كلية : كلية الطب العام

القسم او الفرع : النسائية والتوليد

المرحلة: الخامسة

أستاذ المادة : م.د.آلاء شلال فرحان

اسم المادة باللغة العربية : أمراض النساء

اسم المادة باللغة الإنكليزية : Gynecology

اسم المحاضرة الحادية والثلاثون الثلاثون باللغة العربية: آلام الدورة الشهرية والمتلازمة السابقة الحيض

اسم المحاضرة الحادية والثلاثون باللغة الإنكليزية : Dysmenorrhea and Premenstrual Syndrome

محتوى المحاضرة الثانية والثلاثون

Dysmenorrhea and Premenstrual syndrome

Dysmenorrhoea

Dysmenorrhoea is defined as painful menstruation. It is experienced by 45–95% of women of reproductive age.

Primary dysmenorrhoea describes painful periods since onset of menarche and is unlikely to be associated with pathology. There is some evidence to support the assertion that primary dysmenorrhoea improves after childbirth, and it also appears to decline with increasing age.

Secondary dysmenorrhoea describes painful periods that have developed over time and usually have a secondary cause.

Aetiology of secondary dysmenorrhoea

Aetiology includes:

1.Endometriosis and adenomyosis.

2.Pelvic inflammatory disease.

3.Cervical stenosis and haematometra (rarely).

History and examination

Patients will have different ideas as to what constitutes a painful period. For some patients reassurance that the pain may be normal for her will help. For others the ability to alter the menstrual cycle to avoid having a period during key events, for example school examinations or holidays, will be helpful.

To ascertain the actual severity of the pain, the following questions may be useful:

- Do you need to take painkillers for this pain? Which tablets help?
- Have you needed to take any time off work/school due to the pain?

Some primary dysmenorrhoea is associated with flushing and nausea, which may be prostaglandin related. It is important to distinguish between menstrual pain that precedes the period (a vital clue in endometriosis) and pain that only occurs with bleeding. Other important clues about the aetiology include pain that occurs with passage of clots, in which case medication to reduce flow may be effective.

An abdominal and pelvic examination should be performed (except pelvic examination not done to unmarried). Certain signs associated with endometriosis include a pelvic mass (if an endometrioma is present), a fixed uterus (if adhesions are present) and endometriotic nodules (palpable in the pouch of Douglas or on the uterosacral ligaments). An enlarged uterus may be found with fibroids. Abnormal discharge and tenderness may be seen with PID.

Important points in the expression of dymenorrhoea lead the clinician to suspect serious pathology and include an abnormal cervix on examination, persistent PCB or IMB, which may indicate endometrial or cervical pathology, or a pelvic mass that is not obviously the uterus.

Investigations

1.High vaginal and endocervical swabs.

2. TVUSS (Trans vaginal ultrasound scan) may be useful to detect endometriomas or appearances suggestive of adenomyosis (enlarged uterus with heterogeneous texture) or to image an enlarged uterus.

3.Diagnostic laparoscopy: performed to investigate secondary dysmenorrhoea:

A.when the history is suggestive of endometriosis.

B. when swabs and ultrasound scan are normal, yet symptoms persist.

C.when the patient wants a definite diagnosis or wants reassurance that their pelvis is normal.

Discussion about laparoscopy should include risks and the possibility that this investigation may show no obvious causes for their symptoms.

D.If features in the history suggest cervical stenosis, ultrasound-guided hysteroscopy can be used to investigate further. However, this condition is an infrequent cause of dysmenorrhoea, and this investigation should not be routine.

Laparoscopy for primary dysmenorrhoea should not usually be performed.

Management

1.Non-steroidal anti-inflammatory drugs (NSAIDs): effective in a large proportion of women. Some examples are naproxen, ibuprofen and mefenamic acid.

2. Hormonal contraceptives: COCP is widely used but, surprisingly, a recent review of randomized controlled trials provides little evidence supporting this treatment as being effective for primary dysmenorrhoea. Progestogens, either oral (desogestrol) or parenteral (medroxyprogesterone, etonogestrel) may be useful to cause anovulation and amenorrhoea.

3.LNG-IUS: there is evidence that this is beneficial for dysmenorrhoea and indeed can be an effective treatment for underlying causes, such as endometriosis and adenomyosis. It is often used as a first-line treatment before laparoscopy.

4.Lifestyle changes: there is some evidence to suggest that a low fat, vegetarian diet may improve dysmenorrhoea. There are suggestions that exercise may improve symptoms by improving blood flow to the pelvis.

