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Current Status and Advances in the Diagnosis and Management of Cystitis Glandularis

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Abstract: Cystitis Glandularis is a proliferative and metaplastic lesion of the bladder mucosa. Studies have found that Cystitis Glandularis is associated withthe occurrence and development of bladder cancer. In recent years, its incidence has been increasing, it is prone to recurrence after treatment, and theprognosis is poor. The etiology of Cystitis Glandularis is complex, and its clinical manifestations lack specificity. However, clinical diagnostic and therapeutic strategies have advanced rapidly. This article aims to systematically review the new advances in its diagnosis and treatment, providing a reference for clinical practice.

Cystitis glandularis (CG) is a proliferative and metaplastic disorder of the bladder mucosal epithelium. Previous studies reported a prevalence rate of CG ranging from 0.1% to 1.9% [1]. In recent years, with the application of cystoscopy combined with tissue biopsy and imaging in urological practice, the number of clinically confirmed CG cases has significantly increased, with a higher incidence in women than in men. Cystoscopy reveals lesions primarily located in the bladder trigone, followed by the bladder neck and peripelvic area [2].

1. Etiology and Pathogenesis of Cystitis Glandularis

Cystitis Urinary tract infections (UTIs) are closely associated with adenomatous cystitis, with chronic UTIs being recognized as a significant contributing factor. Clinical studies have demonstrated that CG incidence significantly increases in UTI patients, particularly when combined with lower urinary tract obstruction and infection [3-5]. Inhibition of chronic inflammatory responses has been shown to effectively improve CG conditions [6]. CG etiology is also linked to urinary tract obstructions caused by the following factors [7]: female urethral external meatus malformations, urethral external meatus polyps, hymen fusion syndrome and hymenal hood, parurethral cysts, and male conditions such as benign prostatic hyperplasia, bladder neck hypertrophy, urethral stricture, and urinary calculi [8]. Additionally, most CG patients lack prior UTI, stone, or obstruction history,

suggesting potential exposure to uncommon triggers like vitamin deficiencies, metabolic toxins, hormonal imbalances, allergens, and carcinogens [9]. Recurrent UTI pathogens such as Escherichia coli, Klebsiella, and Proteus species are directly associated with CG development [10]. Chronic viral infections, including human papillomavirus (HPV), have also been confirmed as pathogenic factors [11]. Chronic urinary tract infections and inflammatory irritation lead to hyperplasia of bladder basal layer cells, which proliferate into the submucosal layer to form Von Brunn's nests. When inflammatory stimulation persists, cystic cavities containing clear fluid can be observed, a stage termed cystic cystitis (cystitis cystica, CC). When cyst wall cells evolve into columnar epithelial cells with characteristics of intestinal epithelial cells, this stage is called CG. CC and CG are considered different pathological stages of the same disease [1]. Routine pathological examinations may simultaneously detect Brunn's nests, CC, and CG [12]. Von Brunn's nests are also observed in 85%-95% of healthy individuals, thus regarded as normal proliferative lesions of bladder mucosa [13]. However, they may also be detected in malignant tumors such as inverted papilloma, low-potential malignant inverted urothelial carcinoma, non-invasive urothelial carcinoma, and invasive urothelial carcinoma [14]. Therefore, clinicians should exercise caution in differential diagnosis to prevent misdiagnosis or missed diagnosis. The pathogenesis of CG currently involves three main mechanisms: (1) Epithelial transformation theory: Prolonged exposure to pathogenic factors may induce urothelial epithelial transformation into glandular epithelium and intestinal metaplasia. Currently, the epithelial transformation theory is widely accepted by most scholars; (2) Embryonic origin theory: During bladder development, congenital anatomical or abnormal factors may cause ectopic embryonic remnants to form glands, leading to cystic goiter (CG); (3) Pund's degeneration theory: Epithelial cells lose normal function and regress to a previously differentiated stage [15].

