Interstitial cystitis

Etiology, diagnosis, and treatment

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abstract

OBJECTIVE To review current knowledge about the epidemiology, etiology, diagnosis, and treatment of interstitial cystitis, with special emphasis on management of this condition by family physicians.

QUALITY OF EVIDENCE Articles were identified through MEDLINE and review of abstracts presented at Urology and Interstitial Cystitis meetings during the last decade. Recent reviews were further searched for additional studies and trials. Data were summarized from large epidemiologic studies. Etiologic theories were extracted from current concepts and reviews of scientific studies. Diagnostic criteria described in this review are based on clinical interpretation of National Institutes of Health (NIH) research guidelines, interpretation of data from the NIH Interstitial Cystitis Cohort Study, and recent evidence on use of the potassium sensitivity test. Treatment suggestions are based on six randomized placebo-controlled clinical treatment trials and best available clinical data.

MAIN MESSAGE Interstitial cystitis affects about 0.01% to 0.5% of women. Its etiology is unknown, but might involve microbiologic, immunologic, mucosal, neurogenic, and other yet undefined agents. The diagnosis of interstitial cystitis is a diagnosis of exclusion. It is impossible to provide a purely evidence-based treatment strategy, but review of available evidence suggests that conservative supportive therapy (including diet modification); oral treatment with pentosan polysulfate, amitriptyline, or hydroxyzine; and intravesical treatments with heparinlike medications, dimethyl sulfoxide, or BCG vaccine could benefit some patients.

CONCLUSION Family physicians should have an understanding of interstitial cystitis and be able to make a diagnosis and formulate an evidence-based treatment strategy for their patients.

résumé

OBJECTIF Passer en revue les connaissances actuelles concernant l'épidémiologie, l'étiologie, le diagnostic et le traitement de la cystite interstitielle, en insistant particulièrement sur la prise en charge de cet état pathologique par les médecins de famille.

QUALITÉ DES DONNÉES Des articles ont été identifiés au moyen de MEDLINE et d'une revue des résumés présentés à des conférences sur l'urologie et la cystite interstitielle au cours de la dernière décennie. Les analyses récentes ont fait l'objet d'une recherche plus approfondie pour trouver des études et des essais additionnels. Les données tirées d'importantes études épidémiologiques ont fait l'objet d'une synthèse. Les théories étiologiques ont été extraites des concepts courants et des critiques des études scientifiques. Les critères de diagnostic décrits dans la présente étude se fondent sur l'interprétation clinique des lignes directrices de la recherche des Instituts nationaux de la santé (NIH), sur l'interprétation des données tirées de l'étude de cohortes sur la cystite interstitielle (NIH) et des récentes données probantes sur l'utilisation des épreuves de sensibilité au potassium. Les suggestions de traitement se fondent sur six essais cliniques aléatoires de traitement contrôlé contre placebo et les meilleures données cliniques disponibles.

PRINCIPAL MESSAGE La cystite interstitielle touche environ de 0,01% à 0,5% des femmes. Son étiologie est inconnue, mais elle pourrait mettre en jeu des agents microbiologiques, immunologiques, muqueux, neurogènes et d'autres agents encore non définis. Le diagnostic de la cystite interstitielle en est un d'exclusion. Il est impossible de proposer une stratégie de thérapie purement fondée sur des données probantes, mais une étude des données probantes disponibles suggère qu'une thérapie conservatrice de soutien (y compris une modification du régime alimentaire); un traitement au polysulfate de pentosanne, à l'amitriptyline ou à l'hydroxyzine par voie orale; et les traitements intravésicaux avec des médicaments apparentés à l'héparine, au diméthyl-sulfoxyde ou le vaccin BCG pourraient apporter des avantages à certains patients.

CONCLUSION Les médecins de famille devraient avoir une certaine compréhension de la cystite interstitielle et être en mesure de diagnostiquer et de formuler une stratégie thérapeutique fondée sur des données probantes pour leurs patients.

This article has been peer reviewed.

Cet article a fait l'objet d'une évaluation externe.

