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Advances in Integrated Traditional Chinese and Western Medicine Diagnosis and Treatment of Peritoneal Fibrosis Associated with Peritoneal Dialysis: Mechanisms, Strategies, and Prospects

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Abstract: ***Peritoneal dialysis (PD) is a vital renal replacement therapy for end-stage renal disease (ESRD) and is widely adopted globally because of its advantages, including home-based convenience, preservation of residual renal function, and improved quality of life. However, peritoneal fibrosis (PF) complicating long-term PD significantly compromises the efficacy of the treatment and the quality of survival of patients. Currently, both Western medicine and traditional Chinese medicine (TCM) alone exhibit limitations in managing PF, underscoring the critical importance of an integrated TCM-Western medicine approach. This paper systematically reviews the current status and advances in the integrated Chinese and Western medicine diagnosis and treatment of PD-related peritoneal fibrosis, covering its pathogenesis, Western medical treatment strategies, and Traditional Chinese Medicine syndrome differentiation systems. This review summarizes the research progress in the integrated prevention and treatment of PF, aiming to provide a theoretical basis and practical guidance for clinical practice. The integrated approach plays a vital role in improving the prognosis of patients with PD. Future efforts should focus on further optimizing treatment protocols and deepening research into the mechanisms of action.***

Keywords: Peritoneal dialysis, Peritoneal fibrosis, Integrated Chinese and Western medicine, Diagnostic and therapeutic advances**.**

1. Introduction

Peritoneal dialysis (PD) is a vital renal replacement therapy for end-stage renal disease (ESRD), with over 320, 000 patients globally receiving this treatment, accounting for approximately 13. 5% of the dialysis population worldwide. However, long-term PD often induces peritoneal fibrosis, leading to progressive structural and functional impairment of the peritoneum, ultimately resulting in ultrafiltration failure, the primary cause of PD discontinuation [1]. According to relevant studies, approximately 40% of patients develop peritoneal fibrosis after initiating peritoneal dialysis, with approximately 25% subsequently transitioning to hemodialysis.

The pathological features of peritoneal fibrosis include destruction of the peritoneal mesothelial cell layer, proliferation of the submesothelial matrix, collagen fiber deposition, angiogenesis, and hyalinization of the vascular walls, ultimately resulting in abnormal peritoneal permeability and loss of ultrafiltration function. The pathogenesis of this disease is complex. In recent years, with the rapid advancement of multi-omics technologies, the understanding of peritoneal fibrosis mechanisms has expanded beyond traditional areas, such as inflammatory responses and oxidative stress, to include novel pathways, such as metabolic reprogramming, extracellular vesicle-mediated signaling, and immune microenvironment remodeling. Concurrently, treatment strategies have diversified to include novel biocompatible dialysates, targeted therapeutics, traditional Chinese medicine interventions, and regenerative medicine technologies. Integrated Chinese and Western medicine approaches have unique advantages in slowing PF progression. This review systematically examines the pathophysiology, diagnostic strategies, and therapeutic advances in peritoneal fibrosis from both Chinese and Western medical perspectives, emphasizing novel signaling pathways, biomarkers, and evidence-based integrated treatment protocols to inform clinical practices.

2. Pathogenesis: From Traditional Understanding to Cutting-Edge Exploration

2.1 Advances in Western Medical Mechanism Research

2.1.1 Traditional Mechanistic Understanding

The core mechanism of peritoneal fibrosis involves chronic injury and abnormal repair of the peritoneum following prolonged exposure to biologically incompatible dialysate, uremic toxins, and recurrent infections. Inflammation and cytokine-driven processes are recognized as the central mechanisms. Studies have indicated that prolonged exposure to high-glucose dialysate induces peritoneal mesothelial cells to produce substantial inflammatory mediators, such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6). These factors activate downstream signaling pathways, promoting fibroblast proliferation and extracellular matrix (ECM) deposition [2]. Furthermore, oxidative stress and metabolic abnormalities play significant roles in the development of peritoneal fibrosis. The low pH, high glucose concentration, and accumulation of glucose degradation products, such as advanced glycation end products (AGEs), in conventional dialysates not only directly damage peritoneal tissue but also exacerbate inflammation and fibrosis progression by inducing reactive oxygen species (ROS) production [3]. ECM remodeling and vascular alterations are key pathological features of peritoneal fibrosis. Transforming growth factor-β

