Background: Women with CPP have high incidence of endometriosis, between 71-87% of women with CPP have laparoscopically proven endometriosis. Hormonal therapies and surgeries have failed to restore pain free living; it appears central sensitization and myofascial pain secondary to MTrPs may be another “source of pain initiation, amplification and perpetuation.” Most Gyns are not trained in assessment of myofascial dysfunction – this article targets CPP related to endometriosis from a pain centered perspective, indicating a need for Gyn providers to be trained in a more manual based exam with specific treatments focused away from traditional allopathic medicine.

Neural Mechanism in Endometriosis- Associated Chronic Pelvic Pain: the Dynamic Role of Sensitization:
- Chronic pain is pathological- persists after initial insult has resolved.
- Result of functional and structural rearrangements of CNS which sustains perception of pain and facilitates expansion to distant regions.
- Endometrial lesions: hormonally dependent, inflammatory; engage reproductive, endocrine, vascular, MSK, neuronal systems

Potential mechanism for activation of nervous system by ectopic endo lesions:
- Innervation of lesions through neural sprouting of sensory/sympathetic fibers that innervate nearby blood vessels- allowing for simultaneous invasion of nerves.
- Nerve growth factor (NGF) - found in high levels in: peritoneal, deep adenomyotic, ovarian endo lesions. Greater nerve fiber density is associated in peritoneal endo lesions compared to normal peritoneum.

Direct innervation of ectopic lesions by sensory and sympathetic n. fibers confirmed in rat/humans with endo
* Presence of these nerve fibers correlates with severity of pelvic pain and dysmenorrhea- sensory fibers include C-fiber nociceptors which are sensitive to immune and inflammatory factors prevalent in endo. NGF activates nociceptors and recruits mast cells releasing inflammatory molecules with degranulation.

- Peripheral sensitization- repeated or prolonged activation of nociceptors results in lowering activation threshold
- Central sensitization- nociceptor activation generates an afferent bombardment of noxious signals into dorsal horn of spinal cord inducing structural changes throughout spinal cord with evoked exaggerated responses to peripheral stimuli.
  o Allodynia- pain to a non-noxious stimulus
  o Hyperalgesia- increased pain to noxious stimulus
  o Viscerosomatic convergence (VC)- process which allows spinal neurons that receive visceral input to receive somatosensory input from muscle and skin; impedes specific localization/discrimination of sensory info. VC explains how ongoing noxious visceral input can sensitize multiple areas of spinal cord-leading to broad areas of allodynia, hyperalgesia, referred somatic pain
  o Viscerosomatic reflex- visceral nociceptors converge with
  o Somatic nociceptors onto interneurons in spinal cord activating both alpha/gamma motor neurons to skeletal mm. – can produce increased mm tone/spasm in pain referral pattern, trigger ‘guarding reflexes’ by visceral pain/inflammation leading to PF dysfunction/MTrPs.
Contribution of Active Myofascial Trigger Points to CPP

MTrPs- believed to occur secondary to muscle overload/overuse

Medical conditions: metabolic, visceral, endocrine, infectious, psychological origin
Pelvic floor: previous Gyn surgeries, childbirth, injury, sexual abuse, dyspareunia. Poor mechanics.
Visceral disease: endometriosis, IC/painful bladder syndrome, vulvodynia, IBS, coccygodynia, urethral syndrome

Myofascial pain requires own diagnosis and treatment: Active MTrPs can be source of ongoing nociception, reduction in pain thresholds, enhancement of visceral/referred pain, sensitization of nervous system.

With Endometriosis- MTrPs could sustain pain/dysfunction despite removal/reduction in endo lesions post surgery or hormonal Rx.

Clinical Evaluation:

Pelvic Evaluation/Neuromuscular Pain Assessment
Standard history: endo/CPP Add: pain calendar for menstrual cycle, past & current treatment
Pelvic Exam for MtrPs: single digit: Internal/External pelvis, abdomen, SI joints
  *MTrPs most common with CPP: sphincter ani, levator ani, coccygeus, obturator
    Internus- manifested as: taut bands, tender, span distance of fiber, in spasm
Bimanual exam: central uterine tenderness

Neuro-muscular Exam: Identifies widespread pain, central sensitization, myofascial dysfunction
Dermatomes for allodynia/hyperalgesia
Myotomes for presence of MTrPs.

Pain Management

1. MFR- Physical therapy (PT) and other manual techniques
Research: One small retrospective study: PT benefits up to 63% of patients; there are no RCT comparing PT with standard of care for patients with endometriosis

2. MTrP injection- wet or dry needling
   a. CPP patients with MTrPs in abdomen wall had good response to direct lidocaine injections compared to PT

3. Botulinum Toxin (BTX) Type A: alleviates hypertonicity- efficacy in treating spasticity, dystonia, hemifacial spasm, OAB, migraine well established. Effects of BTX on neuromuscular junctions are irreversible, requiring new motor axon regeneration & restoration of NMJ. Period of efficacy: 3-6 mths.
Safety established over 20 year hx.
Research
   a. Case studies- good results
   b. RCT type A BTX – improvement in non-menstrual pelvic pain unique to treatment group, dry needling may contribute to pain relief of MTrPs.
   c. Other studies – less efficacy
   d. Authors research: Randomized placebo-controlled pilot study, N=7. Both group experienced relief: BTX x 3 weeks, placebo group 1 week. * preliminary date difficult to interpret- these receiving BTX had lower overall pain severity prior to injection than those with placebo group (3.6 vs. 8.5 on VAS scale 0-10)
   e. BTX may be effective in relaxing mm of PF, unlikely to respond to surgery, hormonal therapy or MFR techniques
   f. BTX- highly specific for cholinergic neurons, decrease in release of proinflammatory neuropeptides with resultant decrease in inflammation, alterations in intracellular trafficking patterns
Conclusion: Viscerosomatic convergence may explain the way for pain referral to somatic structures to MTrPs. Painful MTrPs may contribute significantly to CPP. Targeted interventions have the potential to reverse central sensitization and improve pain in patients with endometriosis.

Clinical Application Questions:

1. Does having a better appreciation for the mechanism of central sensitization change your thoughts regarding your patient population with endometriosis driven CCP? Do you feel the authors of this article have a good understanding of pelvic floor PT/MFR?
2. Are your referring providers aware of central sensitization in your patients with CPP? If so, are you working together with injections/dry needling concurrently with MFR/other PT modalities?
3. Do you feel the research sited in this article is biased toward use of BTX? (refer to their pilot study) Or on track as an appropriate modality for those with central sensitization?
4. Has anyone had patients treated with BTX? What was the clinical outcome?