Dysmenorrhoea

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Abstract

Dysmenorrhoea is a medical condition characterized by severe uterine pain during menstruation manifesting as cyclical lower abdominal pain. It is commonly classified into primary dysmenorrhoea in the absence of co-existent pathology and secondary dysmenorrhoea when there is an identifiable pathological condition. About 40–70% of women of reproductive age suffer with dysmenorrhoea along with its associated psychological, physical, behavioural and social distress. It is a leading cause of absenteeism. The exact pathophysiological processes are not fully understood but it probably reflects increased myometrial activity induced by an excessive production of prostaglandin causing ischaemia (uterine 'angina'). History is critical in establishing the diagnosis of dysmenorrhoea and also in differentiating between primary and secondary dysmenorrhoea. Mainstay treatment is generally supportive providing symptomatic relief and more directive surgical treatment is reserved for specific secondary causes of dysmenorrhoea or for refractory cases. Therefore, patients with primary dysmenorrhoea may simply need reassurance and simple analgesics, while those with secondary dysmenorrhoea require investigation and treatment of the underlying organic problem. We present an overview of managing this condition.

Keywords dysmenorrhoea; menstrual disorders; primary; secondary

Background

Dysmenorrhoea is a medical condition characterized by severe uterine pain during menstruation manifesting as cyclical lower abdominal or pelvic pain, which may also radiate to the back and thighs. The term dysmenorrhoea is derived from the Greek words 'dys' meaning difficult, painful or abnormal, 'meno' meaning month and 'rrhea' meaning flow. It is commonly divided into primary dysmenorrhoea, where there is no co-existent pathology, and secondary dysmenorrhoea where there is an identifiable pathological condition known to contribute to painful menstruation. Symptoms of primary dysmenorrhoea begin a few hours before the start of menstruation and are often relieved during the first few days of bleeding. The initial onset of primary dysmenorrhoea is usually shortly after menarche (6–12 months), when ovulatory cycles are established. Secondary dysmenorrhoea can also occur at any time after menarche but is most commonly observed in women in their third and fourth decade of life in association with an existing condition.

Since dysmenorrhoea is a symptom that could be perceived differently by different women, it is difficult to establish its true incidence. However, the reported prevalence is age-related, increasing from around 40% in girls aged 12 years, to 70% in girls at 17 years of age. An epidemiological study showed the prevalence of dysmenorrhoea among adolescent females ranges from 60 to 93%, but decreases with advancing age. One systematic review of community and hospital surveys estimated the overall prevalence to be 45–95% whilst a second age-based systematic review suggested 25–50% of adult women and as many as 75% of adolescents experience pain with menstruation. Pain is significant in 5–20% who report severe dysmenorrhoea or pain that prevents them from participating in their usual activities. One community survey of adolescents aged 12–18 years showed dysmenorrhoea as one of the most common biomedical problems causing health risk behaviours and psychosocial problems, such as cigarette or alcohol use, dieting, infrequent/never seat belt use and feeling depressed. It is one of the most commonly reported reasons for absenteeism from school or work with some studies suggesting that 10–45% of women miss or reduce time at work, school or other activities. In the United States, the annual economic loss has been estimated at 600 million work hours and 2 billion dollars. These reports suggest a significant socio-economic and psychosocial impact of dysmenorrhoea on female health during the reproductive years.

Pathophysiology

The most important physiological event reported with dysmenorrhoea is increased myometrial activity with accompanying uterine ischaemia (uterine 'angina'), which stimulates the type C afferent pain neurones. Doppler flow studies have supported this hypothesis by showing higher uterine and arcuate artery resistance on the first day of menses in women with primary dysmenorrhoea than in controls. While the pathophysiological mechanisms that cause this are not entirely understood, it is thought that this myometrial activity is modulated and augmented by prostaglandin synthesis. This may reflect the increase in circulating levels of prostaglandin F2α, which is a potent myometrial stimulant and vasoconstrictor. It is also found that the levels of prostaglandins in endometrial fluid in the early follicular phase of menstrual cycle are less than levels in late luteal phase of menstrual cycle. (The hypothesis that prostaglandins released from the endometrium at the time of menses contribute to dysmenorrhoea is supported by the observation that endometrial concentrations of PGE2 and PGF2 correlate with the severity of dysmenorrhoea, that cyclo-oxygenase inhibitors decrease menstrual fluid prostaglandin levels and decrease pain, and that the clinical manifestations of dysmenorrhoea are similar to those with prostaglandin induced labour or medical management of miscarriage. In contrast to the uterine contractions in normal menstruation, contractions in women with primary dysmenorrhoea often begin with elevate basal tone, reach higher active pressures (frequently more than 150 mmHg), and are nonrhythmic or in coordinate.) In addition to stimulating uterine contractions, PGF2 and PGE2 can cause contraction of bronchial, bowel, and vascular smooth muscle resulting in bronchoconstriction, nausea, vomiting, diarrhoea and hypertension. Diarrhoea and nausea are commonly associated with primary dysmenorrhoea.