(TGF-β), a pivotal pro-fibrotic factor, regulates matrix metalloproteinases (MMPs) and their inhibitors (TIMPs) by activating the Smad2/3 and non-canonical pathways, such as the PI3K/AKT/mTOR pathway. regulating the expression of matrix metalloproteinases (MMPs) and their inhibitors (TIMPs). This leads to excessive ECM deposition, loss of epithelial characteristics in peritoneal mesothelial cells, their transformation into myofibroblasts, and promotion of neovascularization. Consequently, it accelerates the destruction of the peritoneal structure and functional loss, playing a pivotal role in the development of peritoneal fibrosis [4]. Concurrently, dysbiosis of the gut microbiota (characterized by reduced Lactobacillus and increased Pseudomonas) has also been implicated in fibrosis progression.

2.1.2 Novel Mechanistic Breakthroughs

Metabolic reprogramming and glycolytic regulation are emerging key mechanisms in peritoneal fibrosis. Studies have revealed that mesothelial cells in patients undergoing long-term peritoneal dialysis exhibit significant glycolytic enrichment, which is positively correlated with the extent of mesothelial-mesenchymal transition. In animal models, peritoneal tissues from peritoneal dialysis mice show upregulation of key glycolytic enzymes (e.g., HK2), and correcting excessive glycolysis effectively inhibits the progression of peritoneal fibrosis [5].

Extracellular vesicles also play a crucial role in mediating peritoneal fibrosis. Studies have indicated that extracellular vesicles derived from injured mesothelial cells or macrophages carry miRNAs (e.g., miR-21-5p and miR-155), lncRNAs (e.g., H19), and protein signaling molecules. These are transferred to neighboring cells, inducing epithelial-mesenchymal transition and fibrosis in the heart. Long-term peritoneal dialysis promotes the increased production and secretion of extracellular vesicles in peritoneal mesothelial cells. The integrin-binding kinase protein within these vesicles is delivered to fibroblasts, promoting their activation and forming a vicious “mesothelial cell-fibroblast” cycle [6].

Immune Microenvironment Remodeling: Single-cell sequencing analysis reveals that long-term PD reshapes the peritoneal immune microenvironment, driving antimicrobial MAIT cells toward a proinflammatory phenotype. Activated MAIT cells secrete inflammatory factors such as IL-17, establishing CCL2-CCR2-dependent immune-mesenchymal cell interaction networks with mesothelial cells. In-depth studies have revealedevealed that this interaction promotes mesothelial-to-mesenchymal transition (MMT) by activating the mTORC1 signaling pathway and enhancing glycolytic metabolic activity [7].

Research has uncovered additional complex networks regarding molecular pathways and gene regulation. Yes-related protein depletion in Gli1-expressing cells significantly attenuates peritoneal fibrosis induced by peritoneal dialysis. YAP knockdown and vitilopent treatment reduced fibroblast-mesenchymal transition markers and inhibited smad2/3 phosphorylation, offering novel therapeutic targets.

2.2 Traditional Chinese Medicine (TCM) Pathogenesis

TCM lacks a specific term for “peritoneal fibrosis.” Based on its clinical manifestations, it may be categorized under “abdominal micro-masses” or “mass accumulation.”

