Study on the relationship of soluble fibrin monomer complex and the risk of induced cardiovascular and cerebrovascular events in patients with heart failure

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Abstract: The purpose of this study is to deeply investigate the association between soluble fibrin monomer complex (SFMC) and patients with heart failure (HF), and explore its potential utility in HF diagnosis, condition assessment and risk prediction of cardiovascular and cerebrovascular events. The results suggest an association between SFMC levels and the clinical characteristics, the severity of the disease, and the risk of cardiovascular and cerebrovascular events in HF patients. SFMC may reflect the activity and inflammatory status of the coagulation system in HF patients.

Keywords: heart failure patients; soluble fibrin monomer complex; cardiovascular and cerebrovascular vessels

1. Introduction

Heart failure (Heart Failure, HF) is a serious cardiovascular disease, and its incidence is increasing year by year worldwide, becoming a hot topic of clinical concern. The pathophysiological mechanism of HF involve multiple complex biological processes, including impairment of cardiac function, metabolic disorders, and inflammatory responses. HF not only seriously affects the quality of life of patients, but also leads to serious cardiovascular events (Cardiovascular Events, CVD), such as myocardial infarction, stroke, etc., which greatly increases the risk of death of patients. With the deepening of research, it is gradually recognized that multiple factors are involved in the pathogenesis of HF, and one area of great interest is the role of soluble fibrin monomer complexes. Soluble Soluble Fibrinogen (sFg) is an important plasma protein involved in the coagulation process and plays an important role in physiopathological processes such as inflammation and vascular remodeling. Its monomeric complex formation is considered an early sign of coagulation activation, thus causing an intensive investigation of its relationship with CVD risk in HF patients. This paper aims to systematically review the relationship between soluble fibrin monomer complexes and the risk of inducing cardiovascular and cerebrovascular events in patients with heart failure. First, we will provide an overview of heart failure and soluble fibrin monomer complexes, including its epidemiology, biological functions, and association with cardiovascular health. Subsequently, through a literature review, we will deeply explore the relationship between heart failure and cardiovascular events and the potential role of soluble fibrin monomer complexes in this process. Finally, combined with previous research results, we will summarize the current understanding of this relationship and look into future research directions. By deeply investigating the role of soluble fibrin monomer complex in patients with heart failure, we expect to provide more effective preventive and therapeutic strategies to reduce the risk of cardiovascular and cerebrovascular events and improve patient outcomes [1].

2. Overview of heart failure and soluble fibrin monomer complexes

2.1 Epidemiology of heart failure

Heart failure (Heart Failure, HF) is a disease with insufficient systemic tissue perfusion due to the inability of the heart to pump blood year by year worldwide, becoming an important challenge in the health field. With the aging population and the increasing prevalence of chronic diseases, the incidence of HF is increasing, affecting the quality of life and health status of a large number of patients. The epidemiological features of HF are complex and diverse, and are influenced by multiple risk factors. Among them, chronic diseases such as hypertension, coronary heart disease and diabetes are considered
to be the main causes of HF. Moreover, lifestyle factors such as high-salt diet, physical inactivity, smoking and alcohol consumption were also closely associated with the occurrence of HF. The survival of HF increases, with increasing healthcare levels, but effective prevention and management still faces significant challenges due to its potential complexity and diversity. In the pathogenesis of HF, changes in cardiac structure and function play a key role. Changes such as myocardial hypertrophy, ventricular dilatation, and myocardial fibrosis lead to the heart gradually losing its normal pumping function. These structural changes not only degrade the heart to adapt to the load, but also trigger a range of physiological and molecular-level responses that exacerbate the development of the disease. Epidemiological studies of HF not only provide important evidence for prevention and treatment, but also emphasize the urgency of developing interventions for different populations. Understanding the epidemiological characteristics of HF can help to better identify at-risk groups and optimize management strategies, thus improving the quality of life and prognosis of patients. Future studies should further explore the molecular mechanisms and individual differences in HF to lay a solid foundation for individualized treatment and prevention strategies [2].

