Original article

Mapping chronic urogenital pain in women: review and rationale for a muscle assessment protocol - Part 1

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INTRODUCTION

Chronic urogenital pain (CUP) is a prevalent, yet complex pain phenomenon, affecting women of all ages. CUP is difficult to understand because it exists where there is no visible pathology, continues long after tissue irritation or damage, and persists well beyond the expected time of healing. In simple terms, CUP occurs where there is little, if any, obvious reason for it to exist. CUP syndromes encompass a wide range of pain sites and symptoms, which affect the functioning of the urinary and reproductive systems. Women diagnosed with CUP report symptoms of urge, frequency, suprapubic pressure, nocturia, dyspareunia, abdominal and lower back pain and a range of other comorbidities. The pervasive nature of CUP can be debilitating, interfering with daily activities, relationships, vocational capacity and reproductive potential. Given the high prevalence of these pain disorders,[1,2] it is surprising that even what is considered as a state-of-the-art response in relation to CUP management, is deemed unsatisfactory by clinicians and sufferers.[3]

Women diagnosed with CUP, invariably consult multiple specialists, present with lists of inconclusive investigations, and often have a long history of unsuccessful attempts at treatment. This reflects a notorious lack of response to antibiotics, antifungals, corticosteroids, hormone therapies, antidepressants, and less frequently surgeries. These ineffective management strategies tend to protract suffering and are disparaging to both patient and clinician, not to mention costly to the individual and the health care system. In the last 30 years, vast resources have been expended on the study of CUP and the growing body of scientific literature attests to the complexity and impact these disorders have on women. Yet, one of the key, but elusive tasks, is the need to establish the cause and source of pain.

Reviewing the current management guidelines formulated by various peak associations,[4-6] pain of pelvic muscle origin, though mentioned in literature, has been long overlooked as a potential cause of CUP symptoms.[7] Over the last thirty years evidence linking dysfunctional pelvic muscles with CUP syndromes has increased significantly.[8] Each of the peak associations endorse pelvic muscle assessment as part of diagnostic screening, and recommend muscle related therapies as frontline interventions in the management of CUP.

The authors of this two part series contend that there is an evident need for developing a standardized assessment protocol based on the link between CUP and pain of pelvic muscle origin. The first article introduces the *integrated mapping and assessment protocol* (IMAP) and establishes the rationale for pain mapping in the context of two CUP syndromes, vulvodynia and bladder pain syndrome/interstitial cystitis (BPS/IC). The second article introduces the clinical use of the IMAP in CUP, illustrating its benefits. Together these two articles provide new insights into the origins and mechanisms of pain, thereby expanding the evidence base for improved management of CUP.

Pain Mapping and CUP Syndromes

Pain mapping, sometimes referred to as the cartography or iconography of pain, is an assessment method for identifying the origins of pain. The history of pain mapping is not extensive but its evolving use is encouraging and relevant to CUP.[9]

In recent times Travell and Simons enriched the understanding of pain through their extensive compilation of pain maps linking the origins of pain with body regions to which pain was referred.[10] The mechanisms of pain, proposed by Travel and Simons, focused on changes in myofascial structures that gave rise to trigger points (TrPs) within muscles, ligaments and fascia. TrPs were identified as tender and irritable points within muscle spindles responsible for tension regulation. TrPs have been shown to be sympathetically innervated and the mechanisms by which emotions affect muscle tension were subsequently identified.[11,12] TrPs can be either active, producing constant pain, or latent, leading to referred pain only when provoked by pressure or the activation of muscles. Because TrPs are often latent, pain of myofascial origin can remain unidentified, unless muscles are examined and palpated. It is important to remember that TrPs, whether active or latent, not only refer pain but also compromise the function of muscles and cause autonomic reactions within proximal regions.[10]

The potential contribution of pain mapping to the understanding of CUP arises from its systematic approach to localising the source of pain and ascertaining its quantitative and qualitative characteristics. Theoretically, certain fundamental features of chronic pain can compound any attempts to localise it. Given that pain is a sensory and emotional experience, it can be elusive, and pain pathways difficult to identify. The modulating impact of mental and emotional states affects any attempts to objectively assess pain severity, and the impact of chronic pain syndromes on sufferers.[13] Currently there are no qualitative or quantitative measures available to logically establish the location, intensity and impact of CUP.[14]