2. Classification and Diagnosis of Cystitis Glandularis

Foreign researchers have classified CG into two subtypes based on morphological and behavioral characteristics: classical cystitis glandularis (CGTP) and intestinal type cystitis glandularis (CGIT). CGTP is characterized by columnar epithelial cell nests forming glandular structures within the bladder's intrinsic layer, while CGIT exhibits similar glandular structures in the intrinsic layer but contains abundant mucin-secreting goblet cells in the inner epithelium [16]. Common clinical diagnostic methods include routine urinalysis, urine exfoliative cytology, urinary markers, ultrasound, and cystoscopy. A 2023 study confirmed that multiparametric MRI can achieve non-invasive diagnosis of CG by quantifying characteristic edema signals in the bladder submucosa [17]. Zhou TL et al. recently discovered that single-cell RNA sequencing (scRNA-seq) can accurately differentiate adenocystitis from early-stage bladder cancer (BC) using molecular markers like UPK1A/TP53 [18]. Clinically, cystoscopy combined with tissue biopsy remains the gold standard for CG diagnosis. During examinations, multiple sampling sites should be considered to prevent missed diagnoses due to insufficient sampling. Cystoscopy can further classify CG into low-risk and high-risk types. 1. Low-risk types manifest as: (1) no significant changes or chronic inflammatory alterations; (2) solitary or small-scale follicular changes; (3) rough or granular mucosa. 2. High-risk types manifest as: (1) Papillomatous changes; (2) Extensive intestinal metaplasia with large-scale follicular or pavement-like patterns; (3) Adenomatous intestinal lesions resembling adenomas (known as the erythematous type), which exhibit strong malignant potential [19]. When patients have urinary tract infections, cystoscopy biopsy is not recommended to prevent infection spread. In such cases, imaging studies can provide preliminary diagnosis. These can be categorized into four basic types: (1) Papillary protrusions on the bladder wall with relatively regular margins, showing cystic changes and calcified nodular elevations; (2) Lawn-like localized thickening of the bladder wall,

typically not exceeding 3cm, presenting as smooth or serrated flat-ridge thickening; (3) Diffuse thickening of the bladder wall with uneven thickness and serrated inner wall changes; (4) Mixed type combining diffuse and flat-ridge thickening of the inner wall [20].

3. The Relationship between Cystitis Glandularis and Bladder Cancer

The carcinogenicity of CG has long been debated. Some scholars argue that CG is a benign condition that does not become malignant, while others propose that CG shares common genetic factors with BC and may undergo malignant transformation under carcinogen exposure. Studies have shown elevated expression of P53 and Ki-67 in CGIT compared to ordinary cystitis patients, with reduced P53 and Ki-67 levels in the bladder mucosa of CG patients after treatment [1,21]. Research also indicates that during the progression from CG to BC, hTERT, P53, PCNA, Cox-2, and UCA-1 exhibit increased expression in tissues. Furthermore, CG patients demonstrate significantly higher protein expression levels of CCND1, EGFR, and AR in both CG and BC compared to healthy individuals [22]. Therefore, elevated expression of P53, hTERT, and PCNA in high-risk CG may indicate potential malignancy. Li Zhongxing et al. [16] identified through Spearman's test that increased COX-2 expression correlates with upregulation of anti-apoptotic protein Bcl-2 in CG patients. This suggests that overexpression of COX-2 and Bcl-2 may represent precancerous lesions in CGIT. However, most bladder cancers are urothelial carcinoma, with only a small proportion being squamous cell carcinoma or adenocarcinoma. To date, there are no clinical reports documenting direct transformation of CG into BC. In 2023, Jeon J et al. [23] analyzed 110 patients and found that imaging studies showed that 21 cases (22.3%) had urinary tract stones, 16 cases (17.0%) had hydronephrosis, 6 cases (6.4%) were due to the same CG, 10 cases were due to other causes, and 4 patients developed urinary tract malignancies at 6, 12,17, and 36 months postoperatively, all of which were urothelial carcinomas, possibly due to being overlooked during initial examinationsor developing subsequently.The incidence of primary bladder adenocarcinoma is very low, and most cases are caused by metastatic infiltration. Current evidence suggests that the risk of CG progressing to BC is very low but not zero.

