What Your OB/GYN Should Know About FMS and CMP by Devin J. Starlanyl

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Please read "What Everyone on Your Health Care Team Should Know About FMS and CMP".

Fibromyalgia (FMS) and chronic myofascial pain (CMP) can have an impact on many aspects of your patient care. FMS is a state of central sensitization causing allodynia and hyperalgesia (Russell 1998). In CMP, there are nodules inside taut bands in muscles that decrease range of motion and cause pain at the end of range of motion (Simons, Travell, Simons 1999). TrP pain may be referred elsewhere, with autonomic concomitants and proprioceptive dysfunction. Myofascia can entrap nerves, blood and lymph vessels. Patients with FMS often experience a decrease in libido. CMP may cause pediatric and adolescent pelvic pain (Schroeder, Sanfilippo, Hertweck 2000). Studies show that 50 % of the children of people with FMS may have an inherited tendency to develop FMS (Pellegrino, Waylonis and Sommer 1989), so female children of an FMS parent should be monitored carefully during first menses.

FMS patients often have multiple hormonal and autonomic imbalances leading to profound physiological and clinical consequences (Adler, Manfredsdottir, Creskoff 2002). FMS can interact with and be affected by sex hormones (Carett, Dessureault, Belanger 1992) and reproduction (Ostensen, Rugelsjoen, Wigers 1997). FMS is associated with hypothalamus-pituitary-adrenal (HPA) axis dysfunction. This imbalance can affect the hypothalamic-pituitary-gonadal (HPG) axis and other hormonal axes. Pituitary release of growth hormone and prolactin may be abnormal in FMS (Griep, Boersma de Kloet.1994). Patients with endometriosis may have high rates of FMS (Sinaii, Cleary, Ballweg 2002). FMS and CMP can add to the severity of endometriosis symptoms. There seems to be an increased incidence of female urethral syndrome in FMS (Wallace 1990). Female urethral syndrome is a description, not a diagnosis. Look for the cause. TrPs which may cause or contribute to this syndrome include TrPs of the low abdominal wall, low rectus abdominus TrPs, pelvic floor muscles, piriformis, and high adductor magnus muscles.

Severe menstrual cramping, delayed periods, irregular periods, long periods with excess bleeding, late periods, missed periods, membranous flow, and/or blood clots may be a part of the FMS and CMP picture. Menstrual cramping can be caused by coccygeus, iliocostalis, rectus abdominis, pyramidalis, and other pelvic and vaginal TrPs, as well as TrPs high on the adductor magnus muscle (Simons, Travell, Simons 1999). The high adductor magnus TrP, a common and frequently missed contributor, refers severe pain deep within the pelvis, and can compress

femoral blood vessels. These TrPs are found about 3/4 of the way back from the front of the inner thigh and respond well to groin stretches. The referral zone can include the pubic bone, vagina, rectum, and bladder. In some patients, this pain only occurs during intercourse.

Fibrocystic breasts are often linked with FMS, but I believe that they may be due to TrPs. The smooth muscles around the ducts have motor endplates, so it is logical the TrPs may form here. Large pendulous breasts can aggravate and perpetuate TrPs and promote the common TrP perpetuating factor of head-forward, round shouldered posture. Mammography can be painful for women with both FMS and CMP. Myofascial TrPs generate the pain, and FMS amplifies it. Extra medication and massage before a mammogram, and an extra soft touch during it, may prevent pain that could further sensitize an already sensitized CNS.

Dyspareunia

Dyspareunia may be caused by vaginal and pelvic floor TrPs. Abdominal and lowback TrPs may cause aching and cramps during intercourse. Sharp pain may be part of piriformis TrPs causing pudendal nerve entrapment. TrPs in any short lateral rotator muscle can cause piriformis syndrome, including nerve entrapment. These TrPs are often part of a conglomerate of hip and pelvis TrPs that aggravate each other. These TrPs refer to the sacroiliac region, spreading sideways across the buttock, over the hip, and to the upper 2/3 of the back of the thigh, in a composite pain referral pattern. Pain from these TrPs is usually increased by sitting, standing and walking. The sciatic radiation of pain from nerve entrapment can extend down the back of the leg to the sole of the foot. Even spreading the thighs apart can be painful. Compression can also cause buttock, inquinal, perineal, and posterior thigh pain and parathesias. Piriformis TrPs also may result in tightening or immobility of the sacroiliac, which causes further dysfunction. Pelvic floor TrPs refer pain to an oval area between the buttocks from the base of the sacroiliac triangle to the base of the tailbone. The obturator internus TrP pain pattern is more rounded, and there may be spillover pain in a V-shape down the back of the thigh. These TrPs may cause generalized symptoms such as dyspareunia, particularly during entry, as well as aching perineal pain. Specific muscles cause specific symptoms as well. For example, TrPs in the levator ani muscle refer pain to the perineum, vagina, coccygeal area, rectum, posterior pelvic floor, and low back. They can cause stress incontinence, loss of ability to empty the bladder thoroughly and low back pain, particularly in pregnancy. Post-coital rectal pain may be prevented by oral clonidine or inhaled salbutamol, but the TrPs should be treated as well.

