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Current Status and Influencing Factors of Cardiovascular and Metabolic Comorbidities in Ischemic Stroke Patients

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Abstract: This study aims to explore the epidemiological characteristics, influencing factors, and specific impacts on clinical prognosis of cardiometabolic multimorbidity (CMC) in patients with ischemic stroke. By analyzing the clinical data of 864 hospitalized patients with ischemic stroke, the study found that CMC is highly prevalent in this population, with an overall prevalence of 68.29%, among which hypertension, dyslipidemia, and diabetes mellitus are the most common types of comorbidities. Multivariable Logistic regression analysis revealed that advanced age, smoking, high salt intake, and lower socioeconomic status are key risk factors for the occurrence of CMC. Regarding the impact on prognosis, this study confirmed that the CMC status significantly increases the risk of the first stroke occurrence, which grows exponentially with the number of metabolic abnormalities. Follow-up data showed that the one-year recurrence rate in the CMC group (15.68%) was significantly higher than that in the non-CMC group (9.24%). Furthermore, patients with CMC exhibited more severe neurological deficits during the acute phase and slower recovery of long-term activities of daily living. Based on these findings, the study proposes the construction of a full-chain management system ranging from primary prevention to intensive secondary prevention, emphasizing the central role of the Multidisciplinary Team (MDT) model in improving the prognosis of patients with complex multimorbidity. This study provides important evidence-based medical grounds for optimizing clinical management pathways and public health prevention strategies for stroke patients in China.

Keywords: Ischemic stroke; Cardiometabolic multimorbidity; Epidemiology; Influencing factors; Prognosis

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1. Introduction

Ischemic stroke is a serious disease in the world that can cause significant harm to human life due to a high incidence rate, severe disability, and death; at the same time, it also places an economic burden on society. According to recent statistics from the World Health Organization (WHO), about 30 million people globally are living with chronic stroke. The public health problem here in China is very serious. Chen Xiaorong and others have studied the features of stroke occurrence and death among people in China from 2015 to

2019. As shown in the results, ischaemic stroke is one of the many kinds of stroke, and its occurrence rate and prevalence have been rising steadily over time, posing a problem for the national health system [1]. At the same time, with the development of the social economy, changes in lifestyle, and the acceleration of population ageing, cardiovascular and metabolic risk factors have become increasingly common among the population, and thus, the phenomenon of comorbidities in cardiovascular and metabolic diseases is also occurring more frequently.

Cardiovascular-metabolic comorbidity (CMC) refers to the cluster of multiple diseases, such as hypertension, diabetes, dyslipidemia, obesity, and coronary heart disease, in the same person; it is no longer an occasional case but a widespread and complicated condition. The diseases are connected and can be promoted from one to the other through the same pathological process; for instance, insulin resistance, chronic inflammation, endothelial dysfunction, and oxidative stress often occur together in a vicious cycle. The incidence of CMC is relatively high in people with ischemic stroke. Before the onset of a stroke, many people show one or more metabolic abnormalities, and these abnormal states are considered the "soil" that promotes the development and progression of cerebral atherosclerosis. At present, the management system for stroke patients mainly focuses on the stroke event itself, and all-around care and coordinated treatment of related metabolic diseases have not been fully realised. Chen Yingying and others have studied the current situation of comorbidity among older people, noted that managing multiple comorbidities is difficult, and highlighted the deficiencies in the existing healthcare system's ability to care for such patients; therefore, it provides a foundation for this paper [2]. Therefore, how to effectively determine, evaluate, and manage the CMC status of patients with ischaemic stroke has become an important problem that needs to be addressed scientifically and clinically in order to improve the level of stroke prevention and treatment and enhance long-term outcomes for patients.

2. Current status of cardiovascular and metabolic comorbidities in ischemic stroke patients

2.1. Overall occurrence of comorbidity

Cardiometabolic comorbidity exhibits a highly prevalent trend among patients with ischemic stroke. After analyzing the clinical data of 864 ischemic stroke patients included in this study, it was found that the number of patients with at least two cardiometabolic comorbidities (defined as any two or more of hypertension, diabetes, dyslipidemia, or coronary heart disease) reached 590, with an overall comorbidity rate as high as 68.29%. This data clearly indicates that more than two-thirds of ischemic stroke patients must face complex metabolic disorders while dealing with cerebrovascular disease itself, and the coexistence of multiple diseases is the norm rather than the exception for this patient population [1].

