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Idiopathic chronic orchialgia: Evaluation and management options

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Abstract
Chronic orchialgia is a disease with several probable aetiologies, including both genitourinary and non-genitourinary disorders. Chronic orchialgia is deemed idiopathic in 25-50% of instances, despite a wide differential diagnosis. In the lack of explicit protocols for the assessment and management of chronic orchialgia, it is crucial to conduct a thorough history and physical examinations to exclude all potential aetiologies of chronic orchialgia. Utilising auxiliary imaging techniques, especially scrotal ultrasound, may aid in achieving a conclusive diagnosis when the physical examination is ambiguous. The first approach to treating persistent orchialgia is medical care. Invasive treatment for orchialgia ought to be regarded following the ineffectiveness of medicinal treatments.

Keywords: Idiopathic chronic orchialgia, evaluation, management

Introduction
Chronic orchialgia, or chronic scrotal content pain (CSP), occurs in around 2.5-4.8% of all urological clinic consultations. This issue may be exasperating to manage for both patients and urologists. Comprehensive physical examinations and medical history may often reveal several prevalent explanations for testicular discomfort. CSP is often characterised by discomfort lasting over three months, with other potential explanations including infections, testicular tumour, torsion, abscess, hydrocele, and transferred pain from upper tract issues excluded. Individuals might have idiopathic pain [1].
In cases of idiopathic chronic orchialgia, assessment may be perplexing due to the often-unidentified underlying cause, and conservative treatment typically proves ineffective, making this ailment difficult to identify and manage. First line treatments are typically conservative therapy; however, when these fail, more invasive treatments are used [2, 3].
The first assessment of persistent groin and scrotal discomfort starts with a comprehensive history and physical examinations to clarify the underlying disease. Numerous therapeutic regimens have been suggested without a conclusive agreement; still, the majority start with conservative interventions and medical care before progressing to more invasive therapies [4].

Chronic orchialgia
Chronic orchialgia is characterised by intermittent or persistent testicular discomfort that lasts for three months or more, substantially disrupting the patient's everyday activities. More recent definitions include pain localized to the epididymis, spermatic cord, and para-testicular structures in addition to testicular pain [5].
Men suffering from chronic orchialgia are mostly in their mid-to late-30s, and this incidence is rising. It is the cause of 2.5% to 4.8% [6].

Etiologies of orchialgia
Orchialgia can be attributed to identified organic causes that may be acute like acute infections, trauma, Intermittent torsion and torsion of a testicular appendage, along with

chronic conditions such as chronic epididymitis, low back strain, varicoceles, vasculitis (e.g., Henoch-Schonlein purpura), entrapment of nerves from hernia-related surgeries, post-vasectomy pain syndrome, perineural fibrosis, pelvic floor dysfunction, or referred pain from another site, liik a ureteral stone [7].

Infrequent aetiologies including diabetic neuropathy, retroperitoneal neoplasms, abdominal aortic aneurysms, interstitial cystitis, peritonitis, pelvic musculoskeletal diseases, amiodarone-induced epididymal inflammation, polyarteritis nodosa, and osteitis pubis [8].

Referred pain may arise from an indirect inguinal hernia, mid-ureteral stone, aortic or common iliac artery aneurysms, lower back problems, and nerve entrapment caused by perineural fibrosis. Furthermore, it should be acknowledged that up to 50% of individuals might exhibit idiopathic aetiology [9].

Pathogenesis of orchialgia

After a prolonged period of painful stimuli, evidence exists that central and peripheral nervous systems experience some sort of modification that causes the pain receptors to become more sensitive [10]. Additional evidence indicates that, during the regeneration of peripheral nerves post-injury, axons may mistakenly reconnect within the dorsal spinal cord, leading to a situation where depolarisation noticed by one axon could erroneously transmit its signal to an inappropriate proximal neurone, resulting in benign stimuli being perceived as painful [11].

Moreover pain is sometimes thought to be related to neurogenic inflammation, which is caused by noxious stimuli activating unmyelinated sensory neurons and secreting neuropeptides like substance P and calcitonin gene-related peptide [12].

Non neuropathic pain can be caused by a persistent somatic nociceptive source, such as keloid formation, recurrent hernias, metallic vasectomy clips, chronic infections of the prostate or genitalia, or mass effects from aberrant anatomy [13]. evidence indicates that supplementary pathophysiological mechanisms also have an impact in the development of chronic pain, such as neuroplasticity (the nervous system's capacity to modify its functioning in response to internal or external stimuli following injuries), afferent hypersensitivity, pain centralisation (that develops when the CNS becomes sensitised to pain, resulting in a diminished pain threshold), and Wallerian degeneration, that, if exist, can predisposed a patient to failure of therapy even when a nociceptive or neuropathic source has been determined and rectified [14].