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Interstitial cystitis (IC) is a clinical syndrome characterized by urinary frequency and urgency, nocturia, and suprapubic (bladder and pelvic) pressure and pain without an identifiable cause, such as bacterial infection.[1] This condition has perplexed and frustrated physicians for decades.[2-4] Research has increased awareness of the disease, standardized our diagnostic approach, and improved our management strategies. Family physicians should understand this syndrome and should be willing to diagnose and treat patients with IC.

Quality of evidence

Articles were identified through a MEDLINE search from 1979 to 1999 and through review of abstracts presented at the American Urological Association and National Institutes of Health (NIH) interstitial cystitis meetings. Recent articles were further searched for additional studies and trials. Data were summarized from large epidemiologic studies. Etiologic theories were extracted from current concepts and recent reviews of scientific studies. Diagnostic criteria described in this review are based on clinical interpretation of the NIH research guidelines, interpretation of data from the NIH Interstitial Cystitis Cohort Study, and recent evidence on use of the potassium sensitivity (KCl) test.

No diagnostic studies had control groups. Only six randomized or controlled clinical treatment trials were identified. Treatment suggestions presented in this review are based on best available clinical data, including data from these trials.

Epidemiology

The true prevalence of the constellation of symptoms we call IC is unknown; estimates vary from 10 cases per 100 000 people in Finland[5] to a range of 30 cases[6] to 510[7] cases per 100 000 people in the United States. In the Netherlands,[8] prevalence was estimated at eight to 16 cases per 100 000 female patients seen by urologists.

Women are much more frequently diagnosed with IC than men (10:1). Results of a recent questionnaire mailed to participants in the US Nurses Health Study I and II suggested a prevalence in these women of between 52 and 67 per 100 000.[9] These widely varying estimates are due to the substantial differences in methodology and diagnostic criteria employed. There is, however, no doubt that it is a common enough condition that primary care physicians will see it relatively frequently.

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Etiology and pathogenesis

The etiology and pathogenesis of IC is still undetermined. During the last two decades, however, several attractive (but still hypothetical) hypotheses have been suggested, most having at least some sound scientific basis.

Glycosaminoglycan layer. A deficiency in the surface glycosaminoglycan-mucin layer allows increased amounts of some unspecified toxic substance (ie, potassium) to permeate the bladder wall, causing inflammation and pain.[10-12] While this theory remains popular, it has not yet been proved.[13,14] Perhaps glycosaminoglycan alteration reflects the inflammatory process rather than being the cause of it.[15-20]

Infection. Urinary tract infection (UTI) and IC have many similar clinical characteristics, except that uropathogenic bacteria are cultured from patients with UTIs and these patients subsequently have a far better response to antimicrobial therapy than patients with IC. Patients with IC might have a history of UTIs[21] and probably suffer the same incidence of UTIs as normal female patients. Interstitial cystitis could be due to small numbers of organisms in the urine (low-count bacteriuria), fastidious organisms not thought to be usually associated with clinical symptoms, or even cryptic nonculturable microorganisms.[22] Evidence, however, tends to discount this very attractive theory.[23,24]

Immunologic response. Interstitial cystitis is either a nonspecific or bladder-specific antibody or a cell-mediated autoimmune disorder,[25,26] or related to mast cell activity.[27] The lack of specificity seen in subsequent immunologic studies of IC makes it more likely that such observed responses are secondary to damage to bladder tissues rather than a primary cause of the inflammation,[28] and no evidence supports the theory that IC is a primary mast cell disorder.[29]

Neurogenic cause. A possible neurogenic etiology for IC has evolved from histopathologic examination of the bladder showing neural proliferation and chronic perineuritis in the bladder wall. This could cause the cardinal symptoms of IC: pain and frequency and urgency of urination.[30]

Diagnosis

We once believed that only urologists could recognize and subsequently diagnose the clinical syndrome we have termed IC. Many family physicians are just now becoming familiar enough with IC to make a clinical diagnosis. Patients diagnosed with IC present with a remarkably similar constellation of symptoms (although they vary in frequency and intensity): urinary frequency and urgency, nocturia, suprapubic pain, and perhaps dyspareunia.[4,31,32]

