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Physical Activity, Blood Pressure, Blood Viscosity, and Hypertensive Heart Disease Risk: A Narrative Review

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ABSTRACT

Hypertension is a major cardiovascular risk factor that can induce structural and functional cardiac alterations and ultimately contribute to the development of hypertensive heart disease (HHD). Persistently elevated blood pressure increases cardiac workload, while changes in blood viscosity may affect blood flow characteristics and vascular function. Physical activity is an established nonpharmacological component of hypertension management; however, its effects on blood viscosity and the risk of HHD progression warrant further investigation. This narrative review examines the effects of physical activity on blood pressure and blood viscosity and explores their relationship with mechanisms underlying HHD development. Relevant literature was identified through PubMed, scientific journal and publisher websites, and the reference lists of pertinent publications. Interventional and observational studies, clinical reviews, systematic reviews, and meta-analyses were synthesized descriptively and organized thematically. The available evidence suggests that aerobic exercise, dynamic resistance training, combined training, high-intensity interval training (HIIT), and isometric exercise may reduce resting blood pressure. Improvements in 24-hour ambulatory blood pressure have been reported more consistently with aerobic, combined, and HIIT interventions. Physical activity may also influence hemorheological parameters, including hematocrit, erythrocyte aggregation, and blood viscosity, although findings vary across studies. Exercise-related blood pressure control may alleviate the hemodynamic burden contributing to cardiac structural alterations. Nevertheless, evidence that changes in blood viscosity directly mediate the prevention of HHD remains limited. Physical activity is important for hypertension management, but further research is needed to clarify its effects on blood viscosity and HHD progression.

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INTRODUCTION

Hypertension is a major cardiovascular risk factor that can cause target-organ damage when inadequately controlled. Persistently elevated blood pressure places an increased workload on the heart, particularly the left ventricle, as it maintains systemic

circulation. Over time, this increased pressure load may induce structural and functional alterations collectively described as hypertensive heart disease (HHD). These changes include left ventricular hypertrophy (LVH), impaired ventricular relaxation and filling, myocardial fibrosis, and ultimately heart failure. Disease progression may be gradual and asymptomatic in its early stages. Effective blood pressure management is therefore essential to reduce the hemodynamic burden associated with the development of HHD (Pastore et al., 2024; Yu et al., 2026).

The pathogenesis of HHD extends beyond increased blood pressure and involves several interrelated physiological and cardiovascular mechanisms. Sympathetic nervous system and renin–angiotensin–aldosterone system activation, endothelial dysfunction, oxidative stress, and inflammation can contribute to myocardial structural and functional alterations. These processes are associated with increased cardiac workload, altered vascular stiffness, and cardiac remodeling. In addition to hemodynamic factors, blood flow properties merit consideration, particularly blood viscosity, a hemorheological determinant of resistance to blood flow. Blood viscosity is influenced by hematocrit, plasma viscosity, erythrocyte aggregation, and red blood cell deformability. Although viscosity is physiologically relevant to blood flow, changes in this parameter should not be equated directly with changes in blood pressure or HHD progression (Pastore et al., 2024; Romain et al., 2011; Connes et al., 2013).

Regular physical activity is an important nonpharmacological strategy for managing hypertension. In particular, structured exercise can promote cardiovascular adaptations through changes in vascular function, blood pressure regulation, and other physiological responses. Several exercise modalities have been investigated, including aerobic training, dynamic resistance training, combined aerobic and resistance training, high-intensity interval training (HIIT), and isometric exercise. Meta-analyses incorporating large numbers of randomized controlled trials have shown that these modalities can lower resting blood pressure, although effect sizes differ according to the type of exercise and study characteristics. In addition to resting measurements, 24-hour ambulatory blood pressure monitoring provides a broader assessment of blood pressure responses during daily activities and sleep. Current evidence suggests relatively consistent ambulatory blood pressure benefits with aerobic, combined, and HIIT interventions, whereas the evidence for certain other modalities remains less conclusive (Edwards et al., 2023; Schneider et al., 2026).