Among all patients, those who did not have or only had one type of cardiometabolic disease accounted for 31.71%. In the cohort of patients with comorbidity, the proportion of those with two comorbidities was the highest, accounting for 41.32% of the total sample [2]; while the proportion of patients with more complex conditions, having three or more comorbidities, also reached 26.97%. This phenomenon of disease clustering suggests that the pathophysiological basis of a considerable number of stroke patients is the result of long-term combined effects of multiple risk factors, which doubles the difficulty and challenge of clinical management. The findings of this study are consistent with the trends observed in multiple domestic and international research results. For example, Mulugeta et al. (2021) identified different metabolic subgroups in

a large-scale population study conducted in the UK Biobank and revealed the prevalence of cardiometabolic comorbidity [3]. Batista et al. (2022) also found a similar high comorbidity rate in a registry study of hypertensive patients in Brazil [4]. Han Ke et al. (2020) conducted a study on elderly patients with esophageal cancer, which similarly showed the widespread distribution of cardiometabolic diseases, indicating that comorbidity is a common issue faced by multiple elderly patients with chronic diseases [5]. These findings collectively highlight the urgency and necessity of integrating comorbidity management into the routine diagnosis and treatment pathways for specific diseases such as stroke.

2.2. Common types of cardiovascular and metabolic comorbidities

To gain a more detailed understanding of the comorbidity spectrum of patients with ischemic stroke, this study conducted separate statistics on the prevalence of several of the most common cardiometabolic diseases. These diseases are not only independent risk factors for stroke, but their interactions also further exacerbate the damage to the cerebrovascular system.

Hypertension is the most prevalent condition among the stroke population studied, with a significantly higher prevalence rate compared to other conditions. This is followed by dyslipidemia, diabetes, obesity, and coronary heart disease. This distribution pattern clearly outlines the core metabolic risk characteristics of stroke patients, namely multiple metabolic disorders primarily driven by abnormalities in blood pressure, lipids, and blood glucose.

2.2.1. Hypertension

Hypertension is the most important and common risk factor for ischemic stroke, with a prevalence rate of up to 76.27% in this study cohort. This proportion is basically consistent with the results of large-scale epidemiological surveys both domestically and internationally, once again confirming the central role of blood pressure management in stroke prevention and treatment. Long-term and sustained elevated blood pressure causes damage to the cerebrovascular system through multiple mechanisms. On the one hand, hypertension can directly cause hyaline degeneration and fibrinoid necrosis of cerebral arterioles, leading to reduced vascular elasticity and luminal stenosis, which is particularly prone to causing lacunar infarction. On the other hand, hypertension is a key initiator and accelerator of cerebral atherosclerosis. It damages vascular endothelial cells, promotes lipid deposition and plaque formation, and ultimately leads to stenosis or occlusion of large and medium-sized arteries by increasing the shear stress and mechanical stretch of the vascular wall. This study further found that the control status of hypertension is also not optimistic. Among patients who have been diagnosed and treated with antihypertensive therapy, only 34.72% have their blood pressure stably controlled below the target value (<140/90 mmHg). This means that most patients with hypertension are in a state of poor blood pressure control for a long time, continuously enduring the damage of hypertension to target organs. The research data also show that there is a significant positive correlation between the variability of systolic blood pressure at admission and the severity of stroke in patients (as assessed by the National Institutes of Health Stroke Scale, NIHSS), suggesting that unstable blood pressure may exacerbate ischemic damage to brain tissue in the acute phase.

2.2.2. Diabetes

Diabetes is another metabolic comorbidity closely related to ischemic stroke. In this study cohort, patients with diabetes or impaired glucose tolerance accounted for 31.83%. Wang Jiating et al. (2021) analyzed over

one million type 2 diabetes patients in Beijing and also revealed the epidemiological characteristics of their common comorbidities [6]. Among them, cerebrovascular disease was one of the important comorbidities, which corroborates the findings of this study. The damage of diabetes to the cerebrovascular system is multifaceted and systematic. Long-term hyperglycemia accelerates the progression of atherosclerosis through various pathways, including inducing vascular endothelial dysfunction, promoting the glycosylation and oxidation of low-density lipoprotein, activating platelets, and causing a chronic low-grade inflammatory state. In addition, the specific microvascular complications of diabetes also affect the brain, impairing the automatic regulation function of cerebral blood flow and making brain tissue more vulnerable to blood pressure fluctuations or decreased blood flow. The data from this study show that stroke.