Since IC was first described by Hunner in 1914,[33] there has been confusion as to the exact criteria upon which to make such a diagnosis. In the late 1980s, the NIH convened workshops to develop diagnostic criteria for a research definition of IC.[34] For the next decade, the diagnostic criteria established by this definition stimulated a great deal of research in a homogenous group of patients.[35] Diagnostic criteria for inclusion in an NIH-funded study of IC were based on cystoscopic findings (glomerulation, or the very rare classic Hunner's ulcer) associated with bladder pain or urinary urgency or both and exclusion of other conditions that might cause similar symptoms (ie, UTIs). **Table 1**[3,34] shows inclusion and exclusion criteria.

Patients with IC who meet NIH inclusion criteria are a fairly homogenous population that has proven effective for clinical studies, such that comparative results can be analyzed. Without doubt, however, many patients diagnosed with IC do not meet the strict NIH criteria.[36] In fact, a recent report even showed that the so-called characteristic cystoscopic findings of IC are frequently found in women without symptoms of IC.[37]

Is cystoscopy necessary for making a diagnosis of IC? Since there is no criterion standard cystoscopic findings of clinical IC (Hunner's ulcers are very rarely seen and the glomerulations traditionally described do not seem to be specific for IC), cystoscopy is unnecessary in most cases of suspected IC.[38] In fact, the NIH does not require cystoscopy any more for patients to be included in the Interstitial Cystitis Data Base Study.[36]

Is cystoscopy required to rule out other more life-threatening medical conditions, such as bladder cancer? No evidence in the literature suggests that bladder cancer is any more common in patients with IC. Cystoscopy should be considered for the same indications that it is for patients who do not have IC: acute onset or change of symptoms, hematuria as a dominant symptom, urinary cytology suggesting malignancy, or an unclear diagnosis.

Table 1. Research definition* of interstitial cystitis

INCLUSION CRITERIA
Cystoscopy findings of glomerulation or classic Hunner's ulcer
Symptoms of bladder pain or urinary urgency
EXCLUSION CRITERIA
Younger than 18 years
Urinary frequency while awake less than eight times a day
Nocturia less than twice a night
Maximal bladder capacity greater than 350 mL while awake
Absence of intense urge to void with bladder filled to 100 mL of gas or 150 mL of water at medium filling rate during cystometry
Involuntary bladder contractions on cystometrogram at medium filling rate
Symptoms persisting for less than 9 months
Symptoms relieved by antimicrobial agents (antibiotics, urinary antiseptics), anticholinergic drugs, or antispasmodics
Urinary tract or prostatic infection in the past 3 months
Active general herpes or vaginitis
Urethral diverticulum
Uterine, cervical, vaginal, or urethral cancer within the past 5 years
History of cyclophosphamide or chemical cystitis, or tuberculosis or radiation cystitis
History of bladder tumours (benign or malignant)

**This abbreviated definition derived by the National Institutes of Health (NIH) and the National Institute of Arthritis, Diabetes, Digestive and Kidney Disorders is a research definition only (for inclusion in clinical trials), not necessarily applicable to family physician–oriented diagnoses.*[3]

Some have suggested that a KCl test is better for diagnosing IC. The KCl test can purportedly detect abnormal epithelial permeability.[39] This test, however, does not appear to discriminate between patients with IC and normal patients.[40] It might detect subgroups of patients who might respond to different modes of therapy.[41] It is not recommended at this time for primary care practice.[42]

Family physicians can make a clinical diagnosis of IC. **Table 2** presents diagnostic steps useful in primary care. Physicians should start with a history and focused physical examination. History should rule out other concomitant diseases and include a very careful review of fluid intake (ie, amount of coffee,

Table 2. Primary care evaluation of patients with suspected interstitial cystitis

History: urinary frequency, nocturia, urgency, suprapubic pain
Physical examination: focused (including pelvic examination)
Urine culture: if positive (or even suspect), treat with antibiotics
Urinalysis: if positive (ie, hematuria), evaluate
Cytology: if suspect or atypical, refer to a urologist

colas, and so on) and output (a voiding diary for a day or so is extremely helpful).