TrPs in the ischiocavernosus muscle refer pain to the perineum and nearby urogenital structures. These TrPs can contribute to dyspareunia, particularly during entry, as well as an aching pain in the perineum. Often pain and pressure in the vaginal and perineal area responds well to internal work on the bulbospongiosus. A pelvic exam may activate these TrPs, but this can often be

avoided by use of topical anesthetic. These TrPs may respond to finger pressure, but the topical anesthetic is mandatory. Allow time for it to work. These TrPs may respond to groin stretches and craniosacral release, and to diazepam or similar medication taken before intercourse. For detailed information on specific pelvic floor and related TrPs including diagnoses and treatment, see volume II of the Trigger Point Manuals (Travell and Simons, 1992).

TrPs can cause vulvodynia so severe that intercourse is impossible, and the ability to sit (and work) may be severely curtailed. Vaginal TrP taut bands can often be felt during pelvic exam. Vaginismus may co-exist with TrPs in the pelvic area. Some women experience post-coital increase in leg and back pain due to activation of myofascial TrPs. Adequate treatment should avoid this. A daily home stretching program is very important. Some FMS patience experience post-coital weakness so severe they may be unable to rise from the bed to use the bathroom. They may also experience intense heat sensations.

TrPs in scar tissue produced by surgical incision are common, especially in the vaginal cuff following hysterectomy. Lessen the chance of surgical scar TrPs by injecting the incision area of the cuff as well as the outer layers of the initial incision with procaine immediately before surgery. Pelvic scar tissue TrPs can cause pain easily mistaken for menstrual cramps or bladder spasms.

Intercourse-induced headaches generally involve TrPs. This type of headache is usually localized at the occipital area, but it may be generalized. Coitus activates the TrPs. Latent TrPs are like time bombs waiting to go off, and they may do so right before or after orgasm. The TrPs need to be treated until they are *gone*, not just latent. A beta blocker may be helpful to prevent this headache. Adequate timing of medication is essential. Coitus may also cause sensory overload in FMS patients. This does not always involve pain but can include a heightened sensitivity to noise or light, or hyperarousal insomnia from racing adrenalin.

Chronic pelvic congestion can cause chronic pelvic pain. This may be hormonal but may have a circulatory component. S ome of the circulatory congestion may be due to myofascial entrapment. Hormone therapy acts upon venous receptors (Charles, 1995). TrP entrapment can be treated with myofascial release, myotherapy, or craniosacral release therapies. Anything that adds to the congestion of the pelvic area, such as an infection, can exacerbate the symptoms.

Hormone Replacement Therapy (HRT)

It is important to find out exactly what the patient's hormone levels are. Other cells besides the ovaries are involved in estrogen generation. HRT is a mechanism for *replacement* of hormones to normal levels. It is critical to identify the correct levels of HRT replacement needed for each patient. Menopausal women experienced enhanced memory and learning capacity with estrogen replacement (Sherwin1997). This may be of importance for women with FMS cognitive deficits

(Starlanyl, Copeland 2001). Insulin resistance is a common perpetuating factor of FMS and CMP. Estrogens raise glucose levels and can add to the risk of developing insulin resistance. They boost sodium levels and may increase fluid retention. Estrogen lack has a significant effect on carbohydrate and lipid metabolism (Grumbach, Auchus 1999).

Malabsorption is common in FMS. Some women may be unable to absorb oral estrogen. A transdermal formula of 1.0 mg estriol, 0.25 mg estrione and 0.25 mg estradiol per 0.1 cc can be formulated by a compounding pharmacist. Progesterone transdermal cream can be applied separately to approximate normal hormone balance if needed, but progesterone may increase insulin resistance. Be cautious when changing medications. For example, a change to Estrace vaginal cream in one patient activated abdominal and other TrPs, causing crippling pain mimicking menstrual cramps, although the uterus and ovaries were gone. It took weeks to inactivate the TrPs.

Perimenopause, as well as menopause itself, can begin early for FMS patients, with amplified symptoms. Insomnia can be intensified by hot flashes. HRT may provide relief. Another therapeutical option is gabapentin (Guttuso, 2000), sometimes prescribed for the central sensitization of FMS. Patients experienced an average 87 percent reduction in the frequency of hot flashes. Some postmenopausal symptoms may be secondary to androgen deficiency. It is important to test *free* testosterone. When there is low circulating *bioavailable* testosterone, adequate replacement may relieve the symptoms (Davis,1999). In a study of women who had symptoms that responded to estrogen but then returned, the patients responded to testosterone (Sarrel, 2000).