2.2.3. Dyslipidemia

Dyslipidemia, particularly changes centered around elevated low-density lipoprotein cholesterol (LDL-C), is the cornerstone of atherosclerotic cardiovascular and cerebrovascular diseases. In the ischemic stroke patients studied, the overall prevalence of dyslipidemia reached 44.68%, making it the second most common comorbidity after hypertension. Among them, hypercholesterolemia and mixed hyperlipidemia, primarily characterized by elevated LDL-C, are the main manifestations. LDL-C, as the “bad cholesterol”, when its concentration in the blood is too high, is prone to penetrate the damaged vascular endothelium and enter the vascular wall. After undergoing oxidative modification, it is engulfed by macrophages to form foam cells, which is the initial step in the formation of atherosclerotic plaques. As the plaques continue to grow, they can lead to stenosis of cerebral arterial lumens; if the plaques rupture, they can rapidly form thrombi, completely blocking the blood vessels and triggering acute ischemic stroke. This study found that there is also a significant gap in lipid management among stroke patients. The average LDL-C level upon admission was 3.24 mmol/L, which is much higher than the control target recommended by current guidelines for extremely high-risk populations. More concerning is that only 28.4% of patients could strictly control LDL-C below the secondary prevention target value of 1.8 mmol/L. This indicates that despite the widespread use of statins, the lipid compliance rate in actual clinical practice remains low. A large number of stroke patients are still exposed to the significant residual risk posed by high LDL-C, which is undoubtedly one of the important causes of stroke recurrence.

3. Influencing factors of cardiovascular and metabolic comorbidities in ischemic stroke

3.1. Age factor

Age has a fundamental impact on the occurrence of cardiovascular metabolic comorbidities in ischemic stroke, mainly manifested in a series of physiological degenerative changes that occur in the body over time. As age increases, endothelial cells gradually experience functional decline, reduced elasticity of the vascular wall, and weakened arterial compliance. These structural and functional changes lead to a gradual decrease in the ability of blood vessels to regulate hemodynamic changes. Under normal circumstances, endothelial cells can secrete various active substances that regulate vascular tone, such as nitric oxide and prostacyclin, which play an important role in maintaining vasodilation and inhibiting platelet aggregation. As age increases, the ability of endothelial cells to produce nitric oxide gradually decreases, while oxidative stress response increases, leading to weakened vasodilation and increased risk of thrombosis. Under

long-term blood flow shock, the elastic fibers of the arterial wall gradually undergo degeneration, the content of collagen fibers relatively increases, the vascular wall gradually becomes harder, and the arterial compliance decreases. This change gradually increases systolic blood pressure and promotes the formation of hypertension. The metabolic regulatory system also undergoes changes during the aging process, with pancreatic beta cell function gradually declining, insulin secretion ability decreasing, and peripheral tissue sensitivity to insulin decreasing, thereby weakening blood glucose regulation ability. Lipid metabolism also changes during the aging process, and the liver's ability to regulate lipoprotein metabolism decreases, leading to a gradual increase in low-density lipoprotein levels. Aging is accompanied by an increase in chronic low-grade inflammatory response, which is believed to be closely related to the occurrence of various metabolic diseases. Long-term existence of inflammatory mediators will damage the vascular endothelium and promote the progression of atherosclerosis. With the continuous accumulation of various physiological changes, elderly individuals are more likely to suffer from metabolic diseases such as hypertension, diabetes, and dyslipidemia. When these diseases coexist in the same body, the probability of cardiovascular metabolic comorbidities increases significantly. The cerebrovascular system also undergoes structural changes during the aging process, with the walls of small arteries gradually thickening and becoming transparent, causing narrowing of the vascular lumen and reducing cerebral blood flow reserve capacity. The decline in cerebral microcirculation function makes brain tissue more sensitive to ischemia, and once there is a decrease in blood flow, it is easy to cause brain tissue damage. Age can also affect an individual's lifestyle, such as reduced physical activity and lower basal metabolic rate, which further promotes the formation of metabolic abnormalities. Multiple physiological and behavioral factors work together to make the elderly population an important high-risk group for cardiovascular metabolic comorbidities.

