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Using structural equation model to analyse the effect of psychological pressure on the patients with essential hypertension blood pressure and blood biochemical indicators

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Abstract

Background Essential hypertension (EH) was the result of the combined action of environmental and genetic factors, and its risk factors were numerous, psychological factors were thought to contribute to the development of EH.

Methods We randomized 600 outpatients and inpatients during the year from January 2018 to December 2021 in the Ningbo Medical Treatment Center Li Huili Hospital. Basic information including blood pressure, blood lipids, AngII, aldosterone and Homocysteine was recorded, and the Chinese Perceived Stress Scale (CPSS) questionnaire was completed. We divided them into four groups: high psychological pressure hypertension group(HPPH), low psychological pressure hypertension group(LPPH), high psychological pressure control group (HPPC) and low psychological pressure control group (LPPC). Using SEM to explore the direct and indirect effects of psychological pressure on blood pressure and biochemical indicators.

Result SBP, DBP, TC, TG, LDL-C, Ang II, ALD and Hcy of HPPH were significantly higher than those of LPPH, HPPC and LPPC ($p < 0.05$). SBP, DBP, TC, TG, LDL-C, Ang II, ALD and Hcy of LPPH were significantly higher than those of HPPC and LPPC. SBP, DBP, TC, TG, LDL-C, Ang II, ALD and Hcy of HPPC were significantly higher than those of LPPC. HDL-C were lowest in HPPH group. Psychological pressure had significantly effect on blood pressure, lipids, AngII, ALD and Hcy($P < 0.05$), and it had positive relationship with SBP, DBP, TC, TG, HDL-C, Ang II, ALD, Hcy ($P < 0.05$), and had negative relationship with HDL-C. AngII and ALD had significantly effects on blood pressure, lipids ($P < 0.05$), and it had positive relationship with SBP, DBP, TC, TG, ALD($P < 0.05$), and had negative relationship with HDL-C($P < 0.05$). Blood pressure had significantly effects on Lipids and Hcy ($P < 0.05$), it had positive relationship with TC, TG, LDL-C ($P < 0.05$), and had negative relationship with HDL-C($P < 0.05$).

Conclusion The research demonstrated that psychological pressure plays an important role in the pathophysiological process of essential hypertension and may serve as a potential target for comprehensive hypertension management.

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Introduction

Essential hypertension(EH) was a highly prevalent condition that had been identified as a major risk factor for mortality, ranking first in terms of the global burden of disease [1, 2]. The prevalence of hypertension among adults (18 years and older) in China was reported to be 27.9% [3]. Additionally, it was projected that the global number of adults with hypertension will reach 1.56 billion by 2025 [4]. The significant impact and pervasive nature of hypertension necessitate the identification and characterization of modifiable risk factors for effective public health and clinical interventions.

With the evolution of the "biopsychosocial" medical model, EH was considered as a typical psychosomatic disease. In addition to genetic and physiological factors, psychological factors were also thought to contribute to the development of EH. Long-term depression and anxiety can compromise the quality of life and worsen the prognosis.[5] According to a meta-analysis, the incidence of comorbid hypertension and anxiety was approximately 38% [6]. Yu Pan et al. [7] found that there was a positive association between anxiety and increased risk of hypertension. Anothor research demonstrated that patients with high levels of trait and state anxiety were more prone to arterial hypertension. [8] Psychological stress increases the risk of developing high blood pressure, which, in turn, contributed to the high incidence of psychological and behavioral disorders. The relationship between hypertension and psychological stress was mutually reinforced. and longitudinal data and theoretical literature indicated that mental stress was a risk factor for hypertension. [9] Anxiety itself acted as an independent risk factor for hypertension and can also promote its development and progression. Patients with hypertension were prone to anxiety due to the disease or inefficacious treatment [10], and anxiety disorders were more prevalent in hypertensive patients than in the general population. [11] Accordingly, psychological stress increases the likelihood of developing hypertension.

