Dysmenorrhoea

Derived from the Greek meaning difficult monthly flow, the word dysmenorrhoea has come to mean painful menstruation.

Dysmenorrhoea can be classified as either

1. Primary
2. Secondary

Aetiology:

Primary dysmenorrhoea: In this type there is no pelvic pathology.

Secondary dysmenorrhoea: implies underlying pathology which leads to painful menstruation.

Primary dysmenorrhoea

The prevalence of dysmenorrhoea: is high about 72%, nearly 40% regularly used medication for the pain, 8% stayed absent from work or school at every period.

1. Primary dysmenorrhoea: is associated with uterine hypercontractility characterized by excessive amplitude and frequency of contractions and a high ‘resting’ tone between contractions.
2. During contractions endometrial blood flow is reduced and there seems to be a good correlation between minimal blood flow and maximal colicky pain.
3. Prostaglandin and leukotriene levels elevated.

Secondary dysmenorrhoea:

Secondary dysmenorrhoea may be a symptom of:

1. Uterine fibroids
2. endometriosis.
3. pelvic inflammatory disease
4. adenomyosis
5. Asherman’s syndrome
6. (rarely) cervical stenosis
Primary dysmenorrhoea

The risk factors for primary dysmenorrhoea include:

1. duration of menstrual flow of > 5 days,
2. younger than normal age at menarche,
3. cigarette smoking.

There is some evidence to support the assertion that dysmenorrhoea improves after childbirth, and it also appears to decline with increasing age.

Presentation and assessment

In general primary dysmenorrhoea appears 6–12 months after the menarche when ovulatory cycles begin to become established. The early cycles after the menarche are usually anovular and tend to be painless. The pain usually consists of lower abdominal cramps and backache and there may be associated gastrointestinal disturbances such as diarrhoea and vomiting. Symptoms occur predominantly during the first 2 days of menstruation (8-72 hours).

Diagnosis: primary dysmenorrhoea

- The diagnosis of primary dysmenorrhoea is one of exclusion.
- If symptoms are typical of primary dysmenorrhoea, a therapeutic trial may be embarked on before considering any examination and investigation especially in adolescents.

If clinical evaluation raises suspicion of secondary dysmenorrhoea:

transvaginal sonography (TVS) or magnetic resonance imaging (MRI) or laparoscopy should be considered if symptoms of primary dysmenorrhoea are not alleviated with either NSAIDs or the combined oral contraceptive pill, secondary causes of dysmenorrhoea need to be considered. An endocervical swab for Chlamydia trachomatis and Neisseria gonorrhoea and a high vaginal swab for other pathogens should be taken at this stage.

if an organic cause appears likely, it may be appropriate to perform pelvic ultrasound, followed, if necessary, by laparoscopy to investigate further. If other features in the history suggest the possibility of Asherman’s syndrome or cervical stenosis, hysteroscopy can be used.

Management:

Women will usually seek medical advice when self-help measures such as heat and over the counter NSAIDs have failed. The mainstays of treatment are Non steroidal anti inflammatory drugs(NSAIDs) and the combined oral contraceptive pill, the latter especially when fertility control is required.
1. NON-STEROIDAL ANTI-INFLAMMATORY DRUGS

COX-1 inhibitors: such as mefenamic acid, naproxen, ibuprofen and aspirin are all effective.

Ibuprofen is the preferred analgesic because of its favourable efficacy and safety profiles.

Commencing treatment before the onset of menstruation appears to have no demonstrable advantage over starting treatment when bleeding starts. This observation is compatible with the short plasma half-life of NSAIDs.

2. THE COMBINED ORAL CONTRACEPTIVE PILL

They are thought to act by inhibiting ovulation and decreasing endometrial production of prostaglandins and leukotrienes by inducing endometrial atrophy and therefore reducing the amount of endometrial tissue available to produce these mediators.

3. OTHER HORMONAL METHODS:

Although primarily designed for parous women, the levonorgestrel – intrauterine system (LNG-IUS) may be an effective treatment for women who have a contraindication to either NSAIDs or the combined oral contraceptive. Alternatives include:

- depot progestogens used for contraception Clinically they are effective since they render most women amenorrhoic.

Other medication:

Nifedipine is widely used in Scandinavia, but is not licensed for this indication in the UK.