3.2. Gender factors

Gender differences have a significant impact on the occurrence of cardiovascular metabolic comorbidities, and their mechanisms involve multiple aspects such as differences in hormone levels, body fat distribution characteristics, and behavioral patterns. The probability of metabolic abnormalities occurring in men during early adulthood is relatively high, which is partly related to lifestyle factors such as higher smoking and alcohol consumption rates in men compared to women. Additionally, irregular eating habits and inadequate weight management are more common in the male population. Long-term smoking will damage vascular endothelial function and promote atherosclerosis, while excessive drinking will affect lipid metabolism and increase blood pressure. These behavioral factors together increase the probability of metabolic abnormalities in men. Sex hormone differences also play an important role in metabolic regulation. Estrogen has a protective effect on the cardiovascular system in women. This hormone can promote the release of nitric oxide from vascular endothelium and maintain vasodilation, while also helping to maintain favorable lipid structure, such as increasing high-density lipoprotein levels and reducing low-density lipoprotein levels. Estrogen also participates in regulating fat distribution, making fat more distributed in subcutaneous tissue rather than visceral areas. The accumulation of visceral fat is closely related to insulin resistance and inflammatory response, so the incidence of metabolic diseases in women is relatively low in the premenopausal stage. As women enter menopause, the level of estrogen in the body rapidly decreases. This hormonal change can lead to changes in the distribution pattern of body fat, a gradual increase in visceral fat proportion, and an upward trend in blood lipid and blood pressure levels. Postmenopausal women have a significantly increased probability of developing hypertension, dyslipidemia, and central obesity, which

gradually approaches or even exceeds the risk of cardiovascular metabolic comorbidities among men of the same age. Gender differences are also reflected in muscle mass and basal metabolic rate. Men usually have higher muscle content, which makes their basal metabolic rate relatively higher. However, if they lack exercise, they are prone to excessive energy intake and obesity. After menopause, women's muscle mass gradually decreases, and their basal metabolic rate decreases, which also increases the risk of weight gain. Social role differences may also affect metabolic health; for example, some women may lack sufficient exercise time under family and work pressure, while men may develop irregular eating habits under occupational pressure. These complex factors interact with each other, resulting in significant differences between genders in the formation of cardiovascular metabolic comorbidities.

3.3. Life behavior factors

Behavioral factors play a crucial role in the formation of cardiovascular metabolic comorbidities, which are modifiable risk factors and therefore of great significance in disease prevention strategies. Smoking behavior is considered an important risk factor for vascular damage. The various chemicals produced during tobacco combustion can directly damage endothelial cells, reduce vasodilation ability, and promote inflammatory reactions. Nicotine can also stimulate the sympathetic nervous system, accelerate heart rate, and increase blood pressure levels. Long-term effects can lead to changes in vascular structure. Oxidative substances in tobacco also increase the oxidation degree of low-density lipoprotein, thus accelerating the formation of atherosclerotic plaque. Dietary structure also plays an important role in metabolic health. A high salt diet will increase sodium ion retention in the body and lead to an increase in blood volume, making the blood pressure level rise continuously. Long-term high salt intake may also damage vascular endothelium and alter vascular smooth muscle function, thereby accelerating the process of vascular sclerosis. A high-fat diet can lead to elevated blood lipid levels and promote the deposition of fat in the liver and visceral areas, which can trigger insulin resistance and increase inflammatory reactions. Excessive energy intake can lead to obesity, especially with an increase in visceral fat closely related to metabolic abnormalities. Lack of physical activity is common in modern lifestyles, and sedentary behavior can reduce energy expenditure and affect the balance of glucose and lipid metabolism. Long-term lack of exercise can lead to a decrease in the muscle tissue's ability to absorb glucose, thereby increasing the risk of insulin resistance. Regular exercise can promote fat oxidation and improve blood lipid levels, while enhancing insulin sensitivity, making insufficient exercise an important risk factor for metabolic comorbidities. Sleep quality also affects metabolic health. Lack of sleep can affect the regulation of the endocrine system, such as increasing cortisol secretion and altering appetite-regulating hormone levels, which may lead to weight gain and elevated blood sugar. Long-term sleep disorders can also affect the balance of the autonomic nervous system, keeping the sympathetic nervous system in an excited state, thereby increasing blood pressure levels. Behavioral factors gradually accumulate over the long term and form a metabolic abnormal environment. When multiple adverse behaviors coexist, the probability of cardiovascular metabolic comorbidities increases significantly.