5.Heat: although this may seem a rather old-fashioned method for helping dysmenorrhoea, there is strong evidence to prove its benefit. It appears to be as effective as NSAIDs.

6.GnRH analogues: this is not a first-line treatment nor an option for prolonged management due to the resulting hypo-oestrogenic state. These are best used to manage symptoms if awaiting hysterectomy or as a form of assessment as to the benefits of hysterectomy. If the pain does not settle with the GnRH analogue, it is unlikely to be resolved by hysterectomy.

7.Surgery: signs or symptoms of pathology such as endometriosis may warrant surgical laparoscopy to perform adhesiolysis or treatment of endometriosis/drainage of endometriomas.

Chronic Pelvic Pain

Chronic pelvic pain (CPP) is defined as non-cyclical pain lasting for more than 6 months, localized to the anatomical pelvis and anterior abdominal wall, at or below the umbilicus, or to lumbo-sacral back and buttocks of sufficient severity to cause functional disability or lead to medical intervention. CPP is a broad term with varied presentations and has a significant impact on quality of life. It may present as dysmenorrhoea, dyspareunia, vulvodynia, non-specific pelvic pain, musculoskeletal pain, intestinal cramps or dysuria. CPP is associated with long-standing mental health problems, with reported increased rates of anxiety, depression, somatic disorders, disturbed concentration and insomnia.

Classification of the causes of chronic pelvic pain

1. Inflammatory, infective: chronic salpingitis.
2. Inflammatory, non-infective: endometriosis, vulvodynia with dermatosis.
3. Mechanical: uterine retroversion, adhesions.
4. Functional: pelvic congestion, irritable bowel syndrome.
5. Neuropathic: post-surgical, dysaesthetic vulvodynia, vulval vestibulodynia (‘vestibulitis’).
6. Musculoskeletal: pelvic floor myalgia.

Management

Medical therapy:

Medroxyprogesterone acetate (MPA) has been used extensively but is only effective after 4 months' treatment. MPA plus psychotherapy was effective in terms of pain. Benefit was not sustained following treatment.

Venography scores, symptom and examination scores, mood and sexual function were improved to a greater extent 1 year after treatment with the GnRH analogue goserelin compared with progestogen.

No improvement in pain scores was seen in women taking the selective serotonin reuptake inhibitor sertraline compared with placebo. Study showed a small improvement in the sertraline arm, while other study showed a large fall in the sertraline arm.

Medical options such as antidepressant and anticonvulsant drugs are well tolerated and could therefore be started by a gynaecologist or primary care physician.

Surgical therapy

Previously, **laparoscopic uterosacral nerve ablation (LUNA)** was commonly performed for the treatment of CPP. However, a large and well-designed randomized controlled trial showed that this technique did not lead to any improvement in pain, dysmenorrhoea, dyspareunia or quality of life.

Adhesiolysis:

Intraperitoneal adhesions can form *de novo* or following a surgical procedure. A recent systematic review examining the efficacy of adhesiolysis for the treatment of chronic pain showed that the benefit of intervention varied from 16 to 88%, with the majority of studies reporting pain relief in more than 50% of cases.

Premenstrual syndrome

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% symptoms are severe, causing disruption to everyday life.

Aetiology

PMS is not due to a single factor but its basis is multifactorial, with genetic, environmental and underlying psychological influences being important. This is of course true for all mood disorders but in PMS the ovarian cycle comes into play, with ovulation being almost certainly the key factor.Cyclical ovarian activity and the effects of oestradiol and progesterone on certain neurotransmitters, including serotonin, appear to play a role.

Ovulation and progesterone

The principal cause of PMS is uncertain. There is evidence to suggest that the cyclical endogenous progesterone produced in the luteal phase of the cycle after ovulation is the key provoking factor. Women with PMS appear to be unusually ‘sensitive’ to normal levels of progesterone; differences in progesterone levels have not been demonstrated between women with and without PMS. It has been hypothesized that the mechanism of this increased sensitivity is related to a neurochemical factor, and most evidence points to a dysregulation of serotonin metabolism.

History and examination

The patient is likely to complain of some or all of the following: bloating cyclical weight gain, mastalgia, abdominal cramps, fatigue, headache, depression, irritability. The cyclical nature of PMS is the cornerstone of the diagnosis.

