Dysmenorrhea—myths and facts[1]

Gita P. Gidwani, M.D.

Dysmenorrhea has plagued young women since ancient times. The author discusses its treatment in other ages and cultures, as well as recent research that has led to a better understanding and alleviation of this condition. The necessity of a thorough work-up to exclude mechanical obstruction of the genital tract is emphasized, along with the clinical criteria for the work-up. Recently recognized conditions, e.g., endometriosis, are discussed and newer treatments, such as prostaglandin inhibitors and oral contraceptives, are reviewed.

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The term *dysmenorrhea*, derived from the Greek, means difficult menstrual flow.[1] *Primary* dysmenorrhea is painful menstruation in the absence of gross pathological conditions of the pelvic organs, whereas *secondary* dysmenorrhea denotes painful menstrual periods in the presence of gross pathological conditions of the pelvic organs, e.g., endometriosis, salpingitis, congenital anomalies of the Müllerian system, or pathology caused by an intrauterine device.

A recent report from Gothenburg, Sweden, on a random sample of 19-year-old women showed that 72% reported dysmenorrhea.[2] In 15%, dysmenorrhea limited daily activity and was unrelieved by analgesics. Fifty-one percent of the women suffering from dysmenorrhea had been absent from work or school as a result. Despite these large numbers, only one fifth (21%) consulted a physician. Why did these women not report dysmenorrhea to a physician?

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Perhaps most women accept dysmenorrhea as normal or do not believe treatment is available.

Only in the past decade have the relationship of prostaglandins to dysmenorrhea and the availability of nonsteroidal anti-inflammatory agents renewed interest in this condition. This recently defined etiology of dysmenorrhea has elicited an infinite number of proposed remedies.[3]

Historical review

As discussed by Wright,[3] Hippocrates recommended fumigation of external genitalia by vapors of sweet wine, fennel seed and root, and rose oil. Chinese medicine recommended moxibustion, which involves a cone of wormwood on a slice of ginger placed on the abdomen, set aflame, and allowed to burn down to the skin. Oscar Polano, in 1907, claimed relief of dysmenorrhea by applying suction cups to the breasts, and, in 1865, Dr. Battey, in Georgia, performed bilateral oophorectomy of normal ovaries to cure dysmenorrhea. Ovarian radiation and insertion of stem pessaries are other obsolete measures.

Traditionally attitudes toward menstruation usually have been negative. Cultural taboos and religious beliefs, which persist to the present in some cultures, involve isolation and ritual cleansing.

The Victorians treated menstruation as an illness. Supposedly, dysmenorrhea could be prevented or relieved by seclusion and rest with a minimum of needle work and piano playing and absolutely no singing. Working class women used menses as a refuge from daily drudgery.

The changing status of women in the early part of this century and the reaction against their being labelled the "weaker sex" popularized the psychogenic origin of dysmenorrhea. Paulson and Wood,[4] in 1966, showed that all of the psychiatrists and half of the gynecologists questioned about their attitude toward dysmenorrhea supported the psychogenic theory of its origin. The advent of oral contraception in the 1960s helped to alter this psychogenic theory as women taking oral contraceptives experienced relief of dysmenorrhea.

All contemporary lines of investigation assume that the symptoms of dysmenorrhea, namely, nausea, vomiting, pain, headaches, and dizziness are real and organic. They attempt to explain that the consistent finding of hypercontractility is caused by ischemia of the myometrium.

Studies of tracings of uterine contractions in both normal and dysmenorrheic women by the introduction of intrauterine balloons and catheters have been done. In 1976, the microtransducer catheter was used to measure contractions, patient symptoms, and uterine blood flow. Åkerlund et al[5] showed that the dull continuous component of menstrual pain correlated with recordings of high basal tone and short relaxation intervals between contractions. Sharp pains occurred when contractions of high amplitude were associated with decreased blood flow. These tracings helped assess drug effects so that evaluation of different therapies became possible.

Meanwhile, other investigators were determining causes of uterine contractility. Pickles et al[6] used menstrual extracts to stimulate smooth muscle in vitro. He identified a lipid-soluble acid in these menstrual extracts that later was found in endometrial material obtained by curettage. Chromatography revealed that this acid included two active substances, which, by 1963, had been identified as prostaglandin E_2 (PGE_2) and prostaglandin $F_2\alpha$ ($PGF_2\alpha$).[7] Pickles et al[6] then quantified these substances in menstrual extracts and found that menstrual fluid from dysmenorrheic women has about ten times as much $PGF_2\alpha$ as the secretory endometrium, suggesting that $PGF_2\alpha$ is formed shortly before or during menstruation. In addition, the secretory endometrium seemed to have a higher PGF/PGE ratio than proliferative phase samples.

Research into prostaglandins and their derivation from arachidonic acid and the hypothesis that nonsteroidal anti-inflammatory agents (NSAI) are specific inhibitors of PG synthesis all contributed to the suggestion by Pickles[6] in 1972 that NSAIs might be of value in treating dysmenhorrhea. It has only been since 1974 that Schwartz et al[8] and Massey et al,[9] working independently, chose a NSAI to treat uterine pain because these drugs inhibit prostaglandin synthesis.

Both prostaglandins are produced by endometrial cell destruction through the release of intracellular lysosomes. This endometrial cell breakdown releases phospholipids that are converted to arachidonic acid, the immediate precursor of the prostaglandins. Although prostaglandins are produced in the endometrium, the prostaglandin receptor sites are in the myometrium.[10] Pain in dysmenorrhea results from creased basal tone of the uterus; namely, tonus greater than 50 mm Hg, which is recorded during cramps. Most investigators agree that uterine pain is secondary to myometrial ischemia pro-

duced by high intensity contractions superimposed on a high basal uterine tonus.