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The other reported factors in the causation of increased myometrial activity are increased levels of circulating vasopressin and leukotrienes. This effect is mediated via myometrial oxytocin and vasopressin V1a receptors. The role of vasopressin may relate to prostaglandin synthesis and release leukotrienes, as well as prostaglandin E2, may increase the sensitivity of pain fibres in the uterus. Increased endometrial expression of leukotrienes has been demonstrated in women with primary dysmenorrhoea resistant to prostaglandin antagonists. Psychosocial factors may play a role in the perception and the severity of the pain.

Clinical features

History is critical in establishing the diagnosis of dysmenorrhoea and also in differentiating between primary and secondary dysmenorrhoea. A thorough menstrual history including the age at menarche and at the onset of pain, cycle length and regularity, duration and amount of menstrual flow, and an assessment of the onset, duration, type, cyclicity and severity of pain and associated symptoms such as nausea, vomiting, bloating, diarrhoea and fatigue is essential in the diagnosis of dysmenorrhoea. It is then important to exclude the secondary causes of dysmenorrhoea — such as pelvic infection, sexually transmitted disease, endometriosis, subfertility, abdomino-pelvic surgery and difficult childbirth — which could present with additional symptoms of deep dyspareunia, intermenstrual, postcoital bleeding, and subfertility. One should specifically determine the factors that exacerbate or ameliorate the symptoms, as secondary dysmenorrhoea is more commonly refractory to simple treatments such as non-steroidal anti-inflammatory drugs (NSAIDs) and the combined oral contraceptive (COCP). Social and family history should be considered as they may have direct or indirect bearing on the causation and help in optimizing the treatment (Table 1).

Primary dysmenorrhoea

Primary dysmenorrhoea almost invariably occurs in young women with ovulatory cycles and usually appears within a year after menarche. The pain classically begins just before or with the onset of menstruation lasting through the first 1–2 days and is typically described as spasmodic lower abdominal or pelvic pain superimposed over a constant dull aching pain, which may radiate to the back and along the thighs. Menstrual bleeding is usually normal. Associated symptoms — such as malaise and, fatigue (85%), irritability (72%), dizziness (28%), headache (45%), lower backache (60%), diarrhoea (60%), and nausea and vomiting (89%) — may be present.

The diagnosis of primary dysmenorrhoea is made primarily based on history and then by exclusion of existing pathology. Pelvic or rectal examination where appropriate may be helpful to exclude common problems of young age such as pelvic inflammatory disease and endometriosis, and provide reassurance through a negative assessment. Psychosexual factors may be contributing to dysmenorrhoea and should be considered where medical treatment is unsuccessful.

Secondary dysmenorrhoea

Secondary dysmenorrhoea mainly occurs in the third and fourth decade of reproductive life. The pain is different to that of primary dysmenorrhoea and is described as pelvic heaviness and back pain increasing progressively throughout the late luteal phase and peaking with the onset of menstruation. This pattern of pain is often referred to as 'congestive pain' in contrast to the 'spasmodic' pain seen with primary dysmenorrhoea. The pain associated with secondary dysmenorrhoea is more likely to coincide or be temporally associated to other gynaecological symptoms, such as cycle irregularity, heavy periods, dyspareunia, vaginal discharge, intermenstrual bleeding and postcoital bleeding.

Major causes of secondary dysmenorrhoea

Gynaecologic disorders:

- Endometriosis
- Adenomyosis
- Pelvic inflammatory disease
- Fibroids
- Endometrial polyps
- Ovarian cysts
- Intrauterine contraceptive device
- Intrauterine adhesions
- Congenital obstructive mullerian malformations
- Pelvic congestion syndrome
- Cervical stenosis

Differential characteristics of primary and secondary dysmenorrhoea

	Primary	Secondary
Age (years)	16–25	30–45
Onset of pain	Just prior to menstruation (spasmodic)	Pain often progresses through late luteal (congestive)
Pathophysiology	Excess prostaglandins, vasopressin, leukotrienes	Underlying disorder
Symptoms	Usually self-limiting, lasts for first 1–3 days menstruation	Associated with other features related to underlying disease
	Responds to COCP and NSAIDs	Resistant to COCP and NSAIDs
	Periods normal or light	Periods often heavy
Signs	Unremarkable	Dependent on cause but may include a tender, enlarged, fixed, retroverted uterus with adnexal tenderness and a mass

COCP, combined oral contraceptive; NSAIDs, non-steroidal anti-inflammatory drugs.

Table 1