Numerous scholars propose its core pathogenesis involves “underlying deficiency with superficial excess, a mixture of deficiency and excess.” “Underlying deficiency” primarily refers to spleen-kidney yang deficiency. Impaired spleen function leads to internal retention of water and dampness, while impaired kidney qi transformation results in the internal accumulation of damp turbidity. “Superficial excess” involves pathological products such as water-dampness, phlegm-turbidity, blood stasis, and qi stagnation mutually binding, obstructing the abdominal collaterals, and eventually forming masses. The pathological location is the abdominal collaterals, which are closely related to the spleen and kidneys. Long-term retention of peritoneal dialysis fluid constitutes “external dampness,” which easily obstructs the middle jiao, damages spleen yang, and eventually affects the kidneys over time.

Disease progression unfolds dynamically as follows: Early stage: Predominantly qi stagnation and damp obstruction (impaired abdominal collaterals), clinically manifesting as abdominal distension, poor appetite, and fatigue. The middle stage involves phlegm-stasis mutual entanglement (obstruction and stagnation in the abdominal collaterals), characterized by fixed abdominal stabbing pain and peritoneal thickening. The late stage sees spleen-kidney failure, with hardened masses (collateral collapse), severe deficiency of vital energy, and entrenched pathogenic factors, manifesting as aversion to cold, cold limbs, soreness in the lower back and knees, edema, and loss of ultrafiltration function. Microscopic pathological findings highly correlate with TCM pathogenesis: mesothelial cell detachment and disorganized neovascularization correspond to “vessel injury,” while collagen deposition and vascular sclerosis correspond to “stasis obstruction.”

3. Updates in Western Medical Diagnosis and Treatment Strategies

3.1 Advances in Diagnostic Methods and Biomarkers

Imaging studies: Hold significant value in diagnosing peritoneal fibrosis but lack sufficient sensitivity for early stage lesions. Peritoneal balance tests indirectly reflect changes in peritoneal characteristics by assessing solute transport function and serve as common clinical monitoring tools. Peritoneal biopsy, a pathological examination, is the “gold standard” for diagnosing peritoneal fibrosis but is limited in clinical application due to its invasive nature.

Laboratory Tests: Significant progress has been made in biomarker research in recent years. IL-6 and CA-125 levels in the dialysate reflect peritonitis status, whereas mtDNA levels correlate with the severity of membrane damage. The MAUXI machine learning model predicts the risk of peritoneal dialysis failure, and WNT1 gene expression is significantly correlated with the solute clearance rate. Increased integrin-linked kinase-positive extracellular

vesicles may serve as markers of peritoneal dysfunction in PD patients. These novel biomarkers offer the potential for noninvasive diagnosis and early warning.

3.2 Diversification of Treatment Strategies

3.2.1 Optimization of Dialysis Protocols

Biocompatible dialysate is a fundamental strategy for delaying peritoneal fibrosis. Neutral pH, low GDPs PD dialysate: Multiple studies indicate that neutral pH, low GDPs PD dialysate better preserves peritoneal structure and function during long-term dialysis than traditional acidic solutions [8]. Patients using neutral pH and low GDPs dialysate exhibit higher indicators of peritoneal mesothelial cell integrity (e.g., CA125 levels), suggesting superior preservation of mesothelial cell biological activity [9]. Furthermore, this dialysate effectively improves residual renal function and reduces the decline in peritoneal ultrafiltration capacity during prolonged treatment (>12 months) [10].

Osmotic metabolic approaches represent novel glucose-sparing methods in PD, aiming to reduce the intraperitoneal glucose burden through alternative osmolytes. Currently, two osmotic agents are widely used in clinical practice: eicosane-dextrin and amino acid dialysate. Furthermore, adding alanine glutamine to PD solutions may mitigate the harmful effects of chronic PD on the peritoneum [11], while lithium chloride addition shows potential peritoneal protective effects, although further validation is required [12].

3.2.2 Advances in Pharmacological Therapy

Antifibrotic Agents: Currently, multiple antifibrotic drugs are used clinically to prevent and treat peritoneal fibrosis. The tyrosine kinase inhibitor nintedanib may alleviate PF by inhibiting MMT, inflammation, and angiogenesis, demonstrating its potential for preventing and treating PF in patients with PD [13].

