****

**Premenstrual syndrome**

**Objectives:**

**1-learn the definition ,pathogenesis and clinical symptoms.**

**2-be able to diagnose and manage according to the severity of the disease.**

**Definition**: Premenstrual syndrome (PMS) is the occurrence of cyclical somatic, psychological and emotional symptoms that occur in the luteal (premenstrual) phase of the menstrual cycle and resolve by the time menstruation ceases.

Premenstrual symptoms occur in almost all women of reproductive age. In 3–60 per cent, symptoms are severe, causing disruption to everyday life, in particular interpersonal relationships.

**Pathogenesis**

The etiology of the PMS is not known, although several theories have been proposed, including

1. estrogen–progesterone imbalance, excess aldosterone, hypoglycemia, hyperprolactinemia, and psychogenic factors,
2. physiologic ovarian function is believed to be the trigger. This is supported by the efficacy of ovarian cyclicity suppression, either medically or surgically, in eliminating premenstrual complaints.
3. serotonin (5-hydroxytryptamine [5-HT]), a neurotransmitter, is important in the pathogenesis of PMS. Both estrogen and progesterone have been shown to influence the activity of serotonin and gamma-aminobutyric acid (GABA) centrally. GABA levels are decreased in women with PMS during the late luteal phase compared with normal women.

**Diagnosis**

**History and examination**

History taking of Certain medical conditions (eg, thyroid disease and anemia) with symptoms that can mimic those of PMS must be ruled out.

Symptoms include mood symptoms (irritability, mood swings, depression, anxiety), physical symptoms (bloating, breast tenderness, insomnia, fatigue, hot flushes, appetite changes), and cognitive changes (confusion and poor concentration).

Symptoms must occur in the second half of the menstrual cycle (luteal phase).

There must be a symptom-free period of at least 7 days in the first half of the cycle.

Symptoms must occur in at least 2 consecutive cycles.

Symptoms must be severe enough to require medical advice or treatment. PMS is rarely encountered in adolescents and resolves after menopause.

According to the American College of Obstetricians and Gynecologists criteria, PMS can be diagnosed if the patient reports at least 1 affective symptom (depression, angry outbursts, irritability, anxiety, confusion, or social withdrawal) and somatic symptom (breast tenderness, abdominal bloating, headache, or swelling of extremities) during the 5 days before menses in each of the 3 prior menstrual cycles. These symptoms should be relieved within 4 days of the onset of menses, without recurrence until at least cycle day 13, and should be reproducible during 2 cycles of prospective recording.

**examination**

A careful history and physical examination are most important to exclude organic causes of PMS localized to the reproductive, urinary, or gastrointestinal tracts. Migraine-like headaches may occur, often preceded by visual scotomas and vomiting.

One of the most common symptoms of PMS is mastodynia, or mastalgia (pain, and usually swelling, of the breasts caused by edema and engorgement of the vascular and ductal systems). Examination is always necessary to rule out neoplasm, although most malignant tumors are painless.

A psychiatric history should be obtained, with special attention paid to a personal history of psychiatric problems or a family history of affective disorders. The most common associated psychiatric illness is depression, which generally responds to antidepressant drugs and psychotherapy.

**Medical treatments**

1. ***Combined oral contraceptive pill***

The most effective preparation appears to be Yasmin™, which contains an anti-mineralocorticoid and an anti-androgenic progestogen. The most effective regime appears to be bicycling or tricycling pill packets (i.e. taking two or three packets in a row without a scheduled break).

1. ***Transdermal oestrogen***

This has been shown to significantly reduce PMS symptoms.

1. ***GnRH analogues***

GnRH analogues are a very effective treatment for PMS as ovarian activity is switched off. However, this is generally a short-term treatment. If used for more than six months, HRT should be given to reduce the risk of osteoporosis.

1. ***Selective serotonin reuptake inhibitors***

There is good evidence that this group of drugs significantly improve PMS.

1. ***Hysterectomy with bilateral salpingo-oopherectomy***

This procedure obviously completely removes the ovarian cycle. It should only be performed if all other treatments have failed. It is essential for such patients to have a preoperative trial of GnRH analogue as a ‘test’ to ensure that switching off ovarian function (by removing the ovaries at hysterectomy) will indeed cure the problem.

1. ***Vitamins***

Initial studies suggest that magnesium, calcium and isoflavones may be useful in treating PMS.

1. ***Alternative therapies***

Initial results of St John’s Wort are promising, particularly in improving mood. Although Evening primrose oil is commonly used, there is no evidence to support this treatment for PMS.

1. **Cognitive-behavioural therapy**

effective when combined with selective serotonin reuptake inhibitors (SSRIs).

**DYSMENORRHEA**

Dysmenorrhea, or painful menstruation, is one of the most common complaints of gynecologic patients. Many women experience mild discomfort during menstruation, but the term *dysmenorrhea* is reserved for women whose pain prevents normal activity and requires medication, whether an overthe-counter or a prescription drug.

There are 2 types of dysmenorrhea: (1) primary (no organic cause), (2) secondary (pathologic cause)discussion focuses mainly on primary dysmenorrhea. Secondary dysmenorrhea is discussed later in association with specific diseases and disorders (eg, endometriosis, adenomyosis, pelvic inflammatory disease, cervical stenosis, fibroids, and endometrial polyps).