Beyond blood pressure regulation, physical activity may influence hemorheological properties and blood viscosity. These responses may differ between a single bout of exercise

and sustained training over time. Acute exercise of sufficient intensity can transiently alter hematocrit and blood component concentrations, primarily through fluid shifts and changes in hydration status. In contrast, regular exercise may induce adaptations in hemorheological parameters, including erythrocyte aggregation and blood viscosity. Nevertheless, studies evaluating the effects of exercise on blood viscosity have produced heterogeneous findings. Participant characteristics, exercise type and intensity, intervention duration, laboratory methods, and blood sampling time may all influence reported outcomes. These factors should be considered carefully to avoid the unsupported assumption that all exercise modalities consistently reduce blood viscosity (Romain et al., 2011; Connes et al., 2013).

In Indonesia, physical activity has also been incorporated into community-based hypertension management programs, including antihypertensive exercise sessions, fitness programs for older adults, isometric training, and low-impact aerobic exercise. Several local studies have reported reductions in blood pressure following exercise interventions among people with hypertension. However, these investigations are limited by relatively small sample sizes, variable intervention durations, and study designs that do not consistently permit strong causal inferences. Their findings support the feasibility of implementing exercise within community hypertension management, but they do not establish a direct effect on blood viscosity or the prevention of HHD (Octavia et al., 2025; Veralia et al., 2023; Adiputra et al., 2021; Syavinka & Yuniartika, 2024).

Across the published literature, evidence for the blood pressure–lowering effects of physical activity is more consistent than evidence for changes in blood viscosity. Although improved blood pressure control can physiologically reduce cardiac workload, available clinical studies have not fully established how exercise, hemorheological changes, and the prevention of HHD progression are related. Differences in measured outcomes and study methods further complicate an integrated interpretation of these relationships. A synthesis bringing together evidence on physical activity, blood pressure, blood viscosity, and HHD-related cardiovascular alterations is therefore warranted. This narrative review aims to examine the effects of different exercise modalities on blood pressure and blood viscosity, discuss the underlying physiological mechanisms, and critically appraise their potential relationship with HHD risk and progression in light of the available scientific evidence.

METHODS

This article uses a narrative review approach to examine the effects of physical activity on blood pressure and blood viscosity and their possible relationship with the risk

of hypertensive heart disease (HHD) progression. The literature considered includes studies of physical activity among individuals with hypertension, changes in hemorheological parameters, and physiological mechanisms and alterations in cardiac structure and function relevant to HHD. Relevant publications were identified through PubMed, scientific journal and publisher websites, and screening of reference lists. Search terms included "physical activity," "exercise training," "hypertension," "blood pressure," "blood viscosity," "hemorheology," "hypertensive heart disease," and "left ventricular hypertrophy," along with related terms. The literature encompassed interventional and observational studies, clinical reviews, systematic reviews, meta-analyses, clinical practice guidelines, and experimental investigations relevant to the mechanisms of HHD development.

Literature selection was guided by relevance to the review objectives, with emphasis on the effects of different exercise modalities on resting and 24-hour ambulatory blood pressure, blood viscosity and related hemorheological measures, and the mechanisms linking hypertension to alterations in cardiac structure and function. Indonesian studies were also considered to illustrate the implementation of physical activity interventions in community and primary care settings. Relevant findings were analyzed descriptively and synthesized narratively under themes encompassing blood pressure responses, blood viscosity changes, vascular adaptations, and HHD-related myocardial remodeling. Findings across studies were compared with attention to study design, population characteristics, intervention type, and outcome measures to identify areas of agreement and divergence. No new statistical pooling or systematic risk-of-bias assessment of all included studies was performed. Accordingly, this review seeks to provide an integrated interpretation of the available evidence and its limitations rather than a pooled effect estimate of the kind generated by a meta-analysis.

RESULT AND DISCUSSION

The reviewed literature indicates that the cardiovascular effects of physical activity should be evaluated across distinct outcomes, including resting blood pressure, 24-hour ambulatory blood pressure, hemorheological parameters, vascular function, and cardiac structural alterations. These outcomes provide complementary information but cannot be used interchangeably as evidence of HHD prevention. Overall, the most consistent evidence concerns blood pressure reduction, whereas a causal association between changes in blood viscosity and the prevention of HHD has not been established (Pastore et al., 2024;

Edwards et al., 2023; Schneider et al., 2026; Romain et al., 2011; Connes et al., 2013; de Castro et al., 2020). Key studies are summarized in Table 1.

