Biomedical Data Science
2024, VOL. 4, NO. 3, 52-57
DOI: 10.47297/wspbdsWSP2752-630509.20240403

Blood pressure variability and coronary atherosclerotic plaque formation: a clinical and pathological association

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ABSTRACT

This paper explores the complex clinical and pathological link between blood pressure variability (Blood Pressure Variability, BPV) and coronary atherosclerotic plaque formation. Through a systematic review and analyzing the existing research evidence, this paper reveals the potential mechanism of BPV in the onset and progression of coronary atherosclerosis, and further clarifies its value as an important clinical indicator to predict the severity of coronary atherosclerotic plaque and prevent disease deterioration. The results show that the increase of blood pressure level and the increase of BPV is closely related to the development of atherosclerosis, therefore, close monitoring of BPV not only helps to accurately assess the risk of coronary atherosclerosis plaque, also for developing targeted effective intervention strategy provides an important basis, thus in the prevention and control of cardiovascular disease has a profound significance.

KEYWORDS

blood pressure variability (BPV); coronary atherosclerosis; plaque formation; clinical association; and pathological features

1 Introduction

Coronary artery atherogenic heart disease, commonly known as coronary heart disease, is an extremely important and prevalent type in the field of cardiovascular disease. It shows a surprisingly high incidence and high fatality rate around the world, posing a great threat to human health. With the increasing aging of the global population and the widespread spread of bad lifestyles, coronary heart disease has become the number one killer of human life and health. Its pathological basis, namely the formation of atherosclerotic coronary plaques, is the main culprit for a series of serious cardiovascular events such as myocardial ischemia and myocardial infarction. In view of the serious harm of coronary heart disease, this paper aims to deeply explore the complex relationship between blood pressure variability (BPV) and coronary atherosclerotic plaque formation. We hope that this research can provide a new scientific basis and ideas for the early prevention, accurate diagnosis and effective treatment of coronary heart disease. To achieve this goal, we will comprehensively analyze the clinical data and pathology research results, and strive to reveal the specific mechanism of action of BPV in the process of coronary atherosclerosis. We firmly believe that through this study, we can provide strong support for the development of individualized antihypertensive treatment plan and reduce the risk of cardiovascular events. This study not only has important theoretical value, but also has far-reaching practical significance for improving the public health situation and improving the quality of people's life. We hope that through this research, we can contribute to the prevention and treatment of coronary heart disease, and contribute to the cause of human health.

2 Concept and measurement method of blood pressure variability

2.1 Define the BPV

Blood pressure variability (Blood Pressure Variability, BPV) refers to the degree of individual blood pressure values fluctuating around their average level over a certain period of time, and is an important parameter in the risk assessment of cardiovascular disease. Compared with traditional static BP measurements, BPV better reflects the dynamic changes of blood pressure, including short-term (e. g., within 24 hours) and long-term (e. g., weeks to months) BP fluctuations. This volatility not only correlated with the severity of hypertension, but also independently predicted the risk of cardiovascular events such as myocardial infarction, stroke and heart failure in [1]. Therefore, a thorough understanding of the properties of BPV and its relationship with cardiovascular disease is important for optimizing the management of hypertension and reducing cardiovascular complications.

2.2 Measurement method

BPV is measured in diverse ways designed to capture BP variation at different time scales. Here are several common measurements:

24-hour Ambulatory Blood Pressure Monitoring (Ambulatory Blood Pressure Monitoring, ABPM): ABPM is currently one of the most accurate methods for assessing BPV. By wearing a portable sphygmomanometer, blood pressure values are automatically measured and recorded at set intervals, usually including daytime and nighttime blood pressure. ABPM can provide detailed information on the circadian rhythm of blood pressure, morning peak phenomenon and blood pressure load, which can help to assess the full picture of BPV.

Home self-measured blood pressure: Patients are encouraged to measure blood pressure regularly at home and record the results. Although this method is not as accurate as ABPM, it is convenient for long-term follow-up, helping to detect concealed hypertension or white coat hypertension, while monitoring BPV in daily life.

Inter-clinic BP variation: Changes in BP between clinic visits [2] by measuring BP at different time points (e. g. at each visit). Although this method is affected by many factors (e. g., patient mood, ambient temperature, etc.), it can still be used as a reference indicator to assessing long-term BPV.

