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Pathogenesis of Gout from the Perspective of the Theory of Latent Pathogen: "Phlegm-Blood Stasis Latent in Collaterals and Pain Due to Obstruction"

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Abstract: ***The clinical course of gout is characterized by insidious onset, sudden severe pain, and recurrent episodes. This pattern aligns closely with the pathogenic characteristics of latent pathogen—"hidden without manifestation, waiting for an opportunity to act, and triggered by inducing factors." Therefore, gout can be classified under the category of "latent pathogen causing arthralgia." However, the latent pathogen in gout is not exogenous six pathogenic factors, but rather endogenous phlegm turbidity and blood stasis. Initially, dysfunction of the spleen and kidney leads to impaired ascending of the clear and descending of the turbid, resulting in internal accumulation of dampness-turbidity and subsequent formation of phlegm turbidity. Subsequently, phlegm turbidity flows into the meridians and collaterals, lurking in the joints and bones. Over time, phlegm turbidity gives rise to blood stasis, and the two become intertwined, forming a composite latent pathogen that persists throughout the disease course and progressively deepens. When phlegm and blood stasis remain quiescent, the obstruction of collaterals is mild, and the healthy qi can still restrain them; thus, although pathogenic factors are present, pain does not occur. Once triggered by inducing factors such as dietary indiscretion, overexertion, or exogenous pathogenic influences, the latent pathogen suddenly becomes hyperactive, severely obstructing the collaterals and causing abrupt blockade of qi and blood, leading to the sudden onset of excruciating pain. The principle "pain due to obstruction" precisely captures the critical juncture at which the latent pathogen transforms from a hidden state to an active one, and from silence to outburst.***

Keywords: Gout, Latent pathogen, Phlegm turbidity, Blood stasis, Pain due to obstruction.

1. Introduction

Gout is a metabolic rheumatic disease resulting from purine metabolism disorders and/or impaired uric acid excretion. It is characterized biochemically by hyperuricemia and clinically by recurrent acute episodes of erythematous, swollen, and painful joints [1]. In China, the prevalence of hyperuricemia is approximately 13.3%, and the prevalence of gout ranges from 1% to 3%, with a marked trend toward younger age of onset [2-3]. The natural course of gout exhibits distinct phases: an asymptomatic period that may last for years or even decades; acute episodes that often occur suddenly at night with excruciating pain described as "like being bitten by a tiger"; and, if left untreated, increasingly frequent attacks leading to joint destruction and even renal involvement [4].

In traditional Chinese medicine (TCM), gout falls under the categories of "bi syndrome" and "li jie" [5]. The "Suwen" states: "When wind, cold, and dampness invade the body together, they combine to cause bi syndrome" [6]. Zhu Danxi, in his "Gezhi Yulun", first coined the term "tongfeng" (gout) and noted that "when heat encounters cold, turbid and filthy substances coagulate and cause pain" [7]. However, these classical explanations are insufficient to address a key question: the pathological basis of gout is already established during the asymptomatic phase, yet no exogenous pathogenic factors can be detected, nor are there any discernible symptoms. When an acute attack occurs, endogenous factors such as dietary indiscretion and overexertion can abruptly trigger it, and the condition recurs repeatedly. An exclusive focus on exogenous factors fails to provide a comprehensive explanation.

The theory of latent pathogen may offer a novel perspective. Originating from the "Huangdi Neijing", the core concept of latent pathogen is that "pathogenic qi is concealed and emerges after a period of time"[8]. The *Suwen* states: "Injury by cold in winter inevitably leads to febrile disease in spring" [6], revealing that pathogens can be latently harbored. The "Lingshu" further notes that old pathogens "are concealed within the blood vessels and remain for a long time without being eliminated," and when encountering new infections, they "combine with the old pathogens to cause cold bi" [9]. Subsequent generations of physicians have elaborated on this theory [10-12]. When applied to the course of gout, the asymptomatic period corresponds to "hidden without manifestation," acute episodes correspond to "triggered by inducing factors," and the tendency for recurrence corresponds to "persistent and difficult to eliminate." From this perspective, gout can be classified as a "latent pathogen causing arthralgia." However, the exact nature of the latent pathogen in gout remains to be elucidated. If it were exogenous wind, cold, or dampness, its origin during the asymptomatic period would be unclear. If it were endogenous, the mechanism of its generation and evolution requires further investigation.