Most CUP disorders trace their onset to noxious triggers (acute tissue trauma, infections and inflammatory states), which are known to initiate a cascade of nerve and immune reactions, as well as reflex muscle responses. When these reactions continue beyond the acute phase, they give rise to what ultimately characterises chronic pain syndromes; decreased pain thresholds, increased field of nociceptor reception, increased nociceptor responsiveness (allodynia), increased intensity of response (hyperalgesia), prolonged post stimulus sensations (hyperpathia) and the occurrence of unexplained spontaneous pain, affecting the overall functioning and wellbeing of the individual. Given the

known involvement of pelvic muscles in CUP syndromes, if pelvic muscles are the primary generators of persistent pain, they are easily accessible for assessment and pain mapping. Various functional assessments of pelvic muscles have been published,[15,16] but what is evidently lacking is a standardised pain assessment protocol that would assist with localising pain in the urogenital area. The IMAP has been developed to meet this specific clinical need, and its use and efficacy are illustrated in Part 2 of this series.

The benefits of pain mapping are manifold. In the context of CUP, pain mapping assists with:

- Localising the generators of pain;
- Linking the origins of pain with patient's symptoms;
- Quantifying the severity of pain;
- Ascertaining the sensory qualities of pain (i.e. burning, stabbing pain);
- Authenticating the patient's experience of pain by reproducing what the patient considers to be "their pain";
- Identifying the muscular origins of pain;
- Highlighting the role of peripheral mechanisms of pain;
- Guiding therapy based on the results of each individuals pain profile;
- Creating a practical and tangible pathway for resolution of pain; and
- Providing an objective means for evaluating the effectiveness of therapeutic interventions.

With these benefits, pain mapping facilitates a more pragmatic approach to the assessment and management of CUP. It goes beyond the common but basal question of 'where is the pain?' and addresses the more complex issue of 'where is the pain coming from?' By doing so, it directs the inquiry to the source of pain, and in particular focuses on its muscle origin. Unlike pain diaries and body forms, which are a standard feature of pain questionnaires,[17] pain mapping goes beyond phenotyping,[18] and operationalizes the task of chronic pain management.

Rationale for Muscle Pain Mapping in CUP Disorders

The management of chronic pain is most effective when the pathogenesis and mechanisms are clear and reasonably understood. Two study methodologies are often utilised in investigating pathophysiology; the first looks at the anatomical and functional changes associated with a disease, and the second examines treatments that are effective in halting the disease, enabling the identification of mechanisms by which interventions work. The outcomes of both methodologies are relevant to the study of CUP.

Traditionally, the end-organ specialist has been entrusted with excluding all medical causes of urogenital pain before making a diagnosis, explaining the patient's symptoms and offering treatment and a prognosis.[19] However, given that CUP symptoms are poorly localised and not only affect the bladder, urethra and vulvovaginal area, but also the colorectal, perineal, groin, thigh, suprapubic, abdominal and lower back areas, the absence of pathology led to certain assumptions which gave rise to the development of nomenclature and classification systems that relied almost exclusively on organ-based, symptomatic criteria. As a result bladder and vulvar symptoms were thought to originate from an undiagnosed disease or neurological disorder affecting the organs named in the pain syndrome (*bladder* pain syndrome, *urethral* syndrome, *vulvar vestibulitis* syndrome, etc.). This organ-based classification system, arising from a questionable assumption, gave rise to a further dilemma. Because pain was "…thought to stem from the kidney, bladder… pelvic organs or vulva," treatment was organ directed, until it became very evident that such postulations were erroneous and that "pain felt in these regions may not originate in the organs themselves…".[20] The misleading hypothesis that pain stemmed from the organ at the centre of the syndrome gave rise to a high frequency of failure in organ directed treatments, and confirmed the organ-based approach to be wrong, suggesting that the mechanisms being considered were also wrong.[20]

The organ based labelling of syndromes created the false impression that something was known about the underlying pathology. The terminology itself, gave rise to erroneous thinking and misguided, and at times dangerous therapies.[21] It was assumed that if "the disease" in the organ could be cured, or more radically the "diseased organ" removed, the pain would no longer exist. This proved not to be the case.[22,23] The removal of the presumptively "diseased" end-organs, by means of cystectomies, clitoridectomies, colectomies and hysterectomies, did more to "ingrain and accelerate these pain conditions than to relieve them".[24]