Physical workup must include a careful pelvic examination. During the pelvic examination to rule out pelvic pathology (ie, pelvic mass), a Pap smear can be performed, if indicated (not related to IC diagnosis). Bimanual examination of the bladder is typically uncomfortable for patients. Urinary tract infection (ie, bacterial cystitis) must first be ruled out as the cause of patients' lower urinary tract symptoms. Culture for uropathogenic organisms is an understandably mandatory part of the investigation.

Simple urinalysis and urine cytology will complete a typical evaluation (urine cultures and cytologies can be repeated during later visits). Referral to a urologist for cystoscopy is not required unless patients have gross hematuria (or persistent microhematuria)or atypical cytology results, or unless history (acute onset or change of symptoms) and physical examination suggest some other cause for the symptoms. Shared care, between primary care physicians and urologists, for both diagnosis and treatment, is useful if family physicians are uncomfortable making the diagnosis or subsequently managing IC patients.

Treatment

Therapy for IC is generally acknowledged as empiric, palliative, and in most cases, therapeutically marginal. Only six randomized placebo-controlled or controlled clinical trials are available, and many of these are small or have conflicting results. Hence, therapy is based not only on results of treatments subjected to controlled clinical trials but also on the best available evidence, including that from both pilot studies and case series.

Therapy for IC does not result, in most cases, in quick cures. Commitment to a long-term treatment strategy is required. Physicians managing patients with IC should be aiming at reducing symptoms and improving patients' quality of life.

Conservative therapy. Once the diagnosis of IC is made, supportive therapy and even watchful waiting in many cases is the treatment of choice. Educating and empowering patients embodies this approach.[43] Psychotherapy, stress reduction, reduction in anxiety, and exercise have all been found beneficial for some patients.[6,44] Diet modification is an important, if not the most important, conservative therapy available. Some foods that patients with IC should avoid are listed in **Table 3**. Patients have found that joining an IC support group, such as the Canadian Interstitial Cystitis Association, is extremely valuable. They find out they are not alone, learn coping skills, receive dietary information, are kept up to date on new treatments, and most of all, derive support from other patients who have learned to live with the condition. Two excellent sources of information for patients and physicians are the websites of the Interstitial Cystitis Association (http://www.ichelp.org/) and the Interstitial Cystitis Network (http://www.ic-network.com/).

Table 3. Foods patients with interstitial cystitis should avoid

Coffee, tea, alcohol, cranberry juice, carbonated colas
Chocolate, aged cheeses
Onions, tomatoes
Citrus fruits and juices (other fruits might affect certain patients and must be evaluated)
Aged, canned, cured, or processed meats and fish
Nuts
Spicy seasonings (especially Chinese, Indian, Mexican, and Thai seasonings)

Oral medications. *Antispasmodics*: Anticholinergics and antispasmodics, such as oxybutynin, flavoxate, or tolterodine, might ameliorate the irritative voiding symptoms of IC. While they could be effective for some patients in controlling urgency and frequency, they are usually not effective in improving pain, the cardinal symptom of IC. All reports of use of these agents for IC are anecdotal; there are no controlled studies in the literature.

Pentosan polysulfate: The synthetic sulfated polysaccharide, pentosan polysulfate sodium (PPS), is believed to act by adhering to the luminal side of the bladder mucosa, thus maintaining or enhancing the permeability barrier of the bladder mucosa to various potentially toxic components of urine.[12] In a number of well powered, randomized placebo-controlled trials,

efficacy (defined as percentage of patients with 50% or greater improvement in overall evaluation) ranges from 28% to 42% versus 13% to 20% for placebo.[45,46] A large European controlled study, however, failed to demonstrate efficacy.[47]

Analysis of patients treated in the United States in an open-label compassionate-use study during the decade (1986-1996) before it was generally available[48] showed that PPS was well tolerated over time and that it could be extremely beneficial for many patients. In certain patients (ie, patients who have at least a moderate response) improvement in symptoms can continue for years. In this study, an overall improvement in symptoms was reported by 62% of subjects who received therapy for 6 to 35 months.