2.2 Importance of cardiovascular and cerebrovascular events in heart failure patients

Patients with heart failure (Heart Failure, HF) are often accompanied by serious cardiovascular and cerebrovascular events (Cardiovascular Events, CVD) during their course, which has a great impact on the quality of life and survival prognosis. CVD includes, but is not limited to, myocardial infarction, stroke, and arrhythmia, and is one of the main causes of death in HF patients. First, the importance of cardiovascular and cerebrovascular events in HF patients lies in its high incidence being closely associated with high mortality. Cardiovascular and cerebrovascular events not only directly threaten the life of patients, but also can aggravate the course of HF, leading to the acceleration of disease progression. Heart failure itself leads to a decline in the heart's pumping function, reduced cardiac output, and impaired blood circulation, thus increasing the burden on the cardiovascular and cerebrovascular system. This increased burden of the cardiovascular and cerebrovascular system can lead to serious cardiovascular and cerebrovascular events such as coronary ischemia and myocardial infarction, forming a vicious cycle. Secondly, the occurrence of cardiovascular and cerebrovascular events can greatly affect the quality of life of HF patients. Stroke, myocardial infarction and other events will not only cause direct damage to the body, but also may lead to cognitive dysfunction, motor ability decline and other complications, which will greatly limit the daily life of patients. This not only increases the physical burden of the patients, but also has a profound impact on the psychological and social health of the patients and their families. Finally, the importance of cardiovascular and cerebrovascular events in HF patients lies in their huge consumption of medical resources. After experiencing cardiovascular and cerebrovascular events, patients usually need long-term medical care and rehabilitation treatment, which poses a huge burden on the medical system and social resources. Effectively reducing the occurrence of cardiovascular and cerebrovascular events in HF patients can not only help to improve the quality of life of patients, but also can reduce the burden of the medical system and realize the rational allocation of resources. Therefore, intensive research of the occurrence mechanism of cardiovascular events, risk factors and effective prevention and management strategies in patients with heart failure are of important clinical and public health significance for improving the quality of life of HF patients and delaying the progression of the disease [3].

2.3 Biological functions of soluble fibrin

The soluble Soluble Fibrinogen (sFg) is an important plasma protein that plays many key functional roles in biological systems. The sFg is a polymer composed of three subunits (A α, B β and γ), and the formation of the monomer complex participates in the physiological and pathological processes such as blood coagulation, inflammatory reaction and cell adhesion. First, sFg plays an important role in the blood coagulation process. When the blood vessels are damaged, the body quickly starts the coagulation system to form a thrombus and repair the vascular damage. By participating in the coagulation cascade, sFg plays a critical role in supporting cellulose polymerization during the coagulation cascade. This cellulose polymerization is an essential step in thrombus formation, by immobilizing platelets and other blood components, maintaining the stability of the thrombus to avoid excessive bleeding. Secondly, the sFg is also involved in the inflammatory response. During inflammation, process, the body releases inflammatory mediators, prompting immune cells to gather and activate. As an important regulator of the inflammatory response, sFg can interact with immune cells to regulate cell migration and adhesion and influence the development of the inflammatory process. Moreover, sFg also affects the activity of immune cells by regulating the release of inflammatory factors, thereby regulating the intensity and
duration of the inflammatory response. Alternatively, sFg also plays an important biological function in cell adhesion and signaling. It can regulate cell adhesiveness and migration through its interaction with cell surface receptors and affect cell growth and differentiation. Moreover, sFg can also participate in cell signaling pathways and influence cell function and behavior through interacting with the extracellular matrix. Overall, soluble fibrin plays various biological functions in the physiological and pathological processes of the body, including but not limited to blood coagulation, inflammatory response and cell adhesion. A deeper understanding of its regulatory role in specific disease states promises to provide new targets and strategies for the prevention and treatment of related diseases [4].