Sodium-Glucose Cotransporter 2 (SGLT-2) inhibitors, a novel class of antidiabetic drugs, are also being investigated for the treatment of peritoneal fibrosis. Some studies have indicated that SGLT-2 inhibitors exert hypoglycemic effects in the peritoneum exposed to PD solutions by inhibiting SGLT-2 activity, reducing glucose uptake, and increasing peritoneal ultrafiltration. They may also exert protective effects against high-glucose PD solution-induced PF by inhibiting the TGF-β/Smad signaling pathway. However, other studies suggest that these drugs may not reduce peritoneal glucose uptake and could potentially impair residual renal function in patients undergoing PD [14], leaving their efficacy controversial.

Estrogen Receptor Inhibitors: Tamoxifen effectively inhibits mesothelial-mesenchymal transition, interstitial fibrosis, and neovascularization. It counteracts high-glucose-induced pro-fibrotic effects by reducing H19 levels through suppression of ESR1 transcription [15-16], although current research remains at the basic experimental stage. Following exposure to PD solution, tamoxifen protects the peritoneum by inhibiting the production of matrix components, leptin, and VEGF while preserving its fibrinolytic capacity [17].

Anti-inflammatory drugs: Anti-inflammatory agents play a crucial role in the treatment of peritoneal fibrosis. Inflammation is a key pathogenic mechanism in peritoneal fibrosis, and suppressing inflammatory responses can slow its progression.

L-cysteine: As a PKM2 inhibitor, L-cysteine suppresses high glycolysis in mesothelial cells and alleviates peritoneal fibrosis, offering a novel therapeutic strategy for preventing fibrosis in patients undergoing PD.

Additionally, certain immunosuppressants, such as rapamycin, and cytokine inhibitors, such as pirfenidone, have demonstrated anti-PF efficacy in animal and clinical trials. Rapamycin maintains intracellular lipid homeostasis by reducing cholesterol uptake and increasing cholesterol efflux, thereby exhibiting protective effects against high-glucose peritoneal dialysate-induced fibrosis. However, most of these drugs remain in the research phase, andtheir safety and efficacy require further validation [18]. Sirolimus exerts dual effects by inhibiting both lymphocyte activation and fibroblast proliferation. A randomized controlled trial targeting patients with idiopathic retroperitoneal fibrosis is currently underway. Although this study focused on retroperitoneal fibrosis, its mechanistic research provides insights into the treatment of peritoneal dialysis-associated peritoneal fibrosis.

3.2.3 Novel Therapies

Mesenchymal stem cell therapy offers new hope for the treatment of peritoneal fibrosis. stem cells can inhibit EMT and inflammation, reduce peritoneal mesothelial cell death, enhance cell activity and migration capacity, promote proliferation and tissue repair, thereby suppressing peritoneal mesothelial cell EMT, alleviating peritoneal injury, and reducing fibrosis [19].

Researchers have demonstrated that an AAV1-miRNA triad targeting glycolysis and fibrosis signaling confirms AAV1-mediated miRNA gene therapy as a feasible treatment strategy for inhibiting peritoneal fibrosis. This triple therapy simultaneously expresses microRNA-26a and microRNA-200a while suppressing microRNA-21a, thereby downregulating glycolysis and blocking key signaling molecules in extracellular vesicle transport, ultimately inhibiting peritoneal fibrosis [20].