4. Treatment of Cystitis Glandularis

4.1 Etiological Treatment

The clinical symptoms of CG share certain similarities with those of overactive bladder (OAB). Some patients can be diagnosed with CG through cystoscopy, but the diagnosis of OAB is often overlooked, leading to potential clinical misdiagnosis [24]. Additionally, approximately 40% of patients with pelvic lipomatosis (PL) also have CG. Pelvic lipomatosis is a benign condition; however, due to the compression of surrounding pelvic tissues and organs caused by excessive fat proliferation, it can lead to obstruction of bladder lymphatics and veins, resulting in nonspecific lower urinary tract symptoms [25]. Therefore, while treating CG, it is essential to address OAB, PL, and other conditions that cause lower urinary tract obstruction and infection. Furthermore, Tong Xianli et al. found that after addressing the underlying diseases and eliminating predisposing factors, most patients can recover spontaneously without requiring specific treatment [26]. Thus, it is recommended to actively identify and eliminate the causes and triggers in low-risk CG patients. For patients with chronic infections, anti-inflammatory therapy should be administered [19].

4.2 Operative Treatment

Several surgical approaches are commonly used to treat CG, including transurethral laser ablation of bladder lesions, transurethral electrocautery of bladder lesions, transurethral resection of bladder tumor (TURBT), and partial cystectomy [27]. TURBT is the most frequently used procedure in clinical practice, offering the advantages of high safety, minimal trauma, and rapid postoperative recovery [28]. However, high-risk CG still exhibits a high recurrence rate after transurethral resection. Repeated recurrences and multiple surgeries can easily involve the bilateral ureteral orifices, leading to distal ureteral stenosis and subsequent renal impairment. The currently common clinical treatment involves resection of bladder mucosal lesions combined with indwelling double-J ureteral stents.For cases where locating the ureteral orifice is difficult during surgery, Ma Jiaxing et al. [29] analyzed 42 patients with non-muscle-invasive bladder tumors involving the ureteral orifice and 18 patients with CG. All patients underwent TUR with resection extending deep into the muscle layer. All 18 CG patients underwent ureteral orifice resection. When searching for the ureteral orifice, the resection should be performed layer by layer along the direction of ureteral peristalsis, identifying the "concentric circular" muscle tissue. A Zebra guidewire was then used as a guide to locate the ureteral mucosa, followed by the placement of a double-J stent along the guidewire. None of the patients with indwelling double-J stents experienced complications such as hydronephrosis or ureteral stenosis postoperatively. If the ureteral orifice cannot be identified during surgery and hydronephrosis is significant, protecting renal function should be the primary consideration, and ureteral reimplantation on the affected side may be considered [30].

4.3 Bladder Instillation Chemotherapy

Initially, most scholars regarded CG as a precancerous lesion, and its clinical management mirrored that of bladder cancer, primarily involving surgical treatment combined with intravesical chemotherapy. Commonly used instillation drugs included mitomycin, sodium hyaluronate, and pirarubicin. Glycosaminoglycans (GAG) cover the bladder epithelium to block various irritants, preventing the progression of bladder inflammation. Endogenous hyaluronic acid (HA) is a key component of GAGs. Ni Y et al [31] found that after intravesical instillation of sodium hyaluronate (SH) in patients with CCEG, the patients' bladder epithelial layer was intact, the number of Brunn's nests and cysts decreased, and inflammatory cell infiltration was reduced.However, Wu Shuiqing et al [32] found that TURBT alone for CG had better efficacy compared to TURBT combined with mitomycin instillation chemotherapy. Su Xing et al[33]also concluded that intravesical chemotherapy instillation did not provide significant benefit to patients. Intravesical chemotherapy instillation is considered overtreatment, not only increasing the psychological and economic burden on patients but also causing secondary damage to the bladder, which is detrimental to postoperative recovery. Based on updated evidence-based medicine, traditional intravesical therapies (such as mitomycin C, BCG, etc.) are no longer recommended as first-line treatments for CG due to unclear efficacy and the risk of bladder mucosal damage [19].