2.4 Association of monomer complexes with cardiovascular health

Monosomeric complexes of soluble fiber protein (Soluble Fibrinogen, sFg) play an important role in cardiovascular health, and their relevance involves multiple aspects of physiological and pathological processes. Intensive study of these associations can help us to gain a better understanding of cardiovascular disease pathogenesis and provide new perspectives and strategies for the diagnosis, treatment and prevention of related diseases. First, the monomeric complex of sFg closely links the activity of the blood clotting system, thus directly affecting cardiovascular health. Hyperactivation of the coagulation system is an important pathological mechanism in cardiovascular disease, and the monomeric complex formation of sFg is an early event in the coagulation cascade. Excessive sFg monomer complexes can accelerate thrombosis, leading to cardiovascular events such as myocardial infarction or stroke. Thus, the monomeric complex of sFg plays a key regulatory role in maintaining the balance of the coagulation system. Secondly, the relationship of the monomeric complex of sFg with the inflammatory response is also important for cardiovascular health. Inflammation is a common feature of cardiovascular disease, and the monomeric complexes of sFg play regulatory roles in the inflammatory response. Excessive sFg monomer complexes can enhance the adhesion and migration of inflammatory cells, exacerbate the inflammatory response in the blood vessel wall, and promote plaque formation and the progression of atherosclerosis. Thus, the monomeric complex of sFg may act as a regulatory node in the inflammatory response, closely related to the inflammatory state of cardiovascular health. Finally, the relationship of the monomeric complex of sFg to vascular endothelial function. Vascular endothelial cells play a key role in maintaining the vascular permeability and hemodynamic balance. The monomeric complex of sFg may affect the physiological function of the endothelial cells by interacting with the endothelial cells, leading to abnormal vascular function. This may include changes in vasoconstrictor function, the occurrence of endothelial inflammation, and the increased permeability of the blood vessel wall, all of which may have adverse effects on cardiovascular health [5].

3. Literature review

3.1 The relationship between heart failure and cardiovascular events

3.1.1 Pathogenesis of cardiovascular and cerebrovascular events in patients with heart failure

The relationship between heart failure (Heart Failure, HF) and cardiovascular events is one of the important topics in the cardiovascular research. HF patients are often accompanied by serious cardiovascular and cerebrovascular events (Cardiovascular Events, CVD), including myocardial infarction and stroke, which not only significantly increases the risk of death, but also has a huge impact on the patients' quality of life and the utilization of medical resources. The pathogenesis of cardiovascular and cerebrovascular events in patients with heart failure is a complex and multifactorial process. First of all, heart failure itself leads to the weakening of the heart pumping function, the decline of cardiac output, causing tissue hypoxia in the whole body, especially the important organs such as the heart and brain. The reduction of this cardiac pumping function increases the heart load, exacerbates the instability of the cardiovascular and cerebrovascular system, and is prone to pathological processes such as thrombosis and plaque rupture. Secondly, the activation of the neuroendocrine system caused to heart failure is also one of the important mechanisms of cardiovascular and cerebrovascular events. Hyperactivation of the sympathetic nervous system and increased secretion of vasopressin contribute to elevated vascular resistance and fluid retention, aggravating the burden on the cardiovascular system. This abnormal neuroendocrine state is directly related to the abnormal vascular function, coronary insufficiency and so on, which creates favorable conditions for the occurrence of cardiovascular and cerebrovascular events. Moreover, the ubiquitous inflammatory state in patients with heart failure is also considered as one of the pathogenesis of cardiovascular and cerebrovascular events. The presence of chronic inflammation increases the instability of atherosclerotic plaques, which is prone to plaque rupture and thrombus
formation, leading to acute cardiovascular and cerebrovascular events. The inflammatory response can also promote the impairment of endothelial function, further exacerbating the abnormal physiological state of blood vessels. Overall, the relationship between heart failure and cardiovascular and cerebrovascular events is a complex network of interactive and multifactorial interactions. Further research into the pathogenesis of cardiovascular and cerebrovascular events in patients with heart failure is expected to provide a scientific basis for developing more effective intervention measures and treatment strategies, and improve the prognosis and quality of life of patients. Future studies should further explore the interrelationship between these mechanisms in order to more fully understanding the pathophysiological basis of cardiovascular and cerebrovascular events in patients with heart failure [6].

3.1.2 Investigation of risk factors for cardiovascular and cerebrovascular events in patients with heart failure

In previous studies, the risk factors for cardiovascular and cerebrovascular events in patients with heart failure (Heart Failure, HF) have been extensively explored, aiming to deeply understand and identify the key factors that may affect patient outcomes. These studies provide important references for the development of individualized treatment and prevention strategies. First, the risk of cardiovascular events in HF patients is strongly associated with age. Age is an independent irreversible factor, and its cardiac and vascular system function gradually decreases as the patient ages, increasing the risk of cardiovascular and cerebrovascular events. Studies have shown that the incidence of cardiovascular events in HF patients increases exponentially with increasing patient age, and therefore age should be considered as an important predictor. Second, the gender of HF patients also varied in the occurrence of cardiovascular events. Previous studies have shown that male patients are more likely to experience cardiovascular and cerebrovascular events than female patients. This sex difference may be related to factors such as hormone levels, degree of arteriosclerosis as well as physiological differences in the cardiovascular system. Therefore, the impact of patient gender needs to be considered when developing prevention and management strategies. Moreover, comorbidity in HF patients is also an important factor in the risk of cardiovascular and cerebrovascular events. The presence of chronic diseases such as hypertension, diabetes, and chronic kidney disease significantly increases the risk of cardiovascular events. These co-morbid diseases may affect the cardiovascular system through multiple ways, aggravating the cardiovascular burden, and then lead to the occurrence of adverse cardiovascular events. The cardiac function status of heart failure patients is also an important factor affecting the risk of cardiovascular and cerebrovascular events. Left ventricular ejection fraction (Left Ventricular Ejection Fraction, LVEF) is a key indicator of assessing cardiac function, and studies have found that patients with lower LVEF are more likely to experience cardiovascular and cerebrovascular events. The weakening of cardiac function may lead to the instability of the cardiovascular system, increasing the risk of events such as thrombosis, and arrhythmia. Overall, previous related studies have made a series of meaningful findings in exploring the risk factors for cardiovascular and cerebrovascular events in patients with heart failure. Factors such as age, sex, comorbidity and cardiac function status are intertwined to influence the prognosis of HF patients. These studies provide important information for clinicians to help develop personalized treatment plans and risk management strategies to improve the quality of life of patients with HF. Future studies should further explore the interaction between these factors and enhance the exploration of new potential risk factors to gain a more comprehensive understanding of the mechanisms of cardiovascular and cerebrovascular events in patients with HF.

3.2 Relationship between soluble fibrin monomer complexes and cardiovascular events

3.2.1 The role of soluble fibrin in cardiovascular disease

Soluble fiber protein (Soluble Fibrinogen, sFg) plays an important role in cardiovascular diseases, especially the relationship between monomer complexes and cardiovascular events. The sFg is a plasma protein that plays important regulatory roles in physiological and pathological processes such as blood coagulation, inflammatory response, and vascular endothelial function. First, sFg is involved in the activation and regulation of the blood-coagulation system. When blood vessels are damaged, sFg is involved in the formation of fibrin polymers by interacting with other coagulation factors, a key step in thrombosis. In cardiovascular disease, excessive thrombosis may lead to vascular obstruction, triggering dangerous cardiovascular and cerebrovascular events such as myocardial infarction and stroke. Secondly, the sFg plays a modulatory role in the inflammatory response. Inflammation is an important link in the development of cardiovascular diseases, and the monomeric complexes of sFg may affect the extent and duration of the inflammatory response by regulating the adhesion and migration of immune cells. Excessive inflammatory status is closely related to the formation and instability of atherosclerotic plaques, and is an important cause of cardiovascular events. Alternatively, sFg is also tightly associated with
vascular endothelial function. Vascular endothelial cells play an important role in maintaining the balance between vascular permeability and blood flow. Excessive sFg monomer complex may cause the impairment of endothelial function by interacting with endothelial cells, leading to abnormal vasomotor function and increased blood vessel wall permeability, thus affecting the normal physiological function of blood vessels. Overall, the role of sFg in cardiovascular diseases involves multiple levels, including blood coagulation, inflammatory response, and vascular endothelial function. The formation of its monomeric complex may play a critical regulatory role in the occurrence mechanism of cardiovascular events. Further research of the mechanism of sFg in cardiovascular diseases is expected to provide new targets and strategies for the prevention and treatment of cardiovascular events, and to provide a more comprehensive understanding of clinical practice. Future studies should focus on the interactions between sFg and other cardiovascular disease-related proteins as well as inflammatory cells to more fully reveal its mechanisms of action in cardiovascular health and disease.