4. Traditional Chinese Medicine Pattern Differentiation System and Modern Pharmacological Research

In recent years, TCM has proposed the core pathogenesis of “underlying deficiency with superficial excess, or mixed deficiency-excess patterns” based on theories such as “micro-mass accumulation” and “abdominal micro-mass accumulation.” It emphasizes spleen-kidney deficiency as the root cause and phlegm-stasis-turbid toxins as the superficial manifestation. Through syndrome differentiation and treatment, stage-specific therapy, combined with single-herb and compound preparations, TCM exerts multifaceted effects,

including antifibrotic, anti-inflammatory, and immune-modulating actions. Modern pharmacological research has confirmed that multiple herbal medicines, including Astragalus membranaceus, Salvia miltiorrhiza, Fu Shen Granules, and Shen Kang Injection, can intervene in the PF process by regulating key signaling pathways, such as TGF-β/Smad, PI3K/Akt, and NF-κB.

4.1 Pattern Differentiation and Treatment Principles

Traditional Chinese Medicine emphasizes pattern differentiation and treatment, adhering to holistic diagnosis and therapy. Treatment must address both deficiency and excess, tackle both root and branch, and follow the principle of unblocking and tonifying abdominal collaterals. Strengthening the spleen and tonifying the kidneys treat the root cause, while resolving phlegm and clearing turbidity, promoting blood circulation and removing stasis, and eliminating masses and dispersing nodules address the branch symptoms. Early Stage: Damage to the abdominal collaterals impairs collateral flow. At this stage, pathogenic factors have not deeply invaded. The treatment focuses on strengthening the spleen, promoting qi circulation, and dispersing masses. The formula Er Chen Tang combined with Xiang Su San was used with modifications. Xiangfu (Cyperus rhizoma) disperses stagnation with its pungent aroma to resolve qi stagnation; Chuanxiong (Ligusticum rhizoma) promotes qi and blood circulation to resolve blood stagnation; Cangzhu (Atractylodes rhizoma) strengthens the spleen and dries dampness to resolve dampness stagnation. With phlegm resolved and fluid stagnation eliminated, the triple energizer functioned harmoniously. In the middle stage, stagnation obstructs the abdominal collaterals, impeding collateral flow. Both qi and blood are affected by pathogenic factors, making it difficult to distinguish between masses. Treatment focuses on removing stasis, clearing turbidity, detoxifying, and unblocking the collaterals. The modified Peach Kernel and Four Substances Decoction combined with Five Flavors Detoxifying Decoction was used. Optionally add Four Gentlemen Decoction, Chinese Yam, and Lotus Seed Meat to tonify qi, strengthen the spleen, and transform dampness; White Broad Bean and Amomum Villosum warm the middle burner to regulate qi and strengthen the spleen; Coix Seed fortifies the spleen, drains dampness, and disperses toxins and masses; Lindera Root disperses liver stagnation, relieves depression, promotes spleen function, and resolves food stagnation to regulate qi; Salvia Root, Angelica Root, and Ligusticum Root activate blood circulation, resolve stasis, and unblock collaterals; Vitex Vine, Clematis Vine, and Honeysuckle Vine penetrate meridians and collaterals to expel wind and dampness. In the later stages, the abdominal collaterals become obstructed by masses, and the collaterals themselves deteriorate. At this point, the disease reaches its peak, with severe blood-related pathology that is difficult to resolve. Treatment focuses on warming Yang, promoting diuresis, and gradually resolving masses. The modified Zhenwu Decoction combined with Guizhi Fuling Pill was used. Shu di huang, shan yu rou, and shan yao nourish yin, replenish essence, tonify the kidneys, and strengthen the spleen to nourish the abdominal collaterals. Aconite and Cinnamon warm and tonify the spleen and kidneys, break up blood stasis, and eliminate masses; Alisma and Poria drain dampness and promote diuresis, facilitating triple burner flow; Moutan promotes blood circulation and disperses stasis; Magnolia and Cardamom warm the spleen, dry dampness, transform phlegm, and resolve masses; Tangerine Peel and Wood Fragrance unblock triple burner qi stagnation.

4.2 Advances in Modern Pharmacological Research on Traditional Chinese Medicines

4.2.1 Single Herbs and Their Active Components

Research on the prevention and treatment of peritoneal fibrosis by rhein indicates that it exerts its effects by regulating multiple signaling pathways. Although the specific mechanisms remain unclear, existing evidence suggests that rhein possesses multi-target characteristics in combating tissue fibrosis.

