. [1] It means cramping pain accompanying menstruation.

Patients can be classified into groups for understanding the pathogenesis of this distressing condition.

Types

1. Primary dysmenorrhoea refers to the one that is not associated with any identifiable pelvic pathology. It is now clear that the pathogenesis of pain is attributed to a biochemical derangement. It affects more than 50% postpubescent women in the age group of 18-25 years with ovulatory cycles.

2. Secondary dysmenorrhoea refers to the one associated with the presence of organic pelvic pathology, i.e. fibroids, adenomyosis, PID and endometriosis. Unilateral dysmenorrhoea occurs in a rudimentary horn of a bicornuate uterus. It is also seen in some women wearing IUCD and in cases of cervical stenosis.

Varieties

Dysmenorrhoea is described under three clinical varieties:

1. Spasmodic dysmenorrhoea is the most prevalent one and manifests as cramping pains, generally most pronounced on the first and second day of menstruation.

2. Congestive dysmenorrhoea manifests as increasing pelvic dis comfort and pelvic pain a few days before the start of menses. Thereafter, the patient rapidly experiences relief in symptoms. This variety is commonly seen in PID, IUCD wearers, pelvic endometriosis and fibroids. It is also experienced by women having varicosity of pelvic veins.

3. Membranous dysmenorrhoea is a special group in which the endometrium is shed as a cast at the time of menstruation. The passage of the cast is accompanied by painful uterine cramps. This is a rare variety.[2]

Primary Dysmenorrhoea

It is also known as Spasmodic dysmenorrhoea.

Incidence and Prevalence

The incidence of primary dysmenorrhoea of sufficient magnitude with incapacitation is about 5-10 per cent. The incidence of dysmenorrhoea is affected by social status, occupation and age, so groups of schoolgirls, college students, factory workers and women members of the armed forces each provide different status. The number of girls complaining of incapacitating true dysmenorrhoea has decreased considerably during the last 20-30 years. [3] With the advent of oral contraceptives and non-steroidal anti-inflammatory drugs, there is marked relief of the symptom.

In India, prevalence of dysmenorrhea in female students is 70.2%. Majority of the subjects experience pain for one or 1-2 days during menstruation. 23.2% of the dysmenorrheic girls experience pain for 2-3 days. The most common symptom in both dysmenorrheic and non dysmenorrheic girls during the menstrual periods was tiredness and second most prevalent symptom was back pain.[4]

Causes of pain

The mechanism of initiation of uterine pain in primary dysmenorrhoea is difficult to establish. But the following are too often related.

- Mostly confined to adolescents.
- Almost always confined to ovulatory cycles.
- The pain is usually cured following pregnancy and vaginal delivery.
- The pain is related to dysrhythmic uterine contractions and uterine hypoxia.

Aetiology

Behavioural and Psychological Factors

Just before and during menstruation most women are less efficient physically and more unstable emotionally; these factors alone lower the pain threshold. The expectation of pain may be fostered by overanxious parents and by curtailment of normal activities during menstruation. A dysmenorrhoeic mother usually has a dysmenorrhoeic daughter. A girl who is an only child is more likely than most to suffer from dysmenorrhoea. It is often very difficult to separate the respective contributions of physiological and psychological factors and such factors may make dysmenorrhoea worse even if they do not cause it; these include unhappiness at home or at work, unsatisfied sex urge, fear or loss of employment, and anxiety over examinations. Marriage may cure by removing the tension of a long engagement and by providing happiness and security; on the other hand, if it proves disharmonious, it can cause dysmenorrhoea. During adolescence, lower pain threshold is

often attributed as an aggravating factor inpain perception. This may explain, in part, the disappearance of pain with advancing age.

Muscular Incoordination and Uterine Hyperactivity

Spasmodic dysmenorrhoea could be due to incoordinate muscle action of the uterus as a whole. If so, it could be explained by an imbalance in the autonomic nervous control of muscles, in which an overactive sympathetic system leads to hypertonus of the circular fibres of the isthmus and the internal os. This would fit in with the general nervous instability of many of these patients and also with the other manifestations of autonomic upset-such as bowel and bladder tenesmus-which accompany dysmenorrhoea. It does not, however, explain why dysmenorrhoea is cured by age and by childbearing.

Abnormal anatomical and functional aspect of uterus

Uterine myometrial hyperactivity has been observed in cases with primary dysmenorrhoea.
• Stenosis at the internal os or narrowing of the cervical canal → difficult for the menstrual blood to escape → strong uterine contractions → pain. This may explain relief of pain following dilatation of the cervix.
• Unequal development of Müllerian ducts, such as septate or bicornuate uterus. The pain is due to unequal muscular contractions.
• Uterine hypoplasia inadequate expulsive force.
• Imbalance in the autonomic nervous control of uterine muscle.