Many women currently attending pain clinics present with a history of antecedent pelvic surgery, having undergone major invasive procedures, including hysterectomies, sometimes at an early age, even in their early 20's. The present-day practice of multiple laparoscopies, ultrasounds, MRI's and CT scans produces little evidence of disease or anatomical abnormalities that explain CUP.[25]

Another extreme and equally erroneous assumption gave rise to the belief that the absence of pathology was indicative of underlying psychopathology. The reality of patients pain came into question.[26] Some medical texts suggested that CUP syndromes represented the end-stage of repressed emotional disturbances. However, even allowing for the questionable possibility of a psychiatric aetiology, surgical treatments were advocated for chronic pain syndromes "most of which worsened the illness".[24]

With the undue focus on a range of erroneous assumptions about the causes of chronic pain, the functional role of the musculoskeletal system in CUP was consistently overlooked. How this may have come about is not very clear. However, given that the three major systems of the human body that converge in the pelvic cavity (the reproductive, urinary and digestive systems), are traditionally managed by end-organ specialists (the gynaecologist, urologist and gastroenterologist), in ruling out potential causes of pain, they do not routinely evaluate muscles and muscle dysfunction as a possible source of CUP.[27] Despite evidence that links dysfunctional pelvic muscles with CUP, and the established benefits of muscle oriented therapies,[8, 28, 29] the muscular system as a cause of pain was, and continues to be, systematically overlooked.[7] Yet, dysfunctional pelvic muscles can be both a cause of pain and potentially impact all of the organ systems that converge in the pelvis, providing a common base for a range of comorbidities seen in CUP.

Muscles, both external and internal, together with the supporting soft tissue are integral to the proper function of the pelvis and organ related systems.[30] The bladder and the urethra are themselves muscular structures that are closely integrated with pelvic muscles and fascia, which anchor them within bony pelvis. The urethra is fused to the anterior vagina, and the endopelvic fascia attaches the bladder, urethra and vagina to the pelvic bones through ligaments and muscles that support them in a hammock like fashion.[31,32] During contraction of the levator ani muscle the pelvic organs lift and move anteriorly, and during relaxation they descend into a resting position. Failure to tighten muscles can result in loss of continence and sexual response, while failure to relax can lead to pain. When the postural and pelvic muscles are stressed, traumatized, weak or imbalanced they become a potential source of pain.[33] As a result, the major association guidelines, including those

of the European Association of Urology (EAU), American Urology Association (AUA), the International Continence Society (ICS), and the International Society for the Study of Vulvovaginal Diseases (ISSVD), advocate muscle assessments and the use of myofascial therapies as frontline interventions.[4,5,34,35] Given the need for evidence-based and mechanisms-oriented approaches to the classification and management of chronic pain,[36] pain mapping accords with the current paradigm shift.

From the spectrum of CUP conditions, the two most frequently discussed are vulvodynia and BPS. A brief review of current literature on vulvodynia and BPS is provided, and the role of myofascial variables highlighted.

Vulvodynia. The term *vulvodynia* is a descriptive term for unexplained pain in the vulvar area. Since vulvodynia is considered to be one of the chronic urogenital pain syndromes, the differential diagnoses of urethral syndrome and BPS should be considered given that the pathophysiology and prevalence is similar.[34] Anatomically the vulvar area includes the external portion of the female reproductive organs; the vestibule, hymen, urethral opening, ducts of the of the minor and major vestibular glands, labia minora and majora, the clitoris, mons pubis and the perineum.[37] The ISSVD defines vulvodynia as "vulvar discomfort, most often described as burning pain, occurring in the absence of relevant visible findings or specific, clinically identifiable, neurologic disorder".[38] The definition identifies the location of the pain, its sensory qualities and its unknown, but potentially multi-factorial nature. The quality of pain is most often reported as burning, rawness, itching, or stabbing, and the intensity of pain is rated by the majority of patients as severe.[39]

The reported prevalence of vulvodynia has varied between 4 and 16 per cent. Even though the ISSVD has provided a clear definition of vulvodynia, different methodologies of data collecting, and underreporting of the condition make its true prevalence difficult to establish.[2,34, 40-42] A large study of a cohort of vulvodynia patients showed that the prevalence peaks at age 24, and even though it affects women of all ages, it is a disorder primarily impacting young women, most of whom are in their prime reproductive years and are seeking to enter long-term relationships.[2]

