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Narrative Review

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Interstitial cystitis/bladder pain syndrome: a narrative and scoping review of current evidence

Abstract

Objective: To appraise available evidence on the pathophysiology, diagnosis, and management of interstitial cystitis/bladder pain syndrome (IC/BPS) and to outline an evidence-based approach to clinical care.

Method: This is a narrative and scoping review, structured following the general framework of the PRISMA-ScR guidelines for scoping reviews. PubMed and Google Scholar were searched using key phrases related to interstitial cystitis, bladder pain syndrome, diagnosis, and treatment. Thirty articles, including observational studies, clinical guidelines, systematic reviews, and relevant basic science articles published between 2000 and 2025, were selected after title/abstract screening and full-text review. Articles focusing on acute cystitis or exclusively pediatric populations were excluded. The literature search was iterative and recursive. Relevant data on presentation, pathophysiology, diagnostic approaches, and management of IC/BPS were extracted through content analysis, and a narrative synthesis was performed for each aspect of the disease.

Results: IC/BPS is a chronic, debilitating condition affecting predominantly women (5:1 to 10:1 female-to-male ratio) with a significant impact on quality of life. Multiple pathophysiological mechanisms contribute, including urothelial barrier dysfunction, neurogenic inflammation, central sensitization, and microbiome alterations. Two distinct phenotypes exist: Hunner lesion IC (5-10% of cases) and non-Hunner lesion IC. Diagnosis remains clinical, based on symptom assessment and exclusion of other pathologies. Management requires a multimodal approach including behavioral modifications, oral medications, intravesical therapies, neuromodulation, and, in refractory cases, surgical interventions. Most patients experience symptomatic improvement with appropriate treatment, though complete remission remains rare.

Conclusion: IC/BPS is indeed a complex chronic pain syndrome. Individualized and multimodal therapeutic management requires early recognition, phenotypic stratification, and multidisciplinary care to optimize outcomes and improve the quality of life of affected patients.

Keywords: Interstitial cystitis/bladder pain syndrome (IC/BPS), hunner lesion, neurogenic inflammation, urinary microbiome, intravesical therapy, neuromodulation, multimodal management

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Ahemdi Chrive Ahmed,[1] Moncef El Midaoui, Youssef Maachi,[1] Babty Mouftah,[2] Amine Slaoui,[1] Tariq Karmouni,[1] Abdellatif Koutani,[1] Khalid El Khader[1]

[1]Department of Urology B, CHU Ibn Sina, Faculty of Medicine and Pharmacy, Mohamed V University, Morocco
[2]Urology Department, Friendship Hospital, Mauritania

Correspondence: Dr. Ahemdi Chrive Ahmed, Department of Urology B, CHU Ibn Sina, Faculty of Medicine and Pharmacy of Rabat, Mohamed V University, Rabat, Morocco

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Introduction

Interstitial cystitis/bladder pain syndrome is one of the biggest diagnostic and therapeutic challenges in modern urology. It is a chronic disease defined by symptoms of pelvic pain related to the bladder, frequency, and nocturia without evidence of infection or other pathology. The condition predominantly affects females, with a female-to-male ratio of 5:1 to 10:1.[1]

Prevalence estimates range from 50 to 500 per 100,000 inhabitants, reflecting the challenges in diagnosis and the heterogeneity of the disease.[2] The impact on quality of life is profound and comparable to that seen in severe chronic conditions such as rheumatoid arthritis and inflammatory bowel disease.[3]

Patients typically report significant urinary frequency, up to 40 to 60 voids per day in severe cases, prominent nocturia, and long-standing pelvic pain that worsens with bladder filling. These symptoms lead to significant physical, psychological, social, and occupational disability. Table 1 lists key clinical features.