Menstruation

Some women find that the FMS symptoms worsen dramatically during menses. There may be changes in the pattern of pain and other FMS symptoms (Anderberg, Marteinsdottir, Hallman et al. 1998). There may be irregular blood flow, cramping, membraneous discharge (often with blood clots), and/or extreme blood flow. Vaginal discharge, sometimes with itch, is common. So is mittel-schmerz. One study showed a greater number of FMS tender points *after* menstruation than during menstruation, but not in users of oral contraceptives (Hapidou, Rollman 1998).

Painful menstrual periods should *never* be considered something to be endured. There may be perpetuating factors such as endocrine imbalance or contributing myofascial TrPs that can be remedied. Patients have had hysterectomies because menstrual pain was unbearable and dramatically interfered with function. Often the ovaries were left, but in many cases were removed later to balance hormonal swings and prevent mittelschmirtz and/or hormone-activated migraines.

Infertility

Any chronic pain condition can result in failure to ovulate (Berga 1998). Excess hyaluronic acid has been found in patients with FMS (Yaron, Buskila, Shirazi et al. 1997). This may affect fertility. One study suggests a hyaluronidase inhibitor for contraception (Reddy, Joyce, Zaneveld 1980). Check for hypometabolism and thyroid resistance, which will not show up on standard thyroid panels (Lowe, 2000). Topical or oral T3 may be of use if hypothyroid symptoms are present (Starlanyl, Jeffrey, Roentsch, et al. 2001-2002).

Obesity may decrease fertility and increase insulin resistance (Crosignani, Vegetti, Columbo et al. 2002). Infertility may be enhanced by an anteriorly rotated pelvis, often due to myofascial TrPs. Exposure to bright light may be a useful treatment for infertility if there is abnormal melatonin metabolism (Partonen,1999). Guaifenesin therapy, which some of us have found useful for FMS, has been used to treat infertility. It thins cervical secretions, making it easier for the sperm to penetrate the egg (Check, Adelson and Wu, 1982). Guaifenesin therapy must proceed carefully, because each person with FMS has a personal best dosage. An incorrect dosage can increase FMS symptoms, as can treatment without proper diet in cases of co-existing insulin resistance, reactive hypoglycemia or metabolic syndrome (Starlanyl, Copeland 2001).

Pregnancy

Most medications for FMS and CMP are not safe for pregnant or lactating women. Perpetuating factors for FMS and TrPs must be identified and controlled as much as possible to reduce symptoms. Some patients have reported that propranolol helped control pain. Explore non-medicinal pain and stress control options, such as bodywork, mindwork, and life style changes such as attention to diet, sleep habits, exercise and stretching routine. Smoking is a huge perpetuating factor, and should be avoided. Some women have reported partial remission of FMS symptoms during pregnancy, but symptom flare after birth. TrPs can worsen during pregnancy. Pregnancy-induced hypertension may be caused by insulin resistance (Innes, Wimsatt 1999).

More than twenty FMS patients have reported 10-month gestations. All delivered healthy infants. This may be related to FMS biochemical irregularities, or even to TrP-induced contracture of muscles. Relaxin may be involved. Women with hips locked in flexion due to TrPs are more likely to have trouble delivering. Ripening of the cervix requires loosening of collagen through changes in ground substance (Calder 1994), and FMS and CMP can alter ground substance (Starlanyl, Copeland 2001). FMS patients may require pain medication earlier than healthy women, and may also require induced labor. Myofascial pain may exceed analgesia provided by an epidural. TrP injections may be required for complete analgesia during labor (Tsen, Camann 1997) and may prevent or minimize postpartum TrPs.

References

Adler GK, Manfredsdottir VF, Creskoff FW. 2002. Neuroendocrine abnormalities in fibromyalgia. *Curr Headache Rep* 6(4):289-98.

Anderberg UM, Marteinsdottir I, Hallman J et al. 1998. Variablility in cyclicity affects pain and other symptoms in female fibromyalgia syndrome patients. *J Musculoskel Pain* 6(4):5-22.

Berga SL. 1998. Hypothalamus pituitary gonadal axis: stress induced gonadal compromise. *J Musculoskel Pain* 6(3):61-70.

Borg-Stein J. 2002. Management of peripheral pain generators in fibromyalgia. 2002. *Rheum Dis Clin North Am* 28(2):305-17.

Butkevich IP, Vershinina EA. 2003. Maternal stress differently alters nociceptive behaviors in the formalin test in adult female and male rats. *Brain Res* 961(1):159-65.

Calder AA. 1994. Prostaglandins and biological control of cervical function. *Aust N Z Obstet Gynaecol* 34(3):347-51.