This paper proposes that the latent pathogen in gout is not exogenous wind, cold, or dampness, but rather phlegm turbidity and blood stasis generated by spleen-kidney dysfunction. Phlegm turbidity arises endogenously, lurks in the joints and bones, and over time gives rise to blood stasis through phlegm-stasis interaction, forming a composite latent pathogen that persists throughout the disease course. When phlegm and blood stasis remain quiescent, the obstruction of collaterals is mild, and pain does not occur. Once triggered by inducing factors, phlegm and blood stasis suddenly become hyperactive, severely obstructing the collaterals, and causing the sudden onset of intense pain. The principle "pain due to obstruction" precisely captures the critical transition of the

latent pathogen from a "hidden" to an "active" state. The following sections elaborate on this theoretical framework from three aspects: the theoretical foundation of latent pathogen-induced arthralgia, the formation and evolution of phlegm-blood stasis as the latent pathogen, and the dynamic pathogenesis of "pain due to obstruction."

2. Latent Pathogen Causing Arthralgia: Theoretical Foundation of Gout Pathogenesis

2.1 Origins of the Latent Pathogen Theory

The theory of latent pathogen originated from the "Huangdi Neijing". The "Suwen" states: "Injury by cold in winter inevitably leads to febrile disease in spring" [6], revealing the basic pattern that pathogenic qi can be concealed within the body and emerge after a period of time. The "Lingshu" directly associates this mechanism with bi syndrome: "Those who have been injured by dampness, with the dampness concealed in the blood vessels and intermuscular spaces, remaining for a long time without being eliminated... when they encounter wind and cold, qi and blood coagulate, and combining with the old pathogen, they cause cold bi" [9]. The term "old pathogen" clearly indicates that the pre-existing pathogen concealed within the body, when triggered by a new infection, leads to bi syndrome—this represents the earliest textual evidence for latent pathogen-induced arthralgia. The "Suwen" further notes that "the pathogen attacks the five viscera internally and traverses transversely to the pleuro-diaphragmatic space" [6], suggesting that latent pathogens have specific sites of concealment.

Subsequent physicians have expanded upon this theory. Wang Shuhe, in his "Shanghan Lun", formally proposed the term "fu qi", clearly establishing the temporal gap between pathogen concealment and disease onset [13]. Wu Youke, in his "Wenyi Lun", proposed the concept of "pathogen lurking in the membrane source" [14]. Ye Tianshi suggested a dual triggering mechanism of "latent pathogen concealed internally and triggered by new exogenous pathogens"[15]. Liu Jiren, in his "Fuxie Xinshu", extended the concept to internal miscellaneous diseases, stating: "All cases where six exogenous pathogens are contracted but do not cause immediate disease, and only manifest after a period of time, are collectively referred to as latent pathogen" [16]. Thus, the latent pathogen theory evolved into a concept of universal etiological significance.

Based on a comprehensive review of classical and contemporary literature, the core characteristics of latent pathogen can be summarized as follows: (1) hidden without manifestation—no significant symptoms during the latent phase; (2) waiting for an opportunity to act—disease onset depends on the state of healthy qi and environmental changes; (3) triggered by inducing factors—specific triggers are required to activate the pathogen; and (4) persistent and difficult to eliminate—the root of the pathogen is not eradicated, making recurrence easy [10][17].