Table 1 Key Clinical features of IC/BPS

Clinical domain	Manifestations
Pain	Suprapubic, pelvic, or perineal; bladder-associated; improves with voiding
Urinary Symptoms	Frequency (>8/day), urgency, nocturia (>2/night), small volumes
Duration	Chronic symptoms ≥6 months
QOL Impact	Sleep disruption, sexual dysfunction, depression, work impairment
Associated Conditions	IBS, fibromyalgia, chronic fatigue, pelvic floor dysfunction

Table 2 Diagnostic evaluation of IC/BPS

Component	Specific measures	Purpose
History	Symptoms, duration, QOL impact	Establish pattern, severity
Physical Exam	Abdominal, pelvic, pelvic floor	Identify tenderness, dysfunction
Laboratory	Urinalysis, culture, cytology	Exclude infection, malignancy
Procedures	Cystoscopy, biopsy	Phenotype classification
Questionnaires	ICSI/ICPI, voiding diary	Quantify symptoms

Table 3 Therapeutic Approach to IC/BPS

Treatment level	Interventions	Response rate
First-Line	Education, diet, stress management, PT	40-60%
Second-Line	PPS, amitriptyline, antihistamines	30-50%
Third-Line	GAG replacement, DMSO, heparin	40-70%
Fourth-Line	Neuromodulation, BoNT-A, hydrodistension	50-70%
Fifth-Line	Hunner lesion treatment	70-90% (Hunner only)
Sixth-Line	Major surgery	40-70%

Methods

This is a narrative and scoping review designed to synthesize current evidence on IC/BPS. Given the heterogeneous nature of the available literature, the review followed the general framework outlined by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) to guide the search strategy, article selection, and reporting structure, while retaining a narrative rather than a fully systematic synthesis of findings.

Literature search

PubMed and Google Scholar databases were searched using key phrases including "interstitial cystitis," "bladder pain syndrome," "IC/BPS," "diagnosis," "treatment," and "pathophysiology," combined with Boolean operators. The search was restricted to January 2000-December 2025, a period broad enough to capture both the foundational guidelines and mechanistic studies that established current diagnostic frameworks and the most recent evidence.

Selection criteria

Inclusion: clinical guidelines from major societies (AUA, EAU, CUA, ESSIC), systematic reviews, observational studies, basic science articles, English-language publications, adult populations.

Exclusion

Acute bacterial cystitis, pediatric-only studies, non-English articles, case reports and conference abstracts without accompanying full-text data.

Data analysis

The literature review process was iterative and recursive. Content analysis extracted data on presentation, pathophysiology, diagnosis, and management. A narrative synthesis was constructed, organizing findings thematically.

Results

Epidemiology and pathophysiology

IC/BPS predominantly affects women, with a prevalence of 50-500 per 100,000.[1,2] The pathophysiology involves multifactorial components such as urothelial barrier dysfunction, neurogenic inflammation, central sensitization, and changes in the microbiome.

Urothelial barrier dysfunction

The dominant theory implicates a deficiency in the protective glycosaminoglycan layer covering the urothelium.[4] This allows the urinary irritants potassium and urea to penetrate the submucosa, triggering inflammation. Some studies have demonstrated a reduction in tight junction proteins and increased urothelial permeability.[5]

Neurogenic inflammation and central sensitization

Histamine, tryptase, and cytokines are released by mast cell activation, facilitating inflammation. Bladder hyperalgesia is enhanced by increased neuropeptides,[6,7] such as substance P and CGRP. Functional neuroimaging has shown altered brain activation during bladder filling, supporting the presence of central sensitization mechanisms.[6] Segmental hyperalgesia provides further evidence for changes in pain processing at the spinal cord level.[7]

Microbiome alterations

Culture-independent studies demonstrate changes in bacterial composition among IC/BPS patients, including reduced microbial diversity and altered bacterial taxa.[8,9] Whether these changes are causative or consequential remains undetermined.

