Chronic Pelvic and Vulvar Pain in Women

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SUMMARY POINTS

- Peripheral generators and the central nervous system have a role in the production of visceral and somatic hypersensivities and hyperalgesias.
- More generalised symptoms often exist and efferent activity from the CNS may also be responsible for abnormal visceral and muscular function.
- It is important to dissect out the symptoms and as well as treating peripheral generators to consider interventions of the central mechanisms aimed at the cognitive behavioural aspects of the patients' condition.

Introduction

Pelvic and vulvar pain are common in adult women. Gynaecologists do not always successfully manage women where pain is the main problem and in such circumstances should consider referral to a multidisciplinary pain management service. This article will discuss the epidemiology, pathophysiology and management of these women where pain is the main symptom. The importance of a symptomatic approach to managing the distressing symptoms by a multidisciplinary team will be emphasised. The basic principles of medical management are similar to other pain problems, where attention to the neuro-muscular system is central to management. However, it is also important that the medical staff managing the problem have a full understanding of normal physiology of the urogenital system as well as an understanding of female pelvic pathophysiology. Collaboration with gynaecologists, urologists, uro-gynaecologists, uro-neurophysiologists and uro-radiologists is essential. In view of the psycho-social, sexual and behavioural problems, nurses, psychologists and physiotherapists with specific training in managing these symptoms in urogenital related female pain should be involved in patient care.

Definitions

Chronic pelvic pain is defined as pain in the lower abdomen or pelvis, of at least six months duration, occurring continuously or intermittently, not associated exclusively with menstruation or sexual intercourse. Vulvodynia is defined as a chronic disorder in women, characterised by provoked or constant vulvar pain of varying intensity without obvious concomitant clinical pathology. Two subsets of vulvodynia are recognised: generalised and localised pain subtypes, the latter currently referred to as vestibulodynia or vestibulitis.

Epidemiology of chronic pelvic and vulval or perineal pain in women

A US based telephone survey interviewed respondents aged 18-50 years[1]. Seventeen thousand nine hundred and twenty seven households were contacted, 5325 women agreed to participate, and of these 925 reported pelvic pain of at least 6 months' duration, including pain within the past 3 months. Having excluded those pregnant or postmenopausal and those with only cycle related pain, 773/5263 (14.7%) were identified as suffering from chronic pelvic pain.

A British population survey used a postal sample of 2,016 women randomly selected from the Oxfordshire Health Authority register of 141,400 women aged 18-49 years[2]. Prevalence of chronic pelvic pain was 483/2016 (24.0%). There were significant associations between chronic pelvic pain and the specific symptoms of dysmenorrhoea and dyspareunia.

In a UK Primary Care setting, data from 284,162 women aged 12-70 years who had a general practice contact in 1991 were analysed to identify subsequent contacts over the following five years[3]. The monthly prevalence rate was 21.5/1000 and the monthly incidence rate was 1.58/1000. The authors highlighted the burden of disease represented by these data, pointing out the comparability with migraine, back pain and asthma in primary care.

The presence of symptoms does not necessarily reflect health-care seeking. This was highlighted in the UK population survey described above: of 483 women with chronic pelvic pain, 195 (40.4%) had not sought a medical consultation, 127 (26.3%) reported a past consultation and 139 (28.8%) reported a recent consultation for pain. The US telephone survey also drew attention to the large numbers of women with symptoms who do not seek medical attention. Seventy-

five per cent of this sample had not seen a healthcare provider in the previous three months.

With regard to vulval pain, the single available population based sample survey was undertaken in Boston, USA[4]. Census records were used to sample 4915 women age 18 years to 64 years using a self administered questionnaire. Approximately 16% of respondents reported histories of chronic burning, knife like pain, or pain on contact that lasted for at least 3 months or longer. These symptoms were present in nearly 7% at the time of the survey. As with the pelvic pain surveys discussed above, a substantial proportion of women reported that they had not sought treatment. Of those who did seek health-care, 60% saw three or more doctors but a positive diagnosis was frequently absent.

