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Hypersensory Bladder Disease: Concept and Pathogenetic Basis

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ABSTRACT

This review article is devoted to current concepts of hypersensory bladder. It provides a detailed description of the epidemiological, clinical, and pathophysiological features of overactive bladder and bladder pain syndrome / interstitial cystitis, emphasizing the similarity of their clinical manifestations. The molecular mechanisms of the mechanosensory function of the urothelium under normal conditions and in the presence of hypersensory disorders are thoroughly described. The data presented allow these conditions to be regarded as manifestations of a common pathological process based on increased bladder sensitivity. It is noted that the mechanism of hypersensitivity in overactive bladder and bladder pain syndrome / interstitial cystitis is similar and involves the pathological amplification of afferent signal transmission from the bladder to the central nervous system. This process may be associated with multiple factors, but inflammation is regarded as the main cause of increased afferent activity in the bladder. The role of central sensitization in the development of hypersensory bladder disorders is also highlighted. Given the commonality in symptoms, clinical course, and pathogenesis, the concept of "hypersensory bladder disease" is proposed, uniting the hypersensory phenotypes of overactive bladder and bladder pain syndrome / interstitial cystitis. It is stated that this concept is justified from a pathogenetic standpoint, although many aspects require further investigation and discussion.

Keywords: overactive bladder; bladder pain syndrome; interstitial cystitis; hypersensory bladder; hypersensory bladder disease.

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Гиперсенсорная болезнь мочевого пузыря: концепция и патогенетические основы

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АННОТАЦИЯ

Обзорная статья посвящена современным представлениям о гиперсенсорном мочевом пузыре. Подробно описаны эпидемиологические, клинические и патофизиологические особенности гиперактивного мочевого пузыря и синдрома болезненного мочевого пузыря/интерстициального цистита. Подчеркнута схожесть их клинических проявлений. Подробно описаны молекулярные механизмы механосенсорной функции уротелия в норме и при возникновении гиперсенсорных нарушений. Приведены данные, позволяющие рассматривать эти заболевания как симптом единого патологического процесса, в основе которого лежит повышение чувствительности мочевого пузыря. Отмечено, что механизм гиперсенсорности при гиперактивном мочевом пузыре и синдроме болезненного мочевого пузыря/интерстициального цистита сходен и связан с патологическим усилением передачи афферентных сигналов от мочевого пузыря в центральную нервную систему. Данный процесс может быть связан со многими факторами, но в качестве основной причины повышения афферентной активности мочевого пузыря является воспаление. Показана роль в развитии гиперсенсорных нарушений мочевого пузыря центральной сенситизации. Учитывая общность симптоматики, клинического течения и патогенеза, представлена концепция гиперсенсорной болезни мочевого пузыря, объединяющая гиперсенсорные фенотипы гиперактивного мочевого пузыря и синдрома болезненного мочевого пузыря/интерстициального цистита. Указано, что данная концепция представляется оправданной с патогенетической точки зрения, хотя многие вопросы требуют дополнительных исследований и обсуждений.

Ключевые слова: гиперактивный мочевой пузырь; синдром болезненного мочевого пузыря; интерстициальный цистит; гиперсенсорный мочевой пузырь; гиперсенсорная болезнь мочевого пузыря.

Как цитировать

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INTRODUCTION

Urinary disorders are among the most common complaints among patients with urological dysfunction. The symptoms of urinary disorders are collectively referred to as *lower urinary tract symptoms* (*LUTS*) and are divided into three categories: storage, voiding, and post-micturition. LUTS are observed in various diseases and conditions. The most common urinary disorders include pollakiuria and urge to urinate (urgency). In addition to organic (e.g., tumors, stones) and infectious lesions of the urinary tract, the most typical etiologies include overactive bladder (OAB) and bladder pain syndrome/interstitial cystitis (BPS/IC) [1, 2]. In the latter case, urinary dysfunction is combined with pain in the bladder. Many factors are recognized as being involved in the pathogenesis of OAB and BPS/IC, and most of these factors are common to both conditions [3, 4]. This paper presents data suggesting that OAB and BPS/IC, or at least some of their clinical forms, are manifestations of a single pathological process involving increased bladder sensitivity. This concept is based on the analysis of epidemiological, clinical, and pathophysiological aspects of OAB and BPS/IC.

TERMINOLOGICAL ASPECTS

In 2002, the International Continence Society (ICS) established a definition for OAB as a symptom complex characterized by an urge to urinate (urgency), with or without urinary incontinence, often accompanied by increased daytime and/or nighttime urination in the absence of infections or evident bladder lesions [5]. This definition of OAB is still relevant. Urgency is the leading clinical symptom and determines the severity of other OAB symptoms [6].