Phenotypic classification

Two distinct phenotypes have been described.[10,11]

- Hunner Lesion IC (5-10%): Characteristic inflammatory lesions are seen on cystoscopy. Responds favorably to lesion-directed therapies (fulguration/resection), with 70-90% response rates.
- Non-Hunner Lesion IC (90-95%): Normal cystoscopy or glomerulations post-distension. Likely involves more prominent central sensitization, requiring multimodal systemic approaches.

Diagnosis

Diagnosis remains clinical, based on history, symptom assessment, and exclusion of alternative pathologies.[12] Core criteria include:

- Chronic pelvic pain (≥6 months) perceived as bladder-related
- Associated urinary symptoms (frequency, urgency, nocturia)

- Absence of infection
- Exclusion of other pathologies (cancer, stones, neurological disorders)

Diagnostic evaluation

Mandatory investigations.[13,14]

- Detailed history and physical examination
- Urinalysis and culture (to exclude infection)
- Urinary cytology (if risk factors for malignancy)

Frequently performed

- Cystoscopy ± hydrodistension
- Bladder biopsy (to exclude other pathology and identify Hunner lesions)
- Post-void residual measurement

Validated questionnaires (O'Leary-Sant ICSI/ICPI, voiding diary) facilitate symptom quantification and monitoring.[2]

Treatment approaches

Management requires individualized, multimodal strategies following stepwise progression.

First-line conservative therapies

Behavioral modifications: Patient education, bladder training, stress reduction, dietary modification (avoiding citrus, tomatoes, caffeine, alcohol, spices). Provides relief in 50-70% of patients.[15]

Pelvic floor physical therapy: Manual trigger point release, myofascial techniques, biofeedback. Particularly valuable in patients with pelvic floor dysfunction.[16]

Oral pharmacotherapy

Pentosan Polysulfate Sodium (PPS): The only FDA-approved oral medication, 100 mg three times daily. Clinical improvement after 3-6 months in 30-40% of patients. Recent safety concerns regarding maculopathy require ophthalmological monitoring.[17]

Tricyclic antidepressants: Amitriptyline 10-75 mg nightly. Modulates pain and reduces urgency/frequency; 50-60% of patients report improvement.[15]

Antihistamines: Hydroxyzine 25-75 mg nightly. Targets mast cell-mediated inflammation.[2]

Intravesical therapies

GAG-Replacement: Hyaluronic acid or chondroitin sulfate instillations, weekly for 4-8 weeks then monthly maintenance. Response rates of 40-70%.[18]

DMSO: 50 mL of 50% solution every 1-2 weeks for 6-8 treatments; 50-70% report improvement.[19]

Combination cocktails: Lidocaine, heparin, and sodium bicarbonate provide rapid symptomatic relief.[20]

Neuromodulation

Sacral neuromodulation: Modulates neural pathways controlling bladder function and pain; success rates of 50-70%.[21]

Posterior tibial nerve stimulation: A less invasive alternative, delivered as weekly 30-minute sessions for 12 weeks.[22]

Procedural interventions

Botulinum Toxin A: Intradetrusor injection (100-200 units), with effects lasting 6-12 months; may cause temporary urinary retention.[23]

Hydrodistension: Provides temporary relief in 20-50% of patients, with variable duration.[24]

Hunner lesion treatment: Fulguration/resection produces 70-90% response rates in Hunner lesion patients.

Surgical interventions

Reserved for severe, refractory cases. Options include augmentation cystoplasty or cystectomy with diversion. Pain resolution is inconsistent (40-70%) owing to central sensitization.[15,20]

Emerging therapies

Biologics targeting specific cytokines (TNF-α inhibitors, anti-NGF antibodies) show promise. Stem cell therapies and platelet-rich plasma instillations are under investigation.[25] Medical cannabinoids may offer benefit through modulation of the endocannabinoid system.[15] Novel microbiome-targeted interventions, including probiotics, are also being explored.

