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Pathogenesis and Recent Advances in Diagnosis and Treatment of Coronary Slow Flow Phenomenon

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Abstract: ***The pathogenesis of coronary slow flow phenomenon (CSFP) remains incompletely elucidated. Its clinical manifestations primarily include symptoms such as angina pectoris and chest pain, with complex pathological mechanisms that often coexist with other cardiovascular diseases. Current diagnostic approaches rely on coronary angiography, flow reserve assessment, and cardiac magnetic resonance imaging (MRI). However, existing diagnostic methods still exhibit limitations. In terms of treatment, pharmacological therapy and microvascular interventional therapy are common strategies. Nonetheless, the management of CSFP faces numerous challenges, and long-term prognostic evaluations remain insufficient. This review explores the pathogenesis, clinical characteristics, diagnostic methods, and treatment strategies of CSFP, aiming to provide more precise references for clinical diagnosis and management.***

Keywords: Coronary Slow Flow Phenomenon, Pathogenesis, Diagnostic Methods, Treatment Strategies, Risk Assessment**.**

1. Introduction

The coronary slow flow phenomenon (CSFP) is a condition characterized by reduced blood flow velocity in the coronary arteries in the absence of significant stenosis or occlusion [1]. Although this pathological condition does not directly cause structural changes in the coronary arteries, it can significantly affect myocardial perfusion, leading to symptoms of myocardial ischemia such as angina and chest pain [2]. In recent years, with in-depth research on CSFP, researchers have gradually uncovered its complex pathogenesis, including endothelial dysfunction, microvascular insufficiency, and inflammatory responses [3]. Despite the fact that slow coronary flow typically occurs in patients without significant large-vessel stenosis, it is still closely related to the occurrence of cardiovascular events and may have an important impact on long-term prognosis.

2. Clinical Definition and Characteristics of CSFP

2.1 Clinical Definition of CSFP

CSFP is a unique blood flow abnormality typically detected through coronary angiography. In this condition, the coronary arteries show incomplete opacification with blood flow velocity below normal levels, yet there is no significant stenosis, thrombus formation, or other obvious structural lesions [4]. Slow coronary flow usually occurs in patients without extensive vascular disease, meaning that their coronary arteries do not have significant stenosis or occlusion, and no evident pathological changes can be found through traditional imaging examinations. The definition of CSFP is somewhat challenging due to its complex etiology and the interplay of multiple pathological mechanisms. Clinically, slow coronary flow is considered a functional disorder, closely related to microvascular dysfunction, endothelial damage, and hemodynamic abnormalities [5]. Although patients with slow coronary flow may not exhibit significant organic coronary artery disease, their myocardial perfusion is still impaired. Therefore, this phenomenon is often regarded as a potential cardiovascular risk marker.

2.2 Clinical Features of CSFP

The clinical features of CSFP are mainly manifested as angina pectoris or other symptoms of myocardial ischemia. Patients often experience chest pain, shortness of breath, and fatigue during physical activity or emotional excitement. These symptoms are similar to those of coronary heart disease patients, but they do not align with the absence of significant stenosis or thrombosis observed in coronary angiography [6]. Due to the lack of significant vascular lesions shown by coronary angiography, slow coronary flow is frequently misdiagnosed as functional or atypical angina. Electrocardiogram (ECG) examination is an auxiliary tool for diagnosing slow coronary flow. Patients' ECGs may show features of non-ST-segment elevation myocardial infarction, such as T-wave inversion and slight ST-segment depression, indicating potential myocardial ischemia. However, these findings typically do not match the traditional characteristics of myocardial infarction. The symptoms of slow coronary flow patients may occur intermittently and usually lack the organic lesion manifestations seen in traditional coronary heart disease patients. In addition to common angina, some patients with slow coronary flow may also experience shortness of breath and increased fatigue, especially during physical exertion or emotional excitement. Therefore, the diagnosis of CSFP requires not only reliance on imaging examinations but also a comprehensive evaluation combining clinical manifestations, laboratory test results, and the patient's clinical background.

3. The Pathogenesis of CSFP

3.1 Endothelial Dysfunction

Endothelial dysfunction is one of the key pathogenesis mechanisms of CSFP. Under normal conditions, endothelial cells secrete a variety of bioactive substances, such as nitric oxide (Nitric Oxide, NO), to maintain the vasodilation function and normal blood flow [7]. Dysfunction of endothelial cells leads to the weakening of vascular dilation responses, subsequently increasing vascular resistance and affecting blood flow velocity. Endothelial damage is usually caused by a variety of factors, such as high blood pressure,

hyperglycemia, smoking, inflammation, and oxidative stress. When oxidative stress is excessive, the production of NO by endothelial cells decreases, while the increase in vascular contraction factors like endothelin further leads to vasoconstriction and an unsmooth blood flow. The occurrence of slow coronary flow often accompanies these endothelial dysfunctions, leading to elevated local vascular resistance, suppressed blood flow, and subsequently worsening myocardial ischemia. In addition, endothelial damage can also promote inflammatory reactions and release pro-inflammatory factors. These factors not only further damage the vessel wall, but may also induce microvascular dysfunction, thus forming a vicious circle.

3.2 Microvascular Dysfunction

CSFP is closely related to microvascular dysfunction, especially in patients without significant coronary artery stenosis. As a key part of myocardial blood supply, microvascular dysfunction is an important pathological basis for CSFP. Increased microvascular contractility, vascular wall tension imbalance, and uneven microvascular blood flow can all lead to abnormal blood flow and slowed coronary circulation. Structural abnormalities of microvessels, such as hyperplasia, thickened walls, and endothelial damage, can impede local blood flow even without significant large-vessel narrowing [8]. Microvascular dysfunction can also localize myocardial ischemia, increase the heart's metabolic burden, and potentially trigger ischemic heart disease. These microvascular pathological changes are often overlooked in clinical examinations because coronary angiography mainly reflects the condition of large vessels. The causes of microvascular dysfunction are complex and may be associated with factors like hypertension, diabetes, and arteriosclerosis. Therefore, improving microvascular function and restoring blood flow has become a crucial direction for treating CSFP.

3.3 Inflammatory Response

Inflammatory response is one of the important pathological factors of CSFP. Chronic inflammation not only directly damages the blood vessel wall but also causes endothelial cell dysfunction, thereby further exacerbating microvascular dysfunction. Chronic low-grade inflammation is common in patients with various cardiovascular diseases, especially those with coronary heart disease, hypertension, and diabetes. Inflammatory factors such as C-reactive protein (C-Reactive Protein, CRP), interleukin-6 (Interleukin-6, IL-6), and tumor necrosis factor (Tumor Necrosis Factor-alpha, TNF-α) are often in a persistent high-level state in these patients [9]. These inflammatory factors, through mechanisms such as activating endothelial cells, stimulating the aggregation of immune cells, and increasing blood viscosity, promote vasoconstriction and worsen hemodynamic abnormalities. Moreover, inflammatory response may also induce the progression of atherosclerosis by altering the rheological properties of blood, such as increasing platelet aggregation, and thereby further damage the microvessels and large vessels of the coronary arteries. It has been shown that inflammation is not only involved in pathological changes of the coronary arteries but is also closely related to the occurrence and exacerbation of the slow coronary flow phenomenon [10]. Therefore, controlling inflammatory response may play a positive role in the treatment and prevention of CSFP.

3.4 Atherosclerosis and Coronary Stenosis

Despite the fact that the typical manifestation of CSFP is the absence of significant stenosis on coronary angiography, atherosclerosis and mild stenosis of coronary arteries can still trigger slow coronary flow in some patients. Atherosclerosis is a progressive lesion characterized by the deposition of lipids in the intima of blood vessels, infiltration of inflammatory cells, and the proliferation of fibrous tissue, which gradually forms plaques [11]. Even when the degree of stenosis in the coronary arteries is mild, the presence of atherosclerosis can still have a significant impact on blood flow, especially when combined with microvascular dysfunction, leading to a substantial decrease in blood flow velocity. The presence of atherosclerotic plaques increases the rigidity of the vessel wall and weakens the vasodilation function, thereby causing heterogeneous distribution of coronary blood flow. When the plaque compresses the vessel lumen sufficiently to slow down the local blood flow, slow coronary flow may occur even in the absence of significant stenosis. The occurrence of slow coronary flow may indicate that atherosclerosis has progressed to a certain extent, and even minor lesions that are not visible on coronary angiography may have significant effects on myocardial blood supply.

3.5 Hemodynamic Factors

The reduction in blood flow velocity is not only related to the pathological changes in the blood vessels themselves but is also closely related to the systemic and local hemodynamic conditions. Factors such as hypertension, hypotension, increased blood viscosity, and uneven blood flow can all lead to the slow coronary flow phenomenon [12]. In patients with hypertension, the thickening of the vascular wall and the decrease in vascular elasticity can affect the smoothness of blood flow, thereby causing a slowdown in blood flow. Conversely, in patients with hypotension, the insufficient cardiac output naturally reduces the blood flow velocity, which may induce the slow coronary flow phenomenon. In addition, the rheological properties of blood, especially an increase in blood viscosity, can significantly affect blood flow velocity. Increased blood viscosity is usually associated with factors such as hypercholesterolemia, hyperglycemia, and thrombosis, which not only exacerbate vascular blockages but also increase vascular resistance, thereby further reducing blood flow velocity. Abnormal blood cells and platelets may play an important role in the development of CSFP. Studies have found that the red cell distribution width, platelet count, and mean platelet volume in patients with CSFP are significantly higher than those in the control group [13]. These abnormalities may directly or indirectly promote the development of CSFP by **increasing** blood viscosity, promoting platelet aggregation, and thrombosis.

4. Risk Factors for CSFP

4.1 Hypertension

Hypertension is deemed a key risk factor for CSFP. Persistent hypertension imposes mechanical stress on vessel walls, causing endothelial damage and dysfunction. As normal endothelial function is crucial for maintaining vascular dilation and smooth blood flow, long-term high pressure can injure endothelial cells, impairing the vessel's response to dilating factors. This endothelial decline weakens vasodilation, raises vascular resistance, slows blood flow, and can lead to CSFP [14]. Moreover, hypertension can cause microvascular dysfunction, reduce vessel wall elasticity, and increase contractility, further disrupting coronary hemodynamics. Thus, hypertension is not only an independent risk factor for coronary heart disease but also a significant contributor to CSFP.

4.2 Diabetes

Diabetes is another crucial risk factor for CSFP, especially type 2 diabetes. Diabetic patients often experience endothelial dysfunction and microvascular pathology, which can lead to abnormal hemodynamics in the coronary arteries and microvasculature. In a state of hyperglycemia, the accumulation of glycated hemoglobin and excessive advanced glycation end products accelerates endothelial injury and reduces the production of vasodilatory factors such as nitric oxide (NO), leading to weakened vascular dilation [15]. Additionally, the hyperglycemia in diabetic patients promotes inflammatory responses and oxidative stress, which further exacerbate vascular wall damage and affect vascular elasticity and function.

Microvascular pathology is a common complication in diabetic patients. The injury to the microvascular endothelium can directly lead to microvascular dysfunction and cause blood flow stasis and slow coronary flow. Therefore, due to the long-term effects of hyperglycemia, the coronary microcirculation in diabetic patients is often impaired, increasing the incidence of CSFP.

4.3 Hyperlipidemia

Hyperlipidemia, especially hypercholesterolemia, is a notable risk factor for CSFP. The association between lipid metabolism disorders and atherosclerosis is well-established. Elevated low-density lipoprotein (LDL) and reduced high-density lipoprotein (HDL) cholesterol are closely linked to atherosclerosis, which can lead to coronary artery stenosis. Atherosclerosis also increases lipid deposition and inflammation within blood vessels, damaging their structure and function and causing hemodynamic abnormalities. As it progresses, vessel walls thicken, harden, and may develop localized stenosis. Though not always significant on coronary angiography, this can still slow blood flow. Hyperlipidemia can also increase blood viscosity, further impeding blood flow and exacerbating CSFP. Additionally, hyperlipidemia is linked to endothelial dysfunction and microvascular pathology, which increases the risk of slow coronary flow.

4.4 Cigarette Smoke

The harmful substances in cigarette smoke, particularly nicotine and carbon monoxide, can directly damage the vascular endothelium and cause endothelial dysfunction. As the vascular endothelium is crucial for maintaining normal vessel function and smooth blood flow, its damage can weaken the vessel's dilation ability, increase resistance, and lead to slow coronary flow. Smoking also triggers systemic inflammation and oxidative stress, further aggravating endothelial injury and accelerating atherosclerosis. Additionally, smoking can alter blood rheology by increasing platelet and red blood cell aggregation, thereby raising blood viscosity, slowing blood flow, and promoting CSFP [17]. It also enhances vessel constriction, reducing myocardial blood supply and causing myocardial ischemia. Therefore, quitting smoking is considered an effective measure to reduce CSFP and its complications.

5. Clinical Diagnostic Methods for CSFP

5.1 Coronary Angiography

Coronary angiography remains one of the gold standards for diagnosing CSFP. Even though it cannot directly measure blood flow velocity, it can effectively rule out significant coronary stenosis or occlusion, helping doctors identify the slow-flow phenomenon. Coronary angiography is an imaging examination where a catheter is inserted into the coronary arteries and contrast agent is injected. It clearly shows the shape, patency, and lesions of the coronary arteries. In slow-flow patients, the typical angiographic findings are incomplete vessel opacification and slow blood flow without significant stenosis or occlusion. Thus, while coronary angiography can exclude organic lesions and provide preliminary diagnostic evidence for slow coronary flow [18], it does not offer hemodynamic information. As a result, the definitive diagnosis of slow coronary flow often requires additional methodological assessments. Coronary angiography is highly valuable in clinical practice, particularly for patients with acute chest pain or suspected coronary artery disease, as it offers vital diagnostic clues. For slow-flow patients, angiographic results often show no significant vascular abnormalities, but when accompanied by myocardial ischemia symptoms, the likelihood of slow coronary flow is higher.

5.2 Cardiac Magnetic Resonance Imaging

Cardiac magnetic resonance imaging (MRI) is a non-invasive and precise imaging modality that offers detailed cardiac anatomy and blood flow data, which is crucial for evaluating CSFP. Unlike traditional coronary angiography, cardiac MRI can depict the morphology of coronary arteries and directly observe blood flow dynamics and myocardial perfusion. When assessing CSFP, cardiac MRI uses velocity imaging to quantitatively measure coronary blood flow velocity, offering intuitive data for diagnosing slow coronary flow. This is particularly beneficial for patients with no significant vascular abnormalities on coronary angiography, as cardiac MRI can detect microvascular dysfunction or local blood flow abnormalities through detailed blood flow analysis [19]. Moreover, cardiac MRI can identify myocardial ischemic areas, providing a comprehensive assessment of myocardial health. Despite its complexity in equipment and operation,

cardiac MRI's precise blood flow assessment makes it a key technology for researching and clinically managing the slow coronary flow phenomenon.

5.3 Fractional Flow Reserve (FFR) Assessment

As a hemodynamic test, fractional flow reserve (FFR) assesses the severity of coronary artery disease by measuring blood flow velocity and pressure changes. In CSFP diagnosis, FFR helps doctors distinguish between microvascular and large-vessel diseases, enabling personalized treatment. FFR measurement involves inserting a pressure sensor-equipped catheter into the coronary artery to measure flow pressure and velocity changes under different conditions. For CSFP patients, FFR can rule out large-vessel disease and identify microvascular dysfunction or hemodynamic abnormalities. A reduced FFR value often indicates coronary microvascular dysfunction or insufficient flow reserve, which can slow coronary flow and cause myocardial ischemia [20]. Clinically, FFR not only evaluates coronary lesions precisely but also guides CSFP treatment. In slow-flow patients, FFR determines the need for interventional therapy or management through medication and lifestyle changes.

5.4 Blood Biomarker Testing

In diagnosing CSFP, blood marker tests play an auxiliary role by identifying inflammation, microvascular injury, and myocardial ischemia [21]. Common markers include C-reactive protein (CRP) and high-sensitivity troponin I (hs-TnI). CRP, a liver-synthesized acute-phase protein, assesses systemic inflammation. Since CSFP is often linked to chronic inflammation, especially endothelial and microvascular damage, elevated CRP may signal endothelial injury and hemodynamic disturbances. Hs-TnI, a sensitive marker of heart injury, if elevated in CSFP patients, implies myocardial ischemia or microvascular dysfunction. Blood marker tests not only assist CSFP diagnosis but also offer clues for etiology and clinical management. For instance, in acute coronary syndrome patients, combining these tests with imaging better determines the clinical significance of slow coronary flow and its impact on prognosis. With advancing biomarker research, more specific blood markers for early CSFP diagnosis and risk assessment are expected.

6. Treatment Strategies for CSFP

6.1 Pharmacological Therapy

Pharmacotherapy is fundamental in CSFP management. It aims to address endothelial dysfunction, microvascular pathology, and hemodynamic abnormalities to restore normal blood flow [22]. Common medications include antiplatelet drugs (like aspirin and clopidogrel), lipid-lowering agents (like statins), and calcium channel blockers (like diltiazem and amlodipine). Antiplatelet drugs inhibit platelet aggregation and reduce thrombosis risk. Lipid-lowering drugs decrease cholesterol and slow atherosclerosis. Calcium channel blockers dilate vessels to improve coronary flow and alleviate ischemia. Newer agents, such as antioxidants and anti-inflammatory drugs, are also gaining attention for their potential to reduce endothelial damage and improve microvascular function by targeting oxidative stress and inflammation. However, treatment responses vary among patients, so individualized pharmacotherapy and a combined approach with other treatments are often needed for optimal results.

6.2 Microvascular Interventional Therapy

When pharmacotherapy fails to effectively control CSFP symptoms or improve myocardial ischemia, microvascular interventions, such as percutaneous coronary intervention (PCI), may be considered. PCI involves inserting a catheter-based stent or balloon into the coronary artery to open up the vessel and improve blood flow. While PCI is a well-established technique for treating coronary artery disease, its efficacy in treating CSFP remains controversial. Since slow-flow often occurs without significant coronary stenosis or with only mild narrowing, the intervention may not directly enhance coronary flow velocity. In some cases, even after PCI-induced vessel dilation, slow-flow may recur, indicating that its pathogenesis involves complex factors like endothelial dysfunction and microvascular pathology. Thus, the success of microvascular interventions depends on patient-specific factors, including whether microcirculation function is restored and postoperative hemodynamic changes. However, in certain scenarios, particularly in patients with severe coronary spasms or short-term flow issues, PCI may still help improve symptoms and prevent ischemia. Therefore, the decision to pursue microvascular interventions should weigh the patient's specific condition, treatment response, and postoperative expectations.

6.3 Improving Hemorheology

Hemorheology, which encompasses blood viscosity, platelet aggregability, and red blood cell deformability [23], is crucial in CSFP management. Abnormalities can lead to slow blood flow, thrombosis, and inadequate microvascular perfusion. Optimizing hemorheology can enhance coronary flow velocity and alleviate myocardial ischemia in CSFP patients. Regulating Blood Viscosity: Improving blood fluidity reduces flow resistance and restores normal circulation. Drugs like low-molecular-weight heparin and antiplatelet agents can lower viscosity and blood coagulability, minimizing thrombosis risk. Enhancing Vascular Dilation: Calcium channel blockers and nitrates effectively dilate blood vessels, improving coronary flow. Boosting Microcirculation: Agents like prostaglandins can improve microvascular function and promote microcirculation. Overall, optimizing hemorheology not only relieves CSFP symptoms but also reduces the risk of cardiovascular events.

6.4 Comprehensive Intervention Measures

In addition to pharmacological and microvascular interventional therapies, comprehensive interventions play a significant role in managing CSFP. Lifestyle modifications are fundamental, including a healthy diet, regular exercise, smoking cessation, and weight control, which can lower the incidence of slow coronary flow and related risks. A balanced low-fat, low-salt diet rich in dietary fiber helps improve lipid levels, control blood pressure, and reduce systemic inflammation, slowing the progression of coronary artery disease and flow abnormalities. Moderate exercise aids in

weight management, enhances blood circulation, improves microvascular function, and boosts myocardial blood supply. Smoking cessation reduces the harm from tobacco-derived toxins, improves endothelial function, and lowers atherosclerosis risk. Additionally, managing comorbidities like hypertension and diabetes is crucial. A combination of pharmacological and non-pharmacological approaches ensures a comprehensive treatment and management strategy, which can more effectively alleviate CSFP symptoms and reduce cardiovascular events.

7. Prognosis and Risk Assessment of CSFP

7.1 The Impact of Slow Blood Flow on Long-term Prognosis

CSFP is closely related to clinical symptoms such as angina pectoris, myocardial ischemia, and even myocardial infarction, despite the absence of significant vascular stenosis on coronary angiography. Slow coronary flow not only affects the control of short-term symptoms but also significantly impacts long-term prognosis. It is often associated with myocardial under-perfusion, which increases cardiac workload and induces a variety of adverse cardiovascular events, especially in high-risk patients with comorbidities such as hypertension, diabetes, and a history of smoking [24]. According to research, CSFP is an independent risk factor for cardiovascular events and is associated with increased myocardial injury, heart failure, and cardiovascular mortality [25]. Myocardial ischemia caused by slow coronary flow can lead to frequent angina attacks and even progress to myocardial infarction. Patients with CSFP often experience a reduced quality of life due to the long-term burden of cardiovascular lesions. Clinical studies have indicated that patients with CSFP have higher recurrence rates and are more likely to face complications and poor prognosis. The presence of slow coronary flow indicates that there is an issue with the coronary microcirculation or other cardiovascular functions, which in turn affects overall cardiac health. Early identification and active treatment of CSFP can not only effectively alleviate symptoms but also reduce the risk of cardiovascular events to a certain extent.

7.2 Establishment and Clinical Significance of Risk Assessment Models

In order to accurately assess the prognosis of patients with CSFP and predict the occurrence of future cardiovascular events, researchers have begun to develop a variety of risk-assessment models. Based on the patients' clinical characteristics, laboratory test results, and imaging data, these models integrate multidimensional information to calculate the cardiovascular risk level of patients. Risk-assessment models usually involve patient factors such as age, sex, blood pressure, blood glucose levels, lipid profiles, family history, and lifestyle habits. Moreover, imaging findings (e.g., coronary angiography, cardiac MRI) and ECG presentations are also taken into account. The comprehensive evaluation of these factors can help doctors more accurately predict the likelihood of future cardiovascular events and disease-course progression in patients with CSFP [26]. By applying these assessment models, doctors can develop more scientific and rational treatment plans according to each patient's specific risk situation, choose appropriate interventions, and reduce the incidence of cardiovascular events and improve long-term prognosis.

8. Future Research Directions and Issues for CSFP

8.1 Research on Precise Mechanisms

While there is already some research on CSFP's pathogenesis, its exact pathological process remains unclear. CSFP involves multiple complex factors, including endothelial dysfunction, microvascular changes, and inflammation. Future research should focus on exploring molecular and cellular mechanisms, particularly endothelial cell dysfunction and microvascular adaptations. For instance, endothelial dysfunction can cause excessive vessel contraction and weakened dilation, worsening hemodynamics. Inflammation also plays a key role in CSFP, with inflammatory factors like CRP potentially exacerbating endothelial damage [5]. Besides these local factors, the interaction between genetic and environmental factors is worth studying. The development of genomics enables the exploration of individual genetic susceptibility and the relationship between specific gene variations and CSFP, which may offer new directions for precision medicine.

8.2 Development of New Diagnostic Technologies

The diagnosis of CSFP has long relied on traditional imaging methods, which, despite their effectiveness, still have many limitations. Therefore, innovative diagnostic techniques are urgently needed to improve the early detection rate and accuracy of coronary slow-flow. In recent years, the rapid advancement in medical imaging technology has provided new opportunities for the diagnosis of CSFP. Cardiac MRI, as a non-invasive imaging modality, has shown potential in diagnosing slow-flow. Combined with dynamic flow imaging, cardiac MRI can accurately measure blood velocity, flow rate, and vessel compliance without relying on coronary angiography, offering a detailed assessment of microvascular blood flow for patients [27]. The high-resolution MRI can also clearly display the dynamic changes in blood flow, helping to identify slow-flow phenomena in the subclinical stage and providing a solid basis for early diagnosis.

The emergence of high-resolution ultrasound technology has also brought a breakthrough in the microvascular assessment of coronary slow-flow. With contrast-enhanced ultrasound, researchers can precisely observe blood flow velocity, vessel wall motion, and microvascular response in real-time imaging, thus significantly improving the sensitivity and accuracy of slow-flow diagnosis. Compared with conventional coronary angiography, contrast-enhanced ultrasound can not only assess smaller vessels but also provide direct evidence of microvascular dysfunction for clinical use.

In addition, with the discovery and application of biomarkers, the diagnosis of coronary slow-flow is gradually shifting from imaging-based to a combination with blood-based assessments. In recent years, research has shown that biomarkers such as hs-TnI, CRP, and NO are closely related to the occurrence of coronary slow-flow. These biomarkers

not only reflect the inflammatory status of patients but also closely link with pathological processes such as microvascular dysfunction and endothelial damage [28]. Therefore, in the future, a comprehensive detection strategy combining multiple biomarkers is expected to enhance the early screening and prognostic evaluation of slow-flow, providing support for the development of precision medicine.

In summary, the future diagnosis of coronary slow-flow will increasingly rely on multidimensional assessments combining imaging and blood-based evaluations. By integrating novel technologies such as cardiac MRI, ultrasound imaging, and biomarkers, doctors will be able to comprehensively grasp the hemodynamic changes of patients from multiple levels, thereby realizing personalized and precise diagnostic solutions.

8.3 Personalized Treatment Strategies

The treatment strategies for CSFP still face many challenges in clinical practice. Although existing therapies can relieve symptoms in some patients, they still do not fully meet the needs of all patients. The pathogenesis of CSFP varies from patient to patient. Therefore, future research should be committed to developing individualized treatment plans according to the pathological mechanisms, clinical manifestations, and risk assessment results of different patients. Individualized treatment involves not only the selection and dosage adjustment of pharmacotherapy but also the precise choice of microvascular interventional therapy. For example, some patients may be more affected by microvascular pathology, while others may have blood flow impairment due to severe endothelial dysfunction. Thus, targeted selection of antiplatelet drugs, lipid-lowering agents, or calcium channel blockers can more effectively improve the blood flow status and reduce the recurrence of symptoms in patients. In addition, for patients with poor response to pharmacotherapy, microvascular interventional therapy (such as PCI or microvascular dilation surgery) may be another effective treatment option. In the future, individualized treatment should also be combined with precise molecular marker detection, imaging assessment, and the specific conditions of patients to form an integrated treatment system in order to improve the effectiveness of treatment and reduce the occurrence of adverse reactions.

9. Future Perspectives

With the continuous advancement of medical technology, the diagnosis and treatment methods for CSFP are also evolving. Future research should further uncover the intricate pathogenesis of CSFP, particularly exploring the interactions between inflammation, hemodynamic abnormalities, and genetic factors on the basis of microvascular and endothelial dysfunction. In addition, with the innovation of imaging techniques, new diagnostic methods such as high-resolution cardiac MRI and biomarker detection may offer new breakthroughs for the early diagnosis of CSFP, further enhancing the sensitivity and accuracy of diagnosis. In terms of treatment, individualized treatment strategies will be a key direction in the future. By conducting in-depth research on the clinical characteristics, pathological mechanisms, and risk-assessment results of patients, more precise treatment plans can be tailored for different types of CSFP patients. In summary, future research will focus on in-depth mechanism exploration, technological innovation, and individualized treatment, providing more accurate theoretical and practical support for the early warning, diagnosis, and treatment of CSFP.

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