Management of Pelvic Pain from Dysmenorrhea or Endometriosis

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Many women suffer from pelvic pain, and a great many visit their family doctor for diagnosis and treatment. Two common causes are primary dysmenorrhea and endometriosis. Primary dysmenorrhea is best treated by prostaglandin inhibition from nonsteroidal anti-inflammatory drugs (NSAIDs) and cyclo-oxygenase-2 (COX-2)-specific inhibitors. Oral contraceptives can be added to improve pain control. Endometriosis can be treated with NSAIDs and COX-2-specific inhibitors as well but can also be treated with hormonal manipulation or surgery. Empirc treatment for endometriosis in selected patients is now accepted, making the disorder easier for family physicians to manage. (J Am Board Fam Pract 2004;17:S43–7.)

Pelvic pain is one of the most common problems affecting women of reproductive age. The pain may vary from mildly irritating to incapacitating. Dysmenorrhea and endometriosis are the two most common causes. Nonsteroidal anti-inflammatory drugs (NSAIDs) and cyclo-oxygenase-2 (COX-2)-specific inhibitors are the mainstays of therapy for both disorders. Hormonal manipulation may also be used in treatment. Surgical and alternative treatments are also discussed.

Definitions
In the broadest sense, pelvic pain is considered any visceral pain presenting below the umbilicus. This article focuses on the two most common causes of chronic pelvic pain: dysmenorrhea and endometriosis. Pain in the bowel and bladder are considered to be outside the pelvis, although the astute clinician also takes into account the importance of these contiguous organs as causes of pain. Acute pelvic pain is defined as recent in onset, whereas chronic pelvic pain is that which has lasted greater than 6 months and occurs not solely with menses.

Prevalence
A number of studies have estimated the prevalence of chronic pelvic pain to be similar to that reported for migraine, low back pain, and asthma.1,2 Dysmenorrhea and endometriosis are the two most common causes of pelvic pain. Primary dysmenorrhea is a very common gynecologic problem in menstruating women. Reported prevalence rates are as high as 90%; 1 in 13 sufferers are incapacitated for 1 to 3 days per month, impacting school and work attendance. Primary dysmenorrhea usually presents during adolescence within 3 years of menses. Most women who suffer from dysmenorrhea do not seek medical care.

Endometriosis is seen in 5 to 10% of women in the general population and is thought to be more common in the mature woman, but it can also occur in adolescents and has been reported in girls as young as 10.5 years of age.1 The peak incidence is between the ages of 25 and 30 years.

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Dysmenorrhea
Affected women experience sharp, intermittent spasms associated with their menstrual cycle. It is usually centered in the suprapubic area but may radiate to the back of the legs or the lower back. Systemic symptoms of nausea, vomiting, diarrhea, fatigue, fever, headache, or lightheadedness are fairly common. The pelvic pain of dysmenorrhea has been demonstrated to be mediated through the
The action of prostaglandin factor 2x and is ischemic in nature; therefore, prostaglandin inhibition nearly always diminishes or resolves the pain. Numerous studies have documented the efficacy of standard NSAIDs and COX-2-specific inhibitors acting through prostaglandin synthetase inhibition to control dysmenorrhea (SORT A*). These medications are started 1 to 2 days before menses and continued for 2 days after menses starts. This first-step treatment is effective in 80% of patients. For those who fail to respond, oral contraceptive pills or medroxyprogesterone can be added to achieve control. These medicines are effective in 90% of patients (SORT C). Figure 1 presents an algorithm for the management of primary dysmenorrhea.

Some alternative treatments for primary dysmenorrhea have been studied and have shown some success. Topical heat at 38.9°C used for 12 hours per day has been found to be as beneficial as ibuprofen. Four small studies of 126 patients

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* Levels of evidence using SORT: (1) treatment of pain caused by primary dysmenorrhea with NSAIDs or COX-2-specific inhibitors = A; (2) treatment of pain caused by primary dysmenorrhea with oral contraceptive pills = B; and (4) treatment of endometriosis empirically = C.
showed transcutaneous electrical nerve stimulation (TENS) to give moderate relief in 40% to 60% of patients. Acupuncture, when studied in 43 patients for 1 year, showed a 91% improvement in symptoms and a 41% decrease in analgesic use. Daily thiamine (100 mg) for 90 days in 556 patients yielded an 87% cure rate up to 2 months after treatment and, in adolescents, daily intake of marine ω-3 fatty acids netted significant improvement. Nitroglycerin patches improved pain symptoms in one uncontrolled trial. These alternative treatments can be used alone or as adjuvants to standard therapy.