Over the past 25 years, several definitions of BPS/IC have been proposed by various international organizations. According to ICS, BPS/IC is characterized by pain in the bladder area that intensifies with bladder filling and with the urge to urinate. It is also associated with an increased frequency of daytime and/or nighttime urination, in the absence of a urinary tract infection or other visible cause of these symptoms [5]. Subsequent definitions of this condition were only slightly different from the one above. For example, the European Society for the Study of Interstitial Cystitis/Bladder Pain Syndrome indicated that BPS/IC is characterized by persistent or recurrent chronic pelvic pain, pressure, or discomfort associated with the bladder. This is accompanied by at least one other symptom, such as urgency or pollakiuria, in the absence of other reasons for their occurrence [7]. According to the American Urological Association, BPS/IC is an unpleasant sensation (pain, pressure, or discomfort) that is perceived as being related to the bladder. It is associated with LUTS and lasts more than six weeks in the absence of infection or other causes [8]. The European Association of Urology (EAU) defines BPS/IC as persistent or recurrent pain in the bladder region accompanied by at least one of the following symptoms: increased pain with bladder filling, increased urinary frequency during the day and/or night, and the absence of infection or other obvious local pathology. Additionally, the EAU suggested using the term *primary BPS* in the absence of an obvious cause for the symptoms and *secondary BPS* in the presence of an obvious cause [9].

Notably, in 2002, the ICS recommended using the term *BPS* but did not exclude the term *interstitial cystitis* for patients with typical cystoscopic and histological signs of the disease [5]. Currently, the term *interstitial cystitis* is often used for patients with Hunner lesions, whereas *BPS* is used for nonulcerative forms of the disease [10].

A comparison of the definitions of OAB and BPS/IC reveals notable similarities. Firstly, both conditions are characterized as syndromes (symptom complexes). Secondly, an obvious reason for their development is absent in both cases. Finally, the similarity of their clinical manifestations is evident.

EPIDEMIOLOGY

The prevalence of OAB and BPS/IC is high, and both conditions are among the leading causes of dysuria [2]. A common characteristic of OAB and BPS/IC is the higher prevalence in women compared with men. The estimated prevalence of OAB is 15%–20% among all adults [1, 11]. The greatest difference in the incidence of OAB between women and men is observed in people under the age of 60 years, after which the proportion of men with OAB increases [1]. The prevalence of BPS/IC is evaluated within a wide range. This is due to the use of different diagnostic criteria and research methods for BPS/IC [12]. The incidence of BPS/IC among adults is typically reported in the range from 2.5% to 6.5% [12, 13]. The ratio of women to men with BPS/IC ranges from 9:1 [14] to 5:1 [15], and being female is considered an independent risk factor for developing BPS/IC. In light of these circumstances, the American Urogynecological Society and the International Urogynecological Association have proposed classifying BPS in women as a distinct nosological entity [16].

SYMPTOMS AND SIGNS

Many clinical observations indicate that OAB and BPS/IC symptoms are similar [17]. The leading clinical manifestation of OAB is urgency, whereas that of BPS/IC is bladder pain. Urgency and pain are closely related. According to ICS guidelines, urgency is defined as a sudden and

irresistible desire to empty the bladder [1]. In 2014, the definition of urgency was expanded to include reasons for the difficulty of postponing urination, such as pressure, discomfort, or pain [18]. Importantly, urgency may be accompanied by pain or discomfort, which are the primary clinical symptoms of BPS/IC [1, 7, 8].

For patients with the hypersensory phenotype of OAB, urgency is most often expressed by a non-classical variant with strong and sudden urges to urinate. This variant is characteristic of patients with detrusor hyperactivity of neurogenic or non-neurogenic origin. Patients typically experience a constant, urgent need to urinate due to discomfort in the bladder when it is only partially full, with occasional sudden urges to urinate. Urgency, along with pain, is another key symptom of BPS/IC, similar to that experienced by patients with a hypersensory bladder. Another similarity between these two conditions is that the intensity of symptoms increases as the bladder fills. This feature is specified in the definition of BPS/IC. However, patients with BPS/IC also experience the "classical" urge to urinate. For example, Clemens et al. [19] demonstrated that 60% of women with IC experience sudden and intense urges to urinate. In contrast, urgency is associated with discomfort, pressure, or pain in 42% of women with OAB. Lai et al. [20] reported no significant differences in the severity of increased urination frequency and urgency between patients with OAB and BPS/IC.