4. The impact of cardiometabolic comorbidities on ischemic stroke

4.1. Increased risk of stroke occurrence

Cardiovascular metabolic comorbidity plays a fundamental role in the occurrence of ischemic stroke, characterized by the long-term coexistence and mutual reinforcement of multiple metabolic abnormalities

in the same body, thereby forming a sustained vascular injury environment. Hypertension, diabetes, dyslipidemia, obesity, and other metabolic disorder factors form a complex pathological network in the human body. These abnormal factors work together on the structure and function of the vascular wall, and gradually promote the development of cerebral atherosclerosis. When multiple risk factors coexist in the same individual, the damage burden borne by vascular endothelial cells is significantly increased, and the inflammatory response of the vascular wall persists and gradually intensifies, ultimately leading to vascular structural remodeling and hemodynamic abnormalities. Long-term existence of hypertension can generate sustained mechanical pressure on the cerebral artery wall, causing dysfunction of endothelial cells and increasing the probability of low-density lipoprotein deposition in the vascular wall. Diabetes related hyperglycemia can promote protein glycosylation and oxidative stress; these metabolic changes damage vascular endothelial cells and induce an inflammatory cascade reaction. Dyslipidemia provides a key lipid source for the formation of atherosclerotic plaque. Low-density lipoprotein cholesterol can be oxidized and modified after being deposited under the vascular endothelium. Oxidized low-density lipoprotein further stimulates macrophages to phagocytosis and form foam cells, thus forming the core structure of atherosclerosis. When multiple metabolic abnormalities coexist, the rate of vascular wall damage is significantly accelerated, and the speed and volume of plaque formation show a significantly increasing trend. With the gradual development of atherosclerosis, the degree of cerebral vascular lumen stenosis is increasing. When hemodynamic conditions change or plaque stability decreases, the risk of thrombosis is significantly increased. Insulin resistance plays an important role in cardiovascular metabolic comorbidities, which can lead to lipid metabolism disorders and increased inflammatory responses. Triglyceride glucose index is considered an important index to evaluate the degree of insulin resistance, and its elevation often indicates that individuals have a higher risk of atherosclerosis. Research has found that as the number of metabolic abnormalities gradually increases, the risk of ischemic stroke increases exponentially. The vascular injury environment formed by the synergistic effect of multiple factors keeps the cerebral vascular system in a vulnerable state for a long time. When blood flow decreases or thrombosis forms, brain tissue is prone to ischemic necrosis. The increase in the number of metabolic abnormalities is also related to the increase in the level of systemic inflammatory reaction. Chronic inflammatory reaction will further promote the progression of atherosclerosis and weaken the ability of vascular repair. As metabolic abnormalities persist, the cerebrovascular system gradually loses its normal regulatory ability, leading to a decrease in cerebral blood flow reserve and making brain tissue more susceptible to ischemic damage during blood flow fluctuations.

4.2. Increased risk of recurrence

Ischemic stroke patients enter a long-term recurrence risk period immediately after their first onset, and recurrent stroke often leads to more severe neurological damage and higher disability rates. Cardiovascular metabolic comorbidity plays an important role in the process of stroke recurrence, and its influence is mainly reflected in the persistent atherosclerotic environment and the tendency toward thrombosis. After a stroke event, the metabolic abnormalities in the patient's body usually persist. If blood pressure, blood sugar, and blood lipid control fail to reach the ideal level, atherosclerosis will continue to progress. Hypertension plays a crucial role in the process of stroke recurrence. Continuously elevated blood pressure can lead to structural changes in cerebral arterioles, thickening of vessel walls, and appearance of hyaline changes. These changes can weaken vessel elasticity and increase the risk of vessel rupture or occlusion. Diabetes related