Table 1. Summary of key studies included in the review

Author (year)	Study design	Focus	Key findings/interpretation
Edwards et al. (2023)	Network meta-analysis; 270 RCTs	Exercise modalities and resting blood pressure	All major modalities reduced resting blood pressure; rankings do not establish universal superiority.
Schneider et al. (2026)	Network meta-analysis; 31 trials	24-hour ABPM	Aerobic, combined, and HIIT interventions showed benefits; comparative superiority remains uncertain.
Romain et al. (2011)	Meta-analysis; 11 studies; n = 175	Post-exercise hemorheological parameters	Changes in erythrocyte aggregation, hematocrit, and viscosity; very high heterogeneity.
Connes et al. (2013)	Hemorheology review	Acute versus chronic responses	Inconsistent changes dependent on protocol, hydration, and sampling time.
Pastore et al. (2024)	Clinical review of HHD	Role of exercise across the HHD spectrum	Distinguishes physiological exercise adaptations from pathological hypertensive changes.
Akbar et al. (2023)	Preclinical systematic review	Apoptosis and fibrosis pathways in hypertensive hearts	Promising biological signals that cannot be directly extrapolated to humans.
de Castro et al. (2020)	Systematic review and meta-analysis	Exercise plus antihypertensive therapy in LVH	Cardiac structural outcomes evaluated separately from blood pressure.
Veralia et al. (2023)	Local intervention study	Isometric handgrip training in hypertension	Implementation evidence; design and duration limit generalizability.

Abbreviations: RCT, randomized controlled trial; ABPM, ambulatory blood pressure monitoring; HIIT, high-intensity interval training; LVH, left ventricular hypertrophy. The table presents selected key studies; additional sources inform comparisons and clinical interpretation.

Sustained hypertension increases left ventricular pressure overload and can lead to hypertrophy, geometric remodeling, and impaired myocardial relaxation. Although these alterations fall within the spectrum of HHD, the clinical course varies among individuals

with hypertension. The value of physical activity should therefore be assessed primarily in terms of its ability to modify hemodynamic risk factors, rather than on the assumption that every reduction in blood pressure necessarily reverses established cardiac structural damage (Pastore et al., 2024; Yu et al., 2026).

In a network meta-analysis by Edwards and colleagues comprising 270 randomized controlled trials and 15,827 participants, aerobic training, dynamic resistance training, combined training, HIIT, and isometric training were all associated with reductions in resting blood pressure (Edwards et al., 2023). These findings demonstrate that the benefits of exercise are not restricted to one modality. Nevertheless, rankings derived from network meta-analysis must be interpreted in the context of population characteristics, the number of available comparisons, estimate precision, and the comparability of exercise protocols. Although isometric exercise yielded a particularly large estimated reduction in systolic blood pressure, this does not establish it as the optimal intervention for every patient.

The clinical significance of these findings becomes clearer when considered alongside practice guidelines. The 2024 European Society of Cardiology (ESC) guidelines emphasize aerobic physical activity as a foundation of lifestyle management, with resistance exercise as a complementary intervention; the 2025 American Heart Association/American College of Cardiology (AHA/ACC) guidelines also incorporate different forms of exercise into nonpharmacological management (McEvoy et al., 2024; AHA/ACC, 2025). Consequently, exercise prescription should account for safety, comorbidities, functional capacity, and long-term adherence rather than relying solely on the magnitude of blood pressure reduction reported in meta-analyses. Previous evidence syntheses on resistance training further support its inclusion in comprehensive management programs (Cornelissen & Smart, 2013; MacDonald et al., 2016).

A reduction in blood pressure measured at rest does not necessarily reflect blood pressure responses throughout the day and night. Evidence from 24-hour ambulatory blood pressure monitoring (ABPM) must therefore be evaluated separately. A network meta-analysis by Schneider and colleagues included 31 trials involving 1,345 participants and found the most consistent ambulatory blood pressure benefits with aerobic training, combined training, and HIIT; the findings for dynamic resistance and isometric exercise were less conclusive for this outcome (Schneider et al., 2026). The estimated reductions in ambulatory systolic/diastolic blood pressure were 4.73/2.76 mmHg with aerobic training, 6.18/3.94 mmHg with combined training, and 5.71/4.64 mmHg with HIIT. These estimates should not be interpreted as demonstrating the universal superiority of any one modality.

These results are consistent with a meta-analysis by Saco-Ledo and colleagues involving 15 randomized trials and 910 patients with hypertension, which reported mean reductions in 24-hour ambulatory systolic and diastolic blood pressure of 5.4 and 3.0 mmHg, respectively, compared with control groups (Saco-Ledo et al., 2020). An earlier review also supported the ambulatory blood pressure benefits of endurance exercise (Cornelissen et al., 2013). Although the direction of benefit is relatively consistent, participant characteristics and antihypertensive medication use may influence effect estimates, highlighting the need for individualized assessment. Table 2 compares reported reductions in resting and ambulatory blood pressure from major studies. Because the estimates were derived from different trial populations and measurement settings, they should not be interpreted as direct head-to-head comparisons.

Table 2. Estimated blood pressure reductions by exercise modality

Exercise modality	Resting SBP/DBP (mmHg)	24-hour ABPM SBP/DBP (mmHg)
Aerobic training	−4.49/−2.53	−4.73/−2.76
Dynamic resistance training	−4.55/−3.04	Inconclusive
Combined training	−6.04/−2.54	−6.18/−3.94
HIIT	−4.08/−2.50	−5.71/−4.64
Isometric training	−8.24/−4.00	Inconclusive

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure. Resting blood pressure estimates are derived from Edwards et al. (2023), whereas 24-hour ABPM estimates are derived from Schneider et al. (2026). "Inconclusive" indicates insufficiently robust evidence for that outcome, not evidence that the exercise modality is ineffective.

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After considering the effects of exercise on blood pressure, an important question is whether alterations in blood flow properties contribute to these effects. Blood viscosity is determined by factors including hematocrit, plasma viscosity, erythrocyte aggregation, and erythrocyte deformability. These variables do not necessarily change in parallel, and blood viscosity cannot be inferred from blood pressure alone. Responses to an acute exercise bout should also be distinguished from adaptations to repeated training. During acute exercise, fluid shifts and sweat-induced fluid loss may transiently increase hematocrit and blood viscosity, whereas regular training can produce different hemorheological responses (Romain et al., 2011; Connes et al., 2013).

Direct evidence concerning exercise-induced hemorheological changes remains considerably more limited than evidence regarding blood pressure. A meta-analysis by Romain and colleagues of 11 studies involving 175 participants reported exercise-related changes in erythrocyte aggregation, hematocrit, and blood viscosity, but heterogeneity was very high in several analyses, including an I^2 of approximately 99% for blood viscosity (Romain et al., 2011). A review by Connes and colleagues likewise found inconsistent results, including reductions, increases, or no change in specific hemorheological measures (Connes et al., 2013). Considered together, these findings do not support the blanket claim that exercise invariably decreases blood viscosity. Rather, hemorheological responses depend on the training protocol, participant characteristics, hydration status, blood sampling time, and analytical methods.

The interpretation of blood viscosity becomes particularly important in the context of HHD. Physiologically, changes in viscosity may influence flow characteristics and vascular resistance, but blood pressure is also governed by vessel diameter, cardiac output, and vascular regulation. An observed association with, or alteration in, a hemorheological parameter is therefore insufficient to establish that it mediates exercise-induced blood pressure reduction, much less that it prevents myocardial remodeling. The literature considered in this review does not provide adequate clinical evidence to confirm a causal pathway linking physical activity, reduced blood viscosity, and HHD prevention (Pastore et al., 2024; Romain et al., 2011; Connes et al., 2013).

In addition to effects on blood flow properties, physical activity can promote endothelial and vascular adaptations. Modulation of sympathetic activity, neurohormonal regulation, and nitric oxide bioavailability offers plausible mechanisms for improved blood pressure regulation and vascular responsiveness (Pastore et al., 2024; Yu et al., 2026). A systematic review by Pedralli and colleagues of five studies involving 362 participants identified evidence suggestive of improved flow-mediated dilation (FMD) in hypertension, while a review by Waclawovsky and colleagues of ten trials supported the potential benefits of continuous aerobic exercise for vascular function (Pedralli et al., 2018; Waclawovsky et al., 2021). A 2024 meta-analysis by Liang and colleagues, including 37 studies and 2,801 participants, also reported exercise-related changes in nitric oxide indicators, endothelin-1, and FMD (Liang et al., 2024).

