Managing the pain of primary and secondary dysmenorrhea

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Dysmenorrhoea, pain during menstruation, affects 40–95 per cent of menstruating women, and has been reported as the most common cause of regular absenteeism among young women. There are two types of dysmenorrhoea: primary and secondary. Primary dysmenorrhoea is a painful menstruation with no detectable organic disease and is more common in adolescent women. Secondary dysmenorrhoea is painful menstruation that is frequently associated with a pelvic pathology. The symptoms that present with dysmenorrhoea, the management and treatment options that are available, and the implications for nursing practice are discussed.

Dysmenorrhoea has been identified as affecting between 40 and 95 per cent of menstruating women (Vance et al, 1996).

Andersch and Milsom (1982) studied the prevalence of dysmenorrhoea in a random sample of 19-year-old women from an urban Swedish population. They found that 51 per cent had been absent from work or school as a result of dysmenorrhoea, and eight per cent were absent every menstruation. Extrapolation of these figures illustrates the economic consequences of this disorder on a national level (Andersch and Milsom, 1982).

Hewison and van den Akker (1996) maintain that ‘given its personal and economic effects, and the high percentage of nurses who are female, dysmenorrhoea is likely to have a significant effect on the nursing profession’. Despite considerable distress, many women do not seek medical help so many cases are undocumented (Gould, 1998). This means that it is difficult to know the precise incidence of dysmenorrhoea.

For nurses working in women’s health care, it is essential to be able to explain the events of the normal menstrual cycle in lay terms, including being able to provide a simple account of the conditions that give rise to pain and bleeding (Gould, 1998).

Normal physiology

A woman has two ovaries. These lie within the pelvis, and are suspended behind the uterus and the fallopian tubes. The adult ovary is about the size of a walnut.

The uterus is shaped like an inverted pear and is situated between the urinary bladder and the rectum. Before a first pregnancy the adult uterus is approximately 7.5cm long, 5cm wide, and 2.5cm thick.

The fallopian tubes, also called oviducts, extend laterally from the uterus and transport the ova from the ovaries to the uterus. They are about 10cm long and are positioned between the folds of the broad ligaments of the uterus (Tortora and Anagnostakos, 1990). The infundibulum, which is the funnel shaped open distal end of each tube, lies close to the ovary and is surrounded by a fringe of finger-like projections called fimbriae.

The menstrual cycle is about 28 days long, varying by up to two days either side of this figure. It involves interaction between the hypothalamus, the pituitary gland, the ovaries and the hormones they produce.

The follicular phase

In the follicular phase of the menstrual cycle, which occurs in the ovary, the hypothalamus region of the brain sends gonadotrophin-releasing hormone to the pituitary gland, triggering the gland to release low levels of follicular-stimulating hormone. Follicular-stimulating hormones are soluble glycoproteins and are produced in the anterior lobe of the pituitary gland. Production is increased in the first half of the menstrual cycle and diminishes as oestrogen levels increase. During this time one ovarian follicle will normally become dominant.

During the follicular phase of egg growth the increasing amounts of oestrogen cause the endometrium (the lining of the womb) to start thickening in preparation for a fertilised egg (Fig 1, p42).

To make oestrogen the ovarian follicles require a small amount of luteinising hormone, which is another soluble glycoprotein activated in the pituitary gland by gonadotrophin-releasing hormones.

Luteinising hormone causes the ovary cells directly surrounding the ovarian follicle to manufacture testosterone, which is then transported to the cells on the inside of the follicle. An enzyme converts the testosterone into oestrogen and, as the follicle grows in size, stimulated by follicle-stimulating hormone, the amount of oestrogen increases.

The luteal phase

Following ovulation, when the egg is released from the follicle, the cells from the burst follicle collapse to form a cyst called the corpus luteum. The corpus luteum produces progesterone causing the thickening endometrium to secrete nutrients in preparation for a fertilised egg.