Central arterial pressure and pulse pressure variation: Central arterial pressure and pulse pressure (the difference between systolic and diastolic blood pressure) were measured with special equipment to assess the stiffness of the arterial system and the effects of BPV on the heart and blood vessels. This approach is particularly important for gaining a deeper understanding of the pathophysiology of BPV.

3 Clinical association of blood pressure variability with coronary atherosclerotic plaque formation

3.1 Epidemiological evidence

Extensive epidemiological study data strongly support the strong link between increased hypertension and increased blood pressure variability (BPV) and the formation and development of coronary atherosclerotic plaques. Hypertension, as a core risk factor for coronary heart disease, its existence itself greatly increases the risk of disease [3]. This risk is further amplified when the BPV increases. Specifically, several studies have shown that patients with higher BPV show significant increases in the incidence of coronary atherosclerotic plaques, the severity of lesions, and the rate of

progression compared to patients with lower BPV. In addition, the increase of BPV is positively related with the incidence of cardiovascular events, especially myocardial infarction and unstable angina, which further highlights the importance of controlling BPV in the prevention of coronary heart disease.

3.2 Pathophysiological mechanisms

BPV contributes to the formation and development of coronary atherosclerosis through several complex pathophysiological mechanisms. First, frequent blood pressure fluctuations can cause direct damage to the vascular endothelial cells, leading to endothelial dysfunction. This damage not only increases the vascular permeability, but also provides a favorable environment for the lipid deposition and the inflammatory response. Secondly, an increase in BPV activates the oxidative stress response in the body, producing large amounts of free radicals. These free radicals cause damage to the blood vessel wall and accelerate the process of atherosclerosis. In addition, blood pressure fluctuations can also stimulate the immune system and promote the infiltration of inflammatory cells and the release of inflammatory factors, such as interleukins, tumor necrosis factor, etc. These inflammatory substances play a key role in the promotion of plaque formation and destabilization. Finally, changes in the shear force of the vessel wall due to BPV can affect the hemodynamic stability and promote platelet aggregation and thrombus formation, thus accelerating the growth of atherosclerotic plaque [4]. Together, these mechanisms make BPV an important driver in the formation and development of coronary atherosclerotic plaques.

4 Blood pressure variability with pathological features of coronary atherosclerotic plaque formation

4.1 Pathological findings and its mechanism

When ving into the complex relationship between blood pressure variability (BPV) and atherosclerotic plaque formation in coronary arteries, we have to examine its unique pathological findings in detail. Numerous studies have revealed that the morphological features of coronary atherosclerotic plaques present significant differences under the influence of different BPV levels. Especially in high BPV environments, plaques tend to exhibit larger volumes, thinner fibrous caps, more lipid cores, and a wider range of calcification. These remarkable pathological features, not only reveal the high instability of the plaque, but also greatly enhance the risk of its rupture and the triggering of acute cardiovascular events.

Mechanism of impaired plaque stability: In a high BPV environment, frequent fluctuations in blood pressure lead to an extremely uneven stress distribution within the plaque. This uneven stress distribution makes the weak area easy to form inside the plaque, which is more prone to rupture [5] when subjected to external pressure. This increased risk of rupture has undoubtedly increased the incidence of cardiovascular events.

The process of accelerated lipid deposition: The increase in BPV will significantly promote the permeability of endothelial cells to lipoproteins, allowing lipids to be more easily deposited on the blood vessel wall. This acceleration of deposition not only promotes the formation of the lipid core, but also increases the volume of the plaque, which further aggravates the plaque instability.

Reasons for the reduction in fibrous cap thickness: Continuous blood pressure fluctuations can damage the repair mechanism, leading to their gradual thinning. As the outer protective structure of the plaque, the reduced thickness of the fibrous cap means a reduced resistance to the internal pressure of the plaque, thus making the plaque more prone to rupture.

4.2 The strong association of unstable plaques with cardiovascular events

Rupture of unstable plaques is the main cause of serious cardiovascular events such as acute myocardial infarction. However, through the above multiple mechanisms, BPV significantly aggravates the plaque instability, greatly increasing the risk of plaque rupture. Once the plaque rup, the released lipids and inflammatory substances quickly trigger thrombosis, thus blocking the coronary artery and leading to myocardial ischemia and necrosis. Therefore, effective control of BPV is crucial for preventing the formation and rupture of unstable plaques in [6], as well as for reducing the risk of cardiovascular events. By deeply studying the pathological features between BPV and coronary atherosclerotic plaque formation, we can provide a strong scientific basis for developing more effective preventive and treatment strategies.