Pathophysiology of chronic pelvic and perineal pain in women

There is poor correlation between identifiable pathological processes, the chronicity and severity of pain and the impact of symptoms on quality of life. Essentially the mechanisms can be described under the general headings of nociceptive, neuropathic and central mechanisms. There is a general move to a greater emphasis on the role of the central nervous system and many conditions with peripheral generators will have a central component complicating the picture.

Peripheral generators associated with chronic pelvic pain and vulvar pain in women

Endometriosis

Endometriosis is a condition affecting women predominantly in the reproductive age group and characterised by the presence of endometrial glands and stroma outside the endometrial cavity. The condition is thought to arise mainly by implantation of endometrial tissue following retrograde menstruation via the Fallopian tubes. It presents a clinical spectrum, with endometriotic deposits sometimes observed at laparoscopy in the absence of symptoms or tissue damage, through subfertility apparently associated with endometriosis but in the absence of pain, to chronic pain associated with disabling pain symptoms and often gross damage to the pelvic organs. In a series of asymptomatic multiparous patients undergoing sterilisation the prevalence was 26/ 3384 (3.7 %)[5].

Infection and inflammation

In the clinical setting, it is a frequent observation that chronic pain can develop following infection that has apparently resolved, which may point to processes such as the activation of silent afferents.

Adhesions

Adhesions may form after inflammation due to sepsis, following surgery and may be associated with endometriosis. Innervation of adhesion tissue has been described[6] . However, any causal relationship with pain symptoms is unclear.

Myofascial pain

Pelvic muscle spasm may be a feature of chronic pelvic pain[7] and it is often difficult to establish whether this is the primary problem or is a natural response to the presence of pelvic tenderness arising from another condition such as endometriosis. Spasm of the levators certainly contributes to additional distressing symptoms such as urinary retention, constipation and dyspareunia.

Vascular congestion

A vascular pain mechanism, pelvic venous congestion, has been proposed as a cause of chronic pelvic pain. Relevant endothelially mediated vascular pain mechanisms have been demonstrated in human studies. However, whether there is an association between pain symptoms and particular radiological or ultrasound appearances is unclear[8].

Hormonal factors

Hormonal factors are important mediators of nociception both in animal and human experimental models. Variations of pain threshold and behaviour have been demonstrated in relation to sex hormone exposure in rats at different stages of the oestrous cycle. Meta-analysis of studies of women undergoing experimental exposure to different pain modalities at different stages of the menstrual cycle show a variation in pain sensitivity[9].

Vulval pain

Trauma, whether through childbirth or injury to the pelvis is often suspected as a causal factor in the pathogenesis, however, such episodes may be coincidental. Nerve conduction studies to assess damage to the pudendal innervation have proved of marginal value in clinical practice. A syndrome of entrapment of the pudendal nerve in Alcock's canal has been described[10]. It is not clear whether neuropathic processes are involved in this proposed mechanism, what the impact of surgical intervention is on the conduction properties of afferent fibres and whether central sensitisation is a feature of the clinical presentation.

Immune and inflammatory mediators

These have been considered in the pathogenesis of vulvar vestibulitis or vestibulodynia. As with endometriosis it is unclear whether the primary problem is inflammation or the associated nociceptive processes. Evidence for the latter is the observation in a functional MRI study of increased cerebral neuronal activity during painful vulval vestibular stimulation among patients with vulval vestibulitis syndrome compared with that seen among controls[11]. Previous causative hypotheses around human papillomavirus or herpes genitalis infection have been discounted.

Central mechanisms/central sensitisation

There is no doubt that central sensitisation plays an important role in the Pelvic Pain Syndromes. The result of the now well defined central changes is that stimuli not normally perceived may become perceived (such as a constant urge to void or a sensation of rectal fullness), and sensations normally perceived during normal function may become painful where they are not normally so. As well as changes perceived

in the viscera, musculoskeletal hypersensitivities may occur. As a consequence of these central changes, there are three types of visceral hyperalgesia which may be relevant to the clinical presentation of patients with pelvic pain[12]: (a) visceral hyperalgesia: hyperalgesia of a viscus from inflammation and/or excess stimulation of the same viscera e.g. irritable bowel syndrome; (b) referred hyperalgesia from viscera: hyperalgesia of somatic tissues in the area of referred pain from viscera, e.g. trigger points in body wall tissues; (c) viscero-visceral hyperalgesia: hyperalgesia of a viscus rendered clinically manifest by a painful condition of another viscera e.g. exacerbation of urinary colic pain in patients with urinary calculus plus dysmenorrhoea.