This is not a universally accepted conclusion, as another study[49] evaluating long-term follow-up data in significantly fewer patients with IC found that PPS benefited between 6.2% and 18.7% of patients. The only baseline factor predicting response to PPS in this study was less constant pain. A recent meta-analysis of all major studies comparing PPS with placebo[50] concluded that PPS was more efficacious than placebo for treatment of pain, urgency, and frequency associated with IC, but not significantly different from placebo in treating nocturia associated with IC. Pentosan polysulfate remains the most widely studied drug for IC and the only oral medication with a registered indication for treatment of IC.

Hydroxyzine: As suggested in the section on etiology, there appears to be a role for mast cell activation in the pathogenesis of IC. Early clinical experience with hydroxyzine, a heterocyclic piperazine H_1 receptor antagonist, suggests that this drug has some clinical efficacy for patients with biopsy-documented bladder mastocytosis or mast cell activation and a history of allergies. One study employing increasing titrated doses up to 50 mg at night and 25 mg in the morning showed a 30% improvement in symptoms.[51]

Tricyclic antidepressants: Tricyclic antidepressants have multiple mechanisms of action, including central and peripheral anticholinergic actions, blocking reuptake of serotonin and norepinephrine, and antihistaminic properties that are believed to benefit patients with IC. Success rates using amitriptyline have been reported from 64% to 90%.[52,53]

Other: Other oral agents, such as the calcium channel blocker, nifedipine,[54] and L-arginine,[55] have also been reported to have limited efficacy in initial results. Similar to many treatments for IC, these drugs fail to demonstrate significant efficacy when subjected to randomized placebo-controlled trials.[56] Opioid therapy for chronic pain control in refractory patients is an effective but quite desperate measure. A small case series has shown cimetidine therapy effective for some patients.[57]

Intravesical therapy. Intravesical therapy is generally administered by urologists, but because it is appropriate first-line therapy, family physicians should understand the rationale for its use and its potential benefits for their patients with IC. Treatment is usually given weekly (for 4 to 8 weeks). If it is successful, it can be repeated periodically. It is appropriate for family physicians to consider intravesical therapy for new patients under urologic supervision or for patients who have been successfully treated in this manner but have subsequently relapsed.

Dimethyl sulfoxide: Dimethyl sulfoxide (DMSO) has pharmacologic properties including anti-inflammatory, analgesic, collagen dissolution, muscle relaxant, and effects on mast cell histamine release.[58] A small randomized controlled trial[59] comparing DMSO to saline showed a 93% objective improvement and 53% subjective improvement compared with 35% and 18%, respectively, for saline placebo. Patients frequently relapse and require further treatments at regular intervals.

Heparinlike medications: Intravesical heparin and hyaluronic acid are prescribed for the same reasons as oral PPS (maintain or enhance the activity of the bladder's own mucopolysaccharide lining), and both have been shown in small pilot studies[60-64] to have some benefit. Heparinoid therapies also have other properties: inhibiting adherence of immune complexes to polymorphonuclear cells, inhibiting leukocyte migration and aggregation, regulating fibroblasts and endothelial cell proliferation, enhancing connective tissue healing, and scavenging reactive oxygen species,[62] which might also contribute to their action in IC patients.

Many patients treated successfully with intravesical therapy relapse over time and require further treatments. A combination of intravesical DMSO and heparin is superior to DMSO alone for long-term maintenance of beneficial response.[64] A small (20-patient) study[65] comparing intravesical PPS with placebo suggested that PPS benefits some patients.

Bacille Calmette-Guérin vaccine: Intravesical BCG has shown some promise in clinical trials performed by one group.[66] Results of six weekly treatments demonstrated a 60% response rate to BCG, compared with a 27% response to placebo. Most patients who