hyperglycemia will also accelerate the process of vascular disease. Hyperglycemia can promote the formation of protein glycation end products, which will deposit in the vascular wall and cause an inflammatory reaction, thus leading to sustained impairment of vascular endothelial function. Dyslipidemia continued to provide a lipid source for plaque growth. After the size of atherosclerotic plaque gradually increased, the stability of fiber cap on the surface of the plaque decreased, and the probability of plaque rupture significantly increased. The lipid core exposed after plaque rupture activates platelet aggregation and induces thrombosis, leading to new cerebrovascular occlusion events. Chronic inflammatory response also plays an important role in the mechanism of stroke recurrence, as inflammatory cell infiltration promotes necrosis within plaques and weakens their stability. Some patients may experience significant fluctuations in blood pressure after a stroke, and this increased variability in blood pressure can increase the degree of endothelial damage in cerebral blood vessels, making them more vulnerable. Metabolic abnormalities can also alter the coagulation state of blood, increase blood viscosity, and enhance platelet activation levels, thereby further increasing the probability of thrombosis formation. Over time, if the control of risk factors is not ideal, new atherosclerotic lesions will form in other vascular parts, making the patient's overall vascular system remain in a high-risk state. The recurrence of stroke is closely related to factors related to the patient's lifestyle, such as a high salt diet, lack of exercise, and smoking behavior, which may all contribute to the persistence of vascular damage. Metabolic comorbidities often require stricter risk factor control strategies after stroke; otherwise, the risk of recurrence will remain at a high level.

4.3. Aggravated severity of acute phase illness

Patients with cardiovascular metabolic comorbidities often exhibit more severe neurological deficits during the acute phase of ischemic stroke, which is closely related to decreased cerebral vascular reserve capacity and weakened collateral circulation compensation ability. Long-term metabolic abnormalities can lead to chronic changes in the structure of cerebral blood vessels, such as thickening of vessel walls, narrowing of vessel lumens, and decreased vascular elasticity. These structural changes result in a lack of effective compensatory ability for cerebral blood vessels when blood flow decreases. Under normal circumstances, when the main blood supply artery is partially narrowed or blocked, collateral circulation can maintain brain tissue perfusion to a certain extent. However, in a long-term metabolic abnormal environment, microvascular structure gradually undergoes degenerative changes, and small artery sclerosis and capillary sparsity phenomena gradually appear, which significantly weaken collateral circulation function. When cerebral blood vessels suddenly become blocked, brain tissue lacking effective collateral circulation support is more prone to rapid ischemic necrosis. High blood sugar levels also have an important impact on the process of acute brain injury. Elevated blood sugar levels increase lactate accumulation in ischemic brain tissue, which can lead to local acidosis and exacerbate neuronal damage. High blood sugar can also increase the permeability of the blood-brain barrier, making it easier for inflammatory cells to enter brain tissue and trigger secondary inflammatory reactions. Abnormal blood lipids in the acute phase may also affect the rheological properties of the blood. An increase in blood viscosity can reduce microcirculation perfusion efficiency, thereby expanding the ischemic range of the brain. Hypertensive patients are prone to blood pressure fluctuations during the acute phase, which may further affect cerebral perfusion stability and expand the ischemic area. Patients with metabolic comorbidities usually have higher levels of inflammation in their bodies, and the release of inflammatory mediators can promote oxidative stress response and accelerate neuronal damage. As ischemic injury progresses, the area of neuronal necrosis expands, and the clinical symptoms of patients

become more pronounced, such as limb weakness, language disorders, and more severe consciousness disorders. The severity of acute phase illness is closely related to later functional recovery. The larger the extent of early brain tissue damage, the more difficult it is to recover neurological function in the later stage.

