Dysmenorrhea: Contemporary Perspectives

Classification/definition:
- Dysmenorrhea (painful periods) is traditionally classified as either primary dysmenorrhea – *menstrual pain without pelvic pathology; with onset shortly after menarche*, or secondary dysmenorrhea - *pain associated with secondary pathology, and the onset may be years after menarche*.
- Premenstrual syndrome (PMS) is defined as cyclical mood and behavioral changes occurring during 5 days prior to menses.
- Premenstrual dysphoric disorder (PMDD) is the presence of severe affective symptoms during the luteal phase of the menstrual cycle, which may encompass depression, anxiety, concentration difficulties, appetite changes, and sleep changes that interfere with functioning in work, family, and social settings.

Epidemiology:
- Dysmenorrhea affects 40-90% of women.
- Primary dysmenorrhea is most common between the ages of 15-19 years, declining thereafter.
- 5-14% of women have regular school absenteeism as a result of symptoms.
- 13-51% of women have been absent at least once in their lives from school or work due to dysmenorrhea.
- Many cultures, such as some Mediterranean, Muslim, Hindu, and Chinese, still perceive menstruation as taboo and impure, resulting in reluctance of pain report and failure of health care delivery.

Associated risk factors:
- A low BMI is associated with increased risk of primary dysmenorrhea.
- A negative association has been described between consumption of fruit, eggs, and fish and primary dysmenorrhea, perhaps related to intake of omega-3 fatty acids, calcium, and magnesium.
- Psychosocial determinants are also important, as poor mental health, somatoform symptoms, decreased coping ability, depression, and anxiety have been found to be strong determinants of dysmenorrhea.
- Primary dysmenorrhea often co-occurs with nausea and vomiting, diarrhea, tiredness, and feelings of irritability.
  - Many idiopathic pain disorders (IBS, IC/PBS, vulvodynia, dyspareunia, temporomandibular disorder, and migraines) are frequently co-morbid with primary dysmenorrhea.
- Secondary dysmenorrhea presents in association with endometriosis, presence of an IUD, pelvic inflammatory disease, adenomyosis, uterine myomas and adhesions, or cervical obstruction from mullerian anomalies.
- Smoking has been associated with an increased risk of dysmenorrhea, but alcohol is not consistently linked to dysmenorrhea risk.

Presentation:
- Primary dysmenorrhea pain precedes the onset of a menstrual period and typically lasts 2-3 days.
- Secondary dysmenorrhea pain may start 1-2 weeks before menstrual flow and persist beyond the cessation of bleeding.
- The classic labor-like, suprapubic, colicky pain of dysmenorrhea may radiate to the lumbosacral region or anterior thigh.
- Associated visceral symptoms include nausea, vomiting, or diarrhea.
- On examination, prominent uterine tenderness is found during menstruation, which may also extend outside of menses in secondary dysmenorrhea.

Pathophysiology:
- The exact etiology is unclear, but may reflect upregulated cyclooxygenase (COX) enzyme activity and prostanoid synthase activity, which are normally activated in the late luteal phase through release of progesterone inhibition of arachidonic acid production.
- Endometrial prostaglandin production results in increased uterine contractions and relative myometrial ischemia.
- Abnormal uterine contraction patterns and alterations in uterine blood flow are also noted in some dysmenorrhea sufferers.
• Somatization and poor coping are also positively associated with menstrual pain intensity, suggesting that central factors should also be considered.

Treatments:

• Conservative measures such as non-steroidal anti-inflammatories (NSAIDs) are used as first-line therapy, ideally initiated prior to the onset of menses by 48 hours to decrease COX substrate.
  o A usual trial of 3-6 months of therapy is conducted before additional evaluation for causes of secondary dysmenorrhea.
• Vitamin and mineral supplementation (i.e. fish oil, thiamine, magnesium, or pyridoxine) may also be effective based on small studies.
• Local nerve stimulation (TENS, hot compresses, acupuncture) has also been shown to be effective in small studies.
• If NSAIDs fail, combined oral contraceptives are often employed to inhibit ovulation and suppress endometrial growth. By maintaining an endocrine state of the early proliferative phase, this approach also decreases prostaglandin levels.
• Continuous progestins (oral, intramuscular or via intrauterine device) may be needed to induce anovulation in recalcitrant cases.
  o While androgen derivatives (danocrine) also induce anovulation, their severe virilizing side effects make them less attractive.
• In more severe cases, short-courses of opioids should be considered for managing breakthrough pain.
• Exirpative surgery (laparoscopic excision of endometriotic lesions, or leiomyoma) should be reserved only if the above fail, while nerve destructive procedures should be used only cautiously.
  o While randomized controlled trials demonstrate that presacral neurectomy is an effective treatment for dysmenorrhea, this procedure does have a risk for causing permanent visceral side effects.

References: