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CHRONIC RECURRENT CYSTITIS: ANALYSIS OF THE CURRENT STATE OF THE PROBLEM

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Introduction. As part of this literature review, a comprehensive analysis of the current state of the problem of chronic recurrent cystitis was performed. Key aspects of the disease, including mechanisms of its development, features of the diagnostic work-up, and approaches to therapeutic management, are covered in the review. To provide an objective scientific overview, a systematic search for relevant sources was carried out in major international databases, including Medline, PubMed, and EMBASE. The analysis of the available literature indicates that viral CRC represents a substantial issue in contemporary urological practice and is actively discussed within the professional community. Extensive coverage of the topic in scientific publications and online resources reflects its high relevance and clinical significance.
The close attention of researchers to this problem is driven by a set of interrelated challenges. First, timely and accurate diagnosis remains difficult. Verification of cystitis etiology is often objectively challenging, which complicates differential diagnosis with other inflammatory conditions of the bladder. Another aggravating factor is the lack of unified, widely accepted standards of therapeutic management for chronic recurrent cystitis of any etiology. The clinical course is further complicated by its typical relapsing pattern, with exacerbations alternating with temporary remissions, which may falsely suggest that the treatment is effective.
Aim. Based on literature data identify features of the course of chronic recurrent cystitis and to determine differential diagnostic criteria for the development of an infectious process caused by various agents, taking into account their pathogenic properties.

K e y w o r d s: chronic recurrent cystitis, urinary tract infections, urothelium, viral cystitis

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Introduction. In contemporary uroinfectology, issues related to chronic recurrent cystitis continue to be the subject of intensive scientific debate and clinical research. The problems of etiology, mechanisms of development, diagnosis, and therapeutic management of patients with recurrent urinary tract infections still cannot be regarded as fully resolved, and they require further in-depth study and conceptual refinement.

Substantial progress in diagnostic technologies has had a marked impact on the conceptual understanding of the pathophysiological processes underlying urinary tract infection and bladder injury. The development of highly sensitive molecular genetic methods, expanded microbiological investigations, and imaging technologies has made it possible to revise established views on the mechanisms of onset and progression of these pathological conditions [1].

One of the most significant shifts in the scientific paradigm has been the fundamental reconsideration of the concept of urine sterility. For a long time, the prevailing view in medical practice was that the urine of a healthy individual should be completely free of microorganisms. However, the findings of contemporary studies have convincingly refuted this assumption. It has been established that the urinary tract possesses its own microbiome, which is a complex community of microorganisms that plays a certain role in maintaining local immunity and homeostasis. This fundamental revision of basic concepts has naturally led to a transformation in views on a number of key aspects of urological practice. In particular, concepts regarding the etiological structure of chronic cystitis have changed substantially. Whereas the bacterial theory previously predominated, the multifactorial nature of the disease pathogenesis is now recognized, including the potential role of viral agents, fungal flora, as well as dysbiotic alterations in the bladder microbiome [2, 3].

Such an evolution of scientific views is inevitably reflected in clinical practice as well. Approaches to the diagnosis of chronic recurrent cystitis are being reconsidered from routine laboratory tests to comprehensive investigations that make it possible to assess not only the presence of pathogens, but also the state of local immunity, morphological tissue changes, and biochemical markers of inflammation. Accordingly, therapeutic strategies are also being transformed: from the standard prescription of antibacterial agents to personalized treatment regimens that take into account the individual characteristics of the microbiome, the patient's immune status, and the specific features of the pathogenetic mechanisms underlying the disease [4, 5].

Aim. Based on the published literature, to identify the specific features of the course of chronic recurrent cystitis and to determine the differential diagnostic criteria for the development of an infectious process caused by various agents, taking into account their pathogenic properties.

Literature search strategy. An electronic literature search was carried out using the Medline, PubMed, EMBASE, and the Chinese databases CNKI and Wanfang to identify studies relevant to the analysis of CRC published up to December 2025. The following keywords were used: "chronic recurrent cystitis," "urinary tract infection," "viral cystitis," and "urothelium."

The literature search showed that contemporary medical literature demonstrates sustained interest in the study of chronic recurrent cystitis as a significant clinical problem. Analysis of published studies indicates that the concept of the bacterial nature of the disease has traditionally dominated the scientific community, since most studies focus on the role of pathogenic microflora in the formation of the etiopathogenetic mechanisms of bladder inflammation. At the same time, an important trend has emerged in recent years: the number of studies aimed at investigating the viral component in the development of infectious and inflammatory processes of the urinary tract is increasing. This opens new perspectives for understanding the multifaceted nature of chronic recurrent cystitis and calls for a revision of established diagnostic and therapeutic approaches [1, 2].

From a clinical perspective, chronic recurrent cystitis occupies one of the leading places in everyday urological practice, especially in the outpatient setting. Epidemiological observations show that the disease is most commonly diagnosed in sexually active women of reproductive age. This predilection is explained by a combination of anatomical and physiological features of the female body: a shorter and wider urethra, together with the close proximity of natural reservoirs of opportunistic microflora, namely the vagina and rectum, creates favorable conditions for the easier penetration of infectious agents into the urinary tract. As a result, women have a substantially increased risk of developing both primary episodes of cystitis and its recurrent forms. The course of chronic recurrent cystitis represents a complex, multifaceted pathological process characterized by marked resistance to therapeutic interventions. Even when all clinical recommendations are followed, prescribed medications are taken regularly, and preventive measures are observed, achieving stable, long-term remission often remains a difficult task. This is due to a combination of pathogenetic mechanisms that sustain chronic inflammation in the bladder wall and prevent complete restoration of its functional activity [6, 7].

The clinical presentation of the disease is characterized by marked severity and persistence of symptoms, substantially affecting the patient's quality of life. Intense pain comes to the forefront; it may be constant or fluctuate in intensity, is localized predominantly in the suprapubic region, but may also radiate to the perineum, lower back, or inner thighs. The nature of the pain varies from persistent dragging discomfort to acute burning or cutting episodes, especially aggravated by bladder filling or during urination. Pain intensity often reaches a level that significantly limits daily activity and disturbs sleep and the patient's psychologic and emotional state. Alongside pain, persistent urinary disturbances develop, forming a typical clinical triad of manifestations. Increased urinary frequency becomes an integral feature of the disease. The patient is forced to visit the toilet repeatedly throughout the day, including at night, which disrupts her usual routine and causes pronounced discomfort. Urination is accompanied by painful sensations ranging from mild burning to severe cutting pain, which may lead the woman to consciously restrict fluid intake, thereby aggravating metabolic disturbances in the bladder tissues [8].

Particularly problematic is the phenomenon of urgency, which is sudden, irresistible urges to empty the bladder that arise without warning and create a risk of uncontrolled urination. This not only physically exhausts the patient, but also imposes serious psychological limitations, forcing her to plan any movement with regard to the proximity of sanitary facilities. The clinical picture is further complemented by a persistent sensation of incomplete bladder emptying that remains even after urination has been completed. This sensation creates the illusion of the need for repeated voiding, thereby perpetuating the vicious circle of urinary symptoms [7, 8].

A significant factor aggravating the course of the disease is the state of immunocompromise identified in a considerable proportion of patients with chronic recurrent cystitis. This involves not only systemic immune disturbances, but also local dysfunction of the immune defense mechanisms of the urinary tract. As a result, the body's ability to effectively eliminate pathogenic agents is reduced, regulation of the inflammatory response is impaired, and regeneration of the damaged urothelium is delayed [9].

Immune disturbances form a peculiar vicious circle in pathogenesis: chronic inflammation itself exerts an immunosuppressive effect, further weakening the protective mechanisms of the bladder. This creates favorable conditions for persistence of the infectious or autoimmune process, prevents complete tissue repair, and makes it difficult to achieve clinical improvement even with adequate therapy. As a result, the disease acquires a fluctuating course with alternating periods of exacerbation and incomplete remission, and each subsequent exacerbation often proceeds with greater symptom severity and lower responsiveness to treatment [10, 11].

