What is the effect of exercise on primary dysmenorrhea?

Michael Steele/EMPICS

Denise Lewis during the hurdles discipline at the Athletics World Championship in Seville.

In the past 15 to 20 years, research into the link between physical activity and menstrual disorders has increased significantly. Exercise has been found to affect menstruation in a variety of ways, including inducing amenorrhea in athletes and perhaps alleviating symptoms of premenstrual syndrome and dysmenorrhea. Primary dysmenorrhea is characterized by a range of symptoms, foremost of which are lower abdominal pain that may radiate to the lower back or legs, headache, nausea, and vomiting. Some 75% to 85% of women experiencing dysmenorrhea report that their symptoms are mild, although dysmenorrhea may cause an appreciable number of absences from school and work. The symptoms are thought to stem from raised concentrations of prostaglandins, resulting in uterine contractions and ischemia. The origin of the increase in prostaglandins has not been decisively identified; one likely mechanism is thought to involve a decline in progesterone in the premenstrual phase, which results in the synthesis of prostaglandins in endometrial cells by membrane phospholipids. This theory is supported by the success of prostaglandin synthesis inhibitors in pain relief. As these inhibitors only provide pain relief in 70% to 75% of women, however, other factors are probably also involved. Possible contributors include deficient degradation of prostaglandins as the result of a defect in prostaglandin dehydrogenase, the vasoconstrictive action of antidiuretic hormone, and variations in pelvic blood flow, which may influence the synthesis or breakdown of prostaglandins. Primary dysmenorrhea affects between 47% and 80% of the general population depending on the age group studied; it tends to decrease with age and after pregnancy.[1]

Although exercise is generally thought to alleviate the discomfort associated with dysmenorrhea, the scientific literature on this phenomenon offers mixed evidence. One study identified a decline in the severity of symptoms after a 12-week aerobic training program,[2] and another reported diminished dysmenorrhea in exercising junior high school girls.[3] A number of studies have shown a correlation between life stress and gynecologic symptoms. Similarly, women who train intensively have been found to report fewer symptoms than women who exercise occasionally.[4] A number of other studies, however, have failed to find any relation between dysmenorrhea and levels of physical activity.[5-7] Furthermore, Metheny and Smith reported that after controlling for disposition and mood, exercise is actually associated with higher levels of menstrual discomfort.[8]

An interesting element of the relation between exercise and dysmenorrhea is the involvement of stress. A number of studies have shown a correlation between life stress and gynecological symptoms. Increased concentrations of cortisol have been associated with premenstrual syndrome,[9] and one study showed a correlation between menopausal symptoms and environmental stress.[10] Furthermore, dysmenorrhea has been treated successfully with stress reduction techniques.[11,12] Exercise is widely accepted as a means of moderating stress and stress-related symptoms; it may do this by reducing the perception of stress or inducing biochemical changes in the immune system.[13] A mechanism by which exercise may improve the symptoms of dysmenorrhea by reducing stress has been articulated by Gannon.[13] Menstrual pain probably stems from increased contraction of uterine muscle, which is innervated by the sympathetic nervous system. Stress tends to enhance sympathetic activity and may therefore increase menstrual pain by exacerbating uterine contraction. By relieving stress, exercise may decrease this sympathetic activity, thereby alleviating symptoms.

Because exercise has repeatedly been shown to improve mood, however, reduced dysmenorrhea may simply result from a better mood as opposed to an actual alleviation of physical symptoms. In fact, exercise is known to cause the release of endorphins, substances produced by the brain that raise the pain threshold. Many studies have failed to control for disposition and mood; the controlled study by Metheny and Smith suggests that although exercise may alleviate stress-induced dysmenorrhea, it may simultaneously aggravate symptoms.[8] One explanation

for this aggravation may be that exercise raises somatic awareness: regular exercise has been associated with increased sensitivity to bodily states. Alternatively, if women who exercise reduce their level of activity in the premenstrual phase, the resulting fall in endorphins could intensify dysmenorrhea.[8]