EH was the result of the combined action of environmental and genetic factors, and its risk factors were numerous, and the relationship between each risk factors were complicated. Structural equation model (SEM) was a statistical analysis method used to solve multiple variables and complex relationships in social, psychological, behavioral and economic studies, and also widely applied in clinical medicine recently. Current research showed that SEM can be employed to investigate the associations of dietary patterns with hypertension, and it can also be used to analyze the direct and indirect effects of psychological stress on blood pressure and biochemical indexes [12].

Therefore, this study provided a basis for analyzing the various influencing factors of psychological stress in promoting the occurrence and development of EH in the future, as well as a theoretical basis for the prevention and treatment of EH.

Methods

Research object

A total of 600 outpatients and inpatients were randomly selected from Ningbo Medical Treatment Center Li Huili Hospital between January 2018 and December 2021. The inclusion criteria were based on the diagnosis standard of hypertension and patients' consent to participate. Basic clinical data, including height, weight, body mass index (BMI), age, and gender, were recorded. Blood pressure (systolic blood pressure (SBP)) and (diastolic blood pressure(DBP)), total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), Angiotensin II (AngII), Aldosterone (ALD), Homocysteine (Hcy), and other biochemical markers were measured. All participants also completed the Chinese Perceived Stress Scale (CPSS) at baseline. The purpose and procedures of the study were fully explained to the patients, and written informed consent was obtained in accordance with the Declaration of Helsinki.

Inclusioncriteria of hypertension group

(1) Hypertension patients with SBP ≥ 140 mmHg, and/or DBP ≥ 90 mmHg.
(2) Age ≥ 18 years old.

Exclusioncriteria

(1) Secondary hypertension caused by renal parenchymal and/or renovascular diseases, primary aldosteronism, Cushing's syndrome, phaeochromocytoma, aortic dissection, or aortic coarctation.
(2) Comorbidities including diabetes, chronic hepatitis B/C, active infections, or malignancy (current or history of chemotherapy/radiotherapy).
(3) Severe hypertension-related complications including New York Heart Association (NYHA) class III-IV heart failure, hyperthyroidism, acute cerebral infarction, or hemorrhagic stroke.

(4) Cognitive impairment precluding valid psychological assessment (e.g., dementia, major psychiatric disorders).
(5) Individuals who have recently used drugs (such as RAAS blockers, anti-inflammatory drugs) or consumed foods (such as high-potassium foods, red wine, etc.) that could affect the biochemical markers measured in this study.

Inclusion criteria of the control group

Healthy subjects without EH and metabolic diseases who underwent a physical examination at Ningbo Medical Treatment Center Li Huili Hospital were selected as the control group. The control group's general clinical indicators were matched with those of the hypertension group, and they had no family history of diabetes or coronary heart disease. All subjects in the control group were informed of the study's purpose and provided informed consent.

Case stratification and grouping

Perceived stress scale (PSS) is a kind of self-rating scale for measuring stress, which has been widely recognized and accepted by the international community. It was compiled by Cohen et al. of Carnegie Mellon University in 1983 and has been published in many languages. The Chinese Perceived Stress Scale (CPSS) was developed based on the cultural context of China through the translation and revision of PSS. It has demonstrated good reliability and validity, and is currently the most widely used instrument for measuring perceived stress in China. The CPSS consists of 13 questions related to response tension and sense of time and space. Participants were asked to rate each item on a Likert scale from 0 (never) to 4 (always). Higher scores indicate higher levels of perceived stress. Based on domestic epidemiological surveys, a score > 25 was considered high pressure, and a score ≤ 25 was considered low pressure. The CPSS questionnaire was administered and collected on-site.