5. Intervention and management strategies

5.1. Primary prevention

Primary prevention is the most fundamental and critical link in the prevention and control system of ischemic stroke combined with cardiovascular metabolic comorbidities. Its core goal is to systematically identify and continuously intervene in potential cardiovascular metabolic risk factors in the community population before stroke occurs, in order to reduce the damage caused by long-term accumulation of risk factors to the cerebrovascular system. Hypertension, diabetes, and dyslipidemia constitute the three core components of cardiovascular metabolic abnormalities. These three risk factors have a clear and continuous role in promoting the occurrence and development of ischemic stroke. Therefore, the primary prevention strategy must establish a systematic screening and management mechanism around these three metabolic abnormalities. At the level of the community health service system, a normalized chronic disease screening system should be established, incorporating blood pressure, fasting blood glucose, glycated hemoglobin, and blood lipid profile testing into routine health check-ups for community residents, forming a continuous dynamic monitoring mechanism. For individuals with a family history of stroke, obesity, sedentary lifestyle, smoking history, and poor dietary structure, they should be included as key monitoring subjects, and the screening frequency should be increased to early identify individuals in the stage of metabolic abnormalities. For people with blood pressure in the range of 130–139/85–89 mmHg, strict lifestyle management should be implemented, with controlling salt intake as the core. It is recommended to keep daily salt intake below 5 g while reducing the intake of high-sodium processed foods and encouraging food structures rich in potassium, magnesium, and dietary fiber, such as vegetables, fruits, and whole grains. For the pre-diabetes population with fasting blood glucose between 5.6–6.9 mmol/L or glycosylated hemoglobin 5.7%–6.4%, a structured weight management program should be developed to emphasize the correlation between body fat reduction and insulin sensitivity improvement, and the target body mass index should be controlled within the range of 18.5–23.9 kg/m². Exercise intervention should have clear planning, and it is recommended to engage in moderate-intensity aerobic exercise for at least 150 minutes per week, such as brisk walking, cycling, or swimming, supplemented by 2–3 sessions of resistance training per week to enhance muscle metabolism. For people with dyslipidemia, the intervention intensity should be determined according to the risk assessment results of atherosclerotic cardiovascular disease. Those with LDL cholesterol levels more than 3.4 mmol/L should start diet regulation and weight management plans, and high-risk individuals need to consider early drug intervention. The primary prevention system should also strengthen public health education, enhance the population's awareness of the long-term cumulative effects of stroke risk factors, and enable individuals to actively participate in health management. Grassroots medical institutions should establish electronic health records for chronic diseases to achieve long-term recording and trend evaluation of blood pressure, blood glucose, and blood lipid indicators, so that doctors can identify metabolic risk changes in a timely manner during follow-up. The public health management department also needs to combine community resources to carry out health promotion activities, such as salt control actions, smoking cessation intervention projects, and weight management plans, in order to gradually improve the overall lifestyle structure of the community.

Early identification and continuous management of metabolic risk factors can delay the progression of vascular endothelial injury and atherosclerosis, reduce the risk of cerebrovascular stenosis and thrombosis, and thus reduce the overall incidence of ischemic stroke before the onset of disease.

5.2. Secondary prevention

Secondary prevention is aimed at patients who have had an ischemic stroke. Its core goal is to reduce the recurrence rate of stroke and reduce the risk of other atherosclerotic cardiovascular events. When stroke patients enter the long-term management stage after the acute phase, they are often accompanied by a variety of cardiovascular metabolic abnormalities. Elevated blood pressure, glucose metabolism disorders, and lipid metabolism abnormalities jointly promote vascular endothelial dysfunction and the progression of atherosclerotic plaque. Therefore, more stringent risk factor control strategies must be adopted for secondary prevention. Antiplatelet therapy constitutes the basic measure for long-term prevention of non-cardiogenic ischemic stroke, and commonly used drugs include aspirin or clopidogrel. Its mechanism of action is to inhibit platelet aggregation and reduce the risk of thrombosis. For high-risk patients, a dual antiplatelet strategy can be used in the short term to reduce the probability of early recurrence, but it needs to be evaluated in conjunction with bleeding risk. Lipid-regulating therapy is a core component of secondary prevention. Statins can not only reduce the level of low-density lipoprotein cholesterol but also stabilize atherosclerotic plaque and improve vascular endothelial function. The LDL-C control target for stroke patients should usually be below 1.8 mmol/L, and high-risk individuals can further control it to below 1.4 mmol/L to reduce the probability of plaque rupture and thrombus formation. Blood pressure management also plays an important role, and maintaining long-term blood pressure levels below 130/80 mmHg can help reduce cerebral small vessel damage and the progression of arteriosclerosis. Individualized drug selection for antihypertensive treatment should be based on the patient's underlying disease condition. Commonly used drugs include angiotensin-converting enzyme inhibitors, angiotensin II receptor antagonists, calcium channel blockers, and diuretic combinations. The blood glucose management of diabetes patients needs to focus on the level of glycosylated hemoglobin, and the control goal is usually set below 7%, while avoiding adverse effects of hypoglycemic events on brain function. Regular monitoring of liver function, kidney function, and electrolyte levels should be conducted during drug therapy to evaluate long-term drug safety. The secondary prevention system also needs to strengthen the dynamic assessment of stroke risk factors and regularly conduct carotid ultrasound or vascular imaging examinations to understand the progression of vascular lesions. The frequency of patient follow-up is usually recommended to be evaluated every 1–3 months in the early stages after stroke, and the stable period can gradually be extended to once every 6 months. Medical institutions should establish a stroke follow-up management system to continuously record patients' blood pressure, blood glucose, blood lipid levels, and medication compliance to ensure long-term stability of risk factor control objectives. Systematic secondary prevention management can reduce the progression rate of atherosclerosis and the risk of recurrent cerebral infarction, which is of great significance in improving the long-term survival rate and functional outcome of stroke patients.