Surgical treatments

Surgical treatments aimed at interrupting the nerve pathways from the uterus have been employed, and there is some evidence of their efficacy in the long term. This should be confined to specialist centres for the treatment of women whose condition is unresponsive to other therapies.
Premenstrual syndrome

Introduction

Premenstrual syndrome (PMS) is the medical term most often used and estimated to occur in 5% of women.

Definition

A woman is considered to have premenstrual syndrome if she complains of recurrent psychological or somatic symptoms (or both), occurring specifically during the luteal phase of the menstrual cycle and which resolve in the follicular phase at least by the end of menstruation.

Symptoms

- A wide range of symptoms has been described but it is their timing and severity that are most important, more so than the specific character.
- Depression, irritability, anxiety, tension, aggression, inability to cope and feeling out of control are typical psychological symptoms.
- Bloating, mastalgia and headache are classical physical symptoms.

Diagnosis: premenstrual syndrome

- There are no objective tests (physical, biochemical or endocrine) to assist in making the diagnosis.
- Prospectively completed specific symptom charts are required.

This is partly because retrospective reporting of symptoms is inaccurate and because significant numbers of women who present with PMS have another underlying problem such as:

Condition mimic

PMS (premenstrual syndrome)

- the perimenopause.
- thyroid disorder.
- migraine.
- chronic fatigue syndrome.
- irritable bowel syndrome.

menstrual disorders as well as psychiatric disorders such as depression, bipolar illness, panic disorder, personality disorder and anxiety disorder.
**Diagnosis:**

The confirmation of luteal phase timing with the relief of symptoms by the end of menstruation is diagnostic providing the symptoms are of such severity to impact on the patient’s normal functioning.

**Aetiology of Premenstrual syndrome**

- Premenstrual syndrome is not due to a single factor.
- Genetic,
- environmental,
- psychological are important factors in mood disorders.
- as well as hormonal influence.

The principal cause of PMS is uncertain is strongly considered that the cyclical endogenous progesterone produced in the luteal phase of the cycle is responsible for symptoms in women who are unusually sensitive to normal progesterone levels. It has been hypothesized that the mechanism of this increased sensitivity is related to an abnormal neuroendocrine factor and most evidence points to a dysregulation of serotonin metabolism.

**Women have no PMS**

1. before puberty,
2. during pregnancy or,
3. after the menopause –

these are times where ovarian hormone cycling has not begun or has ceased.

**Suppression of the ovarian endocrine cycle with:**

- danazol,
- following administration of analogues of GnRH or
- by bilateral oophorectomy results in the suppression of PMS symptoms.

Therefore, the hypothesis that ovarian steroids have a role in the pathophysiology of the syndrome.

**Treatment of premenestural syndrome**

1. Non–medical therapies.
2. Medical therapies
3. Surgical therapies
1. Non medical therapy

- exercise,
- yoga,
- acupuncture,
- psychotherapy and many more there is very little evidence that any of these treatments for PMS are effective with the exception of exercise and cognitive behavioural therapy

2. Medical therapies

1. the supplementation of calcium
2. vitamin E.
3. magnesium
4. dietary change.
5. vitamin B6.
6. evening primrose oil

Remember that the proposed aetiology of PMS is that normal post-ovulatory progesterone gives rise to symptoms only in women who have increased sensitivity to progesterone and this is likely to be due to serotonin deficiency.

1. SSRIs: serotonin re uptake inhibitor

Fluoxetine 20 mg daily is usually sufficient to improve symptoms in most women.

Side effects such as loss of libido may be partially avoided by administering the drug only during the luteal phase.

2. Cycle suppression

Suppression of the cycle can be achieved with Oestrogen, Danazol, GnRH agonist analogues or Danazol, even at doses of 200 mg, is particularly effective for most symptoms of PMS but is limited by masculinizing side effects.

3. Surgical therapies

Oophorectomy: Bilateral oophorectomy usually with hysterectomy is almost always too invasive though is the only effective cure for premenstrual syndrome. When removal of the ovaries is considered appropriate, it can be followed by oestrogen replacement without of course the need for cyclical progestagens.

Conclusion:

- Suppression of the ovarian cycle eliminates PMS effectively.

- This can be achieved by GnRH analogues with add back tibolone.

- Oestrogen also suppresses ovulation and eliminates PMS without menopausal side effects.

- Intrauterine progestagen (as levonorgestrel IUS) avoids re-stimulation of premenstrual syndrome at the same time that it protects the endometrium.