Determination of basic data

(1) Blood pressure measurement: Blood pressure was measured using a calibrated sphygmomanometer. Subjects were asked to rest for at least 30 min before measurement. Blood pressure was taken in the sitting position with the arm at heart level. The average of three measurements, taken on both arms with a 2-minute interval, was recorded.
(2) Measurement of height: Subjects' height was measured in centimeters (cm) after removing their shoes and head accessories.
(3) Measurement of body weight: The subject's body weight, without clothing, was measured in kilograms (kg) after removing shoes.
(4) BMI calculation: Body mass index (BMI) was derived using the standard formula: BMI = \frac{weight\ (kg)}{[height\ (m)]^2}

Determination of blood biochemical indexes

(1) Determination of blood lipid: Participants were instructed to follow a low-fat diet for three days prior to blood collection. They were also advised to avoid alcohol and foods/drugs that could affect lipid metabolism within 24 h. Blood samples were collected after a fasting period of 10–12 h and analyzed using the ANAYTECH-640 automatic biochemical analyzer.
(2) Determination of Ang II and ALD: Subjects who had their blood measured must have refrained from using drugs such as isoproterenol and methyldopa (which are catecholamines or metabolically converted to catecholamines), sympathomimetic drugs (e.g. ephedrine), and substances that promote catecholamine release (e.g. coffee, tea, nicotine, vanilla). They were also advised to avoid alpha receptor blockers (e.g. prazosin), alpha receptor agonists (e.g. clonidine), dexamethasone, monoamine oxidase inhibitors, and anticoncipiense. Blood samples were collected between 8:00 am to 10:30 am after a fasting period of at least 10–12 h and stored in test tubes containing ethylenediaminetetraacetic acid (EDTA) at -20℃. The samples were sent for testing within 30 min and then centrifuged at 4℃ for 10 min at 2000r/min.
(3) Determination of Hcy: Subjects were fasting for at least 10–12 h in the morning. Venous blood was collected from the elbow using a heparin anticoagulant tube. After thorough shaking, the blood samples were centrifuged at 2000 r/min for 10 min at 4℃. The upper serum of the venous blood was separated and collected for analysis using the Japanese Olympus AU2700 biochemical analyzer.

Statistical analysis

Statistical analysis was performed using SPSS 23.0 software. Descriptive statistics, including median (iqr), orn/N (%) were used for analyzing the general data across the four groups. Analysis of variance (ANOVA) was used for comparing means of multiple groups, with LSD test used for pairwise comparisons if variances were homogeneous. Nonparametric tests (e.g. Kruskal-Wallis test) were used for skewed data, and chi-square test was used for categorical data.

The influence of psychological stress on blood pressure and biochemical markers was explored through SEM statistical analysis using Amos 21.0 software. The SEM models were fitted using the Maximum Likelihood Estimation method. Model fit measures were obtained to assess how well the proposed models captured the covariance between the measures. The model development process included model setting, parameter estimation, model modification, and subsequent parameter estimation iterations. This iterative process was performed multiple times. A preset model was created by integrating insights from existing literature and combining them with relevant theories. Several model fit indices were assessed, including χ^2, the ratio of chi-square to degrees of freedom (CMIN/DF), Comparative Fit Index (CFI), Root Mean Square Error of Approximation (RMSEA), and goodness of fit index (GFI). Generally, a CFI of ≥ 0.90 and an RMSEA of ≤ 0.08 were considered indicative of a reasonably fitting model. P-values < 0.05 were considered statistically significant.

Result

The demographic, and biochemical indexes of HPPH, LPPH, HPPC, and LPPC groups were showed in Table 1. Pairwise comparison of each groups indicated that there were no significant differences in weight and height ($P > 0.05$). SBP, DBP, TC, TG, LDL-C, Ang II, ALD and Hcy of HPPH were significantly higher than those of LPPH, HPPC and LPPC ($p < 0.05$). SBP, DBP, TC, TG, LDL-C, Ang II, ALD and Hcy of LPPH were significantly higher than those of HPPC and LPPC.SBP, DBP, TC, TG, LDL-C, Ang II, ALD and Hcy of HPPC were significantly higher than those of LPPC. HDL-C were lowest in HPPH group, showed in Table 1.

A preset model was created by integrating insights from existing literature and combining them with relevant theories. Psychological stress (as the independent variable) influences biochemical markers (as the dependent variable) through blood pressure (as a mediating variable), showed in pre-set model.

We have established the SEM model, and the association between variables were presented as arrow lines. The standardized coeffiffifficients for each pathway, indicating the influence of predictors on responses, showed in Fig. 1. Among the variables, psychological stress were directly associated with blood pressure (both SBP and DBP)($P < 0.05$), and blood lipid (TC, TG, LDL-C, HDL-C), AngII, ALD, Hcy($P < 0.05$).