Multimodal management and comorbidities

Treatment selection is guided by phenotypic characterization.[26,27] Optimal management combines multiple interventions addressing different mechanisms,[28] an approach that benefits from multidisciplinary collaboration among urologists, pain specialists, physical therapists, and psychologists.[21,26]

IC/BPS often overlaps with irritable bowel syndrome (30-50%), fibromyalgia (20-30%), chronic fatigue syndrome, and other chronic pain conditions.[3] This clustering suggests shared pathophysiological mechanisms related to central sensitization.[27]

Prognosis

Roughly 50% of patients experience clinically significant improvement, but few achieve complete remission.[2] Predictors of good response include shorter symptom duration, presence of Hunner lesions, and absence of comorbid chronic pain conditions. Impairment in quality of life is significant, with increased rates of depression and anxiety.[20] Sexual dysfunction is nearly universal, requiring targeted interventions.[26]

Guidelines

Multiple societies have published evidence-based guidelines. AUA guidelines propose a six-tier algorithm progressing from conservative to invasive interventions. EAU guidelines emphasize phenotypic classification and multidisciplinary management.[28] CUA guidelines advocate stepwise, multimodal approaches.[29] ESSIC has standardized terminology and diagnostic criteria.[10]

Discussion

IC/BPS represents a multifactorial disorder involving peripheral bladder pathology and central nervous system dysfunction. The relative contribution of each varies among individuals and may evolve with disease duration. Early disease may reflect bladder-based pathology, whereas chronic disease increasingly involves central sensitization that becomes self-sustaining.[4,6,7]

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These factors make diagnosis challenging because of the lack of a specific biomarker that can reliably identify the condition,[27] so IC/BPS remains a diagnosis of exclusion, which contributes to delays in diagnosis. Candidate urinary and serum biomarkers under investigation include antiproliferative factor, inflammatory cytokines such as IL-6, TNF-α, and MCP-1, nerve growth factor, and markers of oxidative stress; several correlate with symptom severity or with the Hunner versus non-Hunner phenotype in individual studies.[31,32] However, none has yet achieved the sensitivity, specificity, or cross-study reproducibility required for routine clinical use, and current guidelines do not recommend any single biomarker as a diagnostic or prognostic tool. Key limitations include small and heterogeneous study cohorts, the absence of standardized assay protocols, limited external validation, and overlap of candidate markers with other chronic pain and inflammatory conditions.[32] Research in this area continues to evolve, particularly around subtype-specific candidates such as BAFF and CXCL10 for Hunner lesion IC and NGF for non-Hunner disease; validated biomarkers could ultimately improve diagnostic efficiency and enable earlier intervention that may help prevent the development of central sensitization.

Recognition of phenotypic heterogeneity informs contemporary management.[1] The Hunner versus non-Hunner distinction has important therapeutic implications, and future refinement incorporating biomarkers or molecular profiles could enable precision-medicine approaches.

Central sensitization appears increasingly important, especially in chronic cases. It explains symptom persistence despite bladder-directed treatment, pain expansion beyond the bladder, and the association with other centralized pain syndromes.[6,7] Recognition of this mechanism shifts treatment toward neuromodulation, centrally-acting medications, and psychological therapies.

Recent research into the microbiome calls conventional understanding into question, at least to some extent.[8,9] Although compositional changes are consistently shown, causality has not been established, and microbiome modulation may represent a potential therapeutic target for further study.

Impaired quality of life extends beyond symptoms alone.[3,20] Comprehensive care should cover psychological, social, sexual, and occupational domains, and quality-of-life patient-reported outcomes should extend symptom scores, rather than merely complement them, when assessing patients with the disease.[21,26]

Current evidence is limited by several factors: randomized controlled trials suffer from heterogeneous populations, variable definitions, high placebo responses, and difficulty in defining endpoints. Meta-analyses are further hindered by study heterogeneity, long-term outcome data remain limited, and comparative-effectiveness research is largely absent.

IC/BPS places a high economic burden through both direct medical costs and indirect costs such as disability and reduced productivity.[26] Improving care should focus on increasing clinician awareness, ensuring timely access to specialist referral, providing multidisciplinary models of care, and offering patient support resources.