At present, cystoscopy is one of the principal methods for the diagnosis and differential diagnosis of various bladder diseases. In chronic recurrent cystitis, characteristic morphological changes in the bladder mucosa are identified, reflecting a chronic inflammatory process with alternating periods of exacerbation and partial remission. The visual picture is usually characterized by polymorphism and heterogeneity of changes across the bladder surface. The mucosa demonstrates signs of chronic inflammation: it loses its normal pale pink color and transparency, acquiring diffuse hyperemia of varying intensity, from moderate pink discoloration to deep red. In some cases, the hyperemia is focal, being concentrated predominantly in the region of the bladder neck, the trigone, or around the ureteral orifices; these areas most susceptible to microbial invasion and mechanical irritation. The mucosal surface often appears edematous and friable, with loss of its usual sheen and smoothness. In areas of pronounced inflammation, granularity may be observed, caused by lymphoid infiltration and hyperplasia of subepithelial elements.

In some areas, the mucosa may appear thickened and swollen, creating an impression of a "velvety" texture during examination in transmitted light [12, 13].

A characteristic feature of chronic recurrent cystitis is the presence of areas of epithelial metaplasia. Squamous metaplasia is the most common type and is visually manifested as whitish, slightly elevated lesions with indistinct borders. These areas may be isolated or may coalesce, forming more extensive fields of altered mucosa. In rare cases, areas of glandular metaplasia are detected, appearing as small cyst-like formations or elevated nodules with a smooth surface. During exacerbation, punctate hemorrhages, petechial bleeding, or small subepithelial hematomas may be identified against the background

of hyperemic and edematous mucosa. In some cases, areas of superficial erosion with a dull base and indistinct margins, lacking a normal epithelial covering, are observed. On contact with the cystoscope, such areas may bleed easily, indicating increased vascular fragility and impaired tissue trophism. The vascular pattern of the mucosa undergoes substantial changes. The normal network of thin, evenly distributed vessels is replaced by chaotically arranged, tortuous, dilated vessels. In zones of pronounced hyperemia, the vascular pattern may be obscured because of the intense coloration of the tissues, whereas at the periphery of inflammatory foci the vessels appear markedly dilated, with multiple anastomoses and glomerulus-like formations. The ureteral orifices usually retain their normal round shape; however, the surrounding mucosa is often edematous and hyperemic. In some cases, slight deformation of the orifices may be observed due to periureteral fibrosis or edema. However, pronounced structural changes such as strictures or gaping are uncharacteristic of chronic cystitis and require exclusion of other diseases [14-16].

In a number of cases, deposits of salts or fibrinous overlays are found on the mucosa, especially in areas of erosion or microtrauma. These deposits may appear as whitish or yellowish films partially covering the mucosal surface. With prolonged disease duration, small pseudopolyps may form as focal mucosal proliferations in the setting of chronic inflammation. They have a smooth surface, a broad base, and usually do not exceed 3-5 mm in diameter.

In the phase of partial remission, the severity of hyperemia and edema decreases; however, signs of structural remodeling of the mucosa persist, including areas of metaplasia, irregularity of the vascular pattern, and mild granularity.

In some cases, small scars or fibrotic areas form at the sites of previous erosions or hemorrhages, giving the mucosa a mosaic appearance [16].

As for treatment, it represents a multilevel, differentiated strategy that requires consideration of the etiopathogenetic mechanisms of the disease, the individual characteristics of the patient, and the dynamics of the clinical process. The key objective of therapy is not episodic symptom relief, but the achievement of stable remission through correction of the underlying disturbances and prevention of recurrent exacerbations. The therapeutic approach is based on comprehensive etiopathogenetic correction. Identification and elimination of predisposing factors are of primary importance, including anatomical abnormalities of the urinary tract, urodynamic disturbances, hormonal imbalance, especially in peri- and postmenopausal women, dysbiosis of the vaginal biotope, and chronic infectious foci in adjacent organs. Without addressing these pathogenetic links, any drug therapy remains symptomatic and does not prevent recurrences [17].