Thus, OAB and BPS/IC have similar symptoms that significantly impact the patients' psychoemotional state and quality of life [21, 22].

CLINICAL PROGRESSION

The clinical courses of OAB and BPS/IC have much in common. They are both wave-like in nature, and the severity of symptoms may change over time [20]. Research has demonstrated that only 7% of patients with BPS/IC experience the full spectrum of subsequent symptoms at the disease onset [23], whereas in 42% of women, BPS/IC manifested as pollakiuria and urgency, rather than pain or discomfort in the bladder [24]. For this reason, many patients with BPS/IC are initially misdiagnosed with OAB. According to some data, up to 50% of women with OAB experience long periods of remission. These periods are more likely and longer in patients without urinary incontinence [25, 26]. A similar trend has been observed in women with BPS/IC. Approximately 50% of patients experience spontaneous remission, which lasts an average of eight months [27].

PATHOGENESIS

Although much about the mechanisms of OAB and BPS/IC occurrence remains unclear, the multifactorial nature of their pathogenesis is evident, with several factors common to both conditions.

According to current concepts, OAB may develop due to neurogenic, myogenic, or urotheliogenic mechanisms [4]. For patients with neurogenic or myogenic OAB, detrusor hyperactivity is most often identified through urodynamic testing. Ischemia of the bladder wall is considered as an independent risk factor for the development of OAB [28, 29]. This mechanism is characteristic of elderly patients and attributed to sclerotic processes in the bladder wall. Urodynamic testing reveals a decrease in bladder wall elasticity [30].

In women with OAB, detrusor hyperactivity and decreased bladder compliance are often not detected. Characteristic urodynamic signs include increased bladder sensitivity and decreased bladder capacity [6]. This phenomenon is defined as OAB without detrusor hyperactivity or hypersensory bladder. According to various estimates, the hypersensory urodynamic phenotype is observed in 64%–86% of women with OAB [6, 31]. In most patients, hypersensory bladder is caused by a urotheliogenic mechanism based on increased afferent activity of the bladder in response to distension [32, 33]. Furthermore, BPS/IC is a heterogeneous disease that is classified into two forms: classical with Hunner lesions (ulcers) and non-classical without such lesions. Similar to OAB, bladder hyperactivity is considered to be the main cause of symptoms in patients with non-ulcerative forms of BPS/IC [34].

Thus, the hypersensitivity mechanisms in OAB and BPS/IC are similar and associated with pathological amplification of afferent signals from the bladder to the central nervous system (CNS) [35]. This process may be associated with many factors; however, inflammation is considered the primary cause of increased afferent bladder activity [4, 36, 37]. A significant proportion of patients with OAB and BPS/IC exhibit histological signs of bladder wall inflammation and elevated levels of laboratory markers of systemic and local inflammation [37–39]. Furthermore, a correlation was observed between the level of inflammatory markers and the severity of symptoms in patients with OAB and BPS/IC [37, 39]. The systemic nature of the inflammatory process is indicated by the association between BPS/IC, especially its nonulcerative form, and the development of autoimmune diseases, allergic reactions, bronchial asthma, and inflammatory bowel diseases [10, 40].

Over the past decade, significant progress has been made in understanding the mechanism of the mechanosensory function of the bladder. It has been established that, in response to mechanical distension, specific ion channels of the TRP (transient receptor potential) and Piezo families open in urotheliocyte cell membranes [41, 42]. Ca^{++} ions enter the cell through these channels, which initiates the release of the neurotransmitter

adenosine triphosphate from urotelial cells into the intercellular space. Receptors for this neurotransmitter are located on afferent nerve fiber terminals [41, 42]. Consequently, the urothelium perceives mechanical stimuli caused by the bladder distension and converts them first into chemical substances (neurotransmitters), which are then converted into electrical signals. These signals deliver information about the state of the urinary tract to higher parts of the CNS. Depending on the intensity of the hypersensory reaction, there may be varying degrees of symptoms of OAB and BPS/IC, such as bladder fullness, urgency, and pain.

HYPERSENSORY BLADDER DISEASE

Due to the similarities in symptoms, clinical course, and pathogenesis, it is reasonable to group the hypersensory phenotypes of OAB and BPS/IC together under the new concept of hypersensory bladder disease (HBD). This concept does not include secondary OAB of neurogenic and myogenic nature, as well as OAB with detrusor hyperactivity and ulcerative forms of BPS/IC. The concept of HBD is schematically presented in Fig. 1.