Table 1. Classification of peripheral causes of chronic pelvic pain

Inflammatory, infective	Chronic salpingitis
Inflammatory, noninfective	Endometriosis Vulvodynia with dermatosis
Mechanical	Uterine retroversion Adhesions
Functional	Pelvic congestion Irritable bowel syndrome
Neuropathic	Postsurgical Dysaesthetic vulvodynia Vulval vestibulodynia
Musculoskeletal	Pelvic floor myalgia Abdominal and pelvic trigger points Postural muscle strain

As well as changes in sensory perception, central nervous system efferent activity may be abnormal. This may produce functional abnormalities such as bladder hyperreflexia or atonic bowel function. These mechanisms are very important in producing the diversity of symptoms seen in patients.

Finally, the central nervous system may not only be of major importance in extending the pain condition from the original peripheral generator to other pelvic organs and local somatic systems, but plays a major part in extending symptoms beyond that. Proposed mechanisms include neuro-endocrine and neuro-immune interactions.

All the above mechanisms must be considered when a patient is referred to a pain management service. Once patients with chronic pelvic and vulvar pain reach the stage of assessment in a pain clinic, it appears that the pattern of symptom impact is similar to that seen in other chronic painful conditions in terms of severity and lifestyle impact. Best clinical practice suggests an approach that recognises both the physical, functional and psychosocial dimensions as appropriate.

Risk factors

Consideration of risk factors for chronic pelvic and perineal pain necessitates a recognition of the complex interplay of pathophysiological processes discussed above, reproductive history and status, the psychological mediators of the impact of adverse life experiences, and co-morbidity such as depression. Typically in the literature the direction of causality is difficult to establish. In the early literature, an attempt was made to separate physical from psychological processes. In more recent studies, the similar prevalence of mood disturbance in those with and without a specific cause for pelvic pain has been established[13].

Abuse as a child, whether sexual or physical, may predispose to chronic pelvic pain. Direct causality cannot be inferred as many individuals who have suffered such abuse do not suffer from chronic pain in later life. A comparison of adverse experiences was made with three groups, two of patients from a tertiary referral multidisciplinary clinic and a group without pain[14]. The two groups of pain patients were those with pelvic pain, and those with other types of pain. Among the pelvic pain group 12 (40%) reported sexual abuse compared to five (17%) in each of the two comparison groups. Experience of physical abuse was the same in all groups.

In vulval vestibulitis syndrome (VVS), researchers have undertaken numerous studies attempting to elucidate the significance of infection as a risk factor for vestibular pain. The role of candidiasis has not been confirmed. Recent investigations, which corroborated referring physician statements or prior laboratory results with patient reports, found VVS risk to be associated with a history of bacterial vaginosis, candida albicans, pelvic inflammatory disease, trichomoniasis, and vulvar dysplasia[15].

Latthe et al[16] undertook a systematic review of forty-eight factors predisposing women to chronic pelvic pain, dyspareunia and dysmenorrhoea. Factors associated in the analysis with non-cyclical pelvic pain were drug or alcohol abuse, miscarriage, prolonged menstrual flow, pelvic inflammatory disease, previous caesarean section, pelvic pathology, abuse (sexual and physical), and psychological co-morbidity. While the review establishes associations of interest, one needs to bear in mind that the assembled data are drawn from very diverse settings ranging from population surveys to tertiary medical facilities[16]. Furthermore some of the associations may be self-fulfilling: the diagnosis of 'pelvic inflammatory disease' is often given to women presenting with pain despite the absence of objective features of infection, and patients with recurrent or 'unexplained' pain may be given a psychological or psychiatric label by default.