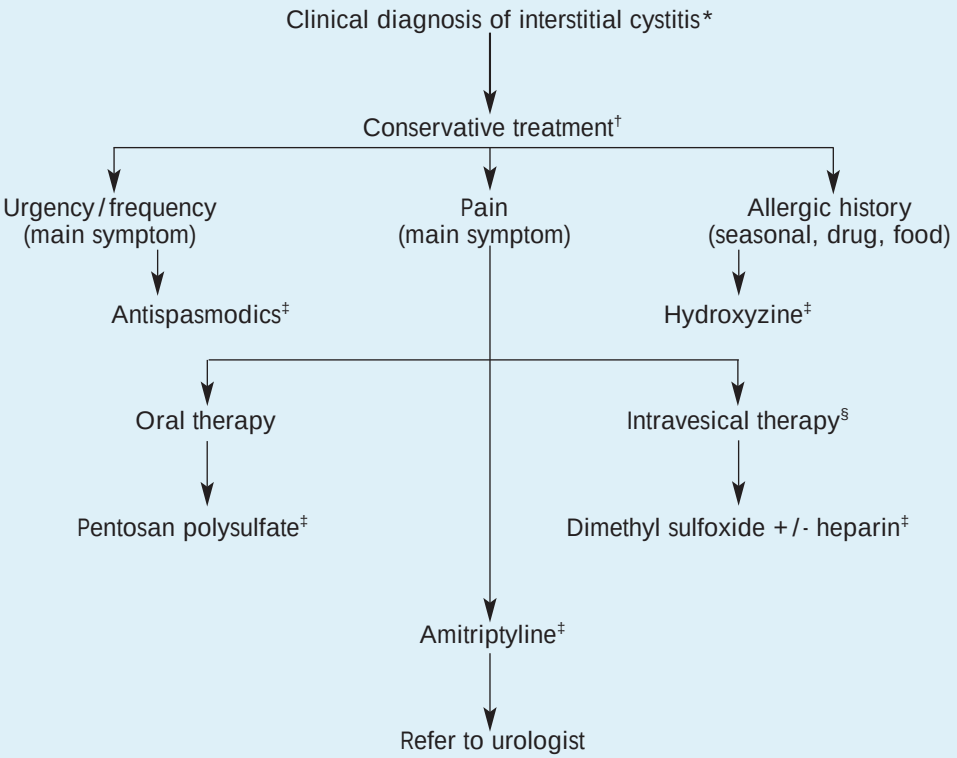
responded favourably continued to have an excellent response with follow up ranging from 24 to 33 months.[67] Preliminary Canadian studies were not encouraging, however (Morales, unpublished observations), and BCG has not become an accepted or common treatment in Canada.

Surgery. Surgical therapy for IC has never been subjected to critical analysis in a controlled clinical trial. Hydrostatic bladder dilation under general anesthesia ameliorates symptoms in about 50% of patients (anecdotal experience of most urologists). The benefits of hydrostatic dilations are short-lived, however, and benefits diminish and risks increase with repeated procedures requiring anesthesia.

Open surgical therapy for IC is absolutely a last resort after every other therapy has been tried and has failed.[68] Most clinicians with experience in IC think the best procedure is a supravesical diversion, usually with a cystectomy. Pelvic pain can persist despite cystectomy[69] (further evidence for a chronic neurogenic etiology for the condition). Patients who have undergone surgery for IC appear to have more long-term complications than patients who have had similar surgery for malignancy.[70] Family physicians should be part of the assessment team that makes the final decision on any type of irreversible surgery.

After diagnosing IC, family physicians can develop a rational treatment plan based on the information presented above. A treatment plan that can be undertaken by primary care physicians is shown in **Figure 1**. Doses and treatment schedules for the various pharmaceutical therapies are listed in **Table 4**. If a patient does not have a favourable clinical

Figure 1. Suggested treatment algorithm for family physicians



*See **Table 2**.
†See **Table 3** and text.
‡See **Table 4**.
§Many family physicians are uncomfortable using intravesical therapy and refer to urologists for this treatment or provide treatment with urologic supervision and advice.

response to treatment, referral to a urologist is indicated. At that time, cystoscopic examination and hydrostatic bladder dilation under general anesthesia might be required.

