



GLOBAL INTERSTITIAL CYSTITIS
BLADDER PAIN SOCIETY

Volume 7 Issue 7 || July 2025

GIBS NEWSLETTER

Urethral Burning – A Urogynecological Enigma

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Relevance

Urethral burning is a frequent yet diagnostically complex complaint among women of varying ages. While urinary tract infection (UTI) is often suspected, many women experience persistent symptoms despite sterile urine cultures. Misdiagnosis can lead to unnecessary antibiotic therapy or invasive procedures without symptom relief, contributing to patient frustration, psychological distress, and reduced quality of life. Greater awareness of non-infectious causes and appropriate management strategies is needed among clinicians (1, 2).

Case Example

A 43-year-old perimenopausal woman presents with a 3-month history of persistent urethral burning, worsened after intercourse. She has been treated empirically with multiple antibiotics with no relief. Urine culture is negative; gynecological exam reveals atrophic vulvovaginal tissues and high pelvic muscle tone. The final diagnosis is hypoestrogenic urethritis and pelvic floor muscle tension. She responds well to topical estrogen and pelvic floor physiotherapy.

Possible Causes

The differential diagnosis of urethral burning in women is broad and includes:

- **Hormonal deficiency:** Common during perimenopause and menopause; estrogen deficiency causes atrophic changes in urethral and vaginal mucosa, leading to dryness and irritation (1).
- **Bladder pain syndrome/interstitial cystitis (BPS/IC):** Presents with suprapubic pain, urgency, frequency, and burning in the absence of infection (1, 2).
- **Urethral pain syndrome (UPS):** Persistent or recurrent urethral pain without proven infection or other pathology (9).

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- **Vulvodynia and vestibulodynia:** Chronic vulvar pain, especially localized at the vestibule, can mimic urethral discomfort (3).
- **Pelvic floor myalgia:** Pelvic floor muscle hypertonicity or trigger points may cause referred urethral pain (4, 7).
- **Mechanical and chemical irritants:** Bicycle riding, soaps, hygiene products, sexual activity, and tight clothing may provoke urethral burning.
- **Iatrogenic trauma:** Catheterization, cystoscopy, or urethral dilation may cause microtrauma or inflammation (8).
- **Contact dermatitis or allergic reactions:** Reactions to pads, lubricants, or laundry detergents.

Treatment traditions – insight from Sweden

A 2019 Swedish study (Ivarsson et al.) surveying 99 clinics treating UPS revealed 19 different treatment modalities. Most common were topical corticosteroids and estrogens, with increasing use of high--potency corticosteroids since 2006. Despite sterile cultures, more than half of clinics still prescribed antibiotics - highlighting ongoing challenges in differentiating pain from infection (9).

Diagnostic evaluation

Accurate diagnosis of urethral burning requires a structured and stepwise approach. Although urinary tract infection is often suspected initially, a broader differential must be explored when symptoms persist despite negative cultures.

Basic testing begins with **urinalysis and urine culture**, which help exclude bacterial infection as a primary cause (1). Additionally, **nucleic acid amplification tests (NAATs) or swabs for sexually transmitted infections (STIs)** such as *Chlamydia trachomatis* and *Neisseria gonorrhoeae* are recommended, particularly in sexually active patients (2).

In women with symptoms suggestive of vaginal atrophy, **vaginal pH testing** and **microbiological evaluation using vaginal smears** can provide insight into the local hormonal

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and microbial environment (1, 4). During physical examination, careful **inspection of the vulva** is crucial to identify signs of atrophy, dermatoses (e.g., lichen sclerosus), or mucosal trauma.

When vulvodynia or neuropathic pain is suspected, a **cotton-swab test** can be employed to assess for **allodynia or hyperalgesia** in the vestibular and periurethral region (3). Evaluation of the **pelvic floor muscles** through palpation may reveal increased tone, tenderness, or trigger points indicative of pelvic floor dysfunction (4, 5, 7).

In selected patients, **assessment of the urethra** is warranted to exclude **periurethral lesions** such as Skene's gland cysts, diverticula, or neoplasms (8). When structural or intravesical pathology is suspected, **cystoscopy** may be indicated to visualize the urethral mucosa, bladder trigone, and bladder lining (1, 9).