The evaluation of SEM: The test results showed that the model: $\chi 2/df = 4.32$, RMSEA = 0.07, TLI = 0.95, CFI = 0.99, GFI = 0.99, and the above indexes were combined with subjective evaluation. Therefore, it can be considered that the hypothesis model fits well, showed in Figs. 1 and 2.

Influence of psychological stress on blood pressure and various biochemical indexes, and the effects among various biochemical indexes

In this SEM model, we have elucidated the effects of psychological stress on blood pressure and various biochemical indexes. Among them, psychological stress was

Table 1 Demographic, and biochemical indexes in each group

	HPPH(*n* = 150)	LPPH(*n* = 150)	HPPC(*n* = 150)	LPPC(*n* = 150)	*p*
Demographic					
age(years)	56(14)	53.5(14)	51(16)	56(17)	0.0010
BMI(kg/m^2)	24.255(0.72)	23.15(1.34)	24.365(0.84)	24.36(1.17)	< 0.0001
height(cm)	165.4(9.6)	171.3(13.5)	165.1(9.4)	165.2(8.6)	< 0.0001
weight(kg)	67(8)	69(11.1)	66.75(8)	66(7.4)	0.9341
male(%)	79(52.67)	91(60.67)	77(51.33)	84(56)	0.3680
Laboratory					
TC, mmol/L	5.34(0.82)	4.375(0.91)	4.35(0.76)	3.93(1.11)	< 0.0001
TG, mmol/L	1.8(1.22)	1.655(0.65)	1.51(0.7)	1.07(0.91)	< 0.0001
HDL-C, mmol/L	1.125(0.37)	1.105(0.37)	1.26(0.28)	1.375(0.46)	< 0.0001
LDL-C, mmol/L	3.59(0.61)	3.41(0.73)	3.295(0.98)	2.74(0.74)	< 0.0001
AngII, ng/dl	86.35(19.27)	72.15(8.87)	65.45(8.70)	60.50(7.30)	< 0.0001
ALD, ng/dl	0.2955(0.06)	0.2305(0.08)	0.187(0.05)	0.1405(0.03)	< 0.0001
Hcy, mmol/L	16.00(14)	16.00(7)	16.00(4)	10.00(5)	< 0.0001
SBP, mmHg	168.4(9.6)	153.3(10)	123.85(10.6)	116.25(8.6)	< 0.0001
DBP, mmHg	94.4(30.4)	76.5(8.6)	79.95(8.6)	77.45(15.8)	< 0.0001

Data are presented as median (iqr), orn/N (%)

p value are calculated with ANOVA test, or chi-square test

HPPH: high psychological pressure hypertension group; LPPH: low psychological pressure hypertension group, HPP: high psychological pressure control group; LPPC: low psychological pressure control group

SBP: systolic blood pressure; DBP: diastolic blood pressure; TC: total cholesterol; TG: trytriglyceride; LDL-C: low density lipoprotein cholesterol; HDL-C: high density lipoprotein cholesterol; AngII: Angiotensin II; ALD: Aldosterone; Hcy: Homocysteine