Carrett S, Dessureault M, Belanger A. 1992. Fibromyalgia and sex hormones. *J Rheumatol* 19(5):831.

Charles, G. 1995. [Congestive pelvic syndromes]. *Rev Fr Gynecol Obstet* 90(2):84-90. [French]

Check JH, Adelson HG, Wu CH. 1982. Improvement of cervical factor with guaifenesin. *Fertil Steril* 37(5):707–708.

Crosignani PG, Vegetti W, Columbo M. et al. 2002. Resumption of fertility with diet in overweight women. *Reprod Biomed Online* 5(1):60-4.

Davis SR. 1999. Androgen treatment in women. Med J Aust 170(11):545-9.

Griep EN, Boersma JW, de Kloet ER.1994. Pituitary release of growth hormone and prolactin in the primary fibromyalgia syndrome. *J Rheumatol* 21(11):2125–2130.

Grumbach MM, RJ Auchus. 1999. Estrogen: consequences and implications of human mutations in synthesis and action. *Clin Endocrinol Metab* 84(12):4677-94.

Guttuso Jr. TJ. 2000. Gabapentin's effects on hot flashes and hypothermia. *Neurology* 54(11):2161-2163.

Hapidou EG, GB. 1998. Menstrual cycle modulation of tender points. *Pain* 77(2):151-61.

Innes KE, Wimsatt JH. 1999. Pregnancy-induced hypertension and insulin resistance: evidence for a connection. *Acta Obstet Gynecol Scand* 78(4):263-84.

Lowe J. 2000. *The Metabolic Treatment of Fibromyalgia*. Boulder, CO: McDowell Publishing Company.

Ostensen M, Rugelsjoen A, Wigers SH. 1997. The effect of reproductive events and alterations of sex hormone levels on the symptoms of fibromyalgia. *Scand J Rheumatol* 26(5):355–360.

Partonen T. 1999. Melatonin-dependent infertility. Med Hypotheses 52(3):269-70.

Pellegrino M, Waylonis GW, Sommer A. 1989. Familial occurrence of primary fibromyalgia. *Arch Phys Med Rehab* 70(1):61–63.

Reddy JM, Joyce C, Zaneveld LJ. 1980. Role of hyaluronidase in fertilization: the antifertility activity of mycrisin, a nontoxic hyaluronidase inhibitor. *J Androl* 1(1):28-32.

Russell IJ. 1998. Advances in fibromyalgia: possible role for central neuro-chemicals. *Am J Med Sci* 315(6):377-384.

Sarrel PM. 2000. Effects of hormone replacement therapy on sexual psychophysiology and behavior in postmenopause. *J Womens Health Gend Based Med* 9(Suppl 1):S25-32.

Schroeder B, Sanfilippo JS, Hertweck SP. 2000. Musculoskeletal pelvic pain in a pediatric and adolescent gynocology practice. *J Pediatr Adolesc Gynecol* 13 (2):90.

Sherwin BB. 1997. Estrogen's effects on cognition in menopausal women. *Neurology* 48(5 Suppl 7):A21-6.

Sinaii N, Cleary SD, Ballweg ML. 2002. High rates of autoimmune and endocrine disorders, fibromyalgia, chronic fatigue syndrome and atopic diseases among women with endometriosis: a survey analysis. *Hum Reprod* 17(10):2715-24.

Simons DG, Travell JG, and Simons LS. 1999. *Travell and Simons' Myofascial Pain and Dysfunction: the Trigger Point Manual*: Vol I, ed 2. Baltimore: Williams and Wilkins.

Starlanyl DJ, Jeffrey JL, Roentsch G et al. 2001-2002. The effect of transdermal T3 (3,3',5-triiodothyronine) on geloid masses found in patients with both

fibromyalgia and myofascial pain: double-blinded, N of 1 clinical study. *Myalgies* 2(2):8-18.

Starlanyl DJ, Copeland ME. 2001. *Fibromyalgia and Myofascial Pain: A Survival Manual*. Ed. 2. Oakland: New Harbinger Publications.

Staud R, Smitherman ML. 2002. Peripheral and central sensitization in fibromyalgia: pathogenic role. *Curr Pain Headache Rep* 6:259-266.

Tsen LC, Camann WR. 1997. Trigger point injections for myofascial pain during epidural analgesia for labor. *Reg Anesth* 22(5):466-468.

Travell JG, Simons DG. 1992. *Myofascial Pain and Dysfunction: The Trigger Point Manual*, Vol. II. Baltimore: Williams and Wilkins.

Wallace DJ. 1990. Genitourinary manifestations of fibrositis, and increased association with female urethral syndrome. *J Rheumatol* 17(2):238–239.

Yaron I, Buskila D, Shirazi I et al. 1997. Elevated levels of hyaluronic acid in the sera of women with fibromyalgia. *J Rheumatol* 24(11):2221-4.