Treatments supported by RCT evidence (Table 2)

There are a number of randomised controlled trials in the management of chronic pelvic pain and vulvar pain in women. Unfortunately, the quality of the studies is not always the best. There is room for much more comprehensive studies which should include measures

Table 2. Evidence for Treatments

Interventions supported by RCT evidence	Laparoscopic surgery for endometriosis Vestibulectomy Pudendal nerve release Multidisciplinary management Counselling with ultrasound scanning Hormonal therapy for ovarian suppression
Interventions with conflicting evidence from RCTs	PSN and LUNA
Treatments supported by non-randomised studies but shown non-effective in RCTs:	Adhesiolysis
Treatments supported by non-randomised studies	Biofeedback Lignocaine ointment for vulvar vestibulitis Tricyclic antidepressants Gabapentin

of function as well as pain scores in accordance with IMMPACT guidelines[17].

Laparoscopic surgery for endometriosis

An early randomised comparison of laparoscopic surgery for mild or moderate endometriosis showed evidence of benefit. However, many patients will continue to complain of pain despite what is considered a surgical success. I am not sure that this statement helps as this study did show an improvement in pain and it is what usually happens now.

Surgery versus cognitive therapy and biofeedback for pelvic muscle relaxation for vulval vestibulitis syndrome.

A twelve week trial of either cognitive behaviour therapy or biofeedback for pelvic muscle relaxation using electromyography was compared with vestibulectomy in 78 women. Post treatment and six month follow up results were superior in the vestibulectomy group, although scores improved in all the groups[18].

Surgery versus conservative management for pudendal nerve entrapment

Sixteen women were randomised to each of two groups, either a group who underwent surgery to decompress the pudendal nerve or a group treated by conservative management. On an intention to treat basis, 50% of the intervention group were improved compared to 6.2% of the control group at three months. Analyzing by actual treatment, 71.4% of the surgery group were improved at 12 months compared to 13.3% of the control group. At four years 8 of those randomized to surgery were still improved (50%). The authors conclude that while the intervention is worthwhile, other modalities of treatment are likely to be needed in addition[19].

Multidisciplinary management

The use of a multidisciplinary approach in the treatment of women with chronic pelvic pain led to a positive outcome in a self-rating scale (OR = 4.15, 95% CI = 1.91-8.99, n = 106) and in daily activity but not in pain scores, by comparison with those receiving 'conventional care'[20].

Counselling supported by ultrasound scanning

Patients with pelvic pain are often anxious about the potential for disease. A randomized comparison of expectant management versus counselling supported by ultrasound scanning to demonstrate normal anatomy showed benefit for the intervention both in terms of pain scores (OR = 6.77, 95% CI = 2.83-16.19, n = 90) and mood[21].

Hormonal therapy for ovarian suppression

Progestogen (medroxyprogesterone acetate) was effective in reducing chronic pelvic pain after four months' treatment as reflected in pain scores (OR 2.64, 95% CI 1.33 to 5.25, n=146) and a self-rating scale (OR 6.81, 95% CI 1.83 to 25.3, n=44), but benefit was not sustained nine months post treatment[22]. Medroxyprogesterone acetate plus psychotherapy was effective in terms of pain scores (OR 3.94, 95% CI 1.2 to 12.96, n= 43) but not in the self rating scale at the end of treatment. Benefit was not sustained post treatment. Venography scores for pelvic congestion, symptom and examination scores, mood and sexual function were improved to a greater extent one year after treatment with goserelin compared to progestogen[23].

Treatments with conflicting evidence from randomized studies

Presacral neurectomy and laparoscopic uterine nerve ablation

Presacral neurectomy (PSN) and laparoscopic uterine nerve ablation (LUNA) are both surgical procedures that involve the disruption of sensory nerve afferents that carry pain stimuli from the pelvis. In LUNA, the utero-sacral ligaments are transacted close to their insertion at the cervix, thus interrupting part of the Lee-Frankenhauser nerve plexus. In PSN, the pre-sacral nerve plexus is isolated and cut proximally and distally. Complications associated with LUNA are rare; there have been isolated cases of uterine prolapse and bladder dysfunction. PSN has been associated with more serious complications such as haematoma formation, major vessel injury, constipation and bladder dysfunction,

though these complications are rare in experienced hands. A number of studies have suggested benefit from LUNA and PSN for primary and secondary dysmenorrhea, including randomized trials[24,25]. However, a large multi-centre study examining the effectiveness of LUNA in pelvic pain (n=487) has recently finished, with indications of negative findings with respect to pain relief (KS Khan, personal communication).