Antibacterial therapy is used strictly when indicated, namely in the presence of confirmed bacteriuria and clinical signs of exacerbation. Drug selection is based on microbiological testing with determination of pathogen susceptibility. Preference is given to agents with high tropism for bladder tissues and the ability to maintain stable concentrations in the urothelium. In the acute phase, courses of standard duration are prescribed (1-5-7 days); however, in recurrent disease, prolonged regimens or low-dose prophylactic antibiotic therapy lasting 3-6 months may be considered. This approach reduces the frequency of exacerbations, but requires careful monitoring to prevent the development of resistance and dysbiosis [18, 19].

One of the key treatment directions is restoration of the protective glycosaminoglycan layer of the urothelium. Deficiency of mucopolysaccharides renders the mucosa vulnerable to bacterial adhesion and chemical irritation. Barrier repair is achieved by instillations of hyaluronic acid, chondroitin sulfate, or heparin preparations, which form a protective film on the epithelial surface. Oral glycosaminoglycan formulations complement the local effect by stimulating endogenous regeneration. This stage of therapy is of long-term importance, since glycocalyx stability directly correlates with the frequency of recurrences [20].

Anti-inflammatory correction is aimed at controlling chronic inflammation and reducing detrusor overactivity. Nonsteroidal anti-inflammatory drugs are used in short courses to reduce edema and pain, although their use is limited by the risk of ulcerogenic effects. A safer option is represented by herbal preparations with antiexudative and antispasmodic effects, such as extracts of bearberry, lingonberry leaf, and cranberry, which complement the main therapy without causing significant adverse reactions [21, 22].

In patients with hormonal disturbances, particularly estrogen deficiency, local estrogen therapy is of fundamental importance. Creams or vaginal suppositories containing estriol restore the trophism of the urogenital epithelium, increase glycogen production, and promote lactobacillus colonization, thereby strengthening local immunity and preventing bacterial invasion. Systemic hormonal correction is prescribed strictly when indicated after consultation with a gynecologist, taking into account contraindications and the individual risk profile [23]. The most extensively studied minimal daily dose of local estriol included in the meta-analyses cited by experts in the international clinical guidelines of IMS 2016 [24], NAMS 2020 [25], and EMAS 2021 [26] is 0.5 mg.

Physiotherapeutic methods are integrated into the treatment regimen to improve microcirculation, stimulate reparative processes, and reduce chronic inflammation. Electrophoresis with anti-inflammatory agents, such as dioxidine and lidocaine, applied to the suprapubic region ensures targeted drug delivery. Magnetotherapy and low-intensity laser radiation activate capillary blood flow and cellular metabolism, thereby accelerating urothelial regeneration. Hyperbaric oxygen therapy improves tissue oxygenation, which is particularly important in the presence of long-standing erosions and areas of metaplasia [27].

Non-pharmacological interventions constitute an integral part of the long-term strategy. An adequate hydration regimen (2-2.5 L of fluid per day) promotes natural bladder flushing and reduces the concentration of irritating metabolites. Dietary restrictions, including avoidance of spicy and acidic foods, caffeine, and alcohol, reduce chemical irritation of the mucosa. Bladder training and pelvic floor muscle exercises performed under biofeedback guidance help correct detrusor dysfunction and eliminate urgent urges to void. Psychotherapeutic support helps reduce anxiety, which often aggravates symptoms through neurogenic spasm [28].

In cases where recurrences are caused by anatomical defects, such as pelvic organ prolapse, urethral strictures,

or bladder diverticula, surgical correction becomes a necessary component of treatment. Surgical interventions are aimed at restoring normal urodynamics and eliminating zones of urinary stasis, which substantially reduces the risk of recurrent infections [29].