Role of Progesterone

Spasmodic dysmenorrhoea has some connection with progesterone stimulus to the uterus. It only occurs in ovular cycles, and it is suggested, but not proved, that the occurrence of anovular menstruation explains the absence of dysmenorrhoea during the few years following the menarche and the occasional painless period even at a later age. Another observation is that progesterone induces high tone in the isthmus and upper cervix. An exaggeration of this could therefore be the basis of the incoordinate action of the uterus.

Role of Prostaglandins

The most favoured view is that dysmenorrhoea is associated with an excess of prostaglandin F2, in the uterus. It has been demonstrated that secretory endometrium contains more prostaglandins than proliferative endometrium. Prostaglandins are known to increase myometrial contractions and constrict small endometrial blood vessels to produce ischaemia and breakdown of the endometrium, bleeding and pain. Increased levels of prostaglandin E_2 are seen in these patients and may increase the sensitivity of the nerve endings to pain.

Role of Vasopressin

There is increased vasopressin release during menstruation in women with primary dysmenorrhoea. Vasopressin increases prostaglandin synthesis and also increases myometrial activity directly. It causes uterine hyperactivity and dysrhythmic contractions, ischaemia and hypoxia leading to pain.

Pathophysiology

The pain of primary dysmenorrhoea is caused by excessive E2 and F2α prostaglandin production within secretory endometrial cells. This prostaglandin release, in turn, causes uterine contractions, uterine muscle ischaemia and increased peripheral nerve sensitivity. Coupled with their elevated prostaglandin levels, dysmenorrhoeic women have higher levels of uterine activity during menstruation compared to asymptomatic women. Menstrual bleeding is triggered by progesterone withdrawal following the demise of the corpus luteum. As progesterone levels drop prior to menstruation, prostaglandin levels increase together with

stimulation of the type-C pain fibres and play a big role in pain, inflammation and other physiological processes regulating body temperature and sleep.

Risk factors

Dysmenorrhoea does not usually occur within the first six months, after the first menstruation. There are a number of risk factors significantly associated with dysmenorrhoea, including the following

- Low body mass index
- Early menarche
- Prolonged menstrual flow for seven days or longer
- Pelvic infections
- Genetic or family history
- Premenstrual somatic complaints
- A history of sexual assault [5]

Clinical features

Patient profile

Primary dysmenorrhoea is predominantly confined to adolescent girls. It usually appears within 2 years of menarche. The mother or her sister may be dysmenorrhoeic. It is more common amongst girls from affluent society.

Symptoms

The pain of primary dysmenorrhea usually begins a few hours before or just after the onset of a menstrual period and may last 48 to 72 hours. The pain is similar to labor, with suprapubic cramping, and may be accompanied by lumbosacral backache, pain radiating down the anterior thigh, nausea, vomiting, diarrhea, and rarely syncopal episodes. The pain of dysmenorrhea is colicky in nature and, unlike abdominal pain that is caused by chemical or infectious peritonitis, is relieved by abdominal massage, counter-pressure, or movement of the body.

Signs

On examination, the vital signs are normal. The suprapubic region may be tender to palpation. Bowel sounds are normal, and there is no upper abdominal tenderness and no abdominal rebound tenderness. Bimanual examination at the time of the dysmenorrheic episode often reveals uterine tenderness; severe pain does not occur with movement of the cervix or palpation of the adnexal structures. The pelvic organs are normal in primary dysmenorrhea.

Diagnosis

To diagnose primary dysmenorrhea, it is necessary to clinically rule out underlying pelvic pathology and confirm the cyclic nature of the pain. During the pelvic examination, the size, shape, and mobility of the uterus; the size and tenderness of adnexal structures; and the nodularity or fibrosis of uterosacral ligaments or rectovaginal septum should be assessed. NAAT for gonorrhea and chlamydia and if relevant, CBC and ESR, help rule out endometritis and subacute PID, Pelvic ultrasound should be performed if symptoms do not resolve with NSAIDs. If no abnormalities are found, a tentative diagnosis of primary dysmenorrhea can be established. Laparoscopy is not necessary at this point. (bn)[6]

Treatment

General measures include improvement of general health and simple psychotherapy in terms of explanation and assurance. During menses, bowel should be kept empty; mild analgesics and antispasmodics may be prescribed. With these simple measures, the pain is relieved in majority.

In refractory or severe cases, drugs like Prostaglandin synthetase inhibitors, Oral contraceptives (combined oestrogen and progestogen), Prostaglandin synthetase inhibitors (PSI) etc are indicated.Transcutaneous

electrical nerve stimulation, Laproscopic presacral neurectomy are the common surgical procedures indicated for relieving pain.

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