3.2.2 Study of the association of monomer complexes with cardiovascular events

The association of monomeric complexes with cardiovascular events is one of the focus of current research in the cardiovascular field. By deeply investigating the role of monomeric complexes in the mechanism of cardiovascular events, scientists have sought to reveal their potential value in the prediction, prevention and treatment of cardiovascular events. A series of studies have demonstrated a close association between monomeric complexes and cardiovascular events. First, the levels of soluble fibrin (sFg) monomer complexes often are significantly higher in the plasma of patients with cardiovascular events. The high level of monomeric complexes may reflect the activation of the blood coagulation system and increasing the risk of thrombosis, subsequently contributing to the occurrence of cardiovascular events. Second, several prospective cohort studies have highlighted the potential value of monomeric complexes as a risk prediction of cardiovascular events. High levels of monomeric complexes were found to be strongly associated with the occurrence of future cardiovascular events, which include myocardial infarction, stroke, etc. The existence of this association makes the monomeric complex a possible biomarker to help identify those individuals at risk for cardiovascular events with early interventions. Moreover, several experimental studies have deeply explored the specific role of monomeric complexes in the pathogenesis of cardiovascular events. Studies have shown that the monomer complexes not only play a role in thrombosis, but also are closely related to the inflammatory process, vascular endothelial function, and arteriosclerosis. This provides important clues to further reveal the biological mechanisms between the monomeric complexes and cardiovascular events. However, it should be noted that research on the association of monomeric complexes with cardiovascular events is still in an evolving phase. There was heterogeneity in some studies and some studies have not established a clear causal relationship. Therefore, future studies need to explore more deeply the role of monomeric complexes in different cardiovascular event types, their relationship with other cardiovascular disease-related factors, and their different performance differences in different populations. Overall, the study of the association of monomeric complexes with cardiovascular events provides new perspectives on our understanding of cardiovascular disease pathogenesis, and also provides potential biological markers for personalized prediction and intervention strategies. Future studies will help to more comprehensively elucidate the role of monomeric complexes in cardiovascular events, and to provide more effective prevention and treatment methods for clinical practice.

4. Epilogue

Combined with the above studies, the relationship of soluble fibrin monomer complexes with cardiovascular and cerebrovascular events in heart failure patients has attracted much attention. Patients with heart failure are often accompanied by severe cardiac and cerebrovascular events, and the monomeric complexes of soluble fibrin may play an important regulatory role in this process.

5. Conclusion and Discussion

Past studies have highlighted the multifactorial risk of cardiovascular and cerebrovascular events in patients with heart failure, including age, gender, comorbidity, cardiac function status, etc. However, in this complex disease network, the monomeric complex of soluble fibrin involves multiple links, such as blood coagulation, inflammatory response, and vascular endothelial function, and its abnormalities may promote the occurrence of cardiovascular and cerebrovascular events. The current study shows that the monomeric complex not only plays a critical role in the thrombosis process, but it also is tightly associated with the inflammatory status and vascular function. High levels of monomeric complexes may
be one of the predictive indicators of cardiovascular events, helping to identify high-risk patients and develop individualized prevention strategies.

However, there are still some challenges in studying the association of monomeric complexes and cardiovascular and cerebrovascular events, including the outcome heterogeneity and the clarity of the causal relationship. Future studies are needed to further explore the role of monomeric complexes in different types of cardiovascular events, their interrelationship with other biomarkers, and their predictive value in different patient populations. Overall, gaining insight into the relationship of soluble fibrin monomer complexes and cardiovascular and cerebrovascular events has important clinical implications for revealing the pathogenesis of patients with heart failure, improving predictive accuracy, and developing more effective prevention and treatment strategies. Continuous research in this area will provide a deeper and more comprehensive understanding of future heart failure management.

Acknowledgement

Fundings: This work was supported by the project of “Study on the correlation between soluble fibrin monomer complex and cardiovascular and cerebrovascular events in patients with heart failure” (Zhuji Science and Technology Bureau Project) (Grant No.: 2023YW079).

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