Mesenchymal stem cell-derived exosomes (MSC-exosomes) pretreated with salvinol A extract target the hsa-miR-27a-5p and STAT3-SHANK2 pathways to enhance antifibrotic therapeutic effects. Salvia miltiorrhiza (Danshen) possesses properties that promote blood circulation, remove blood stasis, cool the blood, and alleviate pain. Both clinical and experimental studies have indicated that Danshen exhibits therapeutic effects in peritoneal fibrosis. Tanshinone IIA improves the microinflammatory state in patients with PD and protects peritoneal function. Experimental studies by Zhou Yao et al. [21] revealed that sodium tanshinone IIA sulfonate injection alleviates damage to human peritoneal mesothelial cells induced by high-glucose peritoneal dialysate-lipopolysaccharide and inhibits PF, potentially by regulating TGF-β1, matrix metalloproteinase inhibitor-1, and matrix metalloproteinase-9, thereby exerting peritoneal mesothelial cell protection and antifibrotic effects. Danshen also modulates TGF-β1 expression levels in peritoneal tissue, inhibits fibroblast proliferation and migration, antagonizes peritoneal mesothelial cell senescence and apoptosis, helps reduce extracellular matrix accumulation, maintains tissue structural integrity, and thereby delays the progression of peritoneal fibrosis.

Astragalus modulates Dnmt3a-mediated ID2 promoter methylation via the PI3K/Akt pathway, thereby improving MMT in peritoneal mesothelial cells (PMCs). These findings provide new insights into the epigenetic regulation of DNA methylation in fibrotic diseases and the pharmacological effects of Astragalus. Astragalus, a commonly used Chinese herbal medicine, possesses qi-tonifying, surface-consolidating, diuretic, and edema-reducing effects. Multiple studies have indicated its efficacy in preventing and treating peritoneal fibrosis. Astragaloside IV, one of the primary active components of Astragalus, inhibits TGF-β1-mediated Akt pathway activation, degrades β-catenin, and suppresses mesothelial cell transdifferentiation [22]. Yu et al. revealed that treating TGF-β1-induced peritoneal mesothelial cells with different concentrations of astragaloside IV inhibited cellular transdifferentiation. Additionally, Astragalus can suppress epithelial-mesenchymal transition in peritoneal mesothelial cells by downregulating β-catenin, thereby slowing peritoneal fibrosis progression. Sheng Meixiao's research team added Astragalus injection to peritoneal dialysate in PD patients, finding improvements in ultrafiltration volume and peritoneal solute clearance capacity,

which may help delay the onset and progression of PF.

4.2.2 Research on Traditional Chinese Medicine Formulas

The formula Shenling Baizhu San (Ginseng-Poria-Atractylodes Powder) exhibits multifaceted effects in the treatment of peritoneal dialysis complications. Studies have indicated that it improves gastrointestinal dysfunction, bidirectionally regulates gastrointestinal motility, alleviates inflammatory states, modulates oxidative stress, and regulates aquaporins. Specific mechanisms include increased mucin and tight junction secretion, enhanced Bcl-2 expression and reduced Bax expression, limited TLR5/MyD88/NF-κB signaling pathways, inhibited NF-κB/p38 signaling pathways, activated G1/PI3K/Akt-Nrf2 signaling pathways, and downregulated aquaporin 1/2/3/4 expression. These actions collectively regulate gastrointestinal dysfunction, inflammation, and protein-energy wasting [23].