4.4 Medication

A case of successful CG treatment with celecoxib was reported by Takizawa N. Takizawa N et al[34] clinically observed a CG patient who experienced recurrence one year after initial treatment with TURBT combined with antibiotics, non-steroidal anti-inflammatory drugs, and hormone pulse therapy. The patient recurred again six months after a second TURBT combined with prednisone treatment. However, no recurrence was observed after the third TURBT combined with celecoxib treatment. Hong Tao et al[35]found that COX-2 (cyclooxygenase-2), PTGS2

(prostaglandin-endoperoxide synthase 2), and HRH1 (histamine H1 receptor) were highly expressed in CG patients. For symptomatic extensive chronic cystitis (CG), Masato Yanagi et al[36] demonstrated the effectiveness of preoperative administration of COX-2 inhibitors as a neoadjuvant treatment strategy. The study showed that a 6-week course of COX-2 inhibitor treatment significantly reduced bladder lesions and created more favorable conditions for subsequent TURBT. Therefore, this regimen offers the dual benefits of symptom relief and disease control, representing an important component in the multimodal treatment of CG. COX-2, a key enzyme in the inflammatory response, catalyzes the conversion of arachidonic acid to prostaglandins, which have vasodilatory effects and promote lymphocyte migration, thereby exacerbating the inflammatory process. Celecoxib, as a selective COX-2 inhibitor, can reduce prostaglandin production by inhibiting this pathway, providing a theoretical basis for its potential application in CG treatment.

A successful case of treating CG with oral steroids was reported by Yuksel OH [37]. A patient who underwent bladder mass resection combined with mitomycin and BCG instillation therapy showed no improvement. Subsequently, oral prednisone was administered for 6 months, and follow-up examination revealed symptomatic improvement. It was suggested that oral steroids might be a good option for patients resistant to other treatments. However, due to its numerous adverse effects, such as osteoporosis and reduced immune response, and the lack of recent reported cases on steroid treatment for CG, the efficacy of steroids for CG still requires further research.

Submucosal bladder injection of botulinum toxin A (BoNT-A) works through the following mechanism: its light chain cleaves synaptosome-associated protein 25 (SNAP-25) in presynaptic neurons, thereby inhibiting the release of the neurotransmitter acetylcholine by disrupting vesicle fusion with the neuronal cell membrane. This leads to flaccid muscle paralysis, effectively relieving urinary frequency and urgency, and improving the bladder urothelial barrier function. Consequently, submucosal BoNT-A injection is regarded as a promising treatment option [38].

Pachymic acid (PA) is a bioactive component isolated from Poria cocos, possessing potential benefits including anti-inflammatory and antioxidant effects. In CG, PA exhibits strong binding affinity for TNF and TP53 proteins. In animal studies, PA treatment reduced levels of IL-1, IL-6, and lactate dehydrogenase content in mouse bladders, downregulated TNF-α, and upregulated TP53 protein. Experimental results identified key anti-CG biological targets and mechanisms of PA. More notably, these key pharmacological targets of PA for CG have been screened and validated through computational and experimental analyses [39]. This research reveals the core biological targets and potential mechanisms of PA action in CG, providing a basis for its therapeutic application.

In recent years, studies have gradually found that Vitamin C (VC) has certain efficacy in treating CG. The anti-CG mechanism of VC is achieved by modulating molecular pathways such as the tumor necrosis factor (TNF) signaling pathway. This hypothesis is further supported by clinical and preclinical research. Significant elevation of inflammatory mediators such as TNF-α, IL-6, and c-Jun was observed in CG patients, accompanied by detectable urinary tract infections. Beneficially, VC supplementation in a CG animal model resulted in the downregulation of endogenous TNF-α, IL-6, and c-Jun expression in the blood and bladder tissues of mice [40], validating the mechanism by which VC exerts its therapeutic effect through the regulation of inflammatory pathways.

5. New Advances in Clinical Diagnosis and Treatment

In recent years, research in the field of urology has significantly focused on chronic granulomatous (CG) disease, delving into the molecular mechanisms of signal pathway regulation by key inflammatory factors (such as IL-6, IL-8, TNF-α) and abnormal expression or epigenetic modifications of specific genes (such as TP53, EGFR). These studies aim to provide a theoretical

foundation for developing novel precision treatment strategies that target inflammatory responses and reverse pathological progression.

Recent basic research has shown that the expression of IL-1, IL-6, and TNF-α in CG lesioned tissues is significantly higher than in normal bladder mucosa, driving the inflammatory cascade [41]. Meanwhile, Kitamura et al. confirmed through animal experiments that rosmarinic acid (RA) can targetedly inhibit the COX-2/IL-6 pathway [42], providing a novel strategy for targeted anti-inflammatory therapy of CG and suggesting its potential efficacy in treating cystitis. However, the clinical application of rosmarinic acid for cystitis has not been effectively implemented, and its specific therapeutic effects require further validation.