**Pathogenesis**

Pain during menstruation has long been known to be associated with ovulatory cycles. The mechanism of pain has been attributed to prostaglandin activity. Advances in the last 3 decades and current understanding suggest that in primary dysmenorrheal there is abnormal and increased prostanoid and possibly eicosanoid secretion, which in turn induces abnormal uterine contractions. The contractions reduce uterine blood flow, leading to uterine hypoxia.

Other studies have confirmed increased leukotriene levels as a contributing factor. Vasopressin was thought to be an aggravating agent, but atosiban, a vasopressin antagonist, has shown no effect on menstrual pain.

Psychological factors may be involved, including attitudes passed from mother to daughter. Girls should receive accurate information about menstruation before menarche; this can be provided by parents, teachers, physicians, or counselors. Emotional anxiety due to academic or social demands may be a cofactor.

**Clinical Findings**

Reactions to pain are subjective, and questioning by the physician should not lead the patient to exaggerate or minimize her discomfort. History taking is most important and should include the following questions: When does the pain occur? What does the patient do about the pain? Are there other symptoms? Do oral contraceptives relieve or intensify the pain? Does the pain become more severe over time? Because dysmenorrhea almost always is associated with ovulatory cycles, it does not usually occur at menarche but rather later in adolescence. As many as 14–26% of adolescents miss school or work as a result of pain. Typically, pain occurs on the first day of the menses, usually about the time the flow begins, but it may not be present until the second day. Nausea and vomiting, diarrhea, and headache may occur. The specific symptoms associated with endometriosis are not present. The physical examination does not reveal any significant pelvic disease. When the patient is symptomatic, she has generalized pelvic tenderness, perhaps more so in the area of the uterus than in the adnexa. Occasionally, ultrasonography or laparoscopy is necessary to rule out pelvic abnormalities such as endometriosis, pelvic inflammatory disease, or an accident in an ovarian cyst.

**Differential Diagnosis**

The most common misdiagnosis of primary dysmenorrhea is secondary dysmenorrhea due to endometriosis. With endometriosis, the pain usually begins 1–2 weeks before the menses, reaches a peak 1–2 days before, and is relieved at the onset of flow or shortly thereafter. Severe pain during sexual intercourse or findings of adnexal tenderness or mass or cul-de-sac nodularity, particularly in

the premenstrual interval, help to confirm the diagnosis . A similar pain pattern occurs with adenomyosis, although in an older age group and in the absence of extrauterine clinical findings.

**Treatment**

NSAIDs or acetaminophen may relieve mild discomfort. Addition of continuous heat to the abdomen in addition to NSAIDs decreases pain significantly. For severe pain, codeine or other stronger analgesics may be needed, and bed rest may be desirable. Occasionally, emergency treatment with parenteral medication is necessary. Analgesics may cause drowsiness at the dosages required.

1. **Antiprostaglandins**

Antiprostaglandins are now used for treatment of dysmenorrhea. The newer, stronger, faster-acting drugs appear to be more useful than aspirin. Ibuprofen and naproxen, NSAIDs that are available over the counter, have been extremely effective in reducing menstrual prostaglandin and relieving dysmenorrhea. More specific cyclooxygenase-2 (COX-2) inhibitors are now available, but concerns

about their adverse effects have recently attracted attention. Rofecoxib, valdecoxib, and lumiracoxib are effective for treating primary dysmenorrhea. Thus far, COX-2 inhibitors are equally effective but not better than naproxen. Given the above considerations, concerns about safety of COX-2 inhibitors, the short duration of therapy for relieving primary dysmenorrhea, and the low costs of NSAIDs, it is prudent to recommend established NSAIDs with track records of long-term safety as the preferred pharmacologic agent. The drug must be used at the earliest onset of symptoms, usually at the onset of, and sometimes 1–2 days prior to, bleeding or cramping. Antiprostaglandins work by blocking prostaglandin synthesis and metabolism. Once the pain has been established, antiprostaglandins are not nearly as effective as with early use.

1. **Oral Contraceptives**

Cyclic administration of oral contraceptives, usually in the lowest dosage but occasionally with increased estrogen, prevents pain in most patients who do not obtain relief from antiprostaglandins or cannot tolerate them. The mechanism of pain relief may be related to absence of ovulation or to altered endometrium resulting in decreased prostaglandin production. In women who do not require

contraception, oral contraceptives are given for 6–12 months. Many women continue to be free of

pain after treatment has been discontinued. NSAIDs act synergistically with oral contraceptive pills to improve dysmenorrhea.

1. **Surgical Treatment**

In a few women, no medication controls dysmenorrhea. Cervical dilatation is of little use. Laparoscopic uterosacral ligament division and presacral neurectomy are infrequently performed, although some physicians consider these procedures to be important adjuncts to conservative operation for endometriosis.

Adenomyosis, endometriosis, or residual pelvic infection unresponsive to medical therapy or conservative surgical therapy eventually may require hysterectomy with or without ovarian removal in extreme cases. Rarely, a patient with no organic source of pain eventually requires hysterectomy to relieve symptoms.

1. **Adjuvant Treatments**

Continuous low-level topical heat therapy has been shown to be as effective as ibuprofen in treating dysmenorrhea, although its practicality in daily life may be questionable. Many studies have indicated that exercise decreases the prevalence and/or improves the symptomatology of dysmenorrhea, although solid evidence is lacking.

 **Best luck**