Management strategy for primary care physicians

How do primary care physicians distil the conflicting evidence as to etiology, diagnosis, and treatment of IC? They must have an understanding of the syndrome, a confident knowledge of how to make the diagnosis, a rational treatment strategy, and a willingness to work with urologic specialists if the management plan does not succeed in ameliorating patients' symptoms. If family physicians remain reluctant to treat the disease at a primary care level, they must be able to at least consider IC in the differential diagnosis of chronic pelvic pain disorders and in patients with irritable voiding symptoms.

Shared care with a urologist is an ideal management strategy for IC. **Table 5** presents the core knowledge of IC distilled from the literature. Primary care and family physicians committed to their patients presenting with bladder irritability and pain can and should take an active role in managing IC.

Table 4. Pharmaceutical agents routinely used in Canada to treat interstitial cystitis

DRUG	ROUTE	DOSE	DOSING INTERVAL
Oxybutynin	Oral	5 mg	3 times daily
Flavoxate	Oral	200 mg	3-4 times daily
Tolterodine	Oral	2 mg	Twice daily
Pentosan polysulfate (PPS)	Oral	100 mg	3 times daily
Hydroxyzine	Oral	25 mg	Twice daily
Amitriptyline	Oral	25-75 mg	Nightly
Dimethyl sulfoxide (DMSO)	Intravesical for 20 min	50 mL of 50% solution	Weekly (6 treatments)*
Heparin	Intravesical	10 000 U in 50 mL normal saline	Weekly (6 treatments)*
Hyaluronic acid	Intravesical	40 mg in 50 mL normal saline	Weekly (4-6 treatments)*

**Initial therapy; maintenance therapy might require less frequent dosing.*

Table 5. Interstitial cystitis: *Core knowledge for family physicians*

EPIDEMIOLOGY
Common
ETIOLOGY
Unknown. Theories include abnormal surface glycosaminoglycan layer, infection, and immunologic and neurogenic causes
DIAGNOSIS
History
Physical examination
Urine culture
Urinalysis
Cytology
TREATMENT
Conservative measures (ie, diet) Oral antispasmodics, PPS, amitriptyline, or hydroxyzine Intravesical* DMSO, heparin, hyaluronic acid, BCG vaccine
Surgery† (last resort)

**Although most family physicians do not feel comfortable using intravesical therapy, they should be aware of its rationale and benefits.*
†Family physicians will not be involved in surgical procedures themselves, but it is important to understand that, except for hydrostatic bladder dilation, surgery is treatment of last resort. Family physicians should be involved in the decision process.

Conclusion

Interstitial cystitis is a relatively common syndrome confronted by primary care physicians. Its etiology is unknown, its diagnosis lacks a criterion standard, and treatment is often empiric and palliative. The etiology of IC is unknown and diagnosis is based on a constellation of symptoms, such as pain and frequency and urgency of urination, after ruling out other causes (especially infection).

A review of available evidence suggests that conservative supportive therapy (including diet modification); oral treatment with PPS, amitriptyline, or hydroxyzine; and intravesical treatments with heparinlike medications, DMSO, or BCG vaccine could be effective for some patients. Surgery is the option of last resort after every therapeutic avenue has been explored. For primary care physicians uncomfortable with diagnosing and treating IC without supervision, shared care with a urologist provides excellent patient care.

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Key points

- Interstitial cystitis is a clinical syndrome characterized by urinary frequency and urgency, nocturia, and suprapubic pressure and pain without an identifiable cause.
- Its etiology is unknown, despite several theories.
- Diagnosis is made based on characteristic clinical signs and symptoms, exclusion of other causes, and not necessarily cystoscopy.
- Therapy is empiric, palliative, and often not very effective, but it can reduce symptoms and improve quality of life.

Points de repère

- La cystite interstitielle est un syndrome clinique qui se caractérise par une fréquence et une urgence de la miction, une polyurie nocturne ainsi qu'une pression et une douleur sus-pubiennes sans cause identifiable.
- Son étiologie reste inconnue malgré l'existence de diverses théories.
- Le diagnostic est posé en se fondant sur des signes et des symptômes cliniques caractéristiques, l'exclusion d'autres causes et n'exige pas nécessairement une cystoscopie.
- La thérapie est empirique, palliative et souvent peu efficace, mais elle peut réduire les symptômes et améliorer la qualité de vie.

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