If fertilisation does not occur the endometrium continues to thicken causing the oestrogen levels to drop substantially. Progesterone levels (which prepare the uterus and breasts for pregnancy) peak during days 21–25. If fertilisation does not take place after 14 days, the
BOX 1. PROSTAGLANDIN CHARACTERISTICS

- Prostaglandins are membrane-associated biologically active lipids, are secreted into the blood in minute quantities and are potent in their action.

- The chemical composition of prostaglandins is that of 20-carbon fatty acids containing five carbon atoms in a cyclopentane ring.

- Prostaglandins are classified into several groups designated by letters A through to I, documented as PGA through to PGI respectively (Tortora and Anagnostakos, 1990). Prostaglandins belonging to the E series (PGE) appear to be associated with dysmenorrhoea (Gould, 1994).

Other symptoms include nausea, vomiting, headache, diarrhoea, dizziness, and in severe cases syncope and collapse. And symptoms often become less severe or disappear after the woman has experienced childbirth for the first time. They also often become less severe with age (Vance et al, 1996).

The association between ovulation and pain has helped to establish the underlying cause of primary dysmenorrhoea. Analysis of women’s menstrual fluid shows that when they have ovulated, high levels of prostaglandins are present (Gould, 1994) (see Box 1).

The pain associated with primary dysmenorrhoea results from uterine contractions that are probably associated with uterine muscle ischaemia and prostaglandins produced by the uterus.

Prostaglandins stimulate uterine contractions but are unable to do so in the presence of high levels of progesterone. Because progesterone levels are high in the latter half of the menstrual cycle, they inhibit the prostaglandins from producing uterine contractions.

If pregnancy does not occur, progesterone levels drop rapidly and prostaglandin production increases. This causes the uterus to contract and slough off its lining, which may result in dysmenorrhoea (Tortora and Anagnostakos, 1990).

Prostaglandin levels vary between individuals and may not even be the same during succeeding episodes of menstruation (Gould, 1994).

Gastrointestinal upsets occur in patients with primary dysmenorrhoea due to the endometrial prostaglandin gaining access to the systemic circulation (Gould, 1994).

This affects the smooth muscle contractions of the gastrointestinal tract contributing to the gastrointestinal symptoms (Shaver et al, 1987).

There are suggestions that heavy bleeding may also be related to increased prostaglandin levels in endometrial tissue at menstruation, one interesting point being that prostaglandin-inhibiting drugs have been found to reduce menorrhagia. It is not known at present why prostaglandin levels are not elevated in all women with dysmenorrhoea (Shaver et al, 1987).

Secondary dysmenorrhoea

In contrast, when a physical abnormality is detected the pain is considered an effect of this primary cause and the dysmenorrhoea is described as being ‘secondary’ (Peck, 1988).

The causes of secondary dysmenorrhoea include endometriosis, pelvic inflammatory disease, inter-uterine devices, uterine tumours, and ovarian cysts.

The clinical presentation of secondary dysmenorrhoea includes a history of pain that is not limited to the menstrual cycle, and the onset of which is more than two years after menarche. The pain may be acyclic or chronic in nature. There may also be a history of dyspareunia (pain with intercourse) (Wolf and Schumann, 1999).

Diagnostic testing for secondary dysmenorrhoea may include ultrasound, hysterosalpingogram (an X-ray examination of the uterus and uterine tubes following the injection of a radio opaque dye) (Kasner and Tindall, 1985), and a laparoscopy, which allows viewing of the abdominal cavity by the passing of an endoscope through the abdominal wall (Kasner and Tindall, 1985).

Direct visualisation may be necessary to confirm the aetiology of the pain and the diagnosis of the disorder (Wolf and Schumann, 1999).

Further causes of secondary dysmenorrhoea have been identified. These include the following: chronic salpingitis, cervical stenosis, sub-mucosal fibroid tumours, uterine myomas, uterine polyps and adenomyosis (Wolf and Schumann, 1999).
Management/treatment

The treatment of secondary dysmenorrhoea is aimed at correction of the primary cause (Tortora and Anagnostakos, 1990). As has already been described, the primary cause of secondary dysmenorrhoea can vary and so its treatment will vary accordingly.