A 2024 study based on network pharmacology identified 16 overlapping genes between fucoidan and CG, clarifying six core targets, including interleukin-6 (IL-6), tumor necrosis factor (TNF), interleukin-1B (IL-1B), matrix metalloproteinase-9 (MMP-9), interleukin-10 (IL-10), and matrix metalloproteinase-2 (MMP-2), as well as the biological processes and signaling pathways through which fucoidan acts against CG. The findings suggest that fucoidan can effectively inhibit inflammatory responses, reduce cytokine infiltration, and enhance immune capacity to combat CG. However, research on fucoidan for treating CG remains at the preclinical stage, and its clinical efficacy requires further validation through in vitro and in vivo experiments as well as randomized controlled trials [43].

According to the latest research progress, low-intensity extracorporeal shock wave therapy (Li-ESWT) [44] activates resident stem cells in bladder tissue to promote their proliferation, differentiation, and migration to inflamed areas. It stimulates adipose-derived stem cells (ADSCs) to secrete vascular endothelial cell growth factor GF (VEGF) and nerve growth factor GF (NGF), thereby improving angiogenesis and nerve regeneration. By downregulating α-sMA and collagen expression, it exerts anti-fibrotic effects. Additionally, it regulates TRPV channels, RhoA/RhoK signaling pathways, and Gq protein to alleviate bladder outlet obstruction (BOO)-related dysfunction. Combined with stem cell therapy, these approaches synergistically promote tissue regeneration. A 2025 systematic review confirmed its significant improvement in urinary frequency and pain symptoms for patients with overactive bladder (OAB) and interstitial cystitis (IC/BPS) [45]. However, the mechanisms of signal pathway interactions and paracrine synergy remain incompletely elucidated, necessitating large-scale validation targeting different subtypes of cystitis.

A 2025 study revealed that pivmecillinam [46] demonstrates potent antibacterial activity against common pathogens like Escherichia coli (E. coli), with favorable clinical cure rates. Its narrow spectrum of activity helps reduce antimicrobial resistance, making it effective for treating Clostridium-related infections caused by E. coli. However, the drug has limited coverage against non-fermenting Gram-negative bacteria and may not be suitable as monotherapy for complex infections. Therefore, clinicians should cautiously expand its use in specific patient populations while addressing the need for cost-effective combination therapies for complex infections.

Glycyrrhetinic Acid (GA) [47] exerts its therapeutic effect on Cystitis Glandularis (CG) by targeting and inhibiting the PTGS2/MUC1 signaling axis, thereby alleviating bladder inflammation. This conclusion is supported by multi-omics evidence: clinical sample analysis revealed significant overexpression of PTGS2 and MUC1 in CG; animal experiments validated that GA effectively reduces inflammatory factors (TNF-α, IL-6) while directly downregulating the expression of these two key targets. This research clarifies GA's mechanism of action at the genetic level, laying a solid theoretical foundation for developing targeted treatment strategies for CG and pointing towards new research directions.

6. Prospects

Given the incomplete understanding of chronic granulomatous (CG) etiology and pathogenesis, coupled with a lack of high-quality evidence-based medical support for treatment, there remains no standardized protocol for CG management. Current clinical practice involves conservative treatment for low-risk CG cases, focusing on addressing obstructive factors and chronic infections. For high-risk CG patients who show poor response to conservative therapy, surgical intervention is recommended followed by postoperative monitoring. However, these approaches neither provide a cure nor prevent recurrence, which significantly impacts patients' quality of life and psychological well-being. Clinicians should prioritize mental health care through timely psychological counseling to alleviate anxiety and depression, with anti-anxiety medications prescribed for severe cases. Novel therapeutic agents for CG remain under investigation, including studies exploring mucosal immune microenvironment analysis, incorporating histological and psychosomatic endpoints in efficacy evaluation, and advancing treatment strategies toward precision-targeted regulation. These research directions require further validation, observation, and long-term follow-up studies.

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