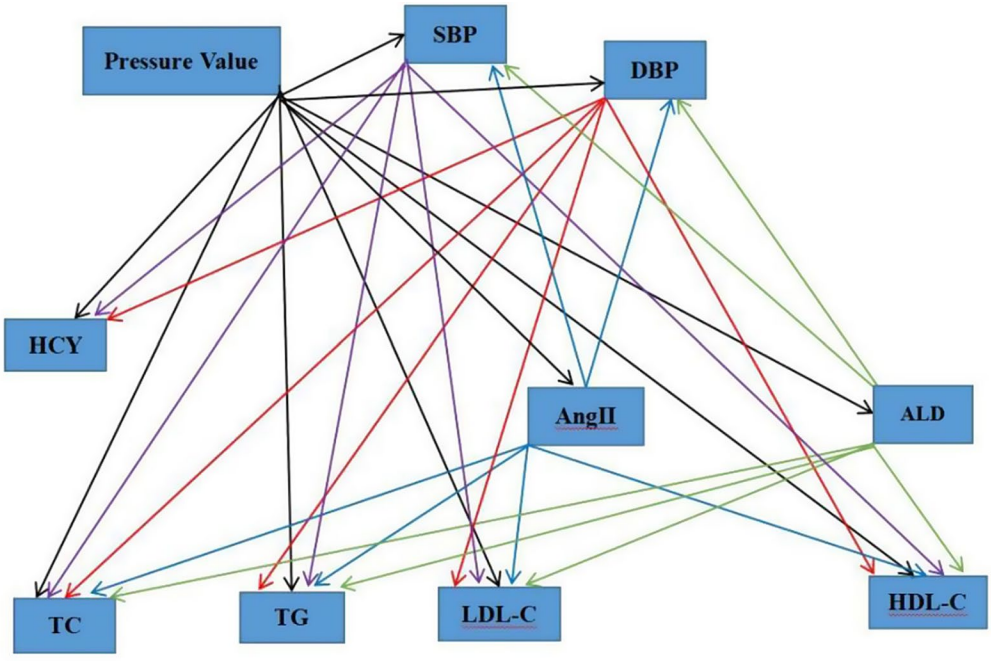

Fig. 1 Pre-set model

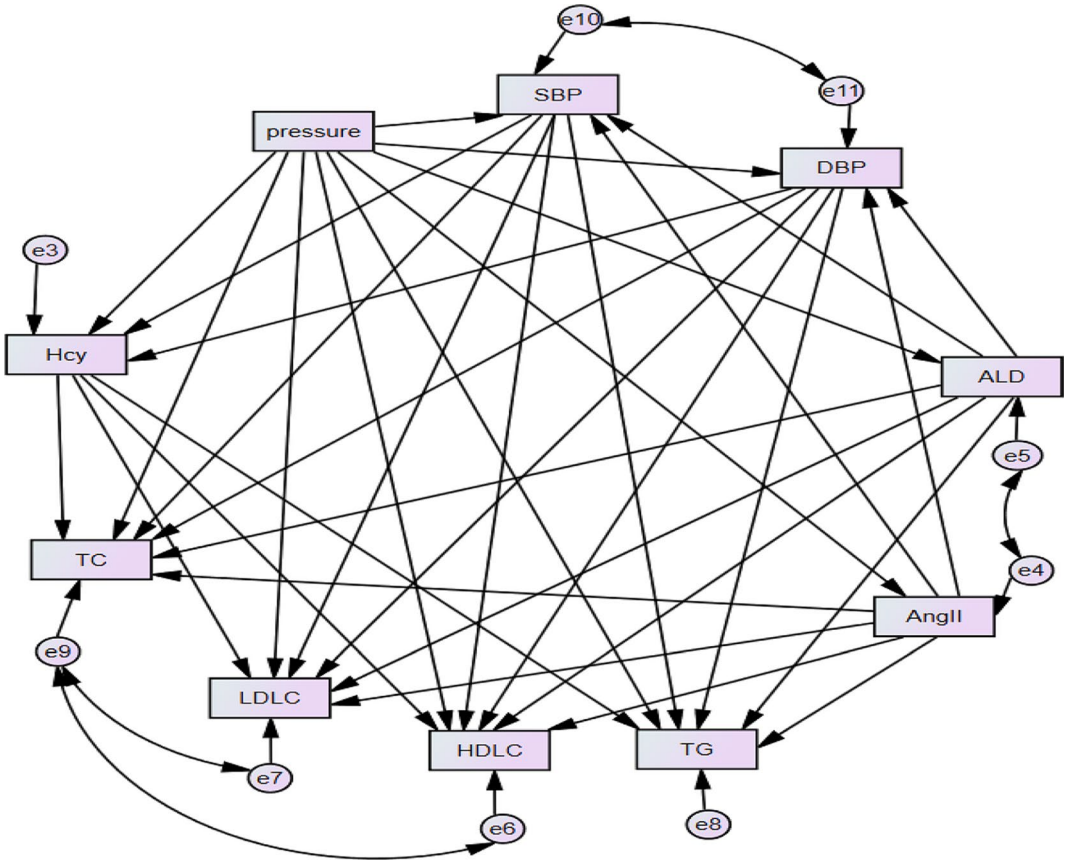

Fig. 2 The SEM model

Table 2 Influence of psychological stress on blood pressure and various biochemical indexes, and the effects among various biochemical indexes