The current approach to the therapy of primary dysmenorrhoea is to inhibit prostaglandin synthesis or to suppress ovulation, which in turn inhibits prostaglandin synthesis (Johnson and Johnson, 1997). This is done through one of a number of different of drugs.

Non-steroidal anti-inflammatory drugs

Prostaglandin synthetase inhibitors such as aspirin, mefenamic acid, and ibuprofen are effective in relieving pain in 80 to 90 per cent of patients (Johnson and Johnson, 1997). Examples of commonly used oral non-steroidal anti-inflammatory drugs (NSAIDs) include aspirin, 600mg six-hourly; ibuprofen, 400–600mg every four to six hours; and mefenamic acid, 500mg eight-hourly (Wolf and Schumann, 1999; Johnson and Johnson, 1997).

Side-effects of NSAIDs include nausea, vomiting, headache, dyspepsia and, less commonly, gastrointestinal bleeding and nephrotoxicity. These drugs are contraindicated in patients with peptic ulcer disease, clotting disorders, aspirin-induced asthma and renal disease. Treatment is started at the onset of menstruation and continued for the duration of the dysmenorrhoea.

Oral contraceptive pill

This should be considered if NSAIDs fail (Wolf and Schumann, 1999). Proctor et al (2004b) maintain that research as early as 1937 showed that dysmenorrhoea responds favourably to ovulation inhibition and that synthetic hormones could be used to treat it. The hormones act by suppressing ovulation and reducing the thickness of the endometrial lining of the uterus. This reduces dysmenorrhoea because the volume of menstrual fluid is reduced as the prostaglandins are produced (Proctor et al, 2004b). Oral contraceptive pills are an effective treatment in 90 per cent of patients with primary dysmenorrhoea. Disadvantages include the need to take them continuously and that they are contraindicated in some women (Johnson and Johnson, 1997).

Oral contraceptives reach maximum effectiveness after several menstrual cycles, the contraceptive of choice being a 30–35µg combined oestrogen–progesterone pill (Wolf and Schumann, 1999).

A ‘tri-cycle regimen’, taking the pill continuously for three months, followed by a week’s break, is most efficacious for the symptoms of endometriosis (Rosevear, 2002). In women who do not require contraception, oral contraceptives are given for six to 12 months (Wolf and Schumann, 1999).

Glyceryl trinitrate patches

Combined oral contraceptives and NSAIDs are not effective in 10 to 20 per cent of patients. Transdermal glyceryl trinitrate, which relaxes uterine contractions, can be used to treat dysmenorrhoea. None of the contraindications of NSAIDs or of the combined oral contraceptive pill apply to transdermal glyceryl trinitrate. The advantages of glyceryl trinitrate are that it has a short half-life and disappears rapidly from the circulation, and that the patches can be applied and removed as required giving patients control over their symptoms (Pitroff et al, 1996).

Transcutaneous electrical nerve stimulation

TENS has been demonstrated as being effective for pain relief in a variety of conditions including dysmenorrhoea. Electrodes are placed on the skin and a pulsating electric
current is passed through at varying rates and intensities. It alters the body’s ability to receive or perceive pain signals (Proctor et al, 2004a).

**Acupuncture**
Acupuncture gives relief from pain and has been found to be effective for the treatment of dysmenorrhoea. It involves puncturing the skin with metal needles, which are manipulated by hand or act as electrodes for electrical stimulation. Acupuncture has long been indicated in traditional Chinese medicine for gynaecological problems such as amenorrhoea and dysmenorrhoea (Proctor et al, 2004a).

**Surgical intervention**
Surgical intervention is appropriate in some cases, usually as a last resort, for the treatment of secondary dysmenorrhoea. For example, pain that is due to endometriosis and adenomyosis will be treated by performing a hysterectomy once childbearing is complete (Rosevear, 2002). In most cases, hysterectomy is seen as a success in terms of relieving women of their presenting symptoms (Walgrove, 2001).