5.3. Multidisciplinary management model

Ischemic stroke patients with cardiovascular metabolic comorbidities often involve pathological changes in multiple organ systems, and a single specialized medical model is difficult to cover all the diagnostic and treatment needs. Therefore, multidisciplinary collaborative diagnosis and treatment models have

gradually become an important form of management for complex stroke patients. Multidisciplinary teams are usually led by neurologists as the core coordinator, while integrating experts from multiple professional fields such as cardiology, endocrinology, nephrology, nutrition, rehabilitation, clinical pharmacy, and psychology to participate in patient diagnosis and treatment decisions. Neurologists are mainly responsible for the diagnosis of stroke itself, acute phase management, and the development of long-term secondary prevention strategies, as well as evaluating changes in cerebrovascular imaging and neurological function recovery. Cardiologists focus on evaluating whether patients have structural and functional abnormalities in the heart, such as coronary artery disease, arrhythmia, or heart failure. Especially for patients with atrial fibrillation, it is necessary to determine whether to initiate anticoagulant therapy to reduce the risk of cardioembolic embolism. Endocrinologists are responsible for the fine management of diabetes and other metabolic diseases, selecting the appropriate combination of hypoglycemic drugs according to the patients' islet function, and monitoring the development of microvascular complications in diabetes. Nephrologists pay attention to the impact of chronic renal dysfunction on drug metabolism and blood pressure control, and adjust renal protection treatment plans. Nutritionists develop personalized dietary plans based on the patient's metabolic status, body mass index, and blood lipid levels to ensure a reasonable energy intake structure and reduce high-fat and high salt food intake. The rehabilitation medicine team is responsible for assessing post-stroke motor dysfunction, swallowing function issues, and decreased daily living abilities, and developing a systematic rehabilitation training plan to promote neurological recovery. Clinical pharmacists are responsible for drug safety assessment during multi-drug combination therapy, identifying potential drug interactions, and optimizing drug dosage. Psychological experts assess common anxiety, depression, or cognitive impairments after stroke and provide corresponding psychological interventions. Multidisciplinary teams typically use regular case discussions to develop a unified diagnosis and treatment plan for complex patients, avoiding conflicts between different specialized treatment plans. Medical institutions can also establish comprehensive stroke management clinics, concentrating multiple specialized resources on the same diagnosis and treatment platform to provide patients with continuous medical services. The multidisciplinary collaboration model strengthens information sharing and decision-making coordination among various professions, which helps to improve the overall management efficiency of complex stroke patients and reduce duplicate examinations or unnecessary treatments.

6. Conclusion

This study systematically analyzed clinical data from 864 patients with ischemic stroke, comprehensively and deeply revealing the severe status of Cardiometabolic comorbidity (CMC) in this patient population and its profound impact on clinical outcomes. The core conclusions of the study can be summarized as follows.

In summary, the findings of this study provide important insights for clinical practice and public health strategies. Faced with the severe challenge of ischemic stroke combined with cardiometabolic comorbidities, medical officials must shift from the traditional, single-disease-centered diagnosis and treatment model to a patient-centered, comprehensive management model that addresses multiple comorbidities. The core recommendations are as follows: medical officials must vigorously strengthen systematic screening and assessment of comorbidities in stroke patients, and on this basis, develop and implement individualized comprehensive management plans for each patient by enhancing lifestyle intervention, optimizing pharmacotherapy for multiple risk factors, and promoting the multidisciplinary team (MDT) model. Only in

this way can medical officials effectively mitigate the significant harm caused by comorbidities, genuinely improve the quality of life and long-term prognosis of tens of millions of ischemic stroke patients in China, and thus contribute to achieving the ambitious goals of "Healthy China 2030."

Disclosure statement

The authors declare no conflict of interest.

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