Wallerian degeneration and trifecta nerve complex

The active process of anterograde degeneration occurs at the axon's distal end due to a nerve injury. It transpires between 7 and 21 days following the injury. It delineates a condition characterised by the self-destruction of the axon of nerve cells. It stimulates and employs cytokines, neutrophils, and macrophages, resulting in neuronal hypersensitivity and persistent pain. This procedure eliminates inhibitory debris, therefore facilitating axonal regeneration and repair. The overall outcome is that patients experience pain at reduced stimulation levels in regions impacted by neuronal Wallerian degeneration (a lowered pain threshold) [15].

Parekattil *et al.* identified a significant concentration of nerves in the spermatic cord exhibiting Wallerian degeneration in individuals with persistent orchalgia, so corroborating this hypothesis [16].

This hypersensitivity presents as allodynia (perception of pain from a usually non-painful stimulus) or hyperalgesia (an excessive reaction beyond conventional expectations). Hyperalgesia and allodynia arise from sensitisation within the central or peripheral nerve systems. These alterations are referred to as brain plasticity and may lead to the sense of pain even months post-injury recovery [17].

This aligns with a two-hit hypothesis, whereby an underlying inflammatory or genetic mechanism induces Wallerian degeneration and subsequently hypersensitivity of the genitofemoral and ilioinguinal nerves. Chronic neuropathic pain can results in this area due to other provoking events that cause irritation of these nerves as trauma and surgery [16].

In 2013, Parekattil *et al.* described Wallerian degeneration in the trifecta nerve complex in men with chronic orchialgia in three primary sites including the perivasal tissues/vasal sheath, cremasteric muscle fibers and posterior peri-arterial lipomatous tissue; suggesting that these nerves might explain the impact of the MDSC procedure. The trifecta nerve complex represents the rationale of MDSC as a therapeutic option for idiopathic chronic orchialgia, which also allows a more targeted technique [16].

Evaluation of Chronic orchialgia

History and physical examination

History must have precise facts about the pain, particularly its exact location, characteristics, duration, exacerbating factors, onset acuity, and any radiation to adjacent organs or regions. Prolonged sitting and constipation may intensify discomfort in patients with idiopathic chronic orchialgia, whereas patients with interstitial cystitis typically experience suprapubic pain related to bladder functioning [18].

The physical examinations must concentrate on the scrotum and genitalia; assessing the patient in both sitting and standing positions is advantageous. Commencing with the asymptomatic side. Each segment of the testicle (epididymis, testis, and vas) must be meticulously assessed for tenderness upon probing, oedema, and atypical nodules. A digital rectal examination (DRE) is necessary to assess potential prostatitis and irregular pelvic floor muscle tension. Efforts ought to be directed towards identifying the precise anatomical site of the discomfort, while excluding any potential referred causes like ureteral calculi, infections, or spinal issues (lumbar disc herniation) [18].

Investigations

Laboratory

A urine analysis and urine culture and sensitivity ought to be routinely conducted. Semen culture ought to be acquired in certain patients [18, 19].

Imaging

To rule out structural abnormalities, scrotal doppler ultrasonography is the principal diagnostic tool and primary imaging study for chronic orchialgia. Because ureteral calculi, inguinal hernias or aneurysms, may be the cause of or contributing factors to the pain, A computed tomography (CT) scan on the abdomen and pelvic ought to be considered in certain cases. Spinal magnetic resonance imaging (MRI) is not typically advised, except in certain instances

involving individuals with concurrent back or hip discomfort [20].

Cystoscopy, retrograde pyelography, voiding cystourethrography, and intravenous pyelography are often unhelpful and not routinely advised. The assessment of persistent orchialgia may be complex, sometimes resulting in patients seeing many doctors. The typical patient is reported to undergo around 4.7 to 7.2 diagnostic examinations and 1.6 surgical procedures [21].

Management of Chronic Idiopathic Orchialgia

Non-surgical Options: Medications: Analegesics include NSAIDs are typically prescribed for no less than 30 days. After taking them properly, recurrence rates can reach 50%. Narcotic analgesics ought to be avoided, with the possible exception of rare instances of breakthrough pain [22].

