Dysmenorrhoea

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Abstract

Dysmenorrhoea is a medical condition characterised 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 very common gynaecological condition affecting anywhere from 41 to 97% of women with one in five cases being severe. Dysmenorrhoea is a medical condition characterised 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. In a large Swedish study 50% of women were absent from work due to dysmenorrhoea and 8% were absent for at least half a day every menstruation. Risk factors are early menarche, nulliparity, family history and cigarette smoking.

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 neurons.

In a normal menstrual cycle, at the end of the luteal phase as the corpus luteum regresses after non-fertilisation of an ovum, there is a decline in progesterone levels. It is this withdrawal of progesterone that leads to the shedding of endometrium and, during its destruction, an increase in inflammatory cytokines, vascular endothelial growth factors and matrix metalloproteinases (MMP's). This leads to a degradation and loss of integrity of blood vessels and destruction of endometrial interstitial matrix and hence the bleeding of menstruation. Uterine contraction and vasoconstriction is caused by the disintegrating endometrial cells releasing $PGF_{2\alpha}$ which is a myometrial stimulant and vasoconstrictor. The uterine contractions and ischaemia that results from decreased blood flow results in uterine pain. Only at the turn of the last century, women were considered neurotic having 'menstrual pain' until it became clear that dysmenorrhoea was associated with uterine hypercontractility leading to uterine ischaemia.

Primary dysmenorrhoea

In women with primary dysmenorrhoea there are increased levels of inflammatory markers such as vasopressin,

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prostaglandins PGE_2 and $PGF_{2\alpha}$ and leukotrienes in endometrial fluid. PGF2α is a potent myometrial stimulant and vasoconstrictor. Leukotrienes increase myometrial stimulation and vasoconstriction as well as increasing the sensitivity of pain fibres. Vasopressin stimulates uterine activity, decreased uterine blood flow and, in vitro, constricts uterine arteries. The primary stimulus for these increased levels is unknown but it is thought that this myometrial activity is modulated and augmented by prostaglandin synthesis. 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 (ref).

Compared with controls, women with primary dysmenorrhoea have increased uterine pressures from increased uterine muscle activity and frequency of contractions (frequently more than 150 mmHg). Doppler flow studies have supported this by showing higher uterine and arcuate artery resistance on the first day of menses in women with primary dysmenorrhoea than in controls. Pain fibres are also stimulated from ischaemia caused by vasoconstriction and anaerobic metabolites produced by an ischaemic endometrium which stimulate type C pain neurons.

Added to this is the possible effect of psychological and behavioural factors which can act centrally and contribute to pain perception. There is an increased prevalence of dysmenorrhoea in women with a history of sexual abuse but there is little research into this compared to other chronic pelvic pain syndromes.

Secondary dysmenorrhoea

Secondary dysmenorrhoea is caused by an underlying pelvic pathology. Endometriosis is the most common cause. However, there is no correlation between the severity of the disease and extent of pain. PID causes both increased inflammatory mediators and scar tissue. Adenomyosis causes tonic contractions through endometrial gland infiltration and polyps, submucous fibroids and IUD's cause increased uterine contractions in order to expel them. All of the less common causes of dysmenorrhoea are due to abnormal uterine contractions. See Table 1 for a comparison of Primary and Secondary dysmenorrhoea.

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, inter-menstrual, post-coital 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 optimising 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.

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