Notably, there were some attempts in the past to consider OAB and BPS/IC together. For example, Frazer et al. proposed the term *hypersensitive bladder* in 1990 to describe idiopathic OAB and the initial stage of IC [43]. Several publications from the late 2000s and early 2010s focused on diagnosing and treating OAB with bladder hypersensitivity in the absence of detrusor hyperactivity [44, 45].

In 2013, Homma recommended using the term *hypersensitive bladder* to describe conditions characterized by increased bladder sensitivity, manifested by frequent urination, with or without bladder pain, in the absence of obvious pathology explaining the onset of these symptoms [46]. The author emphasized that OAB and BPS/IC share similar clinical manifestations, while indicating that the term *hypersensitivity bladder* describes the clinical picture regardless of the reasons for its development [46].

Lai et al. [20] highlighted the significant similarities in symptoms between BPS/IC and OAB, proposing the concept of a *continuum of bladder hypersensitivity syndrome* to describe the three main clinical symptoms: pain, urgency, and urge incontinence. According to this concept, OAB and BPS/IC are two similar but distinct pathological conditions. The severity of bladder pain in OAB is intermediate between that of BPS/IC and that of healthy individuals, whereas the severity of urge incontinence in BPS/IC is intermediate between OAB and healthy individuals. OAB and BPS/IC are located at two poles of a conditional continuum, with various clinical variants between these two conditions, characterized by varying degrees of urinary frequency, urgency, incontinence, and pain.

There are several possible explanations for the similarities in the symptoms and clinical courses of OAB and BPS/IC. One explanation is that patients suffer from both conditions and exhibit the corresponding symptoms. A misdiagnosis is also possible because refractory OAB could actually be BPS/IC [47, 48]. However, increasing evidence suggests that the sensory phenotypes of OAB and BPS/IC have similar developmental pathophysiological mechanisms.

The concept of hypersensory bladder disease considers both the clinical presentation of the symptoms and their underlying cause: increased bladder sensitivity and heightened activity of the afferent link of the micturition reflex. This distinguishes the concept from those proposed by Homma (2013) [46] and Lai et al. (2014) [20]. The proposed model raises an important question. If the pathogenetic mechanism of development is the same, why are there two manifestations of HBD—OAB with a predominance of urinary disorders and BPS/IC, which is manifested by pain? This is presumably related to the phenomenon of central sensitization (sensibilization). The latter is a mechanism that enhances pain perception by increasing the sensitivity of CNS neurons to normal or subthreshold afferent stimulation and reducing the pain threshold (allodynia). Consequently, this phenomenon causes initially "non-painful" stimuli to manifest as painful sensations. In relation to the bladder, central

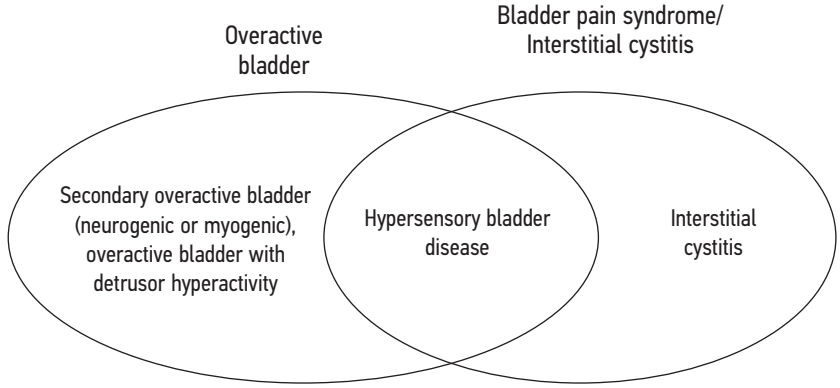

Fig. 1. Concept of hypersensory bladder disease.