The aetiology, diagnosis and management of vulvodynia have not been clearly delineated.[34] It is suggested that multiple pathologies may be involved including immune system up-regulation (increased mast cells), proliferation of local pain fibers (increased peripheral sensitivity) and contraction of the levator ani muscle in response to pain (increased pelvic muscle tone). Assessment predominantly consists of self-report questionnaires and quality of life measures. Clinical evaluations range between medical, psychological and sexual assessments. The treatment of vulvodynia is predominantly focused on prescribed medications in the absence of defined pathology. As a result, management strategies are usually ineffective.[34,43]

The most common diagnostic test is the cotton swab test, sometimes referred to as the Kaufman Q-tip touch test.[44] This swab test is used to localise the pain within the vulva area.[45, 46] The test starts in the thighs and progresses medially to the vestibule. In the vestibule testing follows the perineal clock starting at 2, going onto 4, 6, 8 and 10 o'clock positions and, if pain is present, the patient is asked to quantify the pain as mild, moderate, or severe. The Q-tip test was originally used by Friedrich (1988), who, in a landmark paper, proposed three diagnostic criteria for vestibulodynia, a localised form of vulvodynia (referred to in his paper as vulvar vestibulitis syndrome).[47] The criteria included (i) severe pain on vestibular touch or attempted entry, (ii) tenderness to Q-tip pressure localised within the vulvar vestibule, and (iii) physical findings confined to vestibular erythema of various degrees. The third criterion proved to be unreliable, but patient's self-reporting of pressure-related-pain, and pain with Q-tip test, continue to be widely used in the diagnosis of vulvodynia. From the perspective of pain mapping, two pertinent points arise in relation to vulvodynia; the diagnostic reliability of sensitivity to pressure, and variation in tenderness, as demonstrated by Friedrich's initial pain mapping of the vestibular area.

The consensus statement on definition, diagnosis and management of vulvodynia[34] recommended that in addition to medical tests, a careful urogenital examination should be performed which includes pain mapping and a functional assessment of pelvic muscles. It specifically stated that a thorough pain map should include the vestibule, perineal area and thighs. Furthermore, the pelvic muscles were to be routinely examined with bilateral palpation of the levator ani to assess the potential contribution of referred myofascial pain. Then, the lower third of the anterior vaginal wall was to be examined as part of the assessment to establish any associations with bladder-related comorbidities, which were thought to affect a third of vulvodynia patients.[34] Given the extensive literature linking dysfunctional pelvic muscles with CUP, very few clinicians have followed these recommendation and little has been mentioned about pain mapping with vulvodynia patients. The IMAP addresses each of these points in the mapping of CUP.

Several studies have compared general pain sensitivity between vulvodynia sufferers and asymptomatic controls. A study on quantitative sensory testing used pressure ranging from 0g to 1500g while assessing pain thresholds of the vulva in 23 defined locations.[48] Pain thresholds were significantly lower for vulvodynia cases than controls at all 23 sites tested, with no significant differences between pressure sensitivity at analogous right and left locations. In a study utilising EMG biofeedback and manual therapy, assessment went beyond the Q-tip test of the vestibule, and included appraisal of the role of pelvic muscle function in the aetiology and maintenance of vestibulodynia symptoms.[49] In the vestibulodynia group, muscles were found to be hypertonic but weak and inelastic, thus restricting the vaginal opening. Muscle tension was attributed to pain related guarding, with the suggestion that tension holding created increased pressure at the level of the vestibule and was responsible for perpetuating and exacerbating pain in vestibulodynia. A significant finding of the study was that 90% of women reporting pain with intercourse demonstrated pelvic floor muscle dysfunction. The researchers concluded that regardless of what the primary or adjunct therapy may involve, pelvic floor pathology must be assessed, as it can exacerbate and maintain the pain.