Multiple studies have confirmed that Fusheng Granules exhibit significant therapeutic effects on peritoneal fibrosis. They reduce AGEs and RAGE production and may improve peritoneal fibrosis by inhibiting AGE-RAGE binding, thereby blocking the AGE-RAGE signaling pathway and decreasing the generation of related fibrotic cytokines. Tang et al. [24] indicated that Fusheng Granules may reduce cytokine production and inflammatory cell infiltration by decreasing epidermal growth factor receptor expression. Concurrently, they inhibit the generation and binding of AGEs and RAGE, thereby blocking the AGEs-RAGE signaling pathway and antagonizing peritoneal fibrosis progression. In clinical practice, Fusheng Granules help maintain dialysis efficacy and enhance the quality of life of patients undergoing dialysis.

Research indicates that Shenkang Injection downregulates TGF-β1 expression and inhibits fibronectin (FN) aggregation, thereby improving peritoneal morphological changes in PD-induced rats and delaying PF progression. Renal Kang Injection inhibits the expression of fibrogenic factors such as TNF-α, TGF-β1, VEGF, and connective tissue growth factor (CTGF) in continuous ambulatory peritoneal dialysis (CAPD) mice, demonstrating its clinical significance in preventing and treating PF. Wang Yuxun et al. [25] observed that Renal Kang Injection suppresses the transdifferentiation of peritoneal mesothelial cells, delaying the onset and progression of PF. Its mechanism may involve promoting E-cadherin expression while inhibiting α-smooth muscle actin (α-SMA) expression, thereby suppressing epithelial-mesenchymal transition in peritoneal mesothelial cells.

5. Prospects for Integrated Traditional Chinese and Western Medicine Treatment

Integrated Traditional Chinese and Western medicine holds potential for “complementary advantages and synergistic effects” in treating peritoneal fibrosis; however, it faces several challenges. Collaborative treatment models can adopt corresponding strategies for different disease stages. Early Intervention involves Employing TCM to regulate the constitution and enhance peritoneal resistance before fibrosis becomes evident. Mid-Stage Synergy: Combining anti-inflammatory TCM with biocompatible dialysate to slow progression during mild fibrosis. Late-Stage Support: For patients nearing ultrafiltration failure, TCM is used to fortify the body’s defenses, alleviate symptoms, and improve the quality of life.

6. Summary and Outlook

Peritoneal dialysis-associated fibrosis is a critical bottleneck affecting long-term patient outcomes. Its pathogenesis is complex and involves multidimensional interactions between inflammation, metabolism, immunity, and genetics. Western medicine primarily focuses on optimizing dialysis protocols and pharmacological interventions, but lacks specific therapeutic agents. Traditional Chinese medicine (TCM), grounded in the principles of “the spleen governs the abdomen” and “the theory of collaterals disease,” has established a systematic pattern differentiation framework. Both single herbs and compound formulations have demonstrated promising clinical applications. Integrated Chinese-Western medicine treatment not only synergistically improves symptoms and slows fibrosis progression but also holds promise for regulating the peritoneal microenvironment at its source.

Future research should focus on: Precision medicine and individualized treatment—Establishing peritoneal fibrosis risk prediction models using biomarkers (e. g. , CA125, IL-6, TGF-β1, miRNA profiles) to enable early warning and intervention; In-depth investigation of synergistic mechanisms—Systematically elucidating the multi-component, multi-target, multi-pathway action mechanisms of TCM formulas using genomics, proteomics, and network pharmacology; Integration of novel technologies—Developing nanomedicine delivery systems and exosome-based therapies to enhance the targeting and bioavailability of active TCM components; Real-world studies and long-term follow-up—Strengthening multicenter, large-scale cohort research to evaluate the impact of integrated Chinese and Western medicine on long-term survival and technical survival rates in peritoneal dialysis patients. Through multidisciplinary collaboration, integrating the strengths of both Chinese and Western medicine, we aim to establish an integrated “prevention-intervention-rehabilitation” management system. This approach seeks to achieve the goals of “early detection, early intervention, delaying progression, and preserving function” for peritoneal fibrosis, ultimately improving the quality of life and long-term prognosis of peritoneal dialysis patients.

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