2.2 The Relationship Between Latent Pathogen and Bi Syndrome

Although the "Nei jing" primarily attributes bi syndrome to exogenous wind, cold, and dampness, it does not exclude the possibility of endogenous latent pathogens causing arthralgia. The "Lingshu"'s discussion of "old pathogen" provides direct evidence: the pre-existing pathogen concealed within the body serves as a prerequisite, and the triggering by a new infection leads to bi syndrome—both are indispensable [9]. Zhang Jingyue, in his "Jingyue Quanshu", interpreted "bi" as "obstruction," emphasizing that any pathogen that obstructs the qi and blood of the meridians and collaterals can cause bi syndrome, not necessarily exogenous pathogens [18]. Ye Tianshi's theory of "chronic disease entering the collaterals" proposed that chronic bi pain often results from old pathogens that have deeply penetrated the collaterals and become firmly adherent, requiring insect-based and carminative drugs to dredge them [15]. Wang Qingren, in his "Yilin Gaicuo", proposed the theory of "blood stasis in bi syndrome" and created the "Shen Tong Zhu Yu Tang" to eliminate blood stasis and unblock collaterals [19], pioneering a new approach to treating bi syndrome from the perspective of endogenous blood stasis. Therefore, the etiology of bi syndrome encompasses both "exogenous new infection" and "endogenous latent pathogen" pathways, which complement each other. For recurrent, persistent, and difficult-to-treat bi syndrome, a comprehensive understanding of pathogenesis cannot be achieved without considering the latent pathogen perspective.

When the characteristics of latent pathogen are compared with the clinical course of gout, the correspondence is clear. Gout patients may remain asymptomatic for years, yet the pathological basis is already established—this corresponds to "hidden without manifestation." Acute episodes are often abruptly triggered by dietary indiscretion, overexertion, or cold exposure, with joint swelling and severe pain occurring suddenly, often at night—this corresponds to "triggered by inducing factors." During the intercritical period, although pain subsides, the root cause remains, and recurrence is easily triggered by mild inducing factors, with progressively shorter intervals and broader joint involvement—this corresponds to "persistent and difficult to eliminate" [1,4]

3. Phlegm Turbidity and Blood Stasis: The Substance of Latent Pathogen in Gout

3.1 Endogenous Generation of Phlegm Turbidity

Phlegm turbidity constitutes the initial form of the latent pathogen in gout. Its generation originates from dysfunction of the spleen and kidney, with dietary indiscretion serving as a critical external contributing factor. The "Suwen" states: "Such individuals must have frequently consumed sweet and fatty foods, leading to obesity; fatty foods generate internal heat, and sweet foods cause abdominal fullness" [6]. Chronic consumption of rich, fatty, sweet, and alcoholic foods exceeds the digestive and transport capacity of the spleen and stomach, leading over time to impairment of these organs. The spleen governs the transportation and transformation of fluids, serving as the foundation of acquired constitution and the source of qi and blood generation. When spleen qi is robust, water and grain essences are properly distributed, and fluids are promptly transformed. When spleen qi becomes deficient, transportation and transformation fail, water and grain are not

properly metabolized but instead accumulate as dampness, which over time condenses into phlegm. This is the mechanism underlying the principle that "the spleen is the source of phlegm generation" [20]. Li Zhongzi, in his "Yizong Bidu", emphasized: "When spleen earth is weak, the clear cannot ascend, and the turbid cannot descend; they remain in the middle, stagnate in the diaphragm, and accumulate into phlegm" [20], further elucidating the specific process of phlegm generation due to spleen deficiency.

The kidney also plays a crucial role in phlegm generation. The kidney governs water metabolism, regulates qi transformation, and oversees the body's fluid metabolism. In individuals with congenital deficiency, advanced age, or chronic disease affecting the kidney, the qi transformation function of the kidney declines, fluids cannot be promptly transported to the bladder for excretion, and they remain stagnant in the body, contributing to phlegm generation [21]. Zhang Jingyue provided a precise exposition: "The root of phlegm lies in the kidney, and the activation of phlegm lies in the spleen" [18]. This indicates that although phlegm turbidity is generated by impaired spleen transport function, its root cause is closely related to the kidney's qi transformation function—kidney deficiency leads to loss of water governance, and water overflows as phlegm. Spleen and kidney deficiency often interact: prolonged spleen deficiency can affect the kidney, and kidney deficiency cannot warm the spleen earth, further aggravating spleen deficiency. Together, they contribute to the persistent endogenous generation of phlegm turbidity.

Once generated, phlegm turbidity differs from general dampness in its pathogenic mode. Phlegm turbidity is sticky and heavy, yet it possesses the property of "ascending and descending with qi, reaching everywhere without exception". It can flow with qi and blood into the meridians, deposit in the joints, bones, and fascial spaces, and remain latent without causing significant symptoms for a prolonged period—this is the state of "latent phlegm." The formation of latent phlegm marks the initial stage of the latent pathogen in gout: although the patient already possesses the material basis for disease onset, the phlegm turbidity is still mild, the obstruction is still superficial, and the healthy qi can still maintain basic qi and blood circulation. Therefore, patients may remain in the asymptomatic phase for a long time. However, as long as the latent phlegm remains, the basis for gout onset persists—this is the pathological manifestation of the principle that "latent pathogen causes disease with gradual onset and sudden outburst."

3.2 Secondary Generation of Blood Stasis

Prolonged latent phlegm in the meridians and collaterals is not static; its impact on blood circulation is progressive. Phlegm, by its sticky nature, readily obstructs qi movement. Qi is the commander of blood—when qi moves, blood moves; when qi stagnates, blood slows down. Phlegm turbidity obstructs the meridians, impairs qi movement, and consequently slows blood flow, leading over time to blood stasis [5]. Tang Rongchuan, in his "Xuezheng Lun", provided profound insights: "Phlegm can also transform into blood stasis" and "when blood stasis accumulates for a long time, it can also transform into phlegm water" [22], clearly indicating the inherent tendency for mutual transformation between phlegm and blood stasis. The basic mechanism of phlegm-induced blood stasis is as follows: phlegm turbidity occupies the vascular lumen, narrows the passage for qi and blood circulation, and, due to its adhesive nature, slows the blood flow rate at the site of phlegm obstruction, facilitating the deposition of formed blood elements and leading over time to stasis.

Once blood stasis forms, the vascular lumen becomes even more compromised, and fluids cannot be properly distributed but instead accumulate as phlegm, which in turn aggravates the accumulation of phlegm turbidity—thus forming a self-reinforcing pathological cycle between phlegm and blood stasis. Tang Rongchuan further noted that "the accumulation of phlegm water is caused by blood stasis" [22], accurately describing this feedback loop. Phlegm and blood stasis serve as both cause and effect for each other, mutually reinforcing and generating, transforming the latent pathogen from a single pathological product into a composite one, with correspondingly increased therapeutic resistance.

The clinical manifestations of blood stasis in gout patients typically appear later than those of phlegm turbidity, often emerging after several years of disease progression. However, once established, they are more refractory to treatment. Clinical observations include: dark purple discoloration of the skin over the affected joints; stabbing pain with fixed localization, worsening at night (since blood circulation belongs to yin, and nighttime yang qi withdraws inward, further slowing blood flow and aggravating stasis); purplish dark tongue body with or without petechiae; and tortuous, engorged sublingual veins. These are all external signs of blood stasis becoming evident. When the disease progresses to the stage of tophus formation, hard nodules can be palpated around the joints, which persist for a long time and may even ulcerate, discharging a chalky white substance. This represents the extreme manifestation of phlegm turbidity and blood stasis coalescing into a tangible form. Once tophi form, the phlegm-blood stasis complex has entered a relatively advanced stage, significantly increasing treatment difficulty.

3.3 Phlegm-Blood Stasis Intertwining

Based on the above analysis, the latent pathogen in gout is not a single entity of phlegm turbidity or blood stasis alone, but rather a composite product resulting from their progressive intertwining and amalgamation over the course of the disease. This composite process has a pathological basis: fluids and blood share the same origin [5]; phlegm represents the pathological transformation of fluids, while blood stasis represents the pathological state of blood. They share a natural affinity in terms of their material origin. Once phlegm turbidity and blood stasis coexist in the same location within the meridians and joints, they readily adhere to each other, forming a composite pathological state that is far more refractory than either phlegm turbidity or blood stasis alone. Phlegm augments the power of blood stasis, and blood stasis reinforces phlegm consolidation. With phlegm, blood stasis gains a basis for attachment; with blood stasis, phlegm becomes firmly adherent. They mutually reinforce each other, forming a composite pathogenic unit of "phlegm enveloping blood stasis and blood stasis enveloping phlegm."

The evolution of the phlegm-blood stasis composite latent pathogen follows a progressive deepening trajectory. In the early stage, phlegm turbidity predominates, blood stasis is just beginning to emerge, pathological damage is mainly confined to the meridian level, the extent is limited, and treatment is relatively easier. In the middle stage, phlegm and blood stasis are equally prominent, with deepening intertwining and increasingly significant obstruction of meridian qi and blood, leading to more frequent attacks and involvement of more joints. In the advanced stage, prolonged accumulation of phlegm and blood stasis may generate turbid toxin, with toxin and blood stasis intertwining, deeply penetrating the bones and joints, and internally damaging the liver and kidney [23]. At this stage, joint destruction is evident, renal function may be affected, and treatment becomes extremely challenging. This progressive deepening trajectory spans years or even decades, consistent with the natural course of gout.

Contemporary TCM masters have deeply recognized the dominant role of phlegm and blood stasis in gout pathogenesis. Master Zhu Liangchun proposed the therapeutic principle of "draining turbidity, resolving blood stasis, and regulating the spleen and kidney" for gout treatment [24], emphasizing that treatment should not focus solely on clearing heat and promoting diuresis but must also simultaneously address draining and resolving turbid stasis to capture the essence of the pathogenesis. Professor Lu Zhizheng proposed the academic thought of "regulating the spleen and stomach to treat bi syndrome" [25], also approaching gout treatment from the root cause of spleen deficiency generating phlegm and phlegm leading to blood stasis obstruction. The commonality between these two masters lies in their recognition of phlegm-blood stasis intertwining as the key link in gout pathogenesis, requiring simultaneous treatment of phlegm and blood stasis with attention to both spleen and kidney.

The phlegm-blood stasis composite latent pathogen persists throughout the entire course of gout, with its state varying according to disease stage: during the asymptomatic period, phlegm and blood stasis are latent and mild, not yet reaching the threshold for disease onset; during the acute episode, they suddenly become hyperactive, obstructing the meridians and causing acute pain; during the intercritical period, although reduced, they are not completely eliminated, remaining latent in preparation for the next triggering event. The degree of obstruction of the meridians by phlegm and blood stasis directly determines whether pain occurs and its severity. It is precisely based on these characteristics of the phlegm-blood stasis latent pathogen that the classic principle of "pain due to obstruction" acquires specific and precise pathological significance in gout: what is obstructed are the meridians blocked by the phlegm-blood stasis latent pathogen; what causes pain is the sudden arrest of qi and blood at the site of obstruction.

4. Pain Due to Obstruction: The Pivot of Latent Pathogen Activation

4.1 Theoretical Implications of "Pain Due to Obstruction"

"Pain due to obstruction" is a core proposition in TCM for explaining the pathogenesis of pain, with its theoretical origin in the "Huangdi Neijing". The "Suwen" states: "The meridians flow continuously without cessation, circulating in a never-ending cycle. When cold qi enters the meridians, it causes delay, stagnation, and impaired movement... when it lodges in the vessels, qi becomes obstructed, causing sudden pain" [6]. Although the causes of pain discussed in this chapter vary, the underlying pathogenesis is consistent: all are attributable to impaired qi and blood circulation. Cheng Guopeng, in his "Yixue Xinwu", refined this understanding into the concise formulation: "When there is free flow, there is no pain; when there is pain, there is no free flow" [26], which has become a guiding principle for the diagnosis and treatment of pain.