item		Standardized estimate		*P*
Direct effect of variables				
psychological stress	→	SBP	0.124	0.030
psychological stress	→	DBP	0.085	<0.001
psychological stress	→	TC	0.007	<0.001
psychological stress	→	TG	0.006	0.006
psychological stress	→	LDL-C	0.008	0.001
psychological stress	→	HDL-C	0.003	<0.001
psychological stress	→	AngII	0.75	<0.001
psychological stress	→	ALD	0.001	<0.001
psychological stress	→	Hcy	0.048	0.011
AngII	→	TC	0.002	0.009
AngII	→	TG	0.003	0.022
AngII	→	LDL-C	0.002	0.006
AngII	→	HDL-C	0.002	0.008
AngII	→	SBP	0.069	<0.001
AngII	→	DBP	0.047	<0.001
ALD	→	SBP	8.225	<0.001
ALD	→	DBP	5.660	<0.001
ALD	→	TC	0.430	0.018
ALD	→	TG	0.393	0.019
ALD	→	LDL-C	0.466	0.013
ALD	→	HDL-C	0.399	0.012
SBP	→	TC	0.002	<0.001
SBP	→	TG	0.002	<0.001
SBP	→	LDL-C	0.002	<0.001
SBP	→	HDL-C	0.001	<0.001
SBP	→	Hcy	0.012	<0.001
DBP	→	TC	0.003	<0.001
DBP	→	TG	0.003	0.043
DBP	→	HDL-C	0.001	<0.001
DBP	→	LDL-C	0.004	<0.001
DBP	→	Hcy	0.024	0.046

found to have significant effects on blood pressure (SBP, DBP), it had a positive relationship with blood pressure (SBP, DBP)($P<0.05$), the higher the coefficients value represented a bigger influence between the variables, showed in Table 2.

Additionally, the influence caused by psychological stress on other biochemical indexes has been examined. psychological stress had a positve relationship with blood lipid (TC, TG, LDL-C, HDL-C), AngII, ALD, Hcy ($P<0.05$), it had a positive relationship on blood lipid (TC, TG, LDL-C), AngII, ALD and Hcy($P<0.05$), and had a negative relationship with HDL-C ($P<0.05$), showed in Table 2.

We have elucidated the effects among various biochemical indexes. There were also interactive correlation between various biomedical indexes. Among them, AngII and ALD had significant effects on blood pressure (SBP, DBP), blood lipid (TC, TG, LDL-C, HDL-C) ($P < 0.05$), it had a positive relationship with blood pressure (SBP, DBP), blood lipid (TC, TG, LDL-C) ($P < 0.05$), and had a negative relationship with HDL-C ($P<0.05$), meanwhile, the blood pressure (SBP, DBP) had effects on blood lipid (TC, TG, LDL-C, HDL-C) and Hcy ($P<0.05$), it had a positively correlated with TC, TG, LDL-C, Hcy ($P<0.05$), and had negative correlation with HDL-C ($P<0.05$), showed in Table 2.

Decomposition effect of influence factors of blood pressure and blood lipid

According to the effect of SEM, the degree of effect on blood pressure in descending order was ALD, psychological pressure, AngII. The degree of effect on TC, TG and LDL-C in descending order was ALD, psychological stress, AngII, SBP and DBP. HDL-C was the protective factor of blood pressure, AngII and ALD, showed in Tables 3 and 4.

The higher effect value represented bigger influence between variables. effect value > 0.05 considered big influence. Dash lines means there was no indirect effect in our SEM model.