5 Clinical intervention and management strategies

In the clinical management of blood pressure variability (BPV) and the formation of coronary atherosclerotic plaques, effective interventions are the key to prevent and control disease progression. The following three aspects are elaborated on: drug treatment, non-drug treatment and individualized treatment principles.

5.1 Drug treatment strategies

Drug therapy is the core means of reducing BPV and the prevention and control of coronary atherosclerosis. Calcium channel blockers (CCB), due to its unique antihypertensive mechanism, show a strong antihypertensive and anti-atherosclerotic effects. In addition, drugs such as diuretics, β -blockers, and angiotensin-converting enzyme inhibitors (ACEI) are also commonly used in the management of BPV, [7]. However, when choosing drugs, the patient details must be fully considered, such as age, complications, as well as drug tolerance. For example, older patients may be more suitable for diuretics or ACEI, while younger patients may be more inclined to use CCB or β -blockers. With rational drug selection, BPV can be controlled more effectively and reducing the risk of cardiovascular events.

5.2 Drug treatment method

Non-pharmacological treatment is a basic measure to reduce BPV and prevent atherosclerosis, and its importance cannot be ignored. A low-salt diet helps to reduce sodium intake, which reduces blood volume and effectively controls blood pressure. Moderate exercise can enhance the cardiopulmonary function, improve the vascular endothelial function, and further reduce the BPV. At the same time, smoking cessation and alcohol restriction are also essential measures, which can reduce vascular damage and reduce the risk of cardiovascular disease. In addition, psychological adjustment and maintaining good sleep quality are also important aspects of BPV control. Through the comprehensive application of these non-pharmacological treatments, a more comprehensive health management program can be provided for patients.

5.3 Application of physalizing therapeutic principles

Given the significant individual differences in BPV, the development of individualized treatment options is crucial to improve treatment effectiveness. Doctors should consider the advantages and disadvantages of drug therapy and non-drug therapy according to their BPV level, cardiovascular risk stratification, comorbidities and personal preferences, and develop the most suitable treatment

plan for patients. For patients with high BPV with significant target organ damage, antihypertensive therapy should be intensified and changes in BPV should be closely monitored, to allow timely adjustment of therapeutic strategies. Through the application of individualized treatment principles, we can more effectively control the BPV of patients, reduce the risk of cardiovascular events, and improve the quality of life of patients.

6 Discussion

6.1 Research limitations and future directions

Although the current study has preliminarily revealed the complex association between BPV and coronary atherosclerotic plaque formation, there are still many limitations. For example, the measurement method of BPV has not been standardized, and there are differences in the measurement indicators and assessment criteria adopted between different studies, affecting the comparability of the results. Moreover, the specific molecular mechanisms between BPV and coronary atherosclerosis are not fully understood, and deeper basic studies are needed to elucidate.

Future research should be devoted on the following aspects: first, to optimize the measurement technology of BPV and establish unified evaluation criteria; second, to explore the molecular mechanism of BPV affecting coronary atherosclerosis, especially the interaction with genetic factors; third, to conduct large-scale, multi-center clinical trial [8] to verify the long-term effect of interventions for BPV on the prevention of cardiovascular events.

6.2 Significance in clinical practice

The research results of this paper have an important guiding significance for clinical practice. First, the importance of monitoring BPV in the prevention and treatment of coronary atherosclerosis is emphasized, suggesting that doctors should fully consider BPV when assessing the cardiovascular risk of patients. Secondly, the principle of individualized treatment based on BPV level, which provides scientific basis for clinical decision-making. Finally, the role of non-pharmacological treatment in BPV control and atherosclerosis prevention is highlighted, encouraging patients to actively participate in self-management and improve lifestyle.

In conclusion, as a new dimension of cardiovascular disease risk assessment, the association study between BP variability and coronary atherosclerotic plaque formation not only deepens our understanding of the pathogenesis of cardiovascular disease, but also provides new ideas and strategies for the prevention and treatment of CHD. In the future, with the deepening of research and technological progress, it is reasonable to believe that more refined blood pressure management can effectively reduce the risk of cardiovascular events and improve the quality of life of patients.

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