The meridians serve as the passageways for qi and blood circulation, and the joints and bones depend entirely on nourishment from qi and blood to maintain their normal function. When the meridians are unobstructed and qi and blood circulate continuously, the bones are nourished, the joints are smooth, and there is naturally no pain. When a pathological factor obstructs the meridians and impairs qi and blood circulation, the affected area loses nourishment and function, while qi and blood become congested and stagnant locally. These two factors interact, and pain ensues.

The pathological factors that can cause "obstruction" are diverse, including qi stagnation, blood stasis, phlegm coagulation, cold accumulation, dampness encumbrance, and heat congestion [5]. However, with regard to gout, the material basis of "obstruction" is the phlegm-blood stasis latent pathogen discussed above. The phlegm-blood stasis latent pathogen, concealed in the meridians and joints, already causes some degree of obstruction, yet in the early stage of concealment, it does not trigger pain. This suggests that "free flow" and "obstruction" are not static, mutually exclusive categories, but rather exist along a continuum of dynamic variation in the degree of obstruction.

4.2 Dynamic Evolution from Latent to Active

The key to the transition of phlegm-blood stasis from a quiescent to an active state lies in the accumulation of quantitative changes and the breakthrough of qualitative changes in the degree of obstruction. This process can be understood in three interconnected phases.

4.2.1 Latent Pathogen Accumulation

When phlegm and blood stasis first accumulate in the meridians and joints, their quantity is small and their cohesion is loose. Although some degree of local retention has already formed, the meridian channels have not yet reached the point of severe obstruction. Qi and blood can still pass through, albeit with difficulty or by detours, and therefore the patient experiences no pain. This phase corresponds to the asymptomatic hyperuricemia stage of gout, during which the latent pathogen remains in a "quiescent" state. However, with the persistence of spleen-kidney deficiency and the continuous influence of dietary factors, phlegm turbidity gradually increases and blood stasis progressively deepens. The degree of obstruction accumulates slowly and steadily, approaching the critical threshold for triggering pain [23]. This accumulation phase may last for several years or even more than a decade, reflecting the characteristic of latent

pathogen pathogenesis that "the onset is gradual."

4.2.2 Triggering of Latent Pathogen

The transition of the latent pathogen from a quiescent to an active state always requires an inducing factor. Common clinical triggers include the following. Dietary indiscretion is the most common trigger: excessive eating or alcohol consumption acutely increases the burden on the spleen and stomach, generating dampness and promoting turbidity, thereby rapidly increasing the previously accumulated phlegm pathogen [1]. Overexertion leads to sudden deficiency of healthy qi, impaired defensive function, and inability to restrain the latent pathogen, allowing the pathogen to take advantage of the deficiency and become active [21]. Exogenous wind-cold is also a common trigger: cold causes contraction and stagnation, leading to sudden constriction of the vessels and slowing of qi and blood circulation. As stated in the "Lingshu", "when they encounter wind and cold, qi and blood coagulate, and combining with the old pathogen, they cause cold bi" [39]. The exogenous pathogen combines with the internally latent pathogen, promoting the outbreak of the disease. Emotional dysregulation leads to disorder of qi movement, which can also stir the quiescent phlegm and blood stasis [50]. Although these inducing factors act through different pathways, they converge on the same outcome: they disrupt the quiescent equilibrium of the latent pathogen, allowing the pathogen to cross the threshold from quantitative accumulation to qualitative change.

4.2.3 Sudden Severe Obstruction

Once the latent pathogen is triggered, phlegm and blood stasis suddenly become hyperactive at the site of concealment. The meridians rapidly transition from "mild obstruction" to "severe obstruction," and qi and blood circulation abruptly change from "barely maintained flow" to "complete blockade." At the site of obstruction, qi and blood become congested, and the defensive and nutritive qi become disordered, leading to the rapid development of redness, swelling, heat, and pain [44]. The generation of heat signs can be understood as stagnant heat resulting from prolonged qi and blood stasis and intense struggle between healthy qi and pathogenic factors—the more severe the obstruction, the more intense the resistance of healthy qi, and the greater the local heat. This phase corresponds precisely to the TCM pathogenesis of latent pathogen outburst and sudden meridian obstruction.