Overall, research into the relation between dysmenorrhea and exercise has been hampered by methodologic flaws such as varying definitions of dysmenorrhea and activity, different modes of data collection, disparate study designs, failure to perform blinded studies, and the retrospective reporting of symptoms. Blinded studies are particularly important in this area of research, as it has been shown that women may be influenced by previously held beliefs, taboos, and social expectations involving menstruation when answering questions about menstrual distress.[14] This complication, along with the confounding effects of stress, points to the variety of factors influencing dysmenorrhea and its relation to exercise. More carefully controlled longitudinal studies should help elucidate this complex relation. Research in this area is important, given the high prevalence of dysmenorrhea and the potential benefits of exercise.

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The virus and the hookworm

Hookworm infestation with the nematode *Necator americanus* was endemic in the highlands of Sri Lanka during my internship there. Patients presented profoundly anemic with a characteristic facial appearance that often led to a "spot diagnosis." Indeed, it was common for an intern to tell a colleague in passing, "I see you've got another hookworm coming in."

Hepatitis A or infectious hepatitis, as it was known a few decades ago (to distinguish it from hepatitis B or serum hepatitis), was also a common infection. At any time, the medicine ward would include 3 or 4 patients so afflicted. As inspection of the urine was a better index of jaundice than examination of the eyes, clear glass jars containing a morning specimen of urine could be seen by each patient's bedside. Then, as now, the treatment was largely supportive. As "managed care" was a term yet to come, patients remained in bed for about 3 weeks, and when it was deemed that the patient had convalesced enough, plans for discharge were initiated.

When I was an intern, such a patient taught me a lesson that I shall never forget. Examination of the stools for parasitic ova and cysts was routine for all inpatients, regardless of the reason for admission. Helminthiasis was so prevalent that eradication of asymptomatic infestation was the usual practice. This particular patient's stool had yielded hookworm ova, and on the day before discharge, I ordered the standard dose of trichlorethylene. That was the treatment of the day and, although not as effective as the drugs now available, it had a high success rate in eliminating the parasite. Of course, a recurrence of illness was the rule rather than the exception. There was no way you could tell whether the recurrence was because of incomplete eradication or reinfection, nor did it matter.

On the morning of discharge, my patient was drowsy, and I rather naively attributed his somnolence to a poor night's sleep. The consultant was more impressed by the patient's appearance than by my explanation. He reached for the chart and studied it. "I see that you have prescribed TCE for this patient," he said.

Misinterpreting this as a compliment, I responded, "Yes, because his stools contained hookworm ova, and I thought it best to treat him before he left hospital."

"Do you know the formula for trichlorethylene?" he asked.

With increasing pride, I replied, "Yes, sir. C_2HCl_3."

"And what," he asked, "are the agents used for the experimental induction of hepatic necrosis?"

I still suspected nothing wrong. Remembering an old mnemonic from pathology, P for phosphorus that causes peripheral necrosis, and C for carbon tetrachloride that results in centrilobular necrosis, I answered with some satisfaction, "Phosphorus and carbon tetrachloride."

"And what," continued the consultant, "is the formula of carbon tetrachloride?"

That was when the penny dropped, as did my heart. As I responded, "CCl_4," I knew what was coming next.

"I hope you realize," he said, "that you have administered a highly hepatotoxic drug to a patient whose liver is recovering from hepatitis—a drug that is different by but two atoms from a powerful toxin." I said nothing; what could I say? Then he used the same phrase that had been used before in those circumstances: "Never again."

Results of the thymol flocculation and zinc turbidity—liver function tests used at the time—confirmed what we already knew: a marked deterioration. Fortunately, a few more days of tender loving care resulted in complete recovery. As I saw the patient walk out of the ward, I said to myself, "Never again."

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