Treatments supported by non-randomised studies but shown non-effective in RCTs.

Adhesiolysis

A number of non-randomised studies have reported that division of adhesions is useful in the treatment of pelvic pain[26,27]. Overall, a meta-analysis showed that out of over 600 patients with pelvic pain, 76% would obtain relief from adhesiolysis. However, the two available RCTs are not supportive. The outcome in women undergoing adhesiolysis via laparotomy was not different to that in women who did not undergo surgery on any outcome measure (OR = 1.54, 95% CI = 0.81-2.93, n = 148). However, the small sub-group with dense vascularised adhesions did show a significant benefit for surgery (OR for self rating scale = 16.59, 95% CI = 2.16-127.2, n = 15)[28].

Treatments supported by non-randomised studies

Lidocaine for vulval vestibulitis syndrome (vestibulodynia)

Topical 5% lidocaine ointment applied overnight resulted in significant improvement in a group of 61 participants observed over a mean of seven weeks. Seventy six percent reported being able to have intercourse at the end of the study, compared to 36% before treatment. Fifty seven percent achieved a 50% or greater improvement in symptoms[29].

Tricyclic and SNRI Antidepressants

Tricyclic antidepressants and SNRI antidepressants have been shown to be helpful in neuropathic pain. The question remains as to whether these drugs relieve pelvic pain and vulvodynia. Although there have been anecdotal reports of benefit in small groups of patients with vulvodynia [30,31], there are no published randomized controlled studies to clarify the position.

Anticonvulsants

Gabapentin has been shown to be effective for the treatment of neuropathic pain. A small series of patients with vulvodynia reported a favourable response to gabapentin, but no controlled trials have been published[32] . One small trial has suggested a synergistic effect between amitriptyline and gabapentin[33].

Biofeedback

Twenty –nine patients with moderate to severe vulvar vestibulitis were given electromyographic assessment of the pelvic floor muscles. The patients were then given a portable home trainer biofeedback device and instructions to perform biofeedback assisted pelvic floor muscle rehabilitation exercises. Patients were evaluated on a monthly basis for vestibulodynia and dyspareunia. Fifty one point seven percent demonstrated markedly reduced introital tenderness and 93.3% were able to resume sexual activity without discomfort[34].

Practice advice

Patients with chronic pelvic pain can present a considerable challenge. Often the Cartesian model of pain has been emphasised by the GP and the Gynaecologist. Patients are often confused that the laparoscopy has identified no pathology or the pathology seen has been treated and yet the pain continues. Thirty per cent of patients who undergo a hysterectomy and bilateral salpingoopherectomy for pelvic pain have no resolution of their pain after surgery. The message that an investigative laparoscopy may not reveal the cause of the pain must be communicated by the GP at the first consultation so that the patient realises that this investigation may not cure her symptoms.

Whilst not every patient may need a pain clinic assessment nor indeed a multidisciplinary appointment, the important psychological influences on pain should always be considered. A multidisciplinary approach to pain management offering medical treatment alongside psychological management would seem to be an optimal treatment option for patients with chronic pelvic pain.

The consequences of chronic pelvic pain are now well established with severe physical disability being among them. There is evidence from a number of studies that physiotherapy behavioural interventions combined with psychological interventions may significantly reduce disability.

Conclusions

Understanding pain mechanisms in female pelvic pain has moved on significantly over the last few years. As well as peripheral generators, the role of the central nervous system is now well established as having a role in the production of visceral and somatic hypersensiivities and hyperalgesias. The CNS, via its efferent activity, may also be responsible for abnormal visceral and muscular function as the more generalised symptoms often seen. Research has often focused on peripheral mechanisms, with some benefit on occasions but often with limited results. It is important to dissect out the symptoms and as well as treating peripheral generators to consider interventions of the central mechanisms and aimed at the cognitive behavioural aspects of the patients condition.

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