4.3 The Relationship Between Phlegm-Blood Stasis Obstruction and Qi-Blood Blockade

The pathogenic impact of phlegm turbidity primarily manifests as "stagnation"—stagnation of qi movement, stagnation of fluids, and stagnation of blood circulation, thereby impairing the normal movement of qi, blood, and fluids. The pathogenic impact of blood stasis primarily manifests as "blockade"—blockade of the vascular lumen and obstruction of the collaterals, thereby impeding local qi and blood circulation. When phlegm and blood stasis become intertwined, "stagnation" and "blockade" mutually reinforce each other: "stagnation" creates the conditions for "blockade," and "blockade" exacerbates the degree of "stagnation." [53] Together, they cause severe obstruction of the meridians. At this point, qi and blood are both unable to circulate and deprived of nourishment. The dual factors of "obstruction" and "malnourishment" interact, resulting in excruciating pain.

The "obstruction" caused by phlegm-blood stasis blockade is not an absolute obstruction of the entire meridian system, but rather a relative obstruction of local meridians. When the latent pathogen is mild, qi and blood can still bypass the site, and therefore there is no pain. When the pathogen suddenly becomes hyperactive, the local meridians become nearly completely obstructed, leading to sudden and severe pain. After the acute episode, phlegm and blood stasis gradually dissipate, local circulation is partially restored, and pain subsides accordingly. The principle "pain due to obstruction" precisely captures the critical transition of the latent pathogen from a "hidden" to an "active" state: when the obstruction is mild, it falls within the category of "free flow"; when the obstruction suddenly becomes severe, it crosses into the category of "blockade," and pain ensues.

5. Discussion

This paper systematically explores the TCM pathogenesis of gout from the perspective of latent pathogen theory, proposing the following core insights.

Gout represents a typical disease pattern of "latent pathogen causing arthralgia." The clinical course characterized by "insidious onset—sudden episode—recurrent attacks" aligns closely with the pathogenic characteristics of latent pathogen, i.e., "hidden without manifestation, triggered by inducing factors, and persistent with tendency to recur." This provides a clinical basis for applying latent pathogen theory to explain gout pathogenesis.

Phlegm turbidity and blood stasis constitute the substance of the latent pathogen in gout. Spleen-kidney deficiency and dietary indisposition lead to endogenous generation of phlegm turbidity, which flows into the meridians. Prolonged phlegm obstruction leads to qi stagnation and blood stasis, and phlegm and blood stasis become intertwined, forming a composite latent pathogen. This latent pathogen persists throughout the entire course of gout, evolving along a trajectory of "phlegm turbidity predominance—phlegm and blood stasis equally prominent—prolonged accumulation generating toxin."

"Pain due to obstruction" is the key pivot through which the latent pathogen transitions from a latent to an active state. During the latent pathogen accumulation phase, the obstruction is mild, and qi and blood can still pass through, so no pain is detectable. When triggered by inducing factors such as dietary indiscretion, overexertion, exogenous pathogens, or emotional stress, phlegm and blood stasis suddenly become hyperactive, and the meridians rapidly transition from "mild obstruction" to "severe obstruction." Qi and blood become blocked, and pain suddenly erupts. The dynamic transition between "free flow" and "obstruction" explains the clinical characteristics of gout attacks: sudden onset, severe intensity, fixed localization, and tendency to recur.

In conclusion, the theoretical framework of "phlegm-blood stasis latent pathogen—pain due to obstruction" provides a comprehensive explanation of the pathogenesis and evolution

of gout, offering a unified theoretical foundation for TCM pattern differentiation and treatment. This framework integrates the asymptomatic period, acute episodes, and intercritical periods of gout into a unified pathogenetic explanatory system, contributing to a deeper understanding of the regularity of gout onset and providing a theoretical basis for stage-specific clinical treatment. Future research should further explore objective indicators of the phlegm-blood stasis latent pathogen, investigating its correspondence with modern medical concepts such as hyperuricemia and urate deposition, with the aim of advancing the modern interpretation and clinical translation of the TCM latent pathogen theory.

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