Diagnosis

Here are no objective tests (physical, blood, biochemical, endocrine or imaging) to assist the diagnosis of PMS and so the use of prospectively completed symptom charts is essential. Retrospective reporting of symptoms is inaccurate, but significant numbers of women who present to a PMS clinic have separate under lying problems such as perimenopause, thyroid disorder, migraine, chronic fatigue syndrome, irritable bowel syndrome, seizures, anaemia, endometriosis, and menstrual disorders as well as psychiatric disorders such as depression, bipolar illness, panic disorder, personality disorder and anxiety disorders.

Gonadotrophin-releasing hormone agonists in diagnosis

The use of the so-called gonadotrophin-releasing hormone (GnRH) analogue test may be of benefit in clarifying the diagnosis where there is a mixed or uncertain picture.

It is used extensively by gynaecologists (off-licence and with due discussion with the patient) for the purposes of removing the ovarian cycle to determine which of an individual patient's symptoms are clearly related to the menstrual cycle and which (i.e. those that persist despite suppression of the cycle) are not. It is also a valuable way of demonstrating whether symptoms or medical problems such as premenstrual migraine, asthma and epilepsy are truly related to the cycle or are independent. If her PMS (or other significant premenstrual symptom) is severe and is eradicated by GnRH, it is likely (though not guaranteed) that she would also benefit from removal of ovaries when the hysterectomy is being undertaken.

Non-medical therapies

Claims, mainly unsubstantiated, have been made for the supplementation of calcium, vitamin E, magnesium, dietary change, vitamin B_6, evening primrose oil, 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 very limited evidence for the effects of calcium and magnesium. There is, however, strong evidence to support the use of cognitive behavioural therapy (CBT) for treating PMS and RCOG guidance recommends its use as routine early management.

Medical therapies

Treatment should be achievable either by suppressing ovulation or by enhancing serotonin levels in the central nervous system to ‘reduce the sensitivity to progesterone’.

The first of these is achieved pharmacologically or by surgical intervention, the former by elevating serotonin levels using drugs such as the SSRIs. Although theoretical, treatment based on this option is very useful practically.

Psychotropics: selective serotonin reuptake inhibitors

Elevating serotonin levels can readily be achieved by the use of SSRIs. Such treatment is clearly beneficial, although none of these drugs has a pharmaceutical licence for the management of either PMS in the UK. Fluoxetine 20 mg daily is usually sufficient to improve symptoms in most women.

Ovarian cycle suppression

Suppression of the ovarian cycle can be achieved with oestrogen, danazol, GnRH agonist analogues or bilateral oophorectomy.

Surgery: bilateral oophorectomy

Bilateral oophorectomy with hysterectomy is almost always too invasive for the majority of patients with PMS and, even though it is the only certain cure, its use is rarely justifiable other than as a last-resort measure for the most severely affected women: those who have failed to respond to other measures. When removal of the ovaries and uterus is considered appropriate, oestrogen replacement can follow without the need to consider endometrial protection using progestogen, which inevitably would restimulate the PMS symptoms.

Danazol

When danazol is given orally, even at doses of 200 mg, it is particularly effective for most symptoms of PMS. Its use has been very much limited by anxieties related to the risk of masculinization. Attempts to reduce side effects by prescribing it for use in the luteal phase only (presumably through its direct effect on target tissue rather than ovulation suppression) have shown it be ineffective for nearly all symptoms of PMS with the exception of cyclical mastalgia.

GnRH agonist analogues (and add-back therapy)

GnRH agonist analogues are extremely effective. These are best administered as injected depot preparations (goserelin or leuprorelin) as, unlike nasal preparations, compliance is virtually guaranteed. These drugs are agonist analogues and so omission of nasal doses (or indeed late administration of depot preparations) can result in incomplete suppression and restimulation of cycles. Treatment without add-back therapy will almost always be successful for PMS, but confusion can arise owing to the development of new symptoms usually associated with the menopause. With continuous add-back therapy (particularly tibolone), the analogues remain equally effective for PMS while eliminating menopause symptoms, though not in all patients, hence the resort to surgery in some.

Oestrogen

Suppression of ovulation using oestrogen has significant advantages over oophorectomy and GnRH analogues in that these latter approaches still require the addition of oestrogen to prevent the hypo-oestrogenic effects resulting from the primary treatment approach. Its disadvantage is that in those women who retain their uterus and endometrium, it is necessary to protect from endometrial cancer using potentially ‘PMS-inducing’ progestogens.

THANK YOU