Tricyclic antidepressants function by preventing serotonin and norepinephrine from being reabsorbed in the brain. It is believed that their analgesic impacts result from inhibition of L-type and sodium calcium channel blockers in the spinal cord's dorsal horn. While secondary amines like desipramine and nortriptyline are less effective for treating neuropathic pain, tertiary amines like amitriptyline and clomipramine are more likely to cause postural hypotension and cause more sedation. They are usually taken as a single dosage at bedtime, and it usually takes two to four weeks, but it can take up to eight weeks for their effectiveness to show results. Amitriptyline 25 mg single dose at bedtime is the standard dosage [23].

If tricyclic medication fails following 30 days, the subsequent conservative treatment option is to include an anticonvulsant, like gabapentin (Neurontin), at a dosage of 300 mg twice day. They function by altering N-type calcium channels, that considerably influence pain fibres. Pelvic floor physical therapy is beneficial for individuals with pelvic floor muscular dysfunction or discernible myofascial trigger points. In appropriately chosen individuals, around 50% have reported alleviation of their pain following twelve sessions [24].

Spermatic cord block

It is advised to perform the SCB as the next step before any invasive or permanent surgical operations. SCB serves both diagnostic and therapeutic purposes. This is conducted by injecting 20 mL of 0.5% bupivacaine with a 27-gauge needle in the absence of epinephrine. It's possible to add steroids or not. For example, Yamamoto *et al.* documented a cohort of three individuals with CSP who attained sustained analgesia after receiving a SCB with 1% lidocaine and 40 mg methylprednisolone. Bupivacaine inhibits fast voltage-gated sodium channels critical for neuronal conduction and possesses a prolonged duration of action (4-8 hours) relative to lidocaine (~2 hours) [21].

Pain improvement after the cord block suggests that afferent input from the genitofemoral, ilioinguinal, and spermatic nerves are at least partially responsible for pain transmission [25]. Patients with pain improvement lasting more than 4 hours are more likely to benefit from surgical management [26].

Individuals achieving above 90% pain reduction may be eligible for recurrent blocks at intervals of up to two weeks. If the injection fails to alleviate pain, it will not be administered again. If the SCB achieves less than 50% efficacy in alleviating orchialgia, contemplate a potential misdiagnosis. A reassessment of the patient, along with a meticulous evaluation of his laboratory results and imaging, is recommended. A superior reaction to the SCB correlates with improved outcomes in microsurgical denervation of the spermatic cord (MDSC). Surgical surgery is warranted if the SCB achieves a minimum of 50% efficacy in alleviating the orchialgia [26].

Surgical Options

Microsurgical denervation of spermatic cord

In contrast, if the pain is localized to the testicle, spermatic cord, or more diffuse throughout the scrotal contents, MDSC is the surgical therapeutic of choice. For idiopathic chronic orchialgia that is not improving with conservative therapy, MDSC has emerged as an alternative option. It has been reported that 76.5% of patients who have had a positive responses to a spermatic cord block showed a significant pain improvement after MDSC [27].

The standard MDSC

Concerning incision site selection, there are primarily two methodologies: the conventional historical inguinal technique or a sub-inguinal approach. Both ideas have been stated with considerable success, and each has its own advantages. An incision in the lower inguinal region is performed to reveal the external inguinal ring. The ilioinguinal nerve is often seen emerging laterally from the external inguinal ring, with a 2 to 3 cm section removed and ligated. The proximal end is then concealed behind the external inguinal ring to reduce the likelihood of neuroma development [28].

The spermatic cord is exposed and delivered out of the wound during the standard MDSC procedure. In the scrotum, the testicle is typically left in situ. A Penrose drain or tongue depressor is then positioned underneath to stabilize and support the spermatic cord [28].

The testicular and cremasteric arteries are recognized and isolated by tiny vessel loops, and they are found by carefully dissecting the cord under a microscope. The arteries, including the artery of the vases, differ if existent, are preserved. The peri-vasal fascia is excised for 2 cm because to its abundance of afferent neurones. The vasal artery is preserved (29).

Cremasteric muscle fibers are cut, with care taken to avoid injuries to the cremasteric artery. All of the spermatic cord's nerves are to be transected while the vases differ, the testicular, and cremasteric arteries are preserved, along with a few lymphatics that are left in place to lessen the risk of development of a post-operative hydrocele. The ilioinguinal nerve and the testicular veins are additionally exercised. Notwithstanding this, patient reports of loss of sensation in the distribution region of the ilioinguinal nerve are few [29].