Taken together, these diagnostic tools allow for a comprehensive evaluation of potential infectious, hormonal, neurogenic, musculoskeletal, or anatomical contributors to urethral pain, and help clinicians avoid unnecessary treatment while targeting the underlying cause.

Treatment approaches

Management should be stepwise and personalized, based on etiology and evidence:

Local Therapy:

- o *Estrogen*: Vaginal estrogen improves epithelial integrity in hypoestrogenism (1).
- o *Corticosteroids*: Topical corticosteroids (e.g., clobetasol-propionate) reduce inflammation; widely used in Sweden for UPS (9).
- o *Anesthetics*: Lidocaine gel or urethral instillation eases local pain (9).

- **Systemic pharmacotherapy**: Neuropathic and chronic pain may respond to tricyclic antidepressants (e.g., amitriptyline), SNRIs (e.g., duloxetine), and gabapentinoids (e.g., gabapentin) (2, 4).
- **Physical therapy**: Pelvic floor rehabilitation, myofascial release, and biofeedback are essential for patients with pelvic floor dysfunction or myalgia (4, 5, 7).
- **Lifestyle and dietary modifications**: Avoid irritants; gentle hygiene; sitz baths; avoiding caffeine, alcohol, and spicy food in BPS/IC (2).
- **Psychological support**: Cognitive-behavioral therapy or counseling is valuable for chronic pain with mood comorbidities (3).
- **Interventional options**: Refractory cases may benefit from urethral dilation, bladder instillations, or botulinum toxin injections under specialist guidance (1, 8).

Conclusion

Urethral burning in women is a multifactorial symptom that requires careful diagnostic assessment and a personalized, often conservative, management strategy. A comprehensive, multidisciplinary approach is key to reducing misdiagnosis, avoiding overtreatment, and improving patient-reported outcomes.



DRUG SNIPPET FOR TREATMENT OF ICBPS



Pregabalin

Mechanism of Action

Precise mechanism of action unknown but is a GABA analogue that binds to a subunit of voltage-gated calcium channels in CNS; does not affect sodium channels, opiate receptors, or cyclooxygenase enzyme activity; interactions with descending noradrenergic and serotonergic pathways originating from the brain stem appear to reduce neuropathic pain transmission from spinal cord.

Dosage

Initial : 75 mg PO q12hr (150 mg/day)

Pharmacokinetics

Absorption

Bioavailability : >90%
Peak plasma concentration : 3.2 mcg/mL
(75 mg capsule BID)
Peak plasma time, fasting : 1.5 hr
Peak plasma time, with food : 3 hr
Steady-state is achieved within 24-48 hr

Distribution

Protein bound: None
Although there are no data in humans, pregabalin has been shown to cross the blood brain barrier in mice, rats, and monkeys

Metabolism

Minimal
Elimination
Half-life : 6.3 hr
Clearance : 67-80.9 mL/min
Excretion : Urine

Maintenance: May increase to 150mg q12hr after 1 week as needed; recommended dose is 300-400 mg/day
Owing to dose-dependent adverse reactions, doses >450 mg/day are not recommended

Take orally with or without food When discontinuing treatment, taper gradually over a minimum of 1 week

Adverse Reaction

Peripheral edema may occur; higher frequencies of weight gain and peripheral edema were observed in patients taking both pregabalin and a thiazolidinedione antidiabetic agent compared to patients taking either drug alone; monitor these patients for possible exacerbation of congestive heart failure symptoms when using pregabalin. Pregabalin may cause dizziness and somnolence; inform patients that pregabalin may impair their ability to perform tasks such as driving or operating machinery; concomitant use of pregabalin with other central nervous system (CNS) depressants may exacerbate these effects; for patients 1 month to less than 4 years of age, somnolence includes related terms lethargy, sluggishness, and hypersomnia.

Weight gain may occur; long-term cardiovascular effects of pregabalin-associated weight gain are unknown. Symptoms including, insomnia, nausea, headache, anxiety, and diarrhea were reported following abrupt or rapid discontinuation of treatment; increased seizure frequency may occur in patients with seizure disorders and have rapid discontinued treatment; taper pregabalin gradually over a minimum of 1 week rather than discontinuing the drug abruptly.