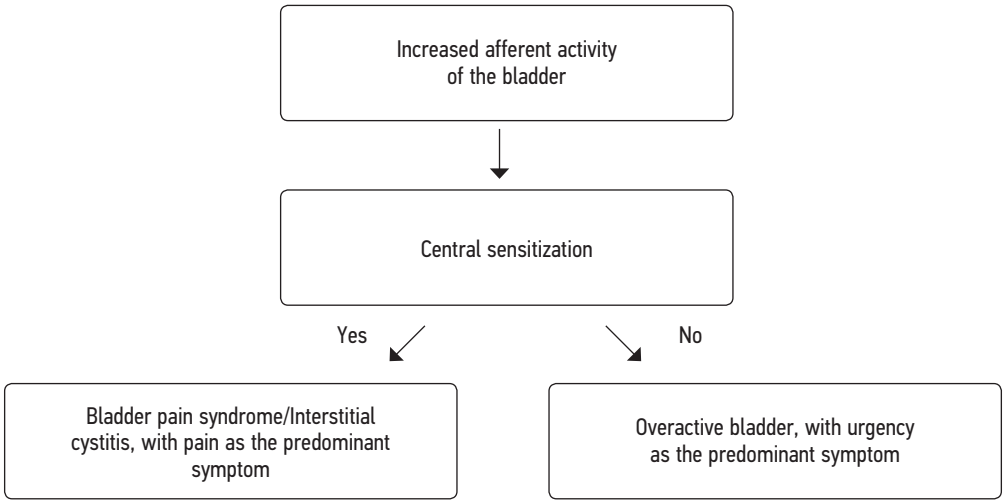

Fig. 2. Concept of the pathogenesis of hypersensory bladder disease.

sensitization amplifies normal, subthreshold, non-painful signals from afferent Aδ and C fibers in central nociceptive neurons. Consequently, the signals exceed the threshold, inducing pain in the absence of initial nociceptive stimulation and prompting a sense of urgency. That is, distension of the bladder wall due to hyperafferentation, in the absence of central sensitization, manifests as symptoms of OAB. In the presence of central sensitization, it manifests as symptoms of BPS/IC (Fig. 2). Remarkably, the severity of central sensitization may vary. If sensitization is low, the clinical symptoms may be "intermediate," manifesting as discomfort or mild pain in patients with OAB. Thu et al. [49] found that 34% of patients with OAB reported varying degrees of bladder pain, and 28% experienced extrapelvic pain. This was apparently due to secondary hyperalgesia accompanied by central sensitization [49].

The proposed concept of HBD may have theoretical and practical significance. Treatment of hypersensory phenotypes has its own characteristics. Specifically, research has shown that anticholinergic therapy is relatively ineffective for women with a hypersensory bladder and sympathetic dysfunction [50]. Moreover, anticholinergic drugs are insufficiently successful in treating women with PBS/IC [51]. However, there are effective treatment options for both OAB and BPS/IC. These include bladder hydrodistension, botulinum therapy, and the prescription of anti-inflammatory drugs [52–56]. The potential for the treatment of hypersensory bladder disorders with beta3-adrenergic receptor agonists is significant [50].

The insufficient effectiveness of M-anticholinergics in patients with hypersensory OAB and BPS/IC is associated with several factors. First, anticholinergic drugs have a limited effect on the afferent purinergic signaling system, which increases bladder sensitivity. Secondly, inflammation of the bladder wall may maintain high levels of afferent activity. Another possible reason is central sensitization. In this case, standard drug therapy targeting the bladder may be ineffective.

CONCLUSION

Thus, the present study indicates that the hypersensory phenotypes of OAB and BPS/IC are similar. These conditions were combined into a common concept, HBD, due to their similar pathophysiological mechanisms of development and clinical manifestations. From a pathogenetic perspective, this concept appears to be substantiated; however, many issues require further research and discussion. This model may be useful for developing personalized treatment approaches for patients with OAB and BPS/IC.

ADDITIONAL INFO

Author contributions: I.V. Kuzmin: conceptualization, investigation, writing—original draft; M.N. Slesarevskaya: investigation, writing—review & editing. All the authors approved the version of the draft to be published and agreed to be accountable for all aspects of the work, ensuring that issues related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Источники финансирования. Отсутствуют.

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Оригинальность. При создании настоящей работы авторы не использовали ранее опубликованные сведения (текст, иллюстрации, данные).

Генеративный искусственный интеллект. При создании настоящей статьи технологии генеративного искусственного интеллекта не использовали.

Рассмотрение и рецензирование. Настоящая работа подана в журнал в инициативном порядке и рассмотрена по обычной процедуре. В рецензировании участвовали один рецензент (член редакционной коллегии, член редакционного совета или внешний рецензент), рецензирование двойное слепое.