Based on published data, it has been shown that in women a chronic inflammatory process in the bladder may be not the cause, but rather the consequence of functional disorders of the lower urinary tract associated with impaired neural regulation of these organs. The development of inflammation in the bladder wall against the background of lower urinary tract dysfunction may, in turn, perpetuate bladder and/or urethral dysfunction. In long-standing lower urinary tract dysfunction, the inflammatory process that has developed progresses over time; accordingly, without correction of impaired bladder and urethral function, antibacterial and anti-inflammatory therapy may prove insufficient. To eliminate functional disorders of the lower urinary tract and improve the function of these organs, drugs affecting the sympathetic and parasympathetic components of the autonomic nervous system may be used. Thus, pathogenetic treatment of chronic cystitis is based on breaking the "vicious cycle" of dysfunction-inflammation [30, 31].

Prevention of recurrence requires long-term monitoring and adaptation of therapy. Regular assessment of the urinary and vaginal microbiocenosis makes it possible to detect imbalance in a timely manner. Preventive courses of herbal preparations or low-dose antibiotics, when individually indicated, reduce the frequency of exacerbations. Intimate hygiene is also important, as is the treatment of concomitant infections, such as vaginitis and pyelonephritis, which may trigger recurrences. Treatment efficiency is assessed using a set of criteria: symptom dynamics, including urinary frequency, pain, and urgent urges to void, microbiological test results, cystoscopic findings, including reduction of hyperemia, edema, and erosions, as well as the patient's quality of life. In the absence of response to a standard regimen, more in-depth diagnostic evaluation, including urothelial biopsy and urodynamic testing, is required to exclude interstitial cystitis, neoplastic processes, or other rare disease entities [29].

Thus, analysis of contemporary data demonstrates that chronic recurrent cystitis cannot be regarded as an isolated inflammation of the bladder. Rather, it is a condition involving numerous factors in its development, including microbial composition, the state of local and systemic immunity, hormonal background, anatomical features of the urinary tract, concomitant diseases, and even the patient's psychologic and emotional status. This multicomponent pathogenesis explains why standardized treatment regimens often prove insufficiently effective and why recurrences occur even in the setting of formally adequate therapy. The issue of diagnosis deserves particular attention. As studies show, routine methods, including urinalysis and urine culture, are often insufficient to identify the true causes of recurrence. A comprehensive approach is required, including advanced microbiological investigation, cystoscopy with targeted biopsy, urodynamic testing, and, where necessary, molecular genetic methods. Only such detailed evaluation makes it possible to differentiate bacterial, interstitial, and mixed variants of chronic recurrent cystitis, which is critically important for selecting an appropriate therapeutic strategy [28].

In the treatment of chronic recurrent cystitis, a clear trend toward personalization has emerged. Moving away from standardized regimens and toward individualized drug selection based on etiology, disease phase, and concomitant conditions substantially increases the likelihood of achieving long-term remission. At the same time, not only pharmacotherapy, including antibacterial, pathogenetic, and symptomatic treatment, but also non-pharmacological methods, such as lifestyle modification, dietary therapy, physiotherapeutic interventions, and bladder training, are of key importance. A multidisciplinary approach is especially important in the management of peri- and postmenopausal patients, where close collaboration between the urologist and the obstetrician-gynecologist is required to correct hormonal disturbances.

It should also be noted that many aspects of chronic recurrent cystitis remain the subject of scientific debate. There is still no consensus regarding the role of viral agents in the genesis of recurrences, and the mechanisms of their formation and their influence on infection persistence remain insufficiently studied. The criteria for selecting patients for long-term prophylactic antibiotic therapy also require further clarification, as do the optimal regimens for the use of agents that restore the glycosaminoglycan layer of the urothelium [22, 23].

Thus, chronic recurrent cystitis represents a clinical reality in which each case requires careful analysis and an individualized approach. In view of all the presented data, it appears logical and necessary to revise the clinical guidelines "Cystitis in Women" so that they address the various possible pathogenetic aspects of CRC development, not only of bacterial origin, with presentation of an expanded diagnostic work-up and options for non-antibacterial drug treatment.

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