Future research priorities include biomarker discovery using omics technologies, deeper mechanistic understanding, phenotypic refinement, novel therapeutics, comparative-effectiveness studies, and long-term outcome data.[30]

Figure 1 synthesizes these threads, mapping the convergence of urothelial barrier dysfunction, neurogenic inflammation, central sensitization, and microbiome alteration onto the clinical picture of pelvic pain, urinary frequency, and impaired quality of life described throughout this review. By visualizing how peripheral bladder pathology and central nervous system mechanisms intersect, the figure underscores the rationale for phenotype-directed, multimodal management rather than therapy aimed at a single causal pathway.

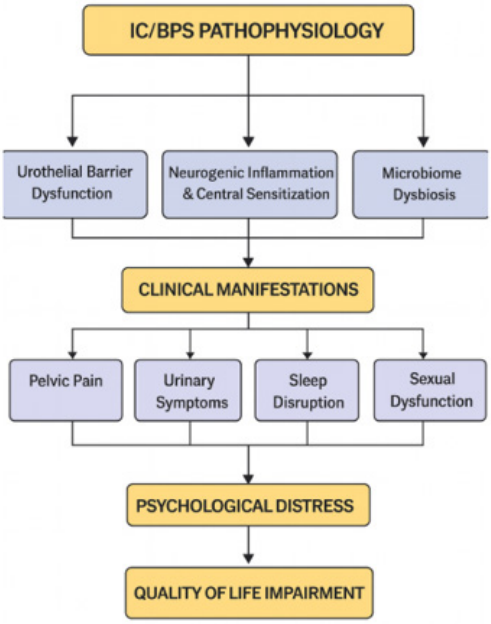

Figure 1 Pathophysiology and clinical impact of IC/BPS.

Conclusion

Interstitial cystitis/bladder pain syndrome is a complex chronic pain condition that requires comprehensive and individualized management. The disease involves various mechanisms such as urothelial barrier dysfunction, neurogenic inflammation, central sensitization, and possibly microbiome alterations. The two phenotypes, Hunner and non-Hunner, show different pathophysiologies and treatment responses.

Diagnosis remains clinical, based on characteristic symptoms and the exclusion of alternative pathologies. No single validated biomarker is yet available for routine clinical use, which contributes to delayed diagnosis.

Management requires multimodal approaches that combine behavioral modifications, pharmacotherapy, intravesical treatments, neuromodulation, and, when appropriate, procedural interventions. Therapy should be phenotype-specific, severity-based, comorbidity-adjusted, and centered on patient preference. About 50% of patients achieve symptomatic improvement, although complete remission remains uncommon.

Comprehensive care should address bladder symptoms, associated comorbidities, psychological wellbeing, sexual function, and overall quality of life. Multidisciplinary collaboration among urologists, pain specialists, physical therapists, psychologists, and primary care physicians optimizes outcomes.

Future research should focus on biomarker discovery, mechanistic understanding, novel therapies, comparative-effectiveness studies, and

long-term outcomes. Recognition of IC/BPS as a substantial source of chronic pelvic pain, improved clinician awareness, accessible specialist referral, and evidence-based multimodal management can substantially improve outcomes and quality of life for affected patients.

Conflicts of interest

There is no conflict of interest between the authors of this work.

Authors contribution

Chrive Ahmed Ahemdi analyzed and interpreted the patient data regarding the subject and were major contributors in writing the manuscript Moncef El Midaoui, Babty Mouftah, Maachi Youssef, Amine Slaoui, Tariq Karmouni, Khalid El Khader, Abdellatif Koutani and Ahmed Ibn Attya Andaloussi read and approved the final manuscript.

Ethics approval and consent to participate

The ethics committee of the Faculty of Medicine of Rabat has given us its agreement. Informed and verbal consent to participate in the study was provided by our patients. The reference number is not applicable.

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