Although these findings strengthen the biological plausibility of exercise-related cardiovascular benefits, endothelial function and vascular biomarkers are intermediate outcomes. They are neither direct measures of blood viscosity nor evidence of reduced HHD incidence. A similar distinction applies to arterial stiffness and wave reflection, as discussed by Ashor and colleagues: measured changes may be influenced by blood pressure at the time of examination and do not necessarily indicate reversal of tissue fibrosis (Ashor et al., 2014). Distinguishing intermediate markers from clinical outcomes is essential to avoid drawing conclusions that exceed the available evidence.

At the myocardial level, the relationship between hypertension and HHD also involves oxidative stress, inflammation, and extracellular matrix remodeling. Zhang and colleagues discussed the potential contribution of macrophages to the progression from hypertension to heart failure (Zhang et al., 2025). In a preclinical systematic review of 11 experimental studies, Akbar and colleagues reported that nine studies described increases in selected cell-survival markers, ten reported reductions in proapoptotic markers, and only two reported fibrosis-related changes (Akbar et al., 2023). These findings provide a

rationale for mechanistic investigation; however, animal data and cellular biomarkers cannot be directly equated with clinical benefits in patients with HHD.

Evidence more directly related to cardiac structure comes from studies assessing left ventricular mass. A meta-analysis by de Castro and colleagues combined five studies involving 1,738 participants and compared exercise plus antihypertensive medication with medication alone. The combined approach was associated with reductions in left ventricular mass and heart rate, but findings for left ventricular mass index, ejection fraction, and blood pressure were not equally robust across analyses (de Castro et al., 2020). These observations extend the discussion beyond blood pressure outcomes, although the limited number of studies and variability in measured endpoints constrain the strength of the conclusions. Fagard also emphasized that physiological cardiac adaptations to exercise should not be confused with pathological hypertrophy arising from hypertension (Fagard, 1997).

When these levels of evidence are considered together, a coherent but nuanced pattern emerges. The strongest evidence supports exercise as a strategy for reducing blood pressure, a major contributor to left ventricular pressure overload. Findings relating to endothelial function, inflammatory markers, and left ventricular mass suggest additional pathways of benefit, but these intermediate measures are distinct from incident heart failure or newly diagnosed HHD. Meanwhile, hemorheological studies have not established that changes in blood viscosity constitute the principal link between exercise and myocardial protection. Thus, the benefits of physical activity for hypertension management are more firmly supported than claims that it prevents HHD specifically through reductions in blood viscosity (Pastore et al., 2024; Edwards et al., 2023; Schneider et al., 2026; Romain et al., 2011; Connes et al., 2013; Akbar et al., 2023; Zhang et al., 2025; de Castro et al., 2020).

Within the Indonesian context, community-based research suggests opportunities for implementing exercise interventions, although the strength of evidence must be assessed according to study design. In a study of 250 participants attending integrated noncommunicable disease health posts (Posbindu), Syamsi reported an association between insufficient physical activity and hypertension (prevalence ratio, 2.85; 95% confidence interval, 1.70–4.77); however, its cross-sectional design does not establish causality (Syamsi, 2019). In contrast, studies by Octavia and colleagues on antihypertensive exercise, Veralia and colleagues on isometric handgrip exercise, and Adiputra and colleagues and Syavinka and Yuniartika on exercise programs for older adults assessed changes following interventions (Octavia et al., 2025; Veralia et al., 2023; Adiputra et al.,

2021; Syavinka & Yuniartika, 2024). The broadly consistent direction of these findings supports the feasibility of exercise programs in primary care but does not confer on pre-experimental or quasi-experimental designs the same evidentiary strength as randomized controlled trials.