Disscusion

In this study, we demonstrated that psychological pressure was associated with elevated blood pressure (both SBP and DBP), in patients with essential hypertension (EH). Furthermore, the severity of psychological pressure appeared to be positively associated with increases in blood pressure. There was a positive correlation between the severity of psychological pressure and the levels of TC, TG, LDL-C, and Hcy, whereas a negative correlation was noted with HDL-C levels. Psychological pressure.

was also associated with elevated levels of AngII and ALD in EH patients, which may indirectly contribute to increased blood pressure and lipid levels. Among these indicators, ALD had the greatest effect on blood pressure.

Table 3 Total, direct and indirect effects of influence factors on blood pressure

Variable	SBP			DBP		
	Total effect	Direct effect	Indirect effect	Total effect	Direct effect	Indirect effect
Psychological stress	2.102	0.408	1.694	1.643	1.136	0.506
AngII	1.087	1.087	---	0.339	0.339	---
ALD	111.743	111.743	---	30.133	30.133	---

Table 4 Decomposition of influencing factors of blood lipid

Variable	TC			TG			LDL-C			HDL-C		
	Total effect	Direct effect	Indirect effect	Total effect	Direct effect	Indirect effect	Total effect	Direct effect	Indirect effect	Total effect	Direct effect	Indirect effect
Psychological stress	0.073	0.021	0.052	0.020	0.003	0.017	0.042	0.025	0.018	-0.006	-0.011	0.004
AngII	0.028	0.006	0.022	0.010	0.001	0.009	0.018	0.004	0.014	-0.010	-0.003	-0.007
ALD	3.212	1.017	2.195	1.283	0.346	0.937	2.638	1.159	1.479	-0.489	-1.238	-0.727
SBP	0.017	0.017	---	0.008	0.008	---	0.015	0.015	---	-0.008	-0.008	---
DBP	0.011	0.011	---	0.002	0.002	---	0.007	0.007	---	-0.006	-0.006	---

Our findings indicated that psychological stress was associated with elevated blood pressure (both SBP and DBP) in patients with EH. The mechanism by which psychological stress leads to hypertension was complex and mediated through various pathways. Among these pathways, the overactivation of the sympathetic nervous system (SNS) and the Renin-Angiotensin-Aldosterone System (RAAS) likely played the most important role. When individuals experienced mental stress, the SNS becomes overactive, resulting in the continuous release of catecholamines, such as norepinephrine and adrenaline. This leads to cardiac output increased and constriction of resistance arterioles, ultimately causing a sustained increase in blood pressure. Chronic activation of the SNS can also contribute to organ damage. Reducing mental stress can help alleviate sympathetic nerve activity and lower blood pressure [13]. Therefore, the excessive stimulation of the SNS was a characteristic feature of persistent hypertension and played a pivotal role in the occurrence and development of hypertension induced by mental stress. Additionally, the RAAS was activated at a higher level in patients with hypertension when exposed to mental stress. Angiotensin-converting enzyme inhibitors can counteract the hypertensive effects of RAAS, indicating that RAAS played a significant role in the mechanism of hypertension caused by mental stress [14]. In addition, hypothalamuspituitary adrenal cortical axis, vascular endothelial system, immuno-inflammatory responses may also be Participated in this process. Study have demonstrated the multi-dimensional nature of the glucocorticoid pressure-raising mechanism [15]. Furthermore, mental stress can reduce serum levels of IFN-γ, weaken cellular immune function, disrupted endocrine regulation, and impaired the body's ability to regulate blood pressure, thus contributing to the development of hypertension [16]. Mental stress can also decrease the production of nitric oxide (NO), leading to vasoconstriction and elevated blood pressure [17].