**Self-help measures**
A number of self-help measures are available for women with dysmenorrhoea.

**Topical heat**
There is a long history of the use of heat pads and hot water bottles for the treatment of menstrual pain (Cassidy, 2001). A study by Akin et al (2001) has demonstrated that continuous low-level topical heat was as effective as oral ibuprofen for the treatment of dysmenorrhoea. For example, pain that is due to endometriosis and adenomyosis will be treated by performing a hysterectomy once childbearing is complete (Rosevear, 2002). In most cases, hysterectomy is seen as a success in terms of relieving women of their presenting symptoms (Walgrove, 2001).

**Diet**
As well as creating a beneficial feeling of well-being, a good diet reduces the chances of constipation. A constipated bowel increases the symptoms of dysmenorrhoea by pressing against the uterus when it swells before menstruation. Standard dietary advice applies: increase the proportion of wholemeal foods, vegetables, salads, fruit and water, and reduce refined carbohydrates, which have a constipating effect (Peck, 1988).

**Exercise**
Exercise is widely accepted as a means of moderating stress and stress-related symptoms because it causes the release of endorphins by the brain. The mood-improving effect of endorphins may indirectly contribute to the lessening of the symptoms of dysmenorrhoea. Endorphins also act directly by raising the pain threshold. (Locke and Warren, 1999).

**Implications for nursing practice**
Nurses are often asked for advice about dysmenorrhoea and are in an ideal position in their role as health educators and health promoters to offer suggestions for self-help (Gould, 1994). Patients should be informed that dysmenorrhoea is a treatable condition and that the prognosis for primary dysmenorrhoea is excellent (Wolf and Schumann, 1999). It is vital, therefore, that nurses understand how the menstrual cycle works and are familiar with the anatomy and physiology of the reproductive system and its associated disorders. This will enable them to give patients a better understanding of their condition.

Patients should be advised to keep a diary of their symptoms and to monitor their pain and bleeding over a few months. This information will be useful when discussing symptoms with the practice nurse or GP. If symptoms deteriorate or an uncharacteristic history presents, secondary dysmenorrhoea should be suspected and treated appropriately (Wolf and Schumann, 1999).

The benefits of the non-pharmacological self-help measures discussed should be presented to the patient.

Other useful measures that should be discussed are the use of a TENS machine or acupuncture to alleviate symptoms. Pharmacological preparations that are available, including their side-effects, should be outlined and women should be encouraged to take any analgesia when discomfort is at a minimum to prevent the pain becoming extreme.

When discussing oral medications it should be explained that if one preparation is not successful a different one can be prescribed. Nurses therefore need to be aware of both the non-pharmacological and pharmacological treatments for dysmenorrhoea and be able to provide a balanced rationale for their use.

Information should also be provided on support groups so that women can avoid feelings of isolation. For some women dysmenorrhoea can be severe and incapacitating and offering a sympathetic approach can do much to alleviate suffering (Johnson and Johnson, 1997).

**Conclusion**
Primary dysmenorrhoea is a common gynaecological disorder that has been reported as being one of the greatest single causes of periodic absenteeism among young women (Sundell et al, 1990). It causes psychological and physical stress, which results in restricted physical activity and even inability to work (Kulshreshtha, 1993).

It is essential that health professionals understand the complexities of this condition so that, as health educators, they are able to offer advice and carry out assessments. Accurate diagnosis and appropriate treatment of both primary and secondary dysmenorrhoea can lead to significant improvements in patients’ quality of life (Wolf and Schumann, 1999).

Further research into this condition is necessary so that there is more understanding of why there are such differences in the severity of symptoms among women.

Although dysmenorrhoea is a common gynaecological disorder, its prevalence is greatly underestimated owing to the large number of women who ‘suffer in silence’. Improved health promotion strategies are required so that women do not put up with painful periods. ■