For example, in a study by Adiputra and colleagues involving 36 older adults, mean systolic blood pressure in the intervention group decreased from 168.1 to 153.4 mmHg, while mean diastolic blood pressure decreased from 95.56 to 82.67 mmHg (Adiputra et al., 2021). Syavinka and Yuniartika reported favorable blood pressure changes following eight weeks of low-impact aerobic exercise in 35 participants, while Veralia and colleagues investigated handgrip training within a chronic disease management program (Syavinka & Yuniartika, 2024; Veralia et al., 2023). Although these reports are relevant to Posbindu, the Chronic Disease Management Program (Prolanis), and community exercise initiatives, small sample sizes, variation in intervention duration, and limitations in control-group design prevent robust comparisons of effect sizes across modalities. None of these local findings establishes a direct effect on blood viscosity or HHD prevention.

The clinical implication of this synthesis is that physical activity should be incorporated into hypertension care as an adjunct to, rather than a replacement for, pharmacological treatment and medical assessment. Moderate-intensity aerobic activity may provide the foundation of an exercise program for many individuals, supplemented by resistance training or combined programs according to individual capacity. HIIT and isometric exercise require appropriate protocol selection and safety assessment. The 2024 ESC guideline recommendations discussed in this review include approximately 150 minutes of moderate-intensity aerobic activity per week and low-to-moderate-intensity resistance exercise two to three times weekly, with gradual progression according to the individual's condition (McEvoy et al., 2024; Piercy et al., 2018). Medication adherence, blood pressure monitoring, and appropriate evaluation of concerning symptoms remain integral to care.

The main limitations of the available evidence include heterogeneity in exercise intensity and duration, concomitant antihypertensive treatment, and methods used to measure blood pressure and hemorheological parameters. Studies of blood viscosity frequently involve smaller samples and variable blood collection conditions, while studies that simultaneously evaluate ABPM, blood viscosity, and cardiac structural changes remain scarce in the literature reviewed (Edwards et al., 2023; Schneider et al., 2026; Romain et al., 2011; Connes et al., 2013; de Castro et al., 2020). Furthermore, some findings concerning apoptosis, macrophage activity, and fibrosis are derived from mechanistic or preclinical

research and should not be presented as established clinical benefits (Akbar et al., 2023; Zhang et al., 2025).

Future research should evaluate exercise interventions using well-defined protocols specifying frequency, intensity, duration, and type, while recording medication use and participant adherence. Simultaneous assessment of resting and ambulatory blood pressure, hydration status and blood sampling time, hemorheological parameters under standardized conditions, and cardiac structure and function using imaging would enable a more comprehensive evaluation of potential mechanistic pathways. Long-term follow-up for clinical outcomes, including HHD progression and incident heart failure, is needed before changes in blood viscosity can be considered a mediator of exercise-related cardioprotection. Accordingly, the present synthesis supports physical activity for hypertension control while underscoring important evidence gaps concerning blood viscosity and HHD prevention (Ashor et al., 2014; de Castro et al., 2020; Almarshad, 2026).

CONCLUSION

Physical activity is an important component of hypertension management. Meta-analyses indicate that aerobic training, dynamic resistance training, combined training, HIIT, and isometric exercise can reduce resting blood pressure. For 24-hour ambulatory blood pressure, the most consistent benefits have been reported with aerobic training, combined training, and HIIT, whereas the evidence for dynamic resistance and isometric exercise remains inconclusive.

Blood pressure reduction and improvements in cardiovascular function provide a biological rationale for the potential role of physical activity in limiting HHD progression. However, evidence regarding exercise-induced changes in blood viscosity remains limited and heterogeneous, and it does not establish blood viscosity as a mediator of this relationship. Direct evidence that exercise prevents cardiac structural changes or incident heart failure is also limited. Physical activity should therefore be integrated with indicated pharmacological treatment and other lifestyle modifications.

The narrative approach permits a broad conceptual synthesis, but the study selection cannot be assumed to be exhaustive. No formal risk-of-bias assessment was undertaken for every article, no GRADE certainty-of-evidence assessment was conducted, and no new meta-analysis was performed. Findings from animal models, observational studies, and human intervention trials were distinguished during interpretation. Future research with standardized protocols, simultaneous assessment of hemorheological and

cardiac structural outcomes, and long-term clinical follow-up is recommended to clarify the mechanistic role of blood viscosity in exercise-related cardioprotection.

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