REFERENCES

1. Milsom I, Abrams P, Cardozo L, et al. How widespread are the symptoms of an overactive bladder and how are they managed? A population-based prevalence study. *BJU Int*. 2001;87(9):760–766. doi:10.1046/j.1464-410X.2001.02228.x EDN: BACBIR

2. Slesarevskaya MN, Ignashov YuA, Kuzmin IV, Al-Shukri SK. Persistent dysuria in women: etiological diagnostics and treatment. *Urology reports (St. Petersburg).* 2021;11(3):195–204. doi: 10.17816/uroved81948 EDN: BDUFWQ

3. Zaytsev AV, Sharov MN, Ibragimov RA, et al. Painful bladder syndrome / interstitial cystitis: modern approaches to diagnosis and treatment. *Ambulance Doctor*. 2018;(8):16–26. EDN: YLJJWX

4. Peyronnet B, Mironska E, Chapple C, et al. A Comprehensive review of overactive bladder pathophysiology: On the way to tailored treatment. *Eur Urol.* 2019;75(6):988–1000. doi: 10.1016/j.eururo.2019.02.038

5. Abrams P, Cardozo L, Fall M, et al. The standartisation of terminology in lower urinary tract function: Report from the standartisation sub-committee of the International Continence Society. *Neurourol Urodyn.* 2002;21(2):167–178. doi: 10.1002/nau.10052

6. Kuzmin IV. *Pathogenesis, clinical course and treatment of overactive bladder* [dissertation]. Saint Petersburg; 2007. EDN: QECRJB (In Russ.)

7. Van de Merwe JP, Nordling J, Bouchelouche P, et al. Diagnostic criteria, classification, and nomenclature for painful bladder syndrome/interstitial cystitis: an ESSIC proposal. *Eur Urol*. 2008;53(1):60–67. doi: 10.1016/j.eururo.2007.09.019

8. Hanno PM, Erickson D, Moldwin R, et al. Diagnosis and treatment of interstitial cystitis/bladder pain syndrome: AUA guideline amendment. *J Urol.* 2015;193(5):1545–1553. doi: 10.1016/j.juro.2015.01.086

9. Engeler D, Baranowski AP, Berghmans B, et al. *EAU guidelines on chronic pelvic pain*. European Association of Urology; 2025.

10. Niimi A, Akiyama Y, Tomonori Y, et al. Clinical manifestations of interstitial cystitis and bladder pain syndrome: Analysis of a patient registry in Japan. *Int J Urol.* 2025;32(1):103–109. doi: 10.1111/iju.15603 EDN: JOTNUC

11. Kuzmin IV. Epidemiological aspects of overactive bladder and urge urinary incontinence. *Urology reports (St. Petersburg).* 2015;5(3):30–34. doi: 10.17816/uroved5330-3 EDN: VHUCCH

12. Kuzmin IV, Slesarevskaya MN, Saveliev MV. Bladder pain syndrome. Terminological and epidemiological aspects. *Experimental and Clinical Urology*. 2025;18(1):175–184. doi: 10.29188/2222-8543-2025-18-1-175-184 EDN: HGNIPW

13. Berry SH, Elliott MN, Suttorp M, et al. Prevalence of symptoms of bladder pain syndrome/interstitial cystitis among adult females in the United States. *J Urol*. 2011;186(2):540–544. doi: 10.1016/j.juro.2011.03.132

14. Oravisto KJ. Epidemiology of interstitial cystitis. *Ann Chir Gynaecol Fenn.* 1975;64(2):75–77.

15. Clemens JQ, Meenan RT, Rosetti MC, et al. Prevalence and incidence of interstitial cystitis in a managed care population. *J Urol*. 2005;173(1):98–102. doi: 10.1097/01.ju.0000146114.53828.82

16. Developed by the Joint Writing Group of the International Urogynecological Association and the American Urogynecologic Society. Joint terminology report: Terminology standardization for female bladder pain syndrome. *Int Urogynecol J*. 2025;36(2):265–277. doi: 10.1007/s00192-024-05923-z

17. Elliott CS, Payne CK. Interstitial cystitis and the overlap with overactive bladder. *Curr Urol Rep*. 2012;13(5):319–326. doi: 10.1007/s11934-012-0264-y EDN: PGMWEN

18. Castro-Diaz D, Cardozo L, Chapple CR, et al. Urgency and pain in patients with overactive bladder and bladder pain syndrome. What are the differences? *Int J Clin Pract*. 2014;68(3):356–362. doi: 10.1111/ijcp.12317

19. Clemens JQ, Bogart LM, Liu K, et al. Perceptions of "urgency" in women with interstitial cystitis / bladder pain syndrome or overactive bladder. *Neurourol Urodyn*. 2011;30(3):402–405. doi: 10.1002/nau.20974

20. Lai HH, Vetter J, Jain S, et al. The overlap and distinction of self-reported symptoms between interstitial cystitis / bladder pain syndrome and overactive bladder: A questionnaire-based analysis. *J Urol*. 2014;192(6):1679–1685. doi: 10.1016/j.juro.2014.05.102

21. Kuzmin IV. Assessment of the quality of life in patients with overactive bladder. *Nephrology (Saint-Petersburg).* 2006;10(4):93–97. EDN: JURDFP