This study showed that psychological stress had an effect on blood lipid. There was a positive correlation between the severity of psychological stress and the levels of TC, TG, LDL-C, whereas there was a negative correlation with the levels of HDL-C. According to the psychophysiological mechanism, abnormal activation of the neuroendocrine system can lead to somatic diseases due to pathological changes in corresponding organ structure and function [18]. Additionally, psychological stress stimulates the secretion of glucocorticoids, which promoting the decomposition of fat cells into free fatty acids. It also activated lipases through the SNS, leading to increased levels of free fatty acids in the blood. This results in the synthesis and secretion of TC and VLDL-C. VLDL-C can further transform into LDL-C, thereby increasing its levels. Furthermore, psychological stress activates the RAS, contributing to the breakdown of fat cells. An experiment demonstrated that long-term injection of AngII in rats led to significantly higher TG levels and corresponding increases in blood pressure. Another study [19] found that elevated blood lipid levels promote the activation of RAS and increased blood AngII levels. Consequently, dyslipidemia played a crucial role in the development of EH.

Our study showed that psychological pressure was associated with elevated levels of AngII and ALD in EH patients, indirectly resulting in blood pressure and lipid levels increased. Psychological stress may have direct and/or indirect effects on SNS, RAS and endocrine system, prompting the secretion of AngII and ALD, meanwhile, making them participate in lipid metabolism, leading to lipid disorders. Studies have shown that the combined use of ACEI and ARB can increase the level of HDL-C [20]. This indirectly suggested that RAS can affect lipid metabolism. An experiment on the relationship between RAS and blood lipid showed [21, 22] that there was a causal relationship between RAS and blood lipid disorders, which may be that patients with EH are often accompanied by blood lipid disorders, while patients with dyslipidemia are usually easy to develop EH, which accelerates the formation of arteriosclerosis. Meanwhile, Psychological pressure interacted with biochemical markers through blood pressure-mediated pathways. We also concluded that there was a positive correlation between the severity of blood pressure and the levels of TC, TG, LDL-C, and Hcy, while a negative correlation was observed with the levels of HDL-C. Therefore, it was speculated that increased blood pressure may

cause damage to the vascular endothelial cells, leading to changes in vascular structure and function. Possible mechanisms include: long-term high blood pressure leading to vascular smooth muscle damaged and accelerated arteriosclerosis. Additionally, blood vessels become more reactive to external factors, causing even minor environmental changes to impact blood lipid homeostasis. Some studies have shown that after accounting for other factors, Hcy was an independent influencing factor for EH [23]. An analysis of Hcy synergistically aggravates the arterial damage factor of hypertension through immune/inflammatory response [24]. This suggested that there may be an association between blood pressure and Hcy levels, although the specific mechanism needs further investigation.

There were several limitations to our study that should be acknowledged. Firstly, the findings were based on a single center study conducted at Ningbo Medical Treatment Center Li Huili Hospital, with a relatively small sample size, which may restrict the generalizability of the results. Secondly, we only included a limited number of variables and did not incorporate other potentially significant biochemical indicators. Thirdly, the use of the CPSS questionnaire may introduce memory bias. Additionally, both outpatient and inpatient data were analyzed, and differences in clinical environments that could influence stress levels were considered in the study design. Outpatients may experience stress related to chronic disease management, while inpatients may encounter acute stress associated with hospitalization. Furthermore, patients receiving RAAS blockers were identified, and this information was incorporated into the data analysis and addressed in the results. Finally, there may be additional concerns raised by other reviewers that are beyond the scope of this study.

Conclusion

We utilized SEM to describe the direct and indirect effects of psychological stress on blood pressure and biochemical markers. Our research demonstrated that psychological pressure was associated with increased blood pressure (both SBP and DBP), as well as elevated blood lipid, AngII, ALD, and Hcy levels in patients with EH. These findings contributed to an in-depth understanding of the various factors influencing psychological stress in the development of EH. Moreover, they offered novel approaches and insights for the analysis and prevention of EH.

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None.

Author contributions
M. H. Z contributed to the conception and design of this manuscript. C. Q. W performed the data analyses and wrote the manuscript. J. N. Z, Y. G, J. L, D. P. C acquired data and helped perform the analysis with constructive discussions. All authors read and approved the final manuscript.

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Data availability
The data underlying the results presented in this study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
The study followed the principles of the Declaration of Helsinki and was approved by the Ningbo Medical Treatment Center Li Huili Hospital (KY2023SL240-01). All participants in the study provided informed consent after being fully informed of the purpose of the research.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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