If a 3- to 4-month trial of anti-inflammatory, hormonal, or alternative treatments has been ineffective, secondary causes of dysmenorrhea and pelvic pain should be considered (Table 1). In one study of 100 women who had inadequate pain relief with NSAIDs and/or oral contraceptives, almost 80% had endometriosis on laparoscopy. Only after these secondary causes have been ruled out would invasive options such as uterosacral nerve ablation, presacral neurectomy, or nerve block procedures, be considered. A Cochrane review did not find sufficient evidence to recommend nerve interruption procedures for the treatment of pelvic pain caused by dysmenorrhea.

**Endometriosis**

Endometriosis typically presents with the triad of pelvic pain, dyspareunia, and infertility. Any of these 3 issues could motivate a woman to seek care; most often, pain is the compelling reason for the visit. Endometriosis can be investigated and treated by laparoscopy or can be treated empirically. The traditional approach has been to perform laparoscopy to visually and pathologically make the diagnosis, with the advantage that any endometriosis found can be surgically treated at the same time. In one placebo-controlled, double-blind, randomized trial of women with stage I, II, or III endometriosis, 40% had alleviation of pain at 6 months that could be attributed to surgical debridement. Progestin, danazol, or gonadotropin-releasing hormone (GnRH) analogs are generally used postoperatively for greater duration of pain relief (Figure 2). Ling showed empiric treatment to be effective in low-risk patients, including women aged 18 to 45 years with regular menses, no previous diagnosis of endometriosis, no hormonal treatment in the prior 3 months, no evidence of gastrointestinal or urinary disease, normal pelvic ultrasound, normal complete blood count, normal urinalysis, negative gonorrhea and chlamydia culture, negative human chorionic gonadotropin, and failure of NSAIDs and doxycycline to improve pain symptoms (SORT B). Of patients treated empirically, 80% experienced significant improvement, including patients without detectable endometriosis at subsequent laparoscopy. The empiric treatment group must be carefully screened to be certain there is no concomitant disease, such as infection or pregnancy.

Whether identified surgically or empirically, endometriosis is treated with one or more of the following: traditional NSAIDs, COX-2-specific inhibitors (SORT C), oral contraceptive pills (OCPs), GnRH agonists, progestins, or danazol. Traditional NSAIDs or COX-2 inhibitors are used initially at maximal or nearly maximal dosage. There is no evidence to support switching from one NSAID to another to improve response, although the practice is frequent. OCPs are used next if pain relief has not been achieved, and they may be used alone or in combination with NSAIDs. Using the “long cycle” approach with oral contraceptive pills (3 months of pills before a week without pills) can reduce the number of menses, thus improving the quality of life. No evidence supports switching from one OCP to another to improve response. High-dose progestins improve endometriosis by deciduation followed by pseudonecrosis and atrophy of lesions. Progestins suppress gonadotropin release and ovarian function; for example, 50 mg/day medroxyprogesterone acetate has been shown to improve symptoms in up to 80% of patients with endometriosis. Other regimens for progesterone

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<tr>
<th>Table 1. Secondary Causes of Dysmenorrhea and Chronic Pelvic Pain</th>
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<tr>
<td>Endometriosis</td>
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<td>Pelvic inflammatory disease</td>
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Dose and delivery including intramuscular depot progesterone are effective as well. Side effects of progestin therapy include weight gain, edema, depression, and headache. Danazol, a testosterone derivative, produces a hypoestrogenic environment and is effective in 80% of patients; however, the high incidence of androgenic side effects, approaching 80%, limits its use. The GnRH agonists (e.g., nafarelin nasal spray and leuprolide depot), suppress ovarian estrogen production causing estrogen deprivation. The typical course of treatment is for 6 months, after which the patient must be monitored for bone loss and consideration given to adding back estrogen or progesterone. In 50% of cases, there is recurrence of symptoms within 6 months after GnRH agonist therapy is discontinued.

If empiric treatment or laparoscopy with local ablation has not been successful, then more invasive treatments must be considered, such as uterosacral nerve ablation, presacral neurectomy, or a nerve block procedure. As a last resort, total abdominal hysterectomy and bilateral salpingo-oophorectomy could be considered.

**Conclusion**

Evidence supports the use of traditional NSAIDs and the COX-2 specific inhibitors in the treatment of pain associated with primary dysmenorrhea and endometriosis. High levels of effectiveness can be expected for both disorders, meaning that many women could find relief. Although endometriosis treatment has traditionally followed a surgical diagnosis, there is also evidence to support treating
endometriosis empirically in carefully screened women. Beyond NSAIDs and COX-2-specific inhibitors, there are several medications that may help. Invasive procedures and surgical cures should be reserved for those who are not improved by the algorithms presented here.

References