22. Slesarevskaya MN, Kuzmin IV, Ignashov YA. Characteristics of symptoms and psychosomatic status in women with chronic pelvic pain syndrome. *Urology reports (St. Petersburg).* 2015;5(3):16–19. doi: 10.17816/uroved5316-19 EDN: VHUCAT

23. Driscoll A, Teichman JM. How do patients with interstitial cystitis present? *J Urol.* 2001;166(6):2118–2120. doi: 10.1016/S0022-5347(05)65517-6

24. Warren JW, Wesselmann U, Greenberg P, Clauw DJ. Urinary symptoms as a prodrome of bladder pain syndrome / interstitial cystitis. *Urology*. 2014;83(5):1035–1040. doi: 10.1016/j.urology.2014.01.012

25. Wennberg A-L, Molander U, Fall M, et al. A longitudinal population-based survey of urinary incontinence, overactive bladder, and other lower urinary tract symptoms in women. *Eur Urol.* 2009;55(4):783–791. doi: 10.1016/j.eururo.2009.01.00

26. Phé V, Gamé X. Definition, epidemiology and impact of non-neurogenic overactive bladder. *Prog Urol*. 2020;30(14):866–872. doi: 10.1016/j.purol.2020.09.002 EDN: ENTZMA

27. Held PJ, Hanno PM, Wein AJ, et al. Epidemiology of interstitial cystitis: 2. In: Hanno PM, Staskin DR, Krane RJ, Wein AJ, editors. *Interstitial cystitis*. London: Springer-Verlag; 1990. P. 29–48. doi: 10.1007/978-1-4471-3293-6_4

28. Thurmond P, Yang J-H, Azadzoi KM. LUTS in pelvic ischemia: a new concept in voiding dysfunction. *Am J Physiol Renal Physiol*. 2016;310(8):F738–F743. doi: 10.1152/ajprenal.00333.201521

29. Al-Shukri SH, Kuzmin IV, Boriskin AG, et al. Correction of microcirculatory disorders in patients with overactive bladder. *Nephrology (Saint-Petersburg).* 2011;15(1):58–64. EDN: NUDVJN

30. Pinggera G-M, Mitterberger M, Steiner E, et al. Association of lower urinary tract symptoms and chronic ischaemia of the lower urinary tract in elderly women and men: assessment using colour Doppler ultrasonography. *BJU Int.* 2008;102(4):470–474. doi: 10.1111/j.1464-410X.2008.07587.x

31. Sekido N, Hinotsu S, Kawai K, et al. How many uncomplicated male and female overactive bladder patients reveal detrusor overactivity during urodynamic study? *Int J Urol.* 2006;13(10):1276–1279. doi: 10.1111/j.1442-2042.2006.01558.x

32. Fry CH, McCloskey KD. Spontaneous Activity and the Urinary Bladder. *Adv Exp Med Biol.* 2019;1124:121–147. doi: 10.1007/978-981-13-5895-1_5

33. Kushida N, Fry CH. On the origin of spontaneous activity in the bladder. *BJU Int.* 2016;117(6):982–992. doi: 10.1111/bju.13240

34. Birder LA, de Groat WC. Mechanisms of disease: involvement of the urothelium in bladder dysfunction. *Nat Clin Pract Urol.* 2007;4(1):46–54. doi: 10.1038/ncpuro0672

35. Yoshimura N, Ogawa T, Miyazato M, et al. Neural mechanisms underlying lower urinary tract dysfunction. *Korean J Urol.* 2014;55(2):81–90. doi: 10.4111/kju.2014.55.2.81

36. Kuzmin IV, Slesarevskaya MN, Romikh VV. Overactive bladder, inflammation and urinary tract infection: pathogenetic parallels. *Urology reports (St. Petersburg).* 2024;14(1):65–79. doi: 10.17816/uroved627461 EDN: GAHDJA

37. Chen L, Xu X, Zhou Y. The association between the systemic inflammation response index and overactive bladder: a cross-sectional study. *Eur J Med Res.* 2025;30(1):481. doi: 10.1186/s40001-025-02773-3

38. Jiang Y-H, Jhang J-F, Hsu Y-H, Kuo H-C. Usefulness of urinary biomarkers for assessing bladder condition and histopathology in patients with interstitial cystitis/bladder pain syndrome. *Int J Mol Sci.* 2022;23(19):12044. doi: 10.3390/ijms231912044 EDN: DIRZMH