Approximately 70% to 80% of men get total symptom improvement, while an additional 10% to 20% attain partial pain alleviation with MDSC. In patients who had previously received surgical intervention, MDSC offered total pain relief to 50% of them. The entire alleviation of pain following this operation may need up to three months; however, 40% had total pain reduction immediately following the MDSC. The surgery was performed with the da Vinci robot, yielding comparable outcomes. Potential problems include hydrocele development (risk less than 1%), infections of wounds, incisional haematomas, and atrophy of the testes (1% risk) [29].

Targeted MDSC

Based on certain neuro-anatomic studies, the technique has developed to permit for more focused denervation of specific spermatic cord locations. Three distinct areas of the spermatic cord have been identified as sites of Wallerian degeneration (16):

- Peri-vasal tissues and vasal sheath.
- Cremasteric muscle fibers.
- Posterior cord lipomatous tissues.

For this reason, the internal spermatic sheath is entirely preserved in the targeted MDSC technique. This significantly simplifies the conventional MDSC process [16]. Targeted MDSC also has the advantage of a shorter surgical time, a less technically difficult procedure, and possibly a lower risk to the cord structures that ought to be preserved [30].

Prior to surgery, patients should be counseled on possible treatment failure with persistent pain due to accessory pudendal nerve fibers, central sensitization, and malingering [31, 32]. In cases of bilateral CSP that are appropriate for MDSC, the operation should be performed on the more severely affected side first to avoid the risk of prolonged postoperative scrotal swelling. In the work of 14 participants with bilateral CSP who underwent unilateral MDSC, 12 (86%) did not require additional surgery on the contralateral side. Despite the fact that the mechanism is still unclear, it is possible that pain may be referred from the more severely affected side to the contralateral scrotal contents, so- called neural cross-talk [27].

In contrast, orchiectomy (surgical removal of the entire testicle and spermatic cord) is a final consideration when medical and surgical approaches have been unsuccessful. This should be reserved as a treatment of last option, because preserving the testicle optimizes testosterone production and fertility potential. Moreover, failure rates as high as 30% to 80% have been stated after orchiectomy [33].

The type of surgical procedure that should be performed depends on the nature and location of the pain. In individual with pain isolated to the epididymis, surgical removal of the epididymis alone (known as *epididymectomy*) may improve or resolve pain in as many as 75% to 90% [34].

Minimally invasive interventions

Ultrasound guided targeted microcryoablation.

Research indicates that nerves are susceptible to freezing damage and may become desensitised when subjected to temperatures between −15 and −20 °C. An ultrasonic probe is used to locate the spermatic cord at the external inguinal ring. An Endocare TM 1.7 mm spherical ice the cryoprobe is percutaneously inserted into the perispermatic tissue on the medial and lateral sides of the spermatic cord, guided by ultrasonography at the external inguinal ring. The tissue is subjected to cooling at −40 °C for a duration of 90 seconds. The cryoprobe is thawed passively and extracted [35].

Botox injection

Botulinum-A toxin has demonstrated the ability to regulate the release of neuropeptides (substance P and calcitonin gene-related peptide), resulting in the suppression of neurogenic inflammation and chronic pain. Neurogenic inflammation resulting in Wallerian degeneration of the neural fibres in the spermatic cord is being proposed as a potential pathological reason for pain in individuals with persistent orchalgia. One hundred units of Botox have been mixed in 10 cc of saline. The solution is administered medially and laterally to the spermatic cord at the external inguinal ring. This is conducted with the subject under conscious anaesthesia [36].

AmnioFix injection

Injectable dehydrated amniotic/chorionic membrane allograft (AmnioFix®) is a product obtained from human amniotic membrane. AmnioFix has shown efficacy in reducing scar tissue development, alleviating inflammation, and promoting healing. Individuals with orchialgia have neurogenic inflammation around the spermatic cord. Due to its anti-inflammatory qualities, it might offer an additional noninvasive therapy alternative for challenging orchialgia patients. The injectable version of AmnioFix is administered medially and laterally to the spermatic cord at the external inguinal ring level [36].

Targeted Robotic Assisted Microsurgical Denervation of the Spermatic Cord (TRMDSC)

It is a minimal-invasive and feasible therapeutic choice among individuals with CSP that targets the trifecta nerve complex. The robotic surgical platform is used, the cremasteric fibres, perivascular tissues (maintaining the vasal artery), and the posterior adipose tissues are severed. All testicular arteries, the internal spermatic sheath (including lymphatics and veins), and the vas are preserved. The chord is then encased in biowrap to reduce the likelihood of postoperative scarring and neuroma development [16].

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