Clinical features of primary dysmenorrhea

Primary dysmenorrhea usually occurs in ovulatory cycles and appears shortly after menarche (about 6–12 months), when ovulatory cycles are established. It is characterized by sharp colicky pain in the suprapubic region radiating to the back and along the thighs. It begins several hours before or immediately after the onset of menstruation and may be accompanied by nausea, vomiting, diarrhea, palpitations. flushing, dizziness, lower backache, and headaches. Syncope and collapse have been reported by patients, and symptoms usually last from a few hours to one day but may last for two to three days.

The differential diagnosis of primary dysmenorrhea includes all causes of secondary dysmenorrhea, but the character and duration of pain, onset immediately after menarche, and a negative pelvic and rectal examination help confirm the diagnosis. Laparoscopy and examination under anesthesia must be done to rule out endometriosis in patients who have a family history of endometriosis or who do not respond to the following therapy.

Therapy of primary dysmenorrhea

1. Analgesics and simple explanation may alleviate anxiety and help the patient cope with mild symptoms. Narcotics should not be prescribed because of the high risk of abuse.

2. Prostaglandin inhibitors, which inhibit accumulation of prostaglandin, e.g., ibuprofen (Motrin) in doses of 400–800 mg, mefenamic acid (Ponstel) 250–500 mg, or naproxen sodium (Anaprox) in doses of 250–500 mg, are used commonly with relief of symptoms in 75%–80% of patients[3] (*Table*). Prostaglandin inhibitors should be used cautiously in patients with gastrointestinal ulcers and in asthmatic patients. Approximately 20% of asthmatic patients are sensitive to aspirin and will have increased bronchoconstriction. Most will experience the same sensitivity to prostaglandin inhibitors.

Table. Available prostaglandin inhibitors

Aspirin
Phenylbutazone (Butazolidin)
Oxyphenbutazone (Tandearil)
Indomethacin (Indocin)
Ibuprofen (Motrin)
Mefenamic acid (Ponstel)
Naproxen (Naprosyn)
Naproxen sodium (Anaprox)
Tolmetin sodium (Tolectin)
Fenoprofen (Nalfon)
Sulindac (Clinoril)

3. Oral contraceptives in a sexually active adolescent effectively control dysmenorrhea. They prevent thickening of the endometrium, thus eliminating the formation of prostaglandins. An oral contraceptive with 30–50 mg of estrogen is usually effective with minimal side effects. If the patient has symptoms in the presence of oral contraceptives, a small dose of NSAI may help.

4. Other measures, e.g., antispasmodics and beta-blockers with vasodilating properties, have not been helpful. Dilation and curettage have no therapeutic effect unless the patient has endometriosis or cervical stenosis. Term pregnancy is curative for dysmenorrhea, probably because the ensuing hypertrophy of blood vessels reduces the relative vascular insufficiency. However, this cannot be recommended for therapy.

Secondary dysmenorrhea

Although primary dysmenorrhea is the most common cause of menstrual pain, endometriosis, pelvic inflammatory disease, and Müllerian abnormalities can cause cyclical and acyclical pain in adolescents.

Endometriosis, once considered a disease of women in the third decade of life, has been found, with the advent of the laparoscope, in adolescents. In one study of patients with pelvic pain, 47% of the adolescents were found to have endometriosis.[11] Similarly, in a series of 43 consecutive laparoscopies in teenagers, Chatman reported an incidence of 65% of endometriosis.[12] An adolescent with dysmenorrhea who has significant positive pelvic findings or does not respond adequately to antiprostaglandins and/or oral contraceptives should undergo laparoscopy. A coagulation of endometrial implants could be performed at that time with a dilatation and curettage. Endometriosis is more common in adolescents with affected mothers or sisters.

Pelvic inflammatory disease can cause dysmenorrhea. The patient may have positive cultures from the endocervix and an elevated white blood cell count and sedimentation rate. Laparoscopy may assist diagnosis.

Müllerian abnormalities, especially noncanalization of a double system, can cause dysmenorrhea. Rectal examination and ultrasound recordings are often helpful. Abnormalities, particularly ab-

sence of a kidney, should alert the examiner to the presence of Müllerian abnormalities.

Workup

Work-up of the patient with dysmenorrhea includes a careful history of the nature and severity of the pain, menstrual history, familial history of endometriosis and dysmenorrhea, sexual history with history of contraceptive use, including the intrauterine device. It is best to take the history from the adolescent both with and without the parent and to assure confidentiality. The physician should explain the anatomy of the pelvic organs and the necessity of performing tests to rule out abnormality. Most teenagers respond well if explanation and examination are done in a confident and unhurried manner. Also, use of a small virginal speculum helps alleviate anxiety. If no abnormality is detected on pelvic and rectal examination, an antiprostaglandin or an oral contraceptive in a sexually active teenager is prescribed. The patient is seen again in about three months. If pain continues in spite of therapy, or if the findings on pelvic examination are abnormal, ultrasound recordings, cultures from the genitals for pathogens, and erythrocyte sedimentation rate should be obtained. The final diagnosis is usually made by examination under anesthesia; hysterosalpingography, and diagnostic laparoscopy may be performed. A psychological consultation is rarely necessary after a thorough work-up fails to reveal pathology. Biofeedback, relaxation techniques, and self-hypnosis have helped these patients.

The overall approach to dysmenorrhea should include proper diagnosis to distinguish primary from secondary dysmenorrhea, reassurance, and adequate therapy.

The myth that "a woman must suffer" should be expunged, and the fact that an adolescent must not be handicapped by dysmenorrhea should be publicized.

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