39. Zwaans BMM, Mota S, Bartolone SN, et al. Evaluating symptom severity and urinary cytokine levels in interstitial cystitis/bladder pain syndrome patients, with and without Hunner's lesions. *Am J Clin Exp Urol.* 2024;12(2):110–118. doi: 10.62347/BLED240

40. Warren JW, Howard FM, Cross RK, et al. Antecedent nonbladder syndromes in case-control study of interstitial cystitis/painful bladder syndrome. *Urology*. 2009;73(1):52–57. doi: 10.1016/j.urology.2008.06.031

41. Vanneste M, Segal A, Voets T, Everaerts W. Transient receptor potential channels in sensory mechanisms of the lower urinary tract. *Nat Rev Urol.* 2021;18(3):139–159. doi: 10.1038/s41585-021-00428-6 EDN: RLNJQR

42. Li Z, Lin D, Luo C, et al. The expression and function of piezo channels in bladder. *Bladder (San Franc).* 2023;10:e21200008. doi: 10.14440/bladder.2023.870 EDN: EPKJIY

43. Frazer MI, Haylen BT, Sissons M. Do women with idiopathic sensory urgency have early interstitial cystitis? *Br J Urol.* 1990;66(3):274–278. doi: 10.1111/j.1464-410x.1990.tb14925.x

44. Yamaguchi O, Honda K, Nomiya M, et al. Defining overactive bladder as hypersensitivity. *Neurourol Urodyn.* 2007;26(S6):904–907. doi: 10.1002/nau.20482

45. Lee SR, Kim HJ, Kim A, Kim JH. Overactive bladder is not only overactive but also hypersensitive. *Urology*. 2010;75(5):1053–1059. doi: 10.1016/j.urology.2009.10.045

46. Homma Y. Hypersensitive bladder: towards clear taxonomy surrounding interstitial cystitis. *Int J Urol*. 2013;20(8):742–743. doi: 10.1111/iju.12143

47. Macdiarmid SA, Sand PK. Diagnosis of interstitial cystitis / painful bladder syndrome in patients with overactive bladder symptoms. *Rev Urol*. 2007;9(1):9–16.

48. Chung MK, Butrick CW, Chung CW. The overlap of interstitial cystitis / painful bladder syndrome and overactive bladder. *JSLS*. 2010;14(1):83–90. doi: 10.4293/108680810X12674612014743

49. Thu JHL, Vetter J, Lai HH. The severity and distribution of nonurologic pain and urogenital pain in overactive bladder are intermediate between interstitial cystitis and controls. *Urology*. 2019;130:59–64. doi: 10.1016/j.urology.2019.03.030

50. Ates E, Ipekci T, Akin Y, et al. Impact of sympathetic dysfunction in the etiology of overactive bladder in women: A preliminary study. *Neurourol Urodyn*. 2016;35(1):26–28. doi: 10.1002/nau.22652

51. Minaglia S, Ozel B, Bizhang R, Mishell DR Jr. Increased prevalence of interstitial cystitis in women with detrusor overactivity refractory to anticholinergic therapy. *Urology*. 2005;66(4):702–706. doi: 10.1016/j.urology.2005.04.042

52. Delaere KPJ, Debruyne FMJ, Moonen WA. The use of indomethacin in the treatment of idiopathic bladder instability. *Urol Int*. 1981;36(2):124–127. doi: 10.1159/000280402

53. Al-Shukri SH, Kuzmin IV, Slesarevskaya MN, Ignashov YuA. Bladder hydrodistension in treating patients with interstytial cystitis / bladder pain syndrome. *Urologiia*. 2018;(1):26–29. doi: 10.18565/urology.2018.1.26-29 EDN: YRSGPY

54. Martov AG, Muzhetskaya NG, Slyukova YuR, Salyukov RV Minimally invasive methods of treatment of interstitial cystitis / painful bladder syndrome. *Urologiia*. 2020(5):93–98. doi: 10.18565/urology.2020.5.93-98 EDN: NKXWXO

55. Slesarevskaya MN, Kuzmin IV, Ignashov YuA. Intravesical injections of triamcynolone and bladder hydrodistension in the treatment of patients with primary bladder pain syndrome. *Experimental and Clinical Urology*. 2023;16(4): 164–171. doi: 10.29188/2222-8543-2023-16-4-164-171 EDN: RCSRQI

56. Krivoborodov GG, Kuzmin IV, Slesarevskaya MN, et al. Botulinum toxin therapy in urology: historical aspect. *Urology reports (St. Petersburg).* 2024;14(2):163–174. doi: 10.17816/uroved633370 EDN: NLRSLD

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