In studies examining the inter-relationship between vulvodynia and BPS/IC, pelvic muscle dysfunction was identified in 87% of BPS/IC cases, 60% of whom also presented with vulvodynia.[50] In a further analysis comparing BPS/IC with vulvodynia, and BPS/IC without vulvodynia, levator ani pain levels were significantly higher in the group with vulvodynia[51] Ratings of levator ani pain were derived from internal palpation of the muscle, and scored on a 10-point Visual Analogue Scale (VAS). Vulvar pain was assessed by palpating the vestibule at the 1, 3, 5, 6, 7, 9, 11 and 12 o'-clock positions. From this comparative analysis, it was evident that the two CUP syndromes, BPS/IC and vulvodynia, appear to be very closely related, and muscle tenderness was a significant feature in both. The connection between these two syndromes is further highlighted by findings that the majority of vulvodynia patients have a positive potassi-

um sensitivity test (a standard test for BPS/IC) and that vulvodynia pain may be referred from the urinary bladder.

A pilot study examining patients diagnosed with clitorodynia (a localized form of vulvodynia) found that palpation of muscles along the paraurethral area reproduced clitoral pain. It was also noted that in some patients, points in the paraurethral area reproduced symptoms of arousal, and in some instances clitoral tumescence, in a subgroup of patients suffering from persistent genital arousal disorder.[53] This study identified the pelvic muscles and paraurethral soft tissue as involved in generating symptoms and pain.

Another study looked at tactile pain sensations using functional magnetic resonance imaging (fMRI). It found augmented genital sensory processing in patients with vestibulodynia.[54] Another found increased sensitivity to touch and pain in the vestibule as a result of physiological arousal.[55] Overall, the evidence points to a generalized sensory abnormality, compounded by increased catastrophizing, hypervigilance, and fear of intercourse and non-intercourse pain.[48, 56, 57]

There are very few studies evaluating the effectiveness of therapy in the management of vulvodynia. A number of electromyographic (EMG) studies confirmed abnormal muscle tone in vulvodynia patients, marked by elevated resting baselines, poor muscle strength, and poor recruitment and recovery.[16] The dysfunctional state of the muscles was significant enough to be of diagnostic value, differentiating between controls, vulvodynia patients and women suffering from incontinence.[58] In studies specifically looking at management of vulvodynia using EMG biofeedback, therapy focused on down-training of hypertonic muscles which resulted in an 83% reduction in symptoms.[58] Very few studies exist reporting benefits of manual therapies in the management of vulvodynia. In studies where dysfunctional pelvic muscles, characterised by high muscle tone, instability, poor contraction and recovery, and tenderness to pressure were noted, soft tissue mobilization and myofascial release techniques produced complete resolution of symptoms or significant improvement.[41, 59]

In summary there is significant evidence linking increased PFM hypertonicity with hyperalgesia and chronic pain. Dysfunctional and tender PFM may be the source and cause of pain, and the vulvar and bladder areas may simply be "innocent bystanders".[60] The extent to which pain of PFM origin gives rise to central sensitization needs to be examined and studied further. It follows that if peripheral mechanisms can give rise to peripheral and central sensitization, reversing the process through the effective rehabilitation of pelvic muscles may be key to reversing the sensitization process and to the management of CUP.

Current literature discusses assessment techniques for evaluating functional pelvic muscle tone but provides no tools for the assessment of pain of pelvic muscle origin.[15] This is a significant oversight which compromises potential outcomes.[61]

Bladder Pain Syndrome. Concepts of bladder pain have varied over time, with different criteria and terminology used to describe pain and associated voiding symptoms.[62] The term Painful Bladder Syndrome/Interstitial Cystitis (PBS/IC) was introduced in 2004 at the inaugural meeting of the Multinational Interstitial Cystitis Association meeting in Rome. Within two years, at the biannual IC conference, PBS was redesignated as BPS to highlight the involvement of the end organ and the neuro-visceral (myopathic) mechanisms.[62] IC came to be considered a subgroup of the syndrome, presenting with a identifiable pathology. The term IC is being phased out, leaving BPS as the label describing chronic pain thought to arise from the bladder.

BPS is defined as "an unpleasant sensation (pain, pressure, discomfort) perceived to be related to the urinary bladder, associated with lower urinary tract symptoms of more than six weeks duration, in the absence of infection or other identifiable causes".[63] The reported prevalence of BPS ranges from 5-16%.[1] Its true prevalence is difficult to establish due to ongoing disparity of definitions, classification, methods of data collection and underreporting.[1,64] Patients present with symptoms of urge, frequency, nocturia, suprapubic pressure, and pelvic, abdominal and back pain, as well as other common comorbidities.[3,64] Though the pain involves the bladder and multiple other body sites in the same patient, it is attributable to a single disorder.[65] The severity of symptoms can vary between patients and can fluctuate widely in the same patient but the quality of life is significantly compromised among all sufferers.[66]

Patients presenting with BPS are often diagnosed on the basis of clinical symptoms, diagnostic tests, such as the potassium sensitivity test, cystoscopy with hydrodistention, and validated questionnaires. Theoretical models hypothesising the pathophysiology of BPS abound, although some clarity is emerging in relation to four potential primary processes. These have been identified as (i) GAG/proteoglycan layer disruption, (ii) immune & inflammatory system up-regulation, (iii) neurological sensitization (central and peripheral), and (iv) pelvic floor dysfunction (PFD).[67]

The link between bladder pain and pelvic muscle dysfunction has been well established.[8,23,64] One of the early studies of BPS reported pain upon digital examination of the levator ani, the sacrum, and anal region. Subsequently, different methods of treatment were compared among participants, including bladder distension, total cystectomy and ultrasound treatment of the perineal area. Of 4 cases treated with total cystectomy, pain continued to persist in 2 of the patients. The authors concluded that pain was partly bladder induced and partly caused by the painful levator muscle. Based on digital examination, the diagnosis of levator ani spasm syndrome was made.[23] However, in a recent pilot study which examined pelvic muscle tenderness and sensitivity in the paraurethral area, the symptoms of bladder pain and urge were fully reproduced in 100% of the cases upon palpation.[68] Urge to void and bladder pain were localized and shown to originate from to the paraurethral area.

Studies of urodynamics and neurostimulation also showed an association between pelvic muscles and symptoms of BPS.[64,69,70] The location of pain varied but included suprapubic, perineal, rectal, and genital pain, and in some cases was reported as radiating to thighs, ankles, and feet. The quality of pain was described as burning, pressure and stabbing, with patients reporting voiding dysfunction, including urgency, frequency, incontinence, straining-to-void, hesitancy, and urinary retention.[64] The functional inability of patients to exercise effective voluntary control over pelvic muscles was a key feature of long standing duration. The authors stated that, "pain and soreness, even inflammation, will develop if the muscle system is functionally abused," suggesting that changes within the bladder can be explained by changes in pelvic muscle function. It was noted that relaxation of pelvic muscles, whether through stimulation or biofeedback-assisted retraining, "can produce immediate relief in the muscle soreness.".[64] The authors recommend that "urologists should be encouraged to deal with pain on as conservative a level as possible" and that "if pelvic muscle dysfunction is not corrected, then the chances are against therapy being successful even with the help of medication".[64]

In a landmark study evaluating the impact of manual therapy on urgency-frequency syndrome, 42 patients who had previously undergone ineffective treatment were examined. Ineffective treatments included antibiotics (55%), urethral dilation (50%), anticholinergics (30%), diazepam (22%), tricyclic antidepressants (15%), α-blockers (12.5%), phenazopyridine hydrochloride (10%), acupuncture (10%) and surgery (5%). Each patient then participated in myofascial treatment and biofeedback-assisted retraining (8). Initial assessment consisted of intravaginal and intrarectal examination of the urinary and anal sphincter, and pelvic muscles in order to identify tightness, tenderness and pain that duplicated symptoms. Treatment consisted of compressing, stretching, strumming of internal muscles, and stretching of external muscles, with heat application to facilitate greater muscle relaxation. In women presenting with tenderness of the urinary sphincter, paraurethral tissue was repeatedly compressed against the pubic bone using increasing pressure. With repeated compressions a reduction in tenderness, softening and thinning of the contracted tissue and decreased sensitivity was noted. Trigger points that were resistant to therapy after 6-8 weeks of treatment underwent trigger point lidocaine injections. As a result of manual therapy, patients with urgency-frequency syndrome experienced an 83% improvement rated moderate to marked.

The study concluded that manual therapy, muscle relaxation techniques using biofeedback, bladder retraining and psychological therapy "arrests the neurogenic trigger leading to bladder changes, decreases central nervous system sensitivity and alleviates pain due to dysfunctional muscles".[8]

In a further study of 70 women with a diagnosis of bladder pain, on examination 87% had levator pain consistent with pelvic floor dysfunction.[50] Of these patients 71% reported dyspareunia, 50% irritable bowel syndrome (IBS) and 36% urge incontinence. Neurostimulation devices were present in 16% of the sample and this subgroup experienced significantly higher levels of pain in the levator ani muscle upon palpation. Given that treatments directed only at the bladder produced disappointing outcomes, the researchers assessed all of the pelvic floor muscles to identify levels of tenderness and pain. Pain from individual muscles was evaluated using the VAS, and pressure applied to pelvic floor muscles elicited pain in the supra-pubic area, perineum, rectum and labia.

Consistent with previous studies which identified 85% of patients as suffering from myofascial pain and hypertonic pelvic floor dysfunction,[8,64] the authors conclude that "the pelvic floor may be a significant source of pain in women with IC, making therapy directed only at the bladder less effective".[50] The authors recommended that muscle directed therapies might provide a new direction for management of BPS/IC in women.

Another study identified myofascial pain in 78.5% of patients with 67.9% having 6 or more separately identifiable trigger points.[71] The most common locations were the obturator internus, puborectalis, arcus tendinineus, and iliococcygeus muscle. The average pain scores for trigger points ranged from 5.2 - 6.8 on a ten-point scale. Suprapubic, back, anal, and vulvar pain were correlated with myofascial trigger points, with the strongest association noted with left sided myofascial trigger points. Urethral pain only correlated with left puborectalis trigger point scores. Patients with a longer duration of BPS had overall higher myofascial trigger point scores. The authors conclude that performing a musculoskeletal examination allows for proper identification of pain, which can be utilised to direct therapy with patients.

In a randomized multicentre clinical trial of physical therapy in women with BPS, those presenting with pelvic floor tenderness during vaginal examination were assigned to either a global therapeutic massage or pelvic floor myofascial physical therapy.[29] Of a total of 81 subjects, 59% reported moderate or marked improvement in the pelvic floor myofascial physical therapy group, while only 26% reported improvement in the global therapeutic massage group. It was noted that 43% in the global therapeutic massage group and 18% in the myofascial therapy group, reported no improvements as a result of treatment. Both treatment groups showed benefits in secondary outcomes of pain, urgency, frequency and quality of life. The authors conclude that myofascial therapy may be beneficial in the management of BPS and pelvic floor tenderness.

The studies on BPS focused primarily on outcomes of various manual, muscle oriented therapies. From the comparative outcomes, some conclusions have been drawn regarding potential mechanisms. Though no causal relationships have been established as yet, the pivotal role of dysfunctional muscles in conditions such as BPS has been recognised.[24]

CONCLUSION

CUP, considered by some as the "black box" of medicine,[33] affects a large number of women with a reported lifetime prevalence of 5-16%. The etiology of CUP is considered to be multifactorial, but often unknown. With the mechanisms of pain poorly understood, the outcomes of current interventions have been notoriously ineffective. Lacking an evidence base, the more invasive therapeutic options potentially cause more physical and emotional harm than good.[24] With growing evidence highlighting the inherent connection between dysfunctional muscles and CUP, greater emphasis needs to be placed on peripheral mechanisms, and less on the end-organ as a cause of symptoms. Though muscle pain is often generalised and poorly localised it can be generated by myofascial changes which not only mimic visceral pain but can induce the pain.[24] Developing a standardized assessment protocol focusing on pain of pelvic muscle origin has become a necessity. The IMAP has been developed to assist with identifying the sources of pain and in doing so providing much needed insight into the mechanisms of CUP.

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INVITED COMMENT: Pelvic Pain Trigger Points

These manuscripts explore an area in desperate need of scientific clarification; pelvic pain disorders. Jantos and colleagues have shown the usefulness of careful pain mapping of the pelvic cavity for myofascial trigger points(TPs). For years my colleagues and I have tried to establish a model for chronic muscular pain by studying muscle structures and their function in chronic pain conditions. Some years ago we published a series of studies showing that myofascial TPs produced high levels of electromyographic activity, even when the adjacent muscle was silent. We further showed that this activity was responsive to psychological stress, was not blocked by cholinergic blockade, but was blocked by alpha sympathetic blockade, establishing a close link between emotions and pain. Through the systematic identification of TP's in the pelvic cavity these two papers demonstrate that not all TP's are equal. The results show that some areas such as the paraurethral region were previously underestimated and drive most of the variance in pain reporting. This type of work, when extended, should help to greatly improve